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OF THE

PRACTICE OF MEDICINE.

EDITED BY DR. H. VON ZIEMSSEN,

PROFESSOR OF CLINICAL MEDICINE IN MUNICH, BAVARIA.

VOL. VI.

DISEASES OF THE CIRCULATORY SYSTEM,

TOGETHER WITH THE CHAPTERS ON

WHOOPING-COUGH, DISEASES OF THE LIPS AND CAVITY OF THE MOUTH, AND DISEASES OF THE SOFT PALATE.

BY PROF. ROSENSTEIN, of Leyden; PROF. SCHROETTER, of Vienna; PROF. LEBERT, of Vevay; PROF. QUINCKE, of Berne; DR. BAUER, of Munich; DR. STEFFEN, of Stettin; PROF. VOGEL, of Dorpat; and PROF. WAGNER, of Leipsic.

U.S.A. MEDICAL

Translated by

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EDITOR OF AMERICAN EDITION.

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BIOGRAPHICAL SKETCHES OF THE AUTHORS.

Samuel Sigmund Rosenstein, son of the rabbi Elkan Rosenstein, was born in Berlin on the 20th of February, 1832. He received his preliminary education in the Friedrichs-Werder Gymnasium, and entered the University of Berlin in 1850. Before commencing the study of medicine, he devoted a year to the study of philology, under the direction of Böckh, Trendelenburg, and others. He passed his examination for the degree of doctor of medicine in 1854, the title of his thesis being "De cyclopia inter animalia observata." Among his instructors were such men as Schoenlein, Traube, and Langenbeck. From 1856 to 1858 he acted as assistant physician in the city hospital of Dantzic. He then returned to Berlin, and continued his medical studies under Virchow and Hoppe-Seyler. Soon afterwards he began his career in that city as a private practitioner. In 1863 he was appointed an instructor in the University of Berlin; in 1865 he was called to Groningen, in Holland, as professor of the medical clinic; and in 1873 he accepted a call to the University of Leyden.

In addition to numerous journal articles, which appeared from time to time in Virchow's Archiv, between the years 1855 and 1873, Rosenstein has written a treatise on the "Pathology and Therapeutics of Kidney Diseases," the first edition of which was published in 1863, the second in 1869. In 1873 a translation of this work was published in the French language.

Leopold Schroetter was born in Gratz, in Steiermark, on the 5th of February, 1837. His father, Anton Schroetter, the discoverer of amorphous phosphorus, was at that time professor of chemistry in the Joanacum. It was here, too, that the son received the greater part of his preliminary education, the balance of it being obtained in the then very flourishing Gymnasium of Vienna, under most excellent teachers. His entire course of medical studies was also completed in Vienna, his chief attention being devoted to the instruction of Schuh and Skoda. In 1861 he received the degree of doctor of medicine and surgery, and, after a service of only fifteen months in the General Hospital as a candidate for the position, he had the good fortune to be appointed a "pupil in operative surgery" in Schuh's surgical clinic, with a yearly stipend from the government. At the same time, so far as the

duties of his position allowed him opportunity, he continued to follow the teachings of Skoda. It was probably owing to the interest thus shown by Schroetter in his lectures, that Skoda soon afterwards chose him as his clinical assistant, a position which he held for a period of six years (from November 1, 1863, to November, 1869). Owing to Skoda's frequent illnesses, Schroetter was often called upon to conduct the clinic alone, and this increase of responsibility stimulated him to great efforts. During this period the following works were published:

1. "A Case of Recovery from Pneumothorax (in a tuberculous patient) without Pleuritic Exudation." Wochenblatt der k. k. Gesellschaft der Aerzte, No. 5, 1865.

2. "Extirpation of a Polypus of the Larynx." Wiener medicin. Presse, No. 34, 1865.

3. "On Narrowing of the Aorta in the Vicinity of the Ductus Arteriosus Botalli." Wochenblatt der k. k. Gesellschaft der Aerzte, No. 43, 1866.

4. "Report of a Case of Cyst of the Epiglottis." Med. Jahrb., XII. Band, 1866.

5. "Echinococcus of the Lungs." Med. Jahrb., XIV., 1867. (This is the first observation on record of echinococcus multilocularis of the lungs.)

6. "Contributions to Laryngoscopical Surgery." Medic. Jahrb., XV., 1868.

7. "A Second Series of Contributions to Laryngoscopical Surgery." Medic. Jahrbücher, XVI. Bd., 1868.

8. "A Third Series of Contributions to Laryngoscopical Surgery." Medic. Jahrb., XVII. Bd., 1869.

9. "On the Body Temperature observed in Croupous Pneumonia." VIII. Bd. d. Sitzungsberichte der k. Academie der Wissenschaften, 1868.

After the publication of this last paper, he was appointed an instructor in diseases of the larynx and chest, in the University of Vienna. On the 4th of March, 1870, he was placed in charge of the newly-created laryngoscopical clinic, thus filling the position which Türck's death had for some time past left vacant. After Oppolzer's death, he was invited to take temporary charge of the first division of the medical clinic, and in 1875 he was raised to the rank of extraordinary professor. During this second period of his career, the following are among the most important of his contributions to medical literature:

"Contribution to our Knowledge of the Changes in Position which the Heart may undergo." Med. Jahrbücher, XX. Bd., 1870; "On the Effects of Digitalis and the Tincture of Veratrum Viride upon the Body Temperature in Croupous Pneumonia." LXII. Bd. der Sitzungsberichte der k. Academie der Wissenschaften. LXVI., B. III. Abth. 1872 (July number); "Laryngoscopical Communications," published by Braumüller in Vienna, 1875; "Contributions to the Treatment of Stenoses of the Larynx." Braumüller, Vienna, 1876; "On Aneurisms of the Aorta." Mittheilungen des Wiener medic. Doctoren-Collegiums, 1876, II. Bd., 12; "On Foreign Bodies in the Naso-pharyngeal Cavity, Larynx, and Trachea." Zeitschrift für Ohrenheilkunde, etc., 1876.

Heinrich Quincke was born in 1842, in Frankfort on the Oder. From 1858 to 1863 he studied medicine in the Universities of Wuertzburg, Heidelberg, and Ber-

lin. In 1866 he was made an assistant in the Bethany Hospital in Berlin, under Wilms. From 1867 to 1871 he served as an assistant in the clinic conducted by von Frerichs, in Berlin. In 1870 he established himself as a private instructor in the University of that city. In 1873 he was called to Berne, in Switzerland, as professor of the medical clinic, and this position he still retains at the present day.

Apart from reports of cases, he has published the following works:

"On a Capillary and a Venous Pulse," Berliner klinisch. Wochenschrift, 1868; "On the Origin of the Sounds of the Heart, and on Heart Murmurs," 1870; "On the Treatment of Pleurisy," 1872; "On Irritation of the Vagus in the Human Being," Reichert und DuBois-Reymond's Archiv für Anatomie und Physiologie, 1875; "On the Excretion of Drugs through the Intestinal Mucous Membrane," 1868; "On the Influence of the Central Nervous System on the Development of Heat," (Naunyn and Quincke), 1869; "On the Circulation of Blood in the Lungs" (Quincke and Pfeiffer), 1871; "A Contribution to the Physiology of the Cerebro-Spinal Fluid," 1872; "On Imbibition," Pflüger's Archiv für Physiologie, 1870; "On the Amount of Hæmoglobin contained in the Blood in Different Diseases," Virchow's Archiv, 1872; "On the Length of the Period of Incubation of Typhoid Fever," Correspondenzblatt für Schweitzer Aerzte, 1875; "On Transudations containing Fat," Das Archiv für klinische Medizin, 1875: "On Pernicious Anæmia," in Volkmann's klinischen Vorträgen, 1876; "Balneologische Tafeln," Berlin, 1872.

OSEF BAUER was born on the first of October, 1845, in Erlhammer, in the northern part of Bavaria. His preliminary education was obtained in part at the Gymnasium in Amberg, and in part at the Ludwigs Gymnasium in Munich. He pursued his medical studies in Munich, and in 1869 received the degree of doctor of medicine, and also the faculty prize for the best essay on "The History of Venesection." He was then appointed an assistant in the General Hospital, under the immediate direction of the late Professor Dr. Jos. von Lindwurm. In the spring of 1871 he passed the state medical examination, and devoted himself for a time to scientific researches in the laboratories. In the spring of 1873 he established himself as a private instructor in clinical medicine in the University of Munich. He next gave courses on physical diagnosis, and also delivered lectures on certain parts of special pathology and therapeutics. After Lindwurm's death the faculty temporarily placed him in charge of the clinic, and when von Ziemssen was called to Munich (in 1874) to conduct it, Bauer still retained his connection with it as clinical assistant. In 1875 he assumed the management of the Propædeutic Clinic, established by von Ziemssen, and in 1876 he became extraordinary professor of the Propædeutic Clinic. The following are the scientific works published by Bauer:

1. "History of Venesection," Munich, 1870. E. Gummi.

2. "On Resorption in the Small and Large Intestines." (Written in conjunction with Professor Dr. Voit.) Zeitschrift für Biologie, 1870.

3. "On the Transformation of Matter in Phosphorus-poisoning." Zeitschrift für Biologie, 1871.

4. "On the Transformation of Matter after Losses of blood." Zeitschrift für Biologie, 1873.

5. "On the Influence of different Remedies (Quinine, Morphine, Alcohol, Digitalis) on the Excretion of Carbonic Acid." Zeitschrift für Biologie, 1874 (written in conjunction with Dr. H. von Boeck).

6. "On the Results of the Cold-water Treatment in Typhoid Fever." Bayerisches aerztliches Intelligenzblatt, 1873.

7. "On the Treatment of Croupous Pneumonia." Deutsches Archiv für klinische Medizin, 1874.

8. "On the Epidemic of Cholera in the Munich Hospital during the year 1873–74." Bericht der Cholera Commission für das deutsche Reich, 1876.

Dr. A. Steffen, son of Privy Medical Counsellor Dr. Steffen, was born at Stettin, in the year 1825. He received his early schooling at St. Mary's High School of that city. In the autumn of 1844 he entered the University; in 1848 he obtained his degree as doctor; and in the spring of 1849 had passed the state examination. After practising medicine in Stettin from the year 1850, he became, in 1851, surgical assistant at the Children's Hospital, where, from the year 1852, he has been physician in charge. The first two volumes of his "Clinic for Children's Diseases" (diseases of the lungs and pleura) were issued between the years 1865 and 1870. His lesser works are to be found scattered through the different periodicals, and are mostly in the Jahrbuch f. Kinderheilkunde, Neue Folge. They are as follows: "On Inhalations in Tussis Convulsiva;" "On Croup and Diphtheria of the Larynx;" "Cases of Cerebral Tumors in Childhood;" "On Some of the Rarer Forms of Diseases of the Brain and its Membranes;" "Diseases of the Œsophagus;" "On Tuberculosis of the Choroid and Acute Miliary Tuberculosis;" "Contribution to the Study of Cardiac Diseases;" "Contribution to the Physiology and Pathology of the Rectum;" "On Striped Pneumonia."

Alfred Vogel, son of the late Dr. August von Vogel, professor of chemistry in Munich, was born in that city, on the 31st of March, 1829. He obtained his preparatory education at the Gymnasium of his native city, and entered the University in 1846. In 1849 he matriculated at the University of Berlin, and followed the clinical instruction of Schoenlein, Traube, Romberg, Langenbeck, and Juengken. During the following year he visited Wuerzburg, and became an enthusiastic pupil and follower of Virchow.

In the year 1851 he entered the Children's Hospital in Munich as assistant surgeon, and in 1852 he passed his final examination for the degree of doctor of medicine, his thesis being on the subject of rachitis (Journal für Kinderkrankheiten). From 1853 to 1855 he acted as clinical assistant to Pfeufer, and while in this position he made his first steps in the career of teacher by giving courses on physical and chemical diagnosis. In 1856 he became installed as an instructor in the University of Munich, his inaugural essay being a Treatise on Typhoid Fever (Erlangen, F. Encke; second edition in 1859). From this time on he conducted a very large private clinic for children's diseases. During the following years he deliv-

ered lectures on diseases of children, physical diagnosis, and certain portions of special pathology. In 1860 he published a Manual of Diseases of Children (Erlangan, F. Encke), which met with a large and rapid sale, was translated into all the principal European languages, and has now reached its seventh edition. Encouraged by a large private practice, and by the extraordinary number of patients who were brought to his public clinic, Vogel continued to devote himself to the special department which he had chosen, and added rapidly to his store of experience. In 1865 he published a paper on "An Optical Test for Milk." In 1866 he was called to Dorpat, Russia, as professor of the medical clinic, a position which he holds at the present time. Since then his publications as well as those of his pupils have appeared in the "Archiv für klinische Medizin." Of these may be mentioned more particularly the following: "An Optical Test for Albumen;" "Inoculations with Tubercle;" "Scabies Crustosa;" "Alterations of the Nails after Acute Diseases;" "Tetanus Rheumaticus;" "The Capabilities of the Cavity of the Mouth for Absorption;" "The 'Etat criblé' of the Cerebellum."

Ernst Leberecht Wagner was born on the 12th of March, 1829, in Dehlitz, near Weissenfels, in Prussia. He received his preliminary education at the Gymnasium in Zeitz. From 1848 to 1850 he pursued his medical studies in Leipzig. The following year he visited Vienna, where he took special delight in the teachings of Oppolzer. After remaining a year in that city, he next went to Prague, and continued his medical studies in that city during the winter-semester of 1851–1852. Returning then to Leipzig, he finally, in November, 1852, passed both the state examination and that for the degree of doctor of medicine. Besides acting as private assistant to several practising physicians, he assisted Wunderlich in this capacity for a period of five years, dating from the autumn of 1853. The works on pathological histology, which were published at that time, and especially those of Virchow, excited Wagner's deepest interest, and eventually led him to accept the appointment, in 1854, of private instructor of pathological anatomy. At first he was associated with Bock in this work, but since 1854 he has been the only lecturer on this subject in the Leipzig University. In 1856 he was made extraordinary professor, and, in 1862, regular professor of general pathology and pathological anatomy. From 1853 to 1858 he also held the position of substitute prosector to the "Jacob's Hospital," but since that time he has been the regular prosector. In 1860 he was appointed director of the Medical (ambulatory) "Poliklinik."

Wagner first published his works in the "Archiv für physiologische Heilkunde," but since 1860 he has published them in the "Archiv der Heilkunde," of which he was at that time (1860), and still is, the editor, in association with Wunderlich and Roser—formerly also with Vierordt and Griesinger. These works relate chiefly to investigations on cancer, tubercle, syphilitic new-growth, colloid metamorphosis, diphtheria, and waxy degeneration. In 1862 appeared the first edition of his "Manual of General Pathology" (published at first by Uhle and Wagner conjointly). Since then the work has been greatly enlarged, and has reached its seventh edition. The "Manual" has also recently been translated into the English language.

EDITOR'S NOTE.

Owing to the great size of volumes VII. and VIII., it has been deemed best to incorporate the article on Whooping-Cough, as well as those on Diseases of the Lips, Cavity of the Mouth, and Soft Palate, into this volume.

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(Translated by George G. Wheelock, M.D.)

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(Translated by J. Solis Cohen, M.D.)

INTRODUCTION

TO

DISEASES OF THE HEART.

ROSENSTEIN.

INTRODUCTION.

BIBLIOGRAPHY: Of the more easily accessible handbooks on anatomy and physiology, those of *Theile-Soemmering*, *Luschka*, *Henle*, *Ludwig*, *Donders*, and *Funke* furnish the most exhaustive information on the heart. For an exact demonstration of the relative positions of the different parts of the heart to one another and to the surrounding organs, the following illustrated works are the most important: *Pirogoff*, Anatome topographica sectionibus per corpus humanum triplici ductione ductis illustrata. Petropoli, 1852.—*Luschka*, Die Brustorgane des Menschen in ihrer Lage. Tübingen, 1857.—*C. E Hoffmann*, Die Eingeweide des Menschen, etc., 1863.—*Rüdinger*, Topographisch-chirurgische Anatomie des Menschen. Stuttgart, 1873.—The *most important* monographs and essays bearing on the questions of anatomy, physiology, and physical diagnosis (the author does not aim at an exhaustive list of literature) are the following:—*Harvey*, Exerc. anatomicæ de motu cordis et sanguinis circulatione, etc., etc. Roterodami, 1660.—*Lower*, Tractatus de corde. Lugd. Batav. 1728.—*J. M. Lancisi*, De motu cordis et aneurysmatibus. Romæ, 1728.—*Alb. de Haller*, Deux mémoires sur le mouvement du sang. Sect. III. Cap. VII. Lausanne, 1754.—*Senac*, Traité de la structure du cœur. Paris, 1777.—*Auenbrugger*, Inventum novum ex percussione thoracis et signo pectoris morbor. deteg. Vienn. 1769; and the same, Traduite et commentée par *Corvisart*. Paris, 1808.—*Corvisart*, Sur les maladies et les lesions organiques du cœur. Paris, 1806.—*R. Laënnec*, Traité de l'auscultation médiate. Paris, 1818, and enlarged in quatrième édition par *Andral*. Paris, 1837.—*Piorry*, De la percussion médiate et des signes obtenus à l'aide de ce nouveau moyen d'exploration dans les maladies des organes thoraciques et abdominaux. Paris, 1827.—*Rouannet*, Nouvelle analyse des bruits du cœur. Thèse, Paris, 1832.—*Corrigan*, On the motions and sounds of the heart, in Dublin Medic. Transac. New series, Vol. I. 1830.

James Hope, Diseases of the Heart, 1830.—*Bizot*, Mem. de la soc. méd. d'observation de Toulouse. Tom. I. 1836.—*Philipp*, Zur Diagnostik der Lungen und Herzkrankheiten mittelst physikalischer Zeichen. Berlin, 1836.—*Corrigan*, Inquiries into a new disease of the heart. Edinb. Jour. 1836.—*Clendinning*, Facts and inferences relative to the condition of vital organs, etc., in Med. Chir. Transact. Vol. XXI. 1838.—*Beau*, Recherches sur les mouvements du cœur

in Archiv génér. de médec. 1835.—*Piorry*, ibid. 1834.—*Bouillaud*, Des Maladies du cœur et des grandes vaisseaux.—Reports of the London and Dublin committees for investigating, etc., in Transact. of British Scientif. Association. Vol. VI. 1837.—*J. Skoda*, Abhandlung über Auskultation und Percussion. Wien, 1839; and 5th edition, Wien, 1854.—*Gendrin*, Lectures sur les maladies du cœur, 1840.—*John Reid*, Tables of the weights of some of the most important organs of the body, etc. Edinb. Monthly Jour. of Med. Science, 1843.—*Kürschner*, Herz und Herzbewegung, in Wagner's Handwörterbuch, Bd. II. Oct. 1844.—*Baumgarten*, Ueber den Mechanismus, etc., in Müller's Archiv für Anat. und Phys. 1843.—*Retzius*, Ueber den Mechanismus des Zuschliessens der halbmondförmigen Klappen, ibid. 1843.—*J. Heine*, Ueber die Mechanik der Herzkammerbewegung, etc., in Zeitschr. f. ration. Med. Bd. I. 1844.—*Volkmann*, Ueber Herztöne und Herzbewegung, in Zeitschr. f. rat. Medic. 1845.—*Zehetmaier*, Die Herzkrankheiten. Wien, 1845.—*Kiwisch*, Neue Theorie des Herzstosses in Prager Vierteljahrschrift, 1846, Bd. IX.—*Hamernyk*, Physiol. Patholo. Untersuchungen über den Mechanismus, etc. in Prager Vierteljahrschrift, 1847.—*Conradi*, Ueber die Grösse und Lage der Brustorgane. Giessen, 1848.—*Gaal*, Physikalische Diagnostik. Wien, 1849.—*Ludwig*, Ueber den Bau und die Bewegungen der Herzventrikel, in Zeitschr. f. rat. Med. 1849.—*Racle*, Remarques sur certains phenomènes d'auscultation in Archiv génér. 1849.—*Levié*, Versuch einer neuer Erläuterung des Herzstosses in Arch. f. phys. Heilk. 1849.—*Rapp*, Beiträge zur Diagnostik der Klappenaffectionen des Herzens. Habilitationsschrift. Würzburg, 1849.—*Volkmann*, Die Hämodynamik nach Versuchen. Leipsig, 1850.—*Kiwisch*, Neue Forschungen über die Schallerzeugung in den Kreislaufsorganen, in den Verhandlungen der Würz. phys. med. Ges. 1850.—*E. H. Weber*, Ueber die Anwendung der Wellenlehre auf die Lehre vom Kreislauf des Blutes, etc., in Müller's Archiv, 1851.—*J. Meier*, Ueber die Lage der einzelnen Herzabschnitte zur Thoraxwand, etc., in Virchow's Archiv für path. Anat. etc. 1851; and Ueber die Grösse und den Grad der normalen Dämpfung in der Präcordialgegend, ibid. Bd. III.—*Traube*, Practische Bemerkungen über den Spitzenstoss des Herzens, in Wiener med. Wochenschr. 1852.—*Nega*, Beiträge zur Kentniss der Function der Atrioventricularklappen des Herzens. Breslau, 1852.—*Strempel*, Beiträge zur physikal. Diagnostik. Habilitationsschrift. Rostock, 1852.—*Knabe* (*Traube*), De venarum pulsatione atque intumescentia Dissert. inaug. Berolini, 1853.—*Scoda*, Ueber die Function der Vorkammern des Herzens, in Zeitschrift der Ges. der Aerzte in Wien, 1853.—*Peacock*, On the weights and dimensions of the heart in health and disease, in Monthly Jour. of Med. Scie. 1854.—*Wachsmuth*, Ueber die Functionen der Vorkammern des Herzens, in Zeitschrift für rat. Med. 1854.—*W. H. Walshe*, A practical treatise on the diseases of the lungs, heart, and aorta. London, 1854.—*Hiffelsheim*, Recherches theoriques et exper. sur la cause de la locomotion du cœur, in Comptes rendus. Tom. 39, 1854.—*A. Heynsius*, Bydrage tot eene physische verklaring van de abnormale geruischen in hel vaatstelsel, in Nederl. Lancet, 1854.—*Th. Weber*, Physikalische und physiologische Experimente über

die Entstehung der Geräusche in den Blutgefässen, in Archiv f. physiol. Heilk. 1855.—*Chauveau* et *Faure*, Recherches experimentales sur les mouvements et les bruits du cœur, in Gaz. méd. de Paris, 1855.—*Vierordt*, Die Lehre vom Arterienpuls in gesunden und krankhaften Zuständen, 1855.—*Drasche*, Ueber Verdopplung und Spaltung der Herztöne. Wiener Wochenschr. 1855.—*Wulff*, Nonnulla de cordis pondere ac dimensionibus, etc. Diss. inaug. Dorpat, 1856.—*F. Ernst*, Studien über die Herzthätigkeit in Virch. Arch. Bd. IX. 1856.—*Bamberger*, Beiträge zur Physiol. und Pathologie des Herzens, in Virchow's Archiv. Bd. IX. 1856.—*Chauveau*, Sur la theorie des pulsations du cœur. Comptes rend. 1857.—*Rüdinger*, Ein Beitrag zur Mechanik der Aorten- und Herzklappen. Erlangen, 1857.—*Hamernyk*, Das Herz und seine Bewegung. Prag, 1858.—*Gerhardt*, Untersuchungen über die Herzdämpfung und die Verschiebung ihrer Grenzen bei Gesunden, im Archiv f. physiol. Heilk. 1858.—*Traube*, Ueber die Herz- und Arterientöne in Krankheiten. Med. Centralzeitung, 1859.—*Marey*, Recherches sur la circulation du sang. Paris, 1859.—*Spring*, Mémoire sur les mouvements du cœur. Mem. de l'Acad. royale de Belgique, 1860.—*C. Gerhard*, Der Stand des Diaphragma, etc. Tübingen, 1860.—*Schoemaker*, Over het outstaan van den ersten toon, etc. Nedel Tydschr. 1860.—*Chauveau* et *Marey*, Determination graphique des rapports de la pulsation cardiaque avec les mouvements de l'oreillette du ventricule. Gaz. Méd. 1861.—*Beau*, Traité expérimental et clinique d'auscultation, etc. Paris, 1856.—*Seitz*, Die Auscultation und Perkussion der Respirationsorgane. Giessen, 1860.—*Sieveking*, On the diagnostic value of murmurs in the pulmonary artery. Lancet, 1860.—*Ringer*, On the influence of change of posture on the character of endocardial murmurs. Edinb. Med. Jour. 1861.—*Markham*, Remarks on the cause of the closure of the valves of the heart. Med. Chir. Transact. 1861.—*Sidney Ringer*, On physical examination of the heart, in Edinb. Med. Jour. 1860.—*Geigel*, Lage und Bewegung des Herzens, in der Würzb. med. Zeitschr. 1862.—*Kornitzer*, Anatom. physiol. Bemerkungen zur Theorie des Herzschlags. Denkschrift der mathemat-naturwissenschaftl. Klasse der Wiener Akademie, 1862.—*Bahr*, Zum Problem des Herzspitzenstosses. Virchow Arch. Bd. XXIII. 1862.—*Scheiber*, Zur Lehre vom Herztosse, ibidem.—*Jacobson*, Beiträge zur Hämodynamik, in Reichert u. Du Bois. Arch. 1862.—*v. Dusch*, Ueber ein eigenthümliches Verhalten der Herzgeräusche für die Auscultation, in Verhandlungen des natur-histor. med. Vereins in Heidelberg, 1862.—*Kobelt*, Ueber Form und Dimensionen der Herzdämpfung, im Archiv der Heilk. 1863.—*A. von Bezold*, Untersuchungen über die Innervation des Herzens. Leipsig, 1863.—*O. Naumann*, Beiträge zur Lehre vom Puls. Zeitschr. für rat. Med. 1863, und Archiv f. Heilk. 1864.—*Loeffler*, Ueber Entstehung des zweiten Ventrikeltones. Wochenblatt der Gesell. der Aerzte, in Wien, 1862.—*Scoda*, Doppelter Puls und doppelte Herztöne, in Allgem. Wiener med. Zeitung, 1863.—*Marey*, Physiologie médicale du sang. Paris, 1863.—*Bamberger*, Beobachtungen über den Venenpuls. Würzb. medic. Zeitschr. 1863. *Geigel*, Ueber den Venenpuls, ibid.—*Helmholtz*, Ueber Muskelgeräusch. Verhandlungen des medic-natur-histor. Vereins in Heidelberg, 1864.—*Gerhard*, Ueber

einige Formen der Herzdämpfung in Prag. Vierteljahrschrift, 1864.—*Hayden*, On the rhythm of the heart's action. Dubl. Quart. Jour. 1865.—*Seidel*, Pulsation der Vena cava inferior bei Insufficienz der Tricuspidalis. Deutsche Klinik, 1865.—*Friedreich*, Ueber den Venenpuls. Deutsche Archiv f. klin. Med. 1865. *O. J. B. Wolff*, Characteristik des Arterienpulses. 1865.— *C. Gerhard*, Lehrbuch der Auscultation und Perkussion. Tübingen, 1866.—*Donders*, Der rhythmus der hartstoonen. Nederl. Arch. voar Geneesk, 1866.—*Landois*, Neue Bestimmung der zeitlichen Verhältnisse der Contraction der Vorhöfe, etc. Centralblatt f. med. Wissenschaft, 1866.—*Parrot*, Etude clinique sur le siège et le mécanisme des murmures cardiaques dites anémiques. Arch. génér. de médec. 1866. —*Potain*, Note sur les redoublements normaux des bruits de cœur. Union medic. Nos. 97, 100, 104–115. 1866.—*Immermann*, Zur Pathogenese und Aetiologie der sichtbaren exspiratorischen Schwellung der Halsvenen. Deutsches Archiv für klin. Medic. 1866.—*G. Valentin*, Versuch einer physiologischen Pathologie des Herzens und der Blutgefässe. 1866.—*Parrot*, Etude sur le siège, le mécanisme et la valeur sémiologique des murmures vasculaires du cou. Arch. génér. de méd. 1867.—*Monneret*, Sur les bruits veineux continus du cou. Gaz. méd. de Paris, 1867.—*A. Leared*, The sounds of the heart in their relation to pathology. Med. Times, 1867.—*N. Friedreich*, Krankheiten des Herzens, 2d edit. Erlangen, 1867.—*Soulier*, Du frémissement arteriel. Gazette de Lyon, 1867.—*A. Leared*, On blood sounds. Med. Times and Gazette, 1868.—*E. Leyden*, Ungleichzeitige Contraction beider Ventrikel. Virchow Arch. 1868.—*Dogiel* und *Ludwig*, Ein neuer Versuch über den ersten Herzton. Berichte der mathem. physikal. Klasse der sächs. Ges. der Wissenschaften, 1868.—*v. Dusch, Th.*, Lehrbuch der Herzkrankheiten, 1868.—*Jaccoud*, Methode cardiographique de Mr. le Professeur Baccelli de Rome. Gaz. hebd. 1868.—*Geigel*, Der gespaltene Herzton, Verhandlungen der Würzburg. Ges. 1868. —*O. Bayer*, Ueber die Entstehung des ersten Herztons, etc. Arch. f. Heilk. 1869. —*The Same*, Casuistischer Beleg für die Nothwendigkeit den ersten Herzton als Muskelton aufzufassen, ibid.—*Guttmann*, Ueber die Entstehung des ersten Herztons. Virchow's Archiv. 1869.—*The Same*, Ueber den gespaltnen diastol. Herzton, etc., ibidem.—*Perls*, Ueber Weite und Schlussfähigkeit der Herzmündungen und ihrer Klappen. Deutsches Archiv für klin. Med. Bd. VI. 1869.— *Thamm*, Beiträge zur Lehre vom Venenpuls und von den Herzgeräuschen. Berl. klin Wochen. 1869.—*A. B. Meier*, Ueber das Hemmungsnervensystem des Herzens, Berlin, 1869.—*Niemeyer, P.*, Entwurf einer einheitlichen Theorie der Herz-, Gefäss- und Lungengeräusche. Deutsches Archiv f. klin. Med. 1870.— *Bayer*, Weitere Beiträge zur Frage über die Entstehung des Herztons. Archiv für Heilk. 1870.—*Michels*, Ueber die Entstehung des ersten Herztons. Diss. Berlin, 1870.—*Quincke*, Beiträge zur Entstehung der Herztöne und Herzgeräusche. Berlin klin. Wochen. 1870.—*Nolet, E. J. M.*, Zur Lehre der Gefässgeräusche. Arch. f. Heilk. 1871.—*H. Jacobson*, Ueber Herzgeräusche. Berlin. klin. Woch. 1871.—*Giese W.*, Versuche über die Entstehung der Herztöne. Deutsche Klinik, 1871.—*Silver*, On functional regurgitant bruit. Med. Times and Ga-

zette, 1871.—*H. W. Fuller*, in St. George Hosp. Reports, 1871.—*Landois, L.*, Die Lehre vom Arterienpuls, etc., 1872.—*Paul Guttmann*, Lehrbuch der klinischen Untersuchungsmethoden, etc. Berlin, 1872.—*E. Hering*, Ueber den Einfluss der Athmung auf den Kreislauf. Wiener acad. Sitzungsberichte, 1872.—*R. Heidenhain*, Ueber arhythmische Herzthätigkeit, in Pflüger's Arch. 1872.—*Schiff*, Versuche über die Innervation des Herzens. Reported in Centralb. f. med. Wissensch. 1872.—*G. Gianuzzi*, Ricerche exquite nel gabinetto di fisiologie, etc., ibid. 1872.—*W. R. Gowers*, On the influence of posture on presystolic cardiac murmurs. The Practitioner, 1873.—*Ph. Knoll*, Ueber den Einfluss des Halsmarks auf die Schlagzahl des Herzens. Wiener acad. Sitzungsber. 1873.—*H. Wilckens*, Ueber die Rotationsbewegungen des Herzens nach einer direkten Beobachtung am lebenden Menschen. D. Arch. für klin. Med. Bd. XIV. 1874.—*M. Jahn*, Ueber Fissura sterai congenita und über die Herzbewegung, insbesondere den Herzstoss. Inaug. Diss. Erlangen, 1874.—*Nuel*, Ueber den Einfluss der Vagusreaction aufs Herz. Plüger's Archiv, Bd. IX. 1874.—*G. W. Balfour*, Clinical lectures on diseases of the heart. Edinb. Med. Journ. 1874.—*Talma*, Beiträge zur Theorie der Herz- und Arterientöne. Deutsch. Arch. f. klin. Med. Bd. XV. 1874.

The heart is situated in the inferior portion of the mediastinum, is capable of free motion, and is attached only to the large vessels. It forms an angle of 60° with the axis of the body, its base looking backwards, upwards, and to the right side, its apex forwards, downwards and to the left. Its plane side, composed chiefly by the left ventricle, lies on the tendinous portion of the diaphragm, while the anterior convex surface, belonging mainly to the right ventricle, together with part of the auricles, faces the thorax wall, being situated behind the sternum and right and left costal cartilages. The anterior and posterior surfaces are separated inferiorly by the sharp border which projects into the groove formed by the diaphragm and thorax, superiorly by a rounded border, which is completely covered by the lungs. However, the anterior surface does not come in its full extent into immediate contact with the thorax, for between the two, the lungs, which surround the heart posteriorly, and over the sides, project their anterior margins. These margins meet at the level of the second rib, and run parallel to one another, down to the sternal end of the fourth rib. Here they separate, the border of the left lung bending down sharply to the left side, and forming a semi-lunar notch, the incisura cardiaca, which extends to the

junction of the fifth costal cartilage with the sternum, and from here again it sends a tongue-shaped process, the linguala, inwards. This process surrounds the border of the apex, and is inserted between it and the thorax. On the size of this cardiac notch, which, even under normal conditions, is very variable, depends the extent of the heart's surface which can come into direct contact with the thorax wall; and that part alone, which comes into this direct contact, is adapted to our powers of examination on the living subject.

The first requisite for the objective examination of the heart in the living subject is an exact knowledge of the positions of the different parts, not only in relation to one another, but also to their surroundings. Many difficulties oppose an absolutely certain determination of these positions—for instance, the presence of the respiratory contractions and expansions of the lungs, the altering condition of the diaphragm, and the varying quantity of blood in the heart itself. Still these have been overcome, and an accurate groundwork has been laid by Pirogoff and Luschka by means of sections through frozen bodies, and by Joseph Meyer, who has used Gendrin's method of puncture on the dead subject. The inquiry of most practical interest is to determine the parts which are wholly or partially covered by the lungs, to what extent they are covered, and where one should look for the vascular foramina. The following are the results of the anatomical investigations on the subject.

The *right auricle*, of which more than half lies external to the right-hand border of the sternum, and only one-third under the bone, is one of the parts which the lungs completely cover. It extends from the middle of the sternal end of the second intercostal space to the sternal end of the cartilage of the fifth right rib. The *right side of the heart*, which is the main part that lies in direct contact with the anterior wall of the chest, is *least* covered by the lungs. Its smaller division, situated behind the sternum, reaches from the end of the third left rib to the base of the xiphoid process, while the other and larger division extends from the middle of the sternal end of the second left intercostal space to below the middle of the cartilage of the sixth left rib.

Only a small segment of the *left ventricle* lies against the

anterior wall of the thorax, the greater portion looking towards the lateral and posterior wall. The anterior segment extends from the middle of the second to the middle of the fifth left intercostal space, its exterior border corresponding with the third, fourth, and fifth left costo-chondroidal sutures. Of the *left auricle* the only part visible on the anterior wall of the chest is the point of the auricular appendage surrounding the pulmonary artery at the level of the second left intercostal space; the auricle itself reaches as high as the upper margin of the second left costal cartilage, and lies quite far back behind and to the left side of the sternum, being covered by the anterior margin of both lungs, by the ascending aorta and the pulmonary artery.

The *venous foramen of the left side of the heart* is situated in the second left intercostal space, and is completely covered by the lungs. The bicuspid valve lies opposite the third left rib, with its attached border projecting more or less into the second, and its unattached more or less deeply into the third intercostal space.

The *venous foramen of the right side of the heart* lies in a line joining the sternal articulation of the fifth right rib with the lateral end of the cartilage of the first left rib. A line, drawn from the third left intercostal space close at the edge of the sternum to the sternal articulation of the fifth right rib, meets the free border of the tricuspid valve. The middle of this border lies between the sternal ends of the fourth pair of ribs. The *right arterial foramen* is situated in the middle of the second left intercostal space, and a needle, piercing perpendicularly to the middle of the second intercostal space, about $\frac{1}{4}$–$\frac{1}{2}$ in. from the border of the sternum, will meet the semi-lunar valves of the pulmonary artery, especially the attached border of the anterior valve.

The *arterial foramen of the left side* lies behind the sternal articulation of the third left rib and a part of the adjoining sternum. A needle entering perpendicularly, opposite the sternal articulation of the third rib, a little to the left of the mid-line of the sternum, pierces the free border of the semi-lunar valves.

The direction of the *aorta ascendens* corresponds to a line drawn from the sternal end of the third left rib towards the sternal end of the second right intercostal space.

The *vena cava*, too, lies directly beneath the anterior wall of the thorax, namely, external to the right sternal border, reaching from the middle of the first costal cartilage to the middle of the second intercostal space, or even to the sternal end of the third costal cartilage.

The outer wall of the heart, as well as the commencements of the large vessels, is firmly enclosed by the pericardium, a serous sac into which the heart is reflected. The inner layer of the sac is so intimately connected with the muscular wall of the heart, that it forms, as it were, its perimysium externum; the external layer is more or less firmly bound up with all the surrounding structures, inferiorly with the diaphragm, in front with the sternum, and laterally with the mediastinum. In addition to the mediastinum, the tendinous sterno-pericardial ligaments, superior and inferior, help to connect it anteriorly, while posteriorly it is connected by loose cellular tissue with the neighboring structures, the œsophagus, the bronchi, and the bronchial glands. A layer of pavement epithelium lines the inner surface of the sac, giving to it its smooth, serous appearance. The anterior and lateral portions are covered by the pericardial pleura, but not completely, for the left costal pleura runs downwards and outwards from the upper border of the sternal end of the fifth rib, thus not extending to the border of the sternum, while the right costal pleura runs down the sternal border as far as the sixth rib, and in this manner their edges bound a triangle of the pericardium (with its base looking downwards, and its apex directed upwards) which is entirely free from serous membrane. This small space, which derives its importance from its bearing on puncture of the pericardium, lies in the level of the sternal end of the fifth and sixth costal cartilages, behind them and the left border of the sternum, and has a breadth of about three centimetres.

The inner surface of both ventricles and auricles is lined by endocardium, which structure also lays the foundation of the valves by sending duplicatures into the auricles and vessels at

their mouths. In order to comprehend the pathological alterations the endocardium undergoes, it is of importance to know that an accurate histological investigation has identified all the elements of a blood-vessel in it. The cellular tissue which connects the internal surface of the heart with the endocardium supplies the part of the adventitious coat, and allows the entrance of the nerves and vessels. The layer next in order corresponds exactly to the media, contractile cells even having been found in it by Schweigger-Seidel; while the innermost layer with its coat of polygonal epithelium represents the intima. In the semi-lunar valves we find blood-vessels which enter the tissue at the attached margin. The prevailing view at present, in opposition to former ones, is that the auriculo-ventricular valves are supplied, richly supplied, with blood-vessels. The vascular supply of the anterior right nodulus of the mitral valve, of the so-called aortic nodulus, is derived from a network common to it and to the neighboring semi-lunar valves. This accounts for the frequent occurrence of disease which attacks both structures simultaneously, and the fact is deserving of particular attention.

The weight and dimensions of the heart have been the subject of repeated investigations, which, however, have never led to entirely harmonious results. The chief reason of this is, that the same method has not always been adopted, the heart being weighed sometimes with the auricles and part of the large vessels, sometimes without; even the fluid or coagulated contents have not been always previously emptied out. In measurements, especially of the thickness of the walls, particular attention should be given to the degree of rigor, and to ascertain in what phase (systole or diastole) death has occurred; further, the quantity of blood present should be noted; and one should be most careful not to include any trabeculæ in the measurement. After these precautions have been taken, the normal measure to be assumed for the length and breadth of the normal adult heart (of such alone my experience allows me to speak) is between 10 and 11 centimetres. The left ventricle, from the insertion of the aorta to the apex, generally measures 10 cm.; the right ventricle, measured from the pulmonary artery, is generally from 3 to 5

mm. shorter. The thickness of the wall of the left ventricle, at the level of the insertion of the mitral valve, measures from 1 to 1.5 cm., at the apex from 0.8 to 1 cm.; that of the right ventricle, at the level of the tricuspid valve, from 6 to 7 mm., at the apex 3 mm.

The weight of the heart varies normally in the adult (as far as my experience goes) from 250 to 370 grammes (from 8 to 11½ oz). This agrees very closely with the weighings which Blosfeld made on hearts of subjects killed by accidents, his results being an average of 346 grms. for males, and 310 grms. for females. The female heart is a little lighter than the male; with increasing age and constant length of body the weight of the heart increases, as has been demonstrated, both by measurement and weighing, by Bizot, Clendinning and Peacock. According to Clendinning's researches the weight of the heart is to the weight of the whole body as 1:158 in the male, and in the female as 1:149. Blosfeld found for males 1:178, and for females 1:153. According to Bizot's universally accepted measurements (reduced to centimetres by Dusch), the breadth of the heart in men, between the ages of 30 and 49, is 10.8 cm. and the length 9.7 cm.; in women the breadth is 9.9 and the length 9.2 cm. The circumference of the ostium venosum dextrum is 10, and that of the o.v. sinistrum 8.5 cm., according to Luschka, who also found the ostia arteriosa dextrum and sinistrum of an equal circumference of 7 cm. My measurements, which agree with those of Peacock, estimate the o. a. d. as larger. Perls, in opposition to Peacock and Chevers, insisted on making a more accurate distinction between the opening proper to the heart, the ostium cardiacum, and the upper point of attachment of the semi-lunar valves, the ostium arteriosum. Carrying out this distinction for the ostia, he found that, under 40 years old, the periphery of the ostium cardiacum was on an average 6.4 mm. greater than that of the ostium arteriosum, while after this age the relations were almost completely reversed. In the pulmonary artery a slight change towards an equalizing of the two takes place, but never a complete reversal, as in the aorta. The averages which Perls found for the peripheries of these ostia are:

	Under 40.	Between 40 and 50.	Over 50.
Ostium aorticum cardiacum	70.9 mm.	74.6 mm.	74.3 mm.
Ostium aorticum arteriosum	63.5 "	72.2 "	80.1 "
Ostium pulmonale cardiacum	81.5 "	84.9 "	85.7 "
Ostium pulmonale arteriosum	71.9 "	74.6 "	81.3 "
Periphery of the ostium venosum sinistrum	102.5 "	105.1 "	102 "
" " " " dextrum	121 "	122 "	122.8 "

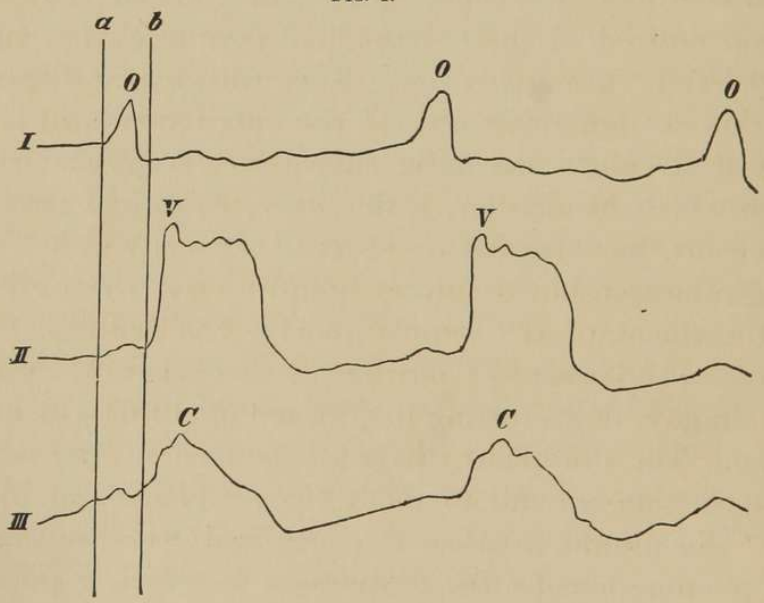
Perls has also measured, by means of a special instrument, the space which the unfolded semi-lunar valves can cover, and thereby has come to the conclusion that in middle age the unfolded valves take up a greater space than that which they must actually cover, while in old age they generally take up a smaller space. Wulff found, too, in the valves of the venous ostia, that the surfaces of the valves considerably outran the surfaces of the ostia, a circumstance of some weight in regard to the question of relative insufficiency.

Under normal conditions, the heart is subject to both active and passive changes of position, the latter accompanying the altered posture of the body. Thus it sinks from 3 to 6 cm. to the left, or from 1.5 to 3 cm. to the right, according as the body is lying on the left or the right side. The still greater displacements, which others have observed, I can only explain, in view of my extended investigations on the subject, as exceptional cases. The heart too retreats slightly, if the sitting or upright position is exchanged for the horizontal. This can be easily observed on the change produced in the cardiac impulse. It is self-evident that the attachment of the pericardium to the centrum tendineum must cause the heart to follow the movements of the diaphragm, which drags it down during inspiration, and raises it during expiration. The difference of level produced in this way in the heart's position extends to about the breadth of an intercostal space. The alteration effected by age is due to the same cause, for in young persons the diaphragm is much higher than in elderly people.

All the other changes of position are active and dependent on the movements connected with the different phases of the heart's action. Auricles and ventricles contract rhythmically (systole), and dilate (diastole), both auricles and both ventricles simultane-

ously. The diastole follows immediately upon the systole. The periodical series of movements is as follows: Towards the end of the ventricular diastole comes the systole of the auricles, lasting a very short time, and followed immediately by the systole of the ventricles; the ventricles then dilate, and a short pause follows, during which the whole organ rests, as both the ventricles and auricles are dilated—the auricles at the close, and the ventricles at the beginning of the diastole. Then follows again the contraction of the auricles, and the motion of the ventricles is repeated, in continuous regularity. During the normal frequency of the pulse, according to Marey's and Donders' investigations, the systole of the ventricles lasts $\frac{2}{5}$ and the diastole $\frac{3}{5}$ of the whole time. If we estimate the duration of one full series of the motions at $\frac{11}{10}$ seconds, the systole of the auricles only lasts $\frac{1}{10}''$. Marey, by means of his cardiograph, has given a very clear, graphic demonstration of both the auricular and ventricular systole, in the following manner.

FIG. 1.



Tracing showing the relations, as regards time, between the systole of the auricles and ventricles and the cardiac impulse (from Marey).

a, Commencement of auricular systole.

b, Commencement of ventricular systole.

O, Highest point of the curve representing the auricular systole.

VV, Highest point of the curve representing the systole of the right ventricle.

C, Highest point of the curve representing the apical pulsation.

The heart, as we shall see, changes both its form and position during each systole and diastole. The auricles undergo least change of all; their contraction beginning at the mouths of the large veins, and moving peristaltically towards the ventricular boundaries. Their longer diameter shortens, and the auricular appendages are flattened out. The ventricles, on the contrary, contract in both directions, in both length and breadth; their antero-posterior diameter alone is lengthened, and the transverse section of the base, which was before elliptical, is transformed into a circle during the systolic increase in firmness. At the same time the ventricular cone, which before occupied an oblique position, now assumes a vertical one (Ludwig). A change of position occurs simultaneously with this change of form. With each systole, the heart, as a whole, moves downwards, and rotates from left to right on its long axis. Although this rotation was known already to Harvey, it did not become an established fact until after the observation made by Skoda and Bamberger, and particularly, in quite recent times, by Wilckens, on persons having ectopia cordis. In addition, Bamberger and Kölliker noticed in their vivisections that the large vessels also straightened themselves out. Kornitzer was the first to show that this straightening out of the spirally twisted commencements of the aorta and pulmonary artery is a necessary mechanism, to which he attributes, not only the downward movement of the heart, but also the spiral-like rotation which it performs.

The phenomenon of the cardiac impulse (or, as Traube has called it, the "apical" impulse, comprehending under apical the whole of the lowermost portion of the heart) is explained by a combination of the changes of form and position just described. On the majority of healthy individuals may be observed a circumscribed bulging of the intercostal space below and to the inner side of the nipple, between the fifth and sixth ribs (seldom between the fourth and fifth), at most one inch broad, and which can be covered with the tips of two fingers.

The explanation of this phenomenon has called forth a series of opinions, of which most are of merely historical interest, and do not need mention here. To this category belongs the theory propounded by Kürschner, in accordance with which the heart per-

forms lever-like movements. Ludwig's theory rests upon a far more solid basis; this observer maintains that, during the systolic change in shape of the heart, its base becomes circular, the apex strives to assume a position at right angles to the centre of this circular base, and in so doing it lifts itself farther away from the base. This lifting of the apex by the systolic hardening of the muscle of the heart is held by Ludwig to be the cause of the apical impulse. Although we must concede that this force mentioned by Ludwig contributes in a measure to the formation of the cardiac impulse, we are yet unable to harmonize his theory with many pathological conditions that come under observation—as, for example, with the absence of the apical impulse in certain valvular defects, like stenosis of the mouth of the aorta.

As a matter of fact, there are other and more important forces at work in producing this impulse, namely, first of all, the principle known in physics as the "*recoil*," and also the systolic *change in position* of the heart. Gutbrod was the first to apply the recoil theory to the explanation of the apical impulse. According to his view, the impulse, which the inferior part of the cardiac wall receives from the blood that flows under a high pressure into the mouths of the arteries, gives rise to a movement of the apex in the opposite direction to that of the current of blood; the process being the same as that which takes place in a Segner's (turbine) water-wheel. Hiffelsheim's experiments on gutta-percha hearts demonstrated the fact that, as regards the recoil, elastic walls behave in the same manner as those which are not elastic, and that the intensity of the recoil is directly dependent on the thickness of the wall, on the diameter of the opening through which the fluid escapes, and on the amount of the fluid. Bamberger's objection, that the contractile force of the heart must exert against the recoil an active resistance of sufficient strength to overcome it, is thus shown to be groundless. A much more serious objection is that which Chauveau raised. This observer noticed that, in horses, the apical impulse was still perceptible after he had placed ligatures around the superior and inferior venæ cavæ; in other words, the impulse was still perceptible in hearts that contained, as he supposed, no blood—a fact which could not be made to harmonize with the recoil theory. M. Jahn,

however, in repeating these experiments, found that the ligature of both venæ cavæ did not immediately cut off the supply of blood to the heart; on the contrary, the left ventricle continued for some little time to receive blood from the pulmonary veins and to drive it into the aorta.

FIG. 2.

No physical objections stand in the way of the application of the law of recoil to the motions of the heart. There can be no doubt, however, that the other force mentioned, *i. e.*, the change in the position of the heart, also contributes its influence, as Kornitzer first demonstrated. In regard to the motion of the heart downwards, and to its rotation about its long axis, as a result of the systolic straightening of the large vessels, it must be remembered—as Wilckens very properly insists—that, in the closed thoracic cavity, where the anterior chest wall offers some resistance to the act of rotation, this combined motion can only take place in the following modified manner: while the upper part of the long axis undergoes displacement backwards, the lower part approaches nearer to the wall of the chest. The apical portion will, therefore, be brought into an oblique position pointing forwards, and at the same time, owing to the downward motion of the heart, it will press downwards against the tissues filling the intercostal space. Hence all the different forces—the changes in form and position, and the recoil—mutually aid each other in producing the apical impulse.

The graphic tracing (fig 2) of the cardiac impulse shows not only the short upward stroke belonging to the contraction of the auricles, but also a well-marked elevation, which likewise belongs to the beginning of the systole, and is produced by the closure of the auriculo-ventricular valves. The vibrations thus produced in the valves afford an explanation of the tremor—observed even in healthy individ-

uals, in whom often the apical impulse cannot be distinguished—which is confined to the region between the third and sixth costal cartilages of the left side, and to which Traube has given the name of "valvular impulse," to distinguish it from the apical impulse. The mechanism of the closure of valves explains, as Wintrich and Traube were the first to show, the origin of the sounds of the heart. On putting the ear to the apex of the heart, we at once hear two sounds, one duller, longer, and less distinct, the other higher, shorter, and more sharply defined. An extremely short interval divides the duller, longer, so-called "first sound" from the higher and shorter "second sound." A longer interval ensues between the second sound, and the following first sound; this is "the pause." Whilst the heart is in diastole, the pointed valves hang down loosely in the ventricles, in the form of a hollow cone. Owing to increasing pressure in the ventricles, to the elastic expansion of their walls, and especially to the contraction of the auricles, the points of the valves approach closer and closer to one another, till they finally touch. At the commencement of the systole the musculi papillares also contract. From each papillary muscle tendinous cords proceed to the different pairs of free borders (those facing each other) of the valves; and as the points of the valves, even before the systole, were so close as to touch one another, the tension, into which the tendons are thrown during the systole, by the contraction of the papillary muscles, brings them still closer together, till the contact is firm and perfect throughout. And, further, these muscles not only prevent the valves slipping back into the auricles, but they act on them in such a manner that they (the valves) project conically into the ventricle, instead of merely lying horizontally over the opening. As soon as the chambers of the heart have emptied their contents into the large vessels, they slacken, and would allow the blood to flow back from the aorta and pulmonary artery, if it were not caught in the hollows of the semilunar valves. The sigmoid valves either lie very slack during the systole, or actually touch the walls of the vessel. But they are thrown into an increasing state of tension (with its maximum at the end of diastole) by the rising pressure of the columns of

blood on them, so that their margins come into contact, and in the shape of a three-rayed star they cut off the connection between the large vessels and the ventricles.

The changes in tension which both the auriculo-ventricular and the semi-lunar valves undergo in the transition from one phase of action to another (from diastole to systole, or the reverse), and the vibrations of the tense membranes thus induced, are the most important causes of the sounds of the heart; that is to say, these sounds are mainly membranous. This is almost universally admitted in the case of the shorter, clearer, and more defined second sound. Not alone that it was heard during the diastole of the heart, but the position, too, where it was best heard, over the mouths of the large vessels, rendered the connection between it and the vibrations of the semi-lunar valves highly probable. But Rouanet, in 1832, was the first who experimentally proved that by sudden tension of the semi-lunar valves a sound was produced, and that after the destruction of these valves the second sound disappears at the mouth of the aorta. Since then the vibrations produced in the semi-lunar valves by a transition from a state of minimum tension, during systole of the heart, to a maximum tension, brought about by the pressure of the blood column on them during diastole, have been unanimously accepted as the cause of the second sound.

As might be expected, even against this explanation some raised their voices, and later Leared tried to derive the sound from the blood itself. Talma, too, has quite lately endeavored to establish Leared's idea on an experimental basis; but neither Leared nor he have adduced sufficient proofs. The weak point in Talma's arguments appears to me to lie in his identifying the terms "short murmur" (kurzes Geräusch) and "sound" (Ton), although in practice we can clearly distinguish a murmur *along with* the sound, and the distinction can by no means be made to rest on the duration of the perception, as Talma presupposes. A murmur, however short, is still a murmur.

As regards the first sound, no one any longer doubts that it is synchronous with the systole of the ventricles. But as to how it is produced, views are very much at variance. Rouanet, Bouillaud, Skoda, and Traube refer it to the vibration of the

auriculo-ventricular valves; but doubt has been thrown anew on this view, already combated by Stokes and others. In Ludwig's and Dogiel's experiments made on dogs, the first sound was still heard after every means had been taken to render the tension of the valves impossible—after all the arterial and venous trunks had been ligatured and almost all the blood expelled from the left side of the heart. This brought Ludwig once more to side with Stokes, who explained the first sound as a result of muscular contraction, as a muscular sound. It is beyond all question that a muscular contraction can produce an acoustical sensation exactly similar to the sensory perception of the first heart sound. In a case of one-sided tetanus of the sterno-cleido-mastoid of long duration, I have had opportunity to listen to this muscular sound, and, by comparing it with the first sound of the heart, have convinced myself and others of the similarity of the sensory perception. Still, the supposition that the first sound is membranous is well-grounded, and conditions enough have already been given for the production of sound-giving vibrations on the auriculo-ventricular valves, in the transition from the low tension at the end of a diastole to the maximum tension during the ventricular systole. A whole series of pathological facts, collected and pointed out in this connection by Traube and Bamberger (the explanation of which, however, would compel us to anticipate the section on pathology), are explicable exactly by assuming the first sound to be membranous. There appears to be so little connection between disease of the cardiac muscle and the intensity of the first sound in the different cases, that from a clinical point of view it is impossible to regard this as a mere muscular sound. In regard to the genesis of this first sound, we may surely consider the contraction of the wall of the ventricle as auxiliary, and in the same category may be placed the vibrations of the tendinous cords, but we should not regard it as a pure muscular sound. The explanation of the longer duration of the first sound, in contradistinction to the second sound, may, perhaps, be found in this prolonged vibration of the papillary tendons, while, on the other hand, the semi-lunar valves are thrown into vibration only at the moment of their closure, and are immediately restored to equilibrium by the

pressure of the blood. We cannot consider Talma's attempt to explain the first sound as a blood murmur as more fortunate than his similar interpretation of the second sound. In the large vessels near the heart, too, we still hear two sounds, the second of which has its origin at the mouths of the arteries, and is transmitted along them. But the first sound is by no means to be considered as the transmitted first sound of the heart, as it is still to be heard after the first heart sound has become inaudible or has been replaced by a murmur. The first vascular sound is also membranous, and is produced by the vibrations excited in the walls of the vessels by the sudden increase of tension due to the systole of the ventricles. Although Talma's interpretation of this first vascular sound as a blood murmur has here much more appearance of probability, still I cannot yet regard it as proved. It is better in the meantime to hold to the view that we can distinguish six different sounds, according to the manner of their origin—two which owe their existence chiefly to the vibrations of the auriculo-ventricular valves, two to the vibrations of the semi-lunar, and two to the vibrations of the walls of the large vessels.

Methods of Examination.

The methods which we adopt to ascertain the symptoms of the normal condition of the heart, or the variations therefrom in case of disease, consist in *inspection*, *palpation*, *percussion*, and *auscultation*. The following are the results given by the several methods for the normal condition:

Inspection of the left thoracical region shows no difference between it and the right side, either in regard to the arching of the wall or the thickness of the layers of skin and muscle. But in the majority of healthy people we perceive a rhythmically occurring protrusion of the fifth, seldom of the fourth, intercostal space, exactly between the mammillary and parasternal lines, internal to and below the nipple, with a maximum breadth of one inch, and two finger-tips deep. This phenomenon, the visible impulse of the heart, may be absent even in quite healthy people, if the soft parts overlying it

are too thick, or if the lung be abnormally spread over the heart; finally, if the apex beat behind a rib. The time of life, too, exerts quite normally a great influence on the position of the impulse, due to the situation of the diaphragm—in young children rendering it perceptible higher up, but still within the mammary line, though not unfrequently external to the nipple (in one case I saw it 3 cm. outside), while in old people it is to be seen lower down. The posture of the person to be examined also alters the position of the impulse in hearts which are otherwise quite normal. As the heart is movable to either side, when the body lies on the left side, we see the impulse move towards the left, and towards the right if the body lie on the right. The motion towards the right is more restricted, but still quite discernible. When its action is excited we see the movement of the heart in several intercostal spaces, and often, especially in lean individuals, a movement traversing the whole region from above and the right, towards below and to the left. The place the wave has just passed sinks for an instant, while the next part rises. Along the left border of the sternum we can observe that the third, fourth, and more rarely the fifth, intercostal spaces sink synchronously with the cardiac impulse, though this be quite in its normal position. The explanation Friedreich offers, that the parts of the heart which lie along the sternum assume a smaller volume than the rest, is founded on an arbitrary assumption, and is even more unsatisfactory than Bamberger's theory, according to which the intercostal spaces, over which the heart has just passed from above downwards with greater rapidity, yield to the atmospheric pressure, and consequently fall in.

In *palpation*, the finger is laid over the region of the heart, and experiences a slight elevation. It is impossible to describe the normal degree of resistance, a matter which can alone be learned by practice. One must not forget, however, that the force of the impulse varies according to the degree in which the lung covers the apex of the heart, according as the patient breathes lightly or heavily, according as he is calm or excited, so that every variation from the normal is not to be regarded immediately as pathological. Palpation is not only of use in estimating the

resistance of the impulse, but serves also as a means of confirming as pulsatory the movements on the anterior surface of the thorax and in the neck, which give the eye an impression of pulsation, and of thus distinguishing apparent from real pulsations. The palpation of the pulse will be specially treated along with the consideration of phenomena dependent on the vessels.

By means of *percussion* we can mark out the boundaries of the anterior portion of the heart, which lies against the thorax wall—either the part uncovered by the lungs, or the whole. The former of these gives a perfectly dead, the latter only a dulled percussion sound. Thus we can distinguish over the heart a region of *absolute dulness* and one of *relative dulness* ("Herzmattheit," and "Herzdämpfung"), or, to use the English terminology, *superficial* and *deep-seated* dulness.

The best mode of percussing is with the fingers, which can be adapted to each intercostal space, and at the same time they can estimate the resistance of the part percussed, thus combining palpation and percussion. If a plessimeter be used, the best form is a vulcanized india-rubber one (that of Seitz), the narrow part of which will fit into an intercostal space. The individual under examination must lie on his back, or sit upright. On account of the above-mentioned displacement caused by lying on the side, this posture must be avoided. The percussion must be light or heavy, according as we wish to hear the superficial or the deep-seated dulness. Where the lung covers the heart strong percussion is necessary to render the dulness of the underlying parts audible; while by light percussion, where the heart is uncovered, we avoid disturbing the equilibrium of the adjacent pneumatic parts. Although the estimation of this relative dulness undoubtedly possesses great value, yet it cannot be concealed that the absolutely accurate determination of the boundaries, superiorly and to the right side, is an impossibility, and that the manifold transitions from one degree of dulness into another can only be recognized by the most practised ear, and that consequently by this method, using all possible precautions and care, we can only attain to an approximate estimate of the size of the heart. Hence determination of the extent of absolute dulness is sufficient for all practical purposes, since, moreover,

all alterations in the extent of the whole heart are attended with proportionate alterations in the extent of the parts uncovered by the lungs. Thus one should always endeavor to obtain the boundaries of the region of relative dulness of the heart; but when it is a matter of hasty general diagnosis, it is better to begin at once with the extent of the absolute dulness, and rest content with this. The best method of finding this latter, is to begin by noting where the apical impulse is visible or palpable. By this means we get the left inferior boundary. Next we find the upper limit by percussing down along the left margin of the sternum. Starting from the left mammillary line, we can then determine exactly the breadth of the dulness, and draw the left boundary line. Over the sternum itself, we get no absolutely dull sound, though the heart lies directly under its lower end. Since the left lobe of the liver is only separated from the sharp inferior border of the heart by the diaphragm, we cannot directly estimate the full extent of the inferior boundary of the area of absolute cardiac dulness. But still we ascertain a part of its circumference, or, as the case may be, of its end, by means of the tympanitic sound of the stomach, where it is sharply divided from the loud lung sound, on the left side; and from the right, by prolonging the line which divides this loud lung sound from the absolute dulness of the liver. If the resulting outline figure of the area of absolute cardiac dulness be projected on the breast, we obtain a triangle in most cases; less frequently, an irregular quadrilateral, when the anterior border of the left lung has more than one curve, thus making the boundary line appear as if bent at an angle. This triangle is so placed that its apex coincides with the junction of the fourth left rib and sternum, its right side runs parallel to the left border of the sternum as far as the sixth rib, and its basis corresponds to the upper border of the sixth rib, while its left border runs either in an outwardly convex curve, cutting the fifth rib, or almost in a straight line down to the point of the impulse, forming, as it were, the hypothenuse of an almost right-angled triangle. The height of the triangle is from 5 to 7 cm., and its greatest breadth from 6 to 8 cm.

The area of relative cardiac dulness sometimes begins as high up as the second intercostal space, especially in childhood, when

we can see and feel the heart's impulse between the fourth and fifth ribs; it is always present in the third intercostal space, and measures from 2.5 to 6.5 cm. in breadth, for it extends as far as the middle line of the sternum. In the fifth space, on the right side, the dull percussion sound can be plainly heard over the inferior part of the sternum, which borders on the xiphoid process, and about two centimetres beyond the right sternal margin. The total breadth of the dulness at this level is from 8 to 13 cm. Kobelt has shown that the area of cardiac dulness is dependent on the age of the individual and the circumference of the thorax, but not on the total length of the body.

If we compare the boundaries of the area of absolute cardiac dulness with those of the area of relative dulness, we shall find that a girdle of dulness, weaker above and to the right, more intense to the left, spreads itself around the triangle of superficial dulness. This is so distributed, that if we project it on the thorax, a cone is produced with its blunt apex generally in the third intercostal space, near the left margin of the sternum, its right boundary generally keeping close to the right sternal margin in the fourth space, and in the fifth passing slightly beyond it; its limit to the left generally remains inside the mammillary line, while a line which unites the superior limits of the liver with the point of the heart's impulse forms its base.

In *auscultation*, which is best executed with the stethoscope, we hear two sounds, as already explained. It only remains to point out where the sounds of the different ostia are to be best heard. From the detailed account of their relative positions, which we have given in a former paragraph, it is clear that they lie within a narrowly bounded space, alongside, sometimes even behind, one another; and moreover that they are all of them more or less covered by the lungs, and so it is only possible to auscultate the sounds of the pulmonary artery and tricuspid valve directly above where they originate, and even then but approximately. Thus, too, we auscultate the sounds produced by the mitral, not at the point corresponding to this valve, but in the region of the apex; that of the tricuspid on the right border of, and over, the sternum at the level of the fifth rib; the sounds of the aorta are to be sought by following the course of the ascending

branch, on the right margin of the sternum, between the second and third ribs, and not where they are produced by its semi-lunar valves behind those of the pulmonary artery; the sounds of the pulmonary artery are best heard in the second left intercostal space close to the border of the sternum. In the majority of healthy people the sounds of the auriculo-ventricular valves beat in the trochaic rhythm, with the accent on the first (— ⏑), while the sounds of the semi-lunar valves of the large vessels follow the iambic metre, with the accent on the second (⏑ —). But it is not necessarily a sign of disease to hear the pointed valves beating in iambic measure.

Physical Symptoms of the Diseases of the Heart.

The same methods which enlighten us on the normal conditions of the heart also afford us the symptoms of its pathological bearing.

First: *Inspection* makes us aware of the abnormalities of the anterior walls of the thorax. If the volume of the heart is increased to a considerable degree, or the pericardium extensively swelled by an abundant exudation into its cavity, we may notice a protrusion of the region of the left breast, which lies over the heart, *i. e.*, the part between the left margin of the sternum and the left nipple, from the third rib down to the seventh; this occurs most frequently in youth while the chest is still flexible. The abnormal protrusion can extend in exceptional cases, particularly in large pericardial exudations, as far as the sternum, and beyond to the right side, thus compelling the retreating left lung to withdraw completely to the upper part of the thorax. This protrusion (*voussure* of French authors) is not to be confounded with asymmetry of the thorax arising from other causes, and especially with the protrusion of the left side due to curvature of the spine, more particularly the form of skoliosis, where the middle dorsal vertebræ are convexly curved to the left. It is only in cases of extraordinarily large pericardial exudations that we find the intercostal spaces themselves projecting convexly forward.

Inspection affords us other important symptoms in regard to

the changes in the apical impulse. This can either be *completely wanting*, or can occur *at an abnormal point*, or exhibit itself *over a greater surface*. We have already learned to account for *its absence* under normal circumstances ; for example, owing to an extraordinarily thick coat of soft parts on the wall of the thorax, very narrow intercostal spaces, or abnormal condition of the incisura cardiaca of the left lung, especially enlargement of the lingula. Amongst the pathological causes we may reckon : *a*, emphysema of the left lung ; *b*, abundant secretion of an elastic or oily fluid in the pericardium ; *c*, adhesion of the heart to the surrounding pericardium ; *d*, certain valvular defects, such as stenosis of the ostium aortæ and stenosis of the ostium venosum sinistrum, if they are well marked.

The cardiac impulse is seen in other than normal conditions (always, of course, provided that the individual to be examined is made to lie on his back, since, as before stated, if he lies on one side, the impulse falls to that side)—

1. *To the left of the mammillary line*, extending an unusual distance in that direction, even as far as the anterior axillary line. The necessary conditions for producing this are : either, *a*, *horizontal position of the heart*, due to abnormal elevation of the diaphragm, without regard to the causes that may have produced this elevation ; or, *b*, *abnormal increase in size and bulk of the muscle of the heart* from valvular defects ; or, *c*, *displacement to the left*, whether it be owing to abnormal pressure on the mediastinum through a collection of elastic or oily fluid, or a tumor in the right pleura, or a vacuum caused by the shrinking of the left lung. Right-sided pleural exudations generally cause a dislocation of the liver rather than of the heart.

2. *Outside the mammillary line to the right.* The impulse of the heart may be seen *on the right border of the sternum between the third and fifth ribs*, corresponding in this case, however, to a section of the base of the heart, not to the apex. This is brought about by dislocation of the heart produced through an accumulation of elastic or oily fluid in the left pleura. The pulsation we see on the right side is rarely to be attributed to the apex, for the heart in its dislocations does not alter its parallels. It is only in congenital transposition of the viscera that

the entire heart, including the apex, may be seen occupying the right half of the chest; this, however, is an occurrence of great rarity. In a case of contraction of the right lung and vicarious enlargement of the left, I found after death the heart displaced to the right, but in an almost vertical position, with the apex looking straight down, below the sternum. The pulsation of the part which lay furthest to the right was to be seen close to the right border of the sternum between the fourth and fifth ribs.

3. The impulse of the heart is seen *lower down*, between the sixth and seventh, or seventh and eighth ribs. This deep situated impulse is at the same time usually located to the left of the mammillary line, and happens in cases of decided increase in size of the left heart, due to valvular disease—for example, disease of the aortic valves. Frequently we merely find a deep-situated impulse, but not outside the mammillary line. This is especially the case in those enlargements of the heart which originate in an abnormal resistance in the peripheral circulation, such as atrophy of the kidneys and sclerosis of the aortic system.

4. The impulse of the heart may be found *higher up*, between the fourth and fifth ribs, or even between the third and fourth ribs, especially in children. This form, too, usually occurs outside the mammillary line. The cause is the abnormal elevation of the diaphragm, which in one series of cases is pushed up by ascites, meteorismus, upward dislocation of the left lobe of the liver, or tumors; in others it is drawn up by contraction of the left lung, or, finally, it may assume a higher position, owing to strongly developed kyphosis, or kyphoscoliosis. The kyphosis must have attained to very large dimensions in order to be considered the cause of this displacement; for the great majority of kyphotic cases do not affect the position of the impulse at all. Pregnancy, too, although at this period the diaphragm is raised, still seldom elevates the heart's impulse. I cannot concur with Walshe's opinion that pericarditis ever causes upward displacement.

5. The majority of cases of displacement of the cardiac impulse is accompanied by an alteration in the size of the surface over which it extends. When hypertrophy is the cause of downward displacement, or when the heart, even without under-

going hypertrophy, comes in direct contact with a larger surface of the thorax wall, the impulse is visible in several intercostal spaces, and increases considerably in volume—in a word, it becomes diffuse. The intensity of the diffuse vibration is directly proportional to the amount of hypertrophy. Dilatation and hypertrophy of the right ventricle can displace the pulsation far to the right; in one case, which came under my notice, it lay 8 cm. beyond the right margin of the sternum.

Instead of the protrusion, which is the normal result of the impulse of the heart, a retraction can sometimes be perceived synchronously with each systole, and in some cases extending to the inferior portion of the sternum and the ribs attached to it. Only such retractions as are confined to the point of apical impulse (and not till this point has been defined by percussion), whether they extend to the sternum or not, are of any diagnostical value ; while retractions of intercostal spaces (which the beginner must not confound with those due to respiration) in the vicinity of the impulse, whether this be present in its normal position or absent, have no significance, and belong to the large class of normal phenomena. If, for instance, the apex beat behind a rib, we very frequently see a contraction of the next upper space. Skoda was the first to point out that simultaneous adhesions between the two folds of pericardium, and between the pericardial and costal pleuræ, are the causes of this phenomenon. However, it is easy to convince one's self, that obliteration of the pericardium can occur without any appearance of this systolic contraction ; and, on the other hand, Traube has demonstrated that a single cord of connective tissue between the heart and pericardium is alone sufficient to produce this symptom. Again, I myself have found a stringy adhesion between the anterior parts of the visceral and parietal pericardia on making the post-mortem examination, where during life there was no trace of a systolic retraction. From this many-sided evidence we may conclude that it would be a great mistake to assume an extended obliteration of the pericardium on the ground of a systolic retraction which was confined to the region of the apical impulse. This diagnosis can only be established with certainty when a *systolic* retraction of the *lower portion of the sternum*, along

with the *left inferior costal cartilages*, and a *diastolic recoil* of these parts take place. Friedreich first pointed out the conditions of this sum of symptoms, and properly assigned, as the main cause, the firm adhesion of the inferior surface of the heart with the diaphragm, accompanied by strong action of the heart; for the diaphragm is drawn up by the systolic shortening of the heart, and consequently it draws inwards those parts of the thorax which correspond with its own insertions.

Besides the abnormal variations already described, we may meet pulsations which are quite unconnected with the apex, and originate in the right side of the heart, or proceed from the large vessels. When, for instance, the diaphragm is unnaturally sunken, a pulsation is usually visible on the border of the left arch of the ribs, exactly isochronous with the apical impulse. It moves downwards and inwards during a deep inspiration, but never crosses to the right of the middle line. This pulsation is designated in general terms "epigastric," and indeed a narrow strip of the epigastric space does pulsate. But still, to avoid confusion with another form, which occurs uniformly right in the middle of the epigastrium, it is better to designate the first-mentioned as "parepigastric." This proceeds from the right ventricle, which, when the diaphragm sinks, assumes a more horizontal position, and projects its sharp border into the groove between the anterior thorax wall and the diaphragm. The fact that in auscultation we hear both cardiac sounds over the pulsating part with the same intensity as over the rest of the heart, and that palpation proves this and the apical impulse to be isochronous, renders the origin from the heart a certainty; in addition, we can in some cases even feel the very muscular hardening. The presence of this pulsation proves nothing but the sunken position of the diaphragm. Where the lungs are large, it may occur (in Holland, for instance, where the lungs are mostly very large, it is of almost universal occurrence) without the presence of the slightest change in the heart, especially in the volume of this organ. Hypertrophy of the right side of the heart, however, can increase the sunken condition of the diaphragm, by increasing the weight, and in this light the above-mentioned pul-

sation may in many cases be regarded as a symptom of right-sided hypertrophy of the heart.

The strictly epigastric pulsation has an altogether different signification. It is the result either of increased innervation of the abdominal aorta or cœliac artery, or else of the improved conduction of the normal pulsations of these vessels. This latter is caused by an increased firmness of the overlying solid parts, whether it be due to a hypertrophied liver, or to the presence of some abnormal tumor springing from the stomach, the retroperitoneum, or any other structure in the epigastrium. Over this pulsation we can only auscultate one sound.

When the liver itself "pulsates" (as in the case of a regurgitation into the hepatic veins, due to tricuspid insufficiency), and does not merely conduct the pulsations of the underlying vessels, then the phenomenon is not confined to the epigastrium; on the contrary it is generally observable over the whole extent of the liver, more especially to the right of the middle line.

Other pulsations connected with the diagnosis of heart diseases can be perceived on the large trunks of the aorta and pulmonary artery, also on the jugular and other cervical vessels. In exceedingly rare cases, and when the subjects are very emaciated, pulsation may be observed in the second intercostal space, close to the right border of the sternum. This can occur under otherwise perfectly normal conditions; it is isochronous with the heart's impulse, and originates in the aorta ascendens. Another visible and palpable pulsation, generally extending over the two first intercostal spaces and likewise close to the right border of the sternum, is due to a dilatation of the aorta ascendens. This dilatation is either connected with an insufficiency of the aortic valves, or occurs as an independent disease of this vessel.

Of more frequent occurrence is a circumscribed pulsation arising from the pulmonary artery, and exhibiting itself one or two centimetres from the left border of the sternum, and also, like the others, in the second intercostal space. From palpation we learn that the stroke occurs in synchronism with the diastole of the heart. If this pulsation corresponds to the normal position of the pulmonary artery, it must be accounted for either by an

abnormally thin thorax, or by a layer of solid parts, such as pulmonary tissue or tumors, which serve to transmit the impulse better. If the pulsation is further removed from the border, say three or four centimetres (the distance varies as the heart has been more or less displaced to the left), it must be regarded as a pathognomonic symptom of contraction of the left lung.

We often observe pulsations of the jugulum when the heart's action is merely excited, but not otherwise altered; and these are especially frequent when the aortic arch lies high up. There is no special signification attached to this, unless it occurs when the individual under examination is perfectly at rest, and unless we can feel it. Under these conditions it points either to an anatomical anomaly (as such I have seen it twice, in the form of a longer and more tortuous arteria innominata than usual), or, as is more usual, it points to dilatation of the aortic arch, which occurs either in connection with insufficiency of the valves of the aorta, or occurs primarily as an aneurism. Constant pulsations in the large cervical arteries, the carotids, is only visible when the action of the heart is considerably increased; while neither the large internal and external jugular veins, nor the small subcutanea colli or thyroidea, etc., are ever visible except when they shine through a very soft skin, as we meet it in some women and children. Consequently we can diagnosticate anomalies in the movements of the contents of the cervical veins (especially of the internal jugular, which usually runs down along the posterior border of the sterno-mastoid), if we see them projecting prominently in an over-filled condition. These anomalies consist in obstructions to the flow of blood towards the right side of the heart, which may be caused by general mechanical difficulties, such as diminished driving power of the heart, diminution of the elasticity of the lungs, and other forces which would tend to raise the pressure on the intrathoracic venous trunks; it can also happen that the obstructions are of a more local nature, such as mediastinal tumors, which directly prevent the veins from emptying themselves. A closer examination is then necessary to establish the special hindrance. While an abnormal dilatation is only a general sign of existing disease, which may arise from the most widely

different causes; on the contrary, a movement in the cervical veins, perceptible both to sight and touch—in a word, a *pulsatory* movement—has a much more specific signification. This pulsatory character is of the most decisive importance, and the first precaution we must take is to see if the motion does not consist of a mere swelling which occurs with every increase of the intra-thoracical pressure (*i.e.*, during expiration)—we must, in fact, make certain that it is not connected with respiration. To exclude respiratory influence alone, is, however, not enough, since movements, which are synchronous with the heart's movements, may be visible in the veins of the neck, without being thereby real pulsations. Every sharp systole of the auricle can by its retreating waves set the contents of the over-filled veins in vibration, and produce undulations in them. And, on the other hand, for a certain diagnosis it is not sufficient to establish merely a pulsating character in general, since a vein, especially the internal jugular, owing to its close relations with the carotid, can exhibit a pulsation which has only been transmitted to it from the artery. Consequently, to establish the venous pulsation as such, and as originally and directly produced by the heart, we must shut out all the possibilities of error mentioned. To effect this, it is only necessary to compress the vein, and thus temporarily cut off the peripheral supply. If the cause of the pulsation be a central one, and if it be not a mere vibration of the over-filled vein, the part beyond the compression ought to continue pulsating, since this peripheral compression has no influence on the auricle and right ventricle. If it was merely undulation, the motion would now cease; if it lay in the pulsations of the adjacent artery, it will be visible above the compression, where the vein is still full, and will cease to appear below, where the vein is empty. It is clear that compression of the carotid would be alone sufficient to decide this point; but it is not always easy to remove the full vein from the artery. This phenomenon of pulsations, which is to be seen both in the large and small cervical and facial veins, was known already to Allan Burns. The investigations of Geigel, von Bamberger and Friedreich, after a lively discussion of the subject, have set it now beyond all ques-

tion. The first condition necessary for the production of a blood-wave running from the heart to the jugular veins, is *inability of the venous valves to close.* These valves normally hinder regurgitation, and where this insufficiency does not exist, no venous pulse is visible. The closure of these valves, and of the circular muscles of the right auricle, is the cause which prevents us from seeing and feeling retreating waves at every auricular systole. While this barrier remains intact, no retreating wave, in whatever way it may be produced, can pass out beyond the sinus venosus. Thus the venous pulse can be produced through insufficiency of the jugular valves alone, however this latter may have come about, whether through anatomical or functional changes; and the direct diagnostic conclusion we are compelled to draw is necessarily insufficiency of the venous valves. But it is self-evident that the venous pulse produced by this defect will be more distinct and intense the larger the retreating wave is, and the greater the force with which it is driven into the veins. These conditions are most completely fulfilled by a defect in the closure of the tricuspid valve, when, with every systole, a portion of the blood, instead of being directed from the heart into the pulmonary artery, is driven by the force of the ventricle into the right auricle, the venæ cava and innominata, and past these out into the cervical veins. But even against the force of the regurgitation, which accompanies tricuspid insufficiency, the venous valves can remain a long time closed. So in some cases this defect of the tricuspid can occur without causing a cervical venous pulsation, and, on the other hand, a most pronounced venous pulse can occur without defect of the tricuspid. This latter may be the result of frequent stagnation of the circulation, due to long fits of coughing (as in bronchial catarrh), affecting the closing power of the valves; or it may be an unclosed foramen ovale, accompanied by insufficiency of the mitral valve, as in Reisch's celebrated case: and thus with every systole a large quantity of blood is driven through the left auricle into the right auricle. I have observed a perfectly analogous case, and the specimen is still in my possession.

Whenever the venous pulse is fully developed, it can not only be seen but also felt; and if the finger be placed on it we feel a

rhythmically repeated stroke. When the phenomenon is only slightly expressed, the pulsation can be seen, but can no longer be felt. Geigel has proved that increased pressure on the vena cava inferior is capable of making the cervical veins pulsate. The venous pulse may be a transitory phenomenon and disappear (especially towards the end of life), though all the conditions for its existence are still present, though even a simultaneous insufficiency of the tricuspid and venous valves is present. This is rendered comprehensible by the fact that the quantity of blood and the rate of circulation both decrease with age, thus impairing the driving power immensely. Cases in which pulsations have been felt to the right of the linea alba in the vena cava inferior are of the rarest occurrence, while a well-developed case of cervical pulsation is almost always accompanied by a similar one in the liver, perceptible both by sight and touch.

Bamberger's and Friedreich's sphygmographic investigations were the first to establish the distinguishing characteristics of the venous and arterial pulse waves.

FIG. 3.

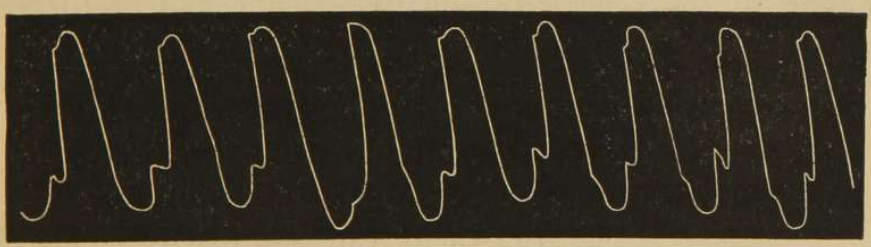


FIG. 4.

ANADICHROTOUS PULSE IN THE INTERNAL JUGULAR VEIN (AFTER FRIEDREICH).

Close inspection of the sphygmographic curve shows that it is anadichrotous. The first and shorter rise in the ascending limb is the result of the contraction of the auricles, and is consequently presystolic; while the second, longer one, corresponds to the systole of the right ventricle. Friedreich has noticed dichrotism as well in the descending limb, and thinks that here

the slight rise at the end should be referred to waves reflected from the inner surface of the right ventricle itself. The presence of a marked degree of anadichrotism or catadichrotism, or the presence simply of marked dichrotism depends upon the energy of the contractions of the auricle and right ventricle, on the one hand, and, on the other, upon the degree of tension in the walls of the veins, or, in other words, on the extent to which they yield.

It should be here remarked, that the sphygmographic tracing taken from the pulsation of the liver does not always appear in the same form. Its ascending limb is indeed generally dichrotous, while we can only now and then perceive a slight dichrotism on the descending limb.

A pulsation which is limited to the lowermost portion of the jugular vein, the so-called pulsation of the bulb, must, as regards its significance, be carefully distinguished from one which is visible more or less along the whole extent of the vein. This kind of pulsation occurs when the valves of the jugular vein, instead of occupying their usual position at the end of the vein, are inserted high up, and when at the same time there is insufficiency of the tricuspid valve. We cannot here attribute the pulsation to any failure of these valves to close properly, for they may even close with such distinctness as to produce a distinct tone upon the ear of the listener—a tone to which Bamberger has given the name of "sound of the jugular valves."

The last phenomenon we have to mention in regard to the cervical vessels, is one to which especial attention has been drawn by Friedreich—this is, that when the pericardium has been obliterated, a sudden collapse is perceptible in the hitherto full veins, occurring simultaneously with the diastole of the heart. When the adherent diaphragm rises with the shortening of the heart during systole, it draws the ribs and sternum inwards, but in the diastole these suddenly regain their position through their own elasticity, and thus at the same time as that in which this movement (which we can both see and feel) takes place, the veins instantly empty their contents. When, along with the adhesion of the pericardium, we find an extensive mediastinitis, which drags upon or compresses the large venæ innomi-

natæ during inspiration, the usual conditions are reversed—the cervical veins, and especially the sinus venosus of the internal jugular, now exhibiting swelling, instead of during expiration. (Kussmaul.)

Palpation in many ways controls ocular impressions, and is consequently used at the same time as inspection. It frequently happens that, by means of the touch, we can find the cardiac impulse in cases where inspection has failed to discover it. In the majority of emphysematous cases the covering of the lung renders the impulse imperceptible to the eye, while, by laying our fingers over it, we feel it at once. On the other hand, when an exudation of oily or elastic fluids in the pericardium is the object which prevents our seeing the movement of the heart, it is here equally imperceptible to the touch. Palpation of the impulse of the heart makes us acquainted especially with its variations in intensity. There is indeed no universally applicable standard for this intensity, but with our fingers we can very well notice any abnormal resistance at the apex, such as that which accompanies hypertrophy of the left ventricle. For those cases, in which the strength of the impulse is so increased that the whole anterior thorax shakes with it, and the head of the auscultator is raised with every systole, and falls back with every diastole, here palpation is not necessary; but just in those cases of secondary hypertrophy of the left ventricle arising from abnormal resistance in the periphery (such as contraction of the kidneys or sclerosis of the aorta), where no special changes are discernible in the position or extent of the heart's impulse, increased resistance is of the greatest diagnostic value. At the same time we must not overlook the conditions which could entail an increased resistance, without any alteration in the heart itself, as for instance, better transmission of the stroke by solid substances whether in the cavity or in the walls of the thorax. This indeed obtains for all pulsatory movements in the thorax, which palpation confirms as such, especially for the pulsations of the aorta ascendens and pulmonalis already mentioned

in the paragraph on inspection. In the case of the pulmonalis especially, palpation supplements the inspection, since, besides the motion visible in synchronism with the systole, we can also feel the closure of the valves answering to diastole, whenever the necessary conditions are present for increased tension in the vessels.

Quite as important as this increase of resistance, is to establish, by palpation, a progressive *decrease* in the power of a once normal heart, from circumstances depending on changes in the substance, such as fatty degeneration, etc., or on a pericardial exudation. The investigator's finger can also discern abnormal vibrations which arise within or upon the heart. When, through hindrances to the stream, owing to changes in the lumen of the channels, whether in the outlets of the heart or in the vessels, eddying movements are produced in the blood, these may attain to such an intensity as to be not only heard, but even felt. This is especially palpable in stenosis of the ostium venosum sinistrum as a whirring sound, which Laënnec has termed "the cat's pur" (*frémissement cataire*); but it can occur in any valvular disease, and is, according to the disease, systolic or diastolic. But palpation can discern, not only those sounds which originate within the heart, but also those which are produced upon its outer surface. When the smooth pericardial surfaces become rough from the fibrinous deposit which takes place as a result of inflammation, and then rub against each other, a rubbing or grating sensation will be felt by the finger. Indeed, it is often an easy matter, by the sense of touch alone, to distinguish this friction sound from the so-called fremitus (*frémissement*). Both of these, however, are chiefly to be discerned when there is increased activity of the heart's action; and at times they vary so greatly in intensity that they may even disappear for a brief time, although a moment before they were quite distinct. To distinguish a pericardial from a pleural friction sound, it is necessary to show distinctly the dependence, in the former case, of the sound on the movements of the heart. The means for distinguishing the two sounds will be more fitly described under the head of auscultation.

This peculiar vibratory sensation (*frémissement*) can also be

felt on the larger vessels, especially on the right internal jugular. The impression is exactly similar to that which we receive from laying one of our fingers on a pianoforte wire, when a note has been struck, and is due to the vibrations of the walls of the vein, imparted to them from the vibrations of the blood. The consideration that the veins have a different-sized lumen along their course and at the termination in the sinus venosus, is quite sufficient to explain the existence of the eddies. Consequently we often feel the vibration even in perfectly healthy people. But slackness of the venous walls and a comparative emptiness of the vessel are the chief conditions which produce an increased intensity, this being the reason why it is chiefly to be heard in chlorotic and anæmic cases; not that it is by any means confined to these diseases, or that it can be regarded as a symptom universally indicative of anæmia. The muscular contraction which takes place in the action of turning the head on its axis, narrows the lumen of the vein, and thus strengthens the sound. For the same reason, light pressure of the finger on the vein will also increase the sound, and even call it into existence, in cases where before it was not perceptible. However, the phenomenon is not altogether confined to the veins of the neck, for we sometimes hear it or feel it in the peripheral veins. Of these, the femoral vein is the one in which the phenomenon is most often observed, not only in constitutional troubles which result in imperfect filling of the veins with blood, and a corresponding flaccid condition of their walls, but also in local conditions or disturbances which produce changes in the lumen of the vessel.

The palpation of the arteries is of great importance in diagnosticating heart diseases; on the one hand, from the judgment we can form thereby, as to the condition of the walls of the vessels; and on the other hand, more important, from the changes which the pulse undergoes as a result of alterations which have taken place in the circulation. The immediate result of diseases of the arterial walls is decrease of elasticity, especially when inflammatory processes have acted on their tissue. This loss of elasticity impairs the resistance the artery can offer to the forward-pressing wave of blood, and thus the vessel undergoes a lasting tension which ultimately dilates it. But as the arteries

are fixed in the direction of their length, the dilatation must result in windings and twistings, and if this assumes large proportions, we can even see it on the relatively superficial vessels, while the slightest trace of it is always palpable. In itself this tortuousness of the arteries signifies nothing more than a loss of vascular elasticity, and can occur to almost any extent, under the name of aneurysma cirsoides, unaccompanied by any atheroma. Endoarteritis, however, is the commonest cause of this loss of elasticity. The inflammation acts chiefly on the intima coat, but is still accompanied by considerable thickenings in the adventitia. The resistance of the vessels is thereby increased, and consequently we feel a peculiar hardness of the wall in addition to its tortuousness. It is highly important to be aware of this condition of the vascular walls, because it creates hinderances in the circulation, the mechanical effects of which must eventually reach the heart; and because, further, similar changes may at the same time attack the aortic valves. By means of Marey's sphygmograph we are enabled to observe these alterations in elasticity, and record them graphically; and Landois has ascertained, by experimental research, the changes which take place in the tracing of the pulse in atheroma. The normal pulse is catadichrotous, *i.e.*, the tracing exhibits a moderately sharp ascent, corresponding to the dilatation of the vessel, and a descent, corresponding to its contraction. The latter is varied by two or more minor ascents, caused partly by the recoil wave, which is excited, as Landois has shown, in the vessels during the systole, and begins with the aortic valves, and partly by the vibrations of the elastic walls. The notches in the curve due to elastic vibrations are altered (*i.e.*, either diminished or obliterated) in diseases which affect the vascular elasticity; and the elevations corresponding to the recoil are also affected, since they, too, are partly under the influence of this elasticity. After some time, general extension of the process produces secondary changes in the heart, through which, in the same space, a larger quantity of blood flows into the aorta with every systole. This, and the diminution in height of the elevations arising from loss of elasticity, produce a curve for atheroma which differs from the normal curve principally in the circumstance that a plateau-

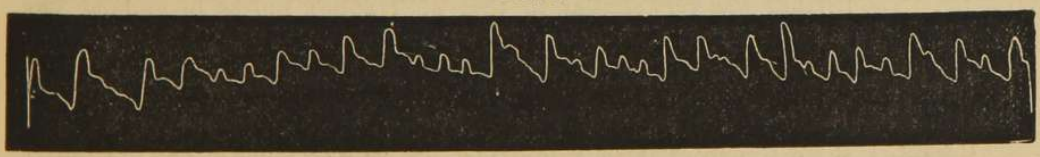
like elevation shows itself on the ascending limb—the curve, in other words, is anachrotic—while on the descending limb there is only a very small elevation, produced by the recoil.

FIG. 5.

In the vascular changes, just described, we have found that tortuousness and hardness were easiest distinguished by palpation, whereas the graphic method brought the variations of the pulse most clearly before our eyes. But diseases of the valves and cardiac muscle also produce changes in the character of the pulse, which are directly perceptible to the touch; these are chiefly such as concern its duration, frequency, and tension. It is perfectly plain that similar and as marked variations in the pulse can also be produced by other diseases besides heart and pericardial diseases. A description of all the causes which produce the various divergences would require a separate treatise on the pulse, and is here quite out of place. We shall accordingly confine ourselves to those forms which occur as symptoms of heart disease.

Alterations of rhythm occur as *inequality*, when waves of even dimensions do not follow one another—as *irregularity*, when the waves do not follow at regular intervals.

FIG. 6.



Unequal pulse.

FIG. 7.

Unequal and irregular pulse.

The irregular pulse may be subdivided, according as entire contractions of the heart are completely wanting and no eleva-

tion of the artery at all can be felt (pulsus deficiens), or as some beats of the heart are too weak to make the wave palpable at the periphery where we usually feel for it (p. intermittens). Sometimes it feels as if, between two equal and regularly following elevations, a dissimilar one were inserted (p. intercurrens or incidens); sometimes two or more beats of the heart are combined in forming a double or trebly compound pulse (p. coturnisans). Amongst the last-mentioned variations from the normal rhythm especial interest attaches to that form which Traube has described under the name of *pulsus bigeminus*. This consists of two short beats, followed by a long pause, and each of the two beats corresponding to a contraction of the heart, in contradistinction to a dichrotous pulse, in which the double beat belongs to a single contraction of the heart. Traube's conclusion, based on his experimental investigations, is that this form occurs when the heart is freed from the influence of the spinal division of its inhibitory nervous system, while the cardiac division, still intact, is irritated to a greater degree than usual.[1] This pulse may consequently be regarded as a very unfavorable prognostic symptom. The *pulsus alternans* (a secondary form of the bigeminus, and also described by Traube) differs from the ordinary rhythm in as far as a low pulsation follows on every high one, and a pause after the low pulsation is longer than that preceding it.[2]

The following case, which came under my treatment, seems worth communicating. The rhythm was very similar to that of Traube's pulsus bigeminus, but still it is not perfectly analogous. Otherwise, however, as may be seen from comparing the cardiac impulse, it was a phenomenon dependent on the heart's action, and cannot be referred to mere changes in the vascular walls. This is not affected by the fact that the tracing was only taken on one side—namely, from the radial artery of the paralyzed arm. The case itself was as follows:

Welling, aged 28, a painter, was dismissed nine years previously from military

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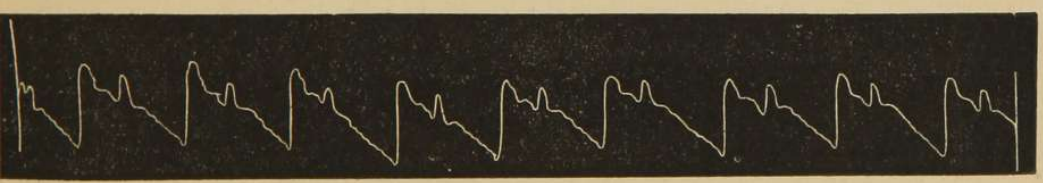
[1] Ph. Knoll disputes this explanation of the pulsus bigeminus, because he saw it continuing after he had injected a large dose of atropine. He seeks to explain it by the premature occurrence of another systole, before the heart, which has emptied its contents in the preceding vigorous systole, has time to take in its normal quantum of blood in diastole.

[2] Comp. Berliner klinische Wochenschrift, 1872, No. 16.

service, on account of "palpitations of the heart," as he alleges. During these nine years he was generally able to work, till one day, three months before being received into hospital, he fell down suddenly, losing consciousness, but without any symptoms of paralysis. After three weeks' confinement to bed he went to work again. Yesterday, October 30th, felt ill while standing on a scaffold, came down to the ground, but then fell, and was paralyzed on one side. He was received into hospital in the following condition: Powerfully built, large bones, and well-developed muscles. Mucous membranes rather pale. Patient is confused, but not perfectly unconscious. When loudly addressed he answers, incomprehensibly indeed, but still showing that he has heard the question. Right angle of the mouth lower than left, puts out his tongue straight, soft palate normal, motion of the eyes intact, pupils evenly dilated. The left arm is completely and the right partially paralyzed. Sensibility likewise diminished on the right side. Thorax normally curved, the type of respiration abdomino-costal; twenty respirations to the minute. Rhythm regular. Cardiac impulse strong—can be seen and felt between sixth and seventh ribs, within and without the mammillary lines of both sides. Rhythm of cardiac impulse is abnormal—on one impulse follows a second weaker one, divided by a longer pause from the next beat than from the previous one. The dulness begins on the lower border of the third rib, is 9 cm. high, 14 broad, extending externally to the left mammillary line. With the stethoscope over the apex of the heart, a loud systolic and weaker presystolic blowing sound is audible. Over the ostia of the larger vessels the second sound is double. The left radial artery of medium calibre, slightly tortuous; the pulse full, and 56 beats to the minute. The annexed curve gives the rhythm.

Fig. 8.



(No tracing could be taken of the pulse on the unparalyzed arm, on account of incessant convulsive movements.) In regard to other organs, the lungs were normal, the liver enlarged. Urine contains a great deal of albumen. Next day the pulse grew more rapid, the respirations intermittent, and the rhythm of the pulse less distinct, the paralytic symptoms unchanged. On the third day patient died. Of the results of the autopsy I shall only mention those relating to the brain and heart: Calvarium normal, vessels of the dura mater injected; after removal of dura superficial extravasations of blood are visible, about a centimetre in breadth. Pia mater easily removable. Gyri flat, and sulci badly expressed. On the right side the substance of the brain at a depth of one centimetre is infiltrated with blood, and as we go deeper the extravasation becomes more plentiful. The right lateral ventricle is completely filled with a coagulum. Corpus striatum and thalamus opticus on this

side are converted into a pulp of blood. The left side is free from extravasation. The arteries of the Sylvian fissure show nothing abnormal.

The heart is 11 cm. long, 12 broad, and 27 in circumference. The free border of the mitral valve is thickened, wrinkled, and beset with a fringe of long excrescences. The opening allows three fingers to pass through easily. The points of the papillary muscles are converted into connective tissue. The borders of the aortic valves are slightly thickened, and along the line of closure beset with small excrescences. Other valves are normal.

Changes of the normal rhythm, too, are generally connected with anomalous frequency of the cardiac pulsations. Both the slow pulse (p. rarus), and the hurried (p. frequens), and at the same time irregular, are most commonly observed in diseases of the cardiac substance and of the vessels which supply it (the coronary arteries), and more rarely in valvular diseases and general atheroma. But these irregularities also occur independently of any perceptible pathological anatomical changes in the heart, expressing pure neurosis, as in violent reflex excitement of the vaso-motor centre. Thus, in a case of intercostal neuralgia, I have observed a long duration of an unequal and irregular pulse, which, when accompanied by an accidental murmur, aroused suspicion of valvular disease. Whether their occurrence in infectious diseases, especially typhoid fever, is unconnected with the coarser changes of the cardiac substance, remains doubtful. Changes of frequency without similar ones of rhythm are brought about by more general influences—age, posture (horizontal or standing), time of day, excitement, fever, etc.

The changes in *celerity* affect the relations of duration in which the diastole and systole stand to one another. The abnormality consists in the shortening of the time during which the vessel remains dilated by the wave of blood driven into it with the cardiac systole; the explanation is, that the maximum of dilatation is quickly reached and equally quickly followed by the contraction of the vessel. Vierordt, and, still more accurately, Landois, have expressed the normal relations in celerity for the different arteries in actual numbers; no estimates, however, have yet been made of these relations in disease. Insufficiency of the aortic valves is the disease, *par excellence*, which

unites all the conditions favorable to the production of this form of pulse. The physical causes are, on the one hand, the enormous quantity of blood which the hypertrophied and abundantly-filled left ventricle drives into the vessels with each systole, and, on the other hand, the easy regurgitation allowed by insufficient valves. The reversed physical conditions prevail in stenosis of the ostium aortæ ; in this condition, in which a smaller quantity of blood is driven with each systole into a tense system of vessels, the pulsus tardus is encountered.

By the force we have to counteract in trying to compress the artery, we can measure the changes in its *resistance*, or in the *tension* of the pulse, and thus distinguish between a hard and a soft pulse. These changes relate to the tension of the artery during its diastole and systole. The tension is directly dependent, as a rule, on the amount of blood in the vessels, and yet it does not always bear a fixed relation to it. The same affections of the venous and of the arterial cardiac ostia produce exactly opposite effects upon this tension. Thus, for example, stenosis of the left ostium venosum renders the pulse small and soft, while stenosis of the aortic ostium renders it small and hard.

Kussmaul has described, under Griesinger's name of "*paradoxical*" pulse, a peculiar form which has been repeatedly observed in cases of excrescent mediastino-pericarditis. For inflammation sometimes attacks not only the serous, but also the fibrous stratum of the pericardium, and produces fibrous formations projecting into the mediastinal connective tissue, and thus throwing tendinous strings from the reflection of the pericardium to the large vessels, such as the aortic arch and the venæ innominatæ. In such cases it often happens that the traction which the sternum exerts on these cords during inspiration produces a displacement and stretching of the vessels, sometimes even a compression ; this renders the pulse very small and sometimes even stops a beat. The peculiarity of this clinical phenomenon is that, without any appearance of disturbance in the cardiac action, we have an apparently irregular pulse, which becomes smaller, or disappears during inspiration. The irregularity is, however, merely apparent, for the pulse acts in the same way during every inspiration.

Though the explanation of this clinical symptom by the participation of the mediastinum in the pericarditis is well founded, still we must not ignore the fact that this pulse can be produced by other diseases besides this one. Apart, too, from the influence of increased negative pressure on the left ventricle, arising from stenosis of the respiratory tract, and displaying itself, according to its intensity, by diminution or interruption of the pulse during inspiration, the pulsus paradoxus can be produced by chronic pericarditis alone, without any complication of the mediastinum; cases of this kind have been observed by both Traube and Baeumler. For these cases, also, Baeumler endeavors to find an explanation in the alteration in intrathoracic pressure and in the resulting variations in the amount of blood which the vessels contain.

Percussion enables us to detect the abnormal symptoms produced by alterations in the volume or position of the heart. Owing to the great freedom of lateral motion allowed to this organ, even under normal conditions, alterations take place in its position, when one turns over on his side, after lying on his back. So the first necessary condition for forming a correct estimate of the results of percussion, whether disease be present or not, is to put the patient in the recumbent position (lying on his back) during examination. A second and equally important precaution, in making a diagnosis of the changes in the area of superficial dulness, is to test the movableness of the anterior borders of the lungs. When one or both of these borders is adherent, where the lung is contracted, or where that part which lies next the pericardium is infiltrated, then we may find the area of superficial dulness (according to the point at which the adherence has taken place) greater or smaller than normal, but still unaccompanied by any change in the size of the heart. The height of the diaphragm, too, must always be estimated and taken into account; for the extent of the anterior surface of the heart coming into contact with the chest is increased or diminished according as the diaphragm stands higher or lower, and thus variations in the

size of the area of absolute dulness are produced, which are not at all the result of variations of the heart's volume. Although this *apparent* atrophy and hypertrophy of the heart, produced by the causes mentioned above, may be disregarded and excluded from the diagnosis, the symptoms of *actual enlargement*, on the other hand, vary in their significance according to the region in which they preponderate. When the left side of the heart is enlarged, both the superficial and deep-seated dulnesses are intensified below and to the left; the same takes place over the sternum and beyond its right border when the right side of the heart is enlarged. It must not be forgotten that the absence of all percussional symptoms does not at all exclude the possibility of hypertrophy. Even very considerable hypertrophy (especially of the right side of the heart) can escape detection by percussion, if the lungs are emphysematous; and that, too, when along with palpation we percuss heavily, and proportionately to the resistance. And besides, where the right heart is enlarged, the limits of dulness on percussion may be somewhat extended toward the left, owing to the more horizontal position which the organ then assumes; this is observed, for instance, in stenosis of the left ostium venosum.

The shape of the normal area of dulness becomes somewhat modified when there is fluid in the pericardial sac. The shape assumed will then be a triangle, with its obtuse apex pointing upwards, and its base below; for the heart, on account of its higher specific gravity, occupies the lower space, and the collection of fluid takes place at first in the upper part, corresponding to the junction of the pericardium and the large vessels. If the exudation is large, provided there was no pre-existing adhesion of the edges of the lungs, the apex of this triangle may reach up to the first intercostal space; usually it does not reach beyond the second space. The right side extends downwards from the third rib and out beyond the right sternal border, and may even go as far as the right nipple; the left side stretches down over the left nipple till it reaches the axillary line. Height and breadth of both regions of dulness are altered, as well as their figure. Gerhardt has called attention to the influence exerted by change of posture in these relations as well as in the others. He

has shown that in the standing posture the region of superficial dulness is more extensive than in the recumbent posture. Small quantities of fluid, so much even as 3 or 4 oz., may be present in the pericardium, though we are unable to detect it by percussion.

Diminution in the area of superficial cardiac dulness is, in the majority of the cases where it occurs, merely apparent, and is produced by interference of the lungs, especially in connection with a sunken diaphragm. When the stomach is greatly distended, the transmission of its tympanitic sound induces an upward displacement of the limits of the deep-seated dulness, and thus makes it appear as though these were reduced. When the pericardium contains gas (pneumo-pericardium), the superficial dulness entirely disappears, and is replaced by a sound, which is generally metallic in character. Since fluid very soon follows the gas, a line may be found with a sharp metallic sound on one side, and a dull sound on the other; the position of this line will naturally shift as the patient changes his posture.

When *auscultating* the heart in disease, we shall meet with many alterations in those sounds which we have already studied in their normal condition. The alterations affect either the purity of the sounds, their clearness, the limits of their extent, their intensity, or their timbre; but the most important point is, that we shall find the sounds accompanied or replaced by murmurs.

The *definition* of the sounds depends chiefly on the uninjured vibratory powers of the valves. Even trifling structural changes in these, which have no material influence on their function, can make one or the other cardiac sound appear less distinct and defined to the ear, and hence give it the name of indefinite. The changes in clearness are quite different, and have, like those of intensity, a complicated origin. Besides vibratory power, the valves must also have a certain tension, and the parts which lie over the heart must be good conductors of sound. Any varia-

tion of a single one of these conditions will render the cardiac sounds clear or dull, plainly or feebly audible. As a general rule, a clear sound is plainly heard, and a dull sound feebly; but still a dull sound may be plainly heard, since bad conduction will dull even an intense sound, and, on the contrary, an originally weak sound can be rendered distinctly audible by improved conduction. All sounds of the heart are rendered abnormally loud, or strengthened, when the work of the whole heart is increased, as, for instance, after physical exertion. The first sound is heard particularly loud at the apex, when the difference of tension in the auriculo-ventricular valves, between the end of the diastole and the beginning of the systole, is very great; this is most marked in stenosis of the left ostium venosum. In cases of very marked hypertrophy of the left ventricle, the first sound is very loud over the apex, accompanied by a metallic click (*cliquetis metallique*). The muscle sound probably contributes in a measure to this increased loudness. We hear the second sound very often much louder than usual over the ostia of the large vessels. Provided we exclude the possibility of an improved conducting medium, such as infiltrated pulmonary tissue, or a vessel brought into close contact with the chest, by contraction of the lung overlying it, this loud second sound must be construed as a symptom of greater tension in that vessel, over the ostium of which it is heard. The second sound over the aortic ostium is, as a rule, relatively louder than that over the pulmonary artery. Hence the increased intensity of the second sound of the pulmonalis implies an increased tension in the pulmonary artery, due to the existence of an obstruction in the lesser circulation.

Increase of the intensity of the second aortic sound points to abnormally high tension in the aorta, as results from increase of power in the left ventricle, provided, of course, that the semilunar valves are still capable of performing their functions.

When the sounds display an increase in intensity, they are also audible over a greater *area*. To hear the normal cardiac sounds on the back (especially in the left interscapular space) is only possible in the case of slenderly-built children and women, while the sounds of a generally hypertrophied heart are audible on the back in every case. The fact that we can hear the cardiac

sounds over a large surface in phthisical cases depends entirely on the presence of infiltrated pulmonary tissue as a better conducting medium.

All sounds of the heart are abnormally *weak*, when this organ contracts but weakly, as in the case of disease of its substance due to inflammation or fatty degeneration. The sounds are especially dull when the conduction of the sounds is deadened—by extensive emphysema of the overlying parts of the lungs, by intervention of fluid between the pericardium and heart, by œdematous infiltration of the external supracardial soft parts, or, finally, by a large panniculus adiposus.

Abnormal weakness of the first sound over the apex occurs either when the tension of the auriculo-ventricular valves is very small, which may be caused by general disturbances, such as typhus or typhoid fever, since these produce changes in the contraction of the papillary muscles, or it may occur when the difference between the final diastolic tension of these valves and their commencing systolic tension is extremely small, as happens when the aortic valves are defective. In this case, the first sound over the apex can disappear completely.

Abnormal weakness of the first sound on the large vessels occurs when their tension is diminished; in the case of the aorta, for instance, it occurs when the mitral valve is defective, and does not allow so much blood to be propelled into the vessel by the contracting ventricle. The second sound is either heard feebly or not at all over the large vessels, when there is stenosis of their ostia.

A change in the *timbre* of the heart sounds, especially one to a metallic timbre, will be observed when at the same time a large space filled with air, and provided with smooth walls, happens to be in the immediate neighborhood of the heart; such a space may be found in the pericardium itself (pneumo-pericardium), or at times in the left pleural cavity (pneumo-thorax), or, most rarely of all, in a pretty large cavity, occupying the substance of the lung, and lying in close proximity to the heart. In pneumo-pericardium, fluid usually soon makes its appearance in the pericardial sac in addition to the air, and so gives rise to splashing sounds of a metallic character, resembling the sounds

produced by water when it is allowed to fall in drops from a certain height into a hollow vessel. I once heard these sounds with peculiar distinctness in a case of pneumo-pericardium, due to the existence of a communication between the pericardial sac and the stomach, through the diaphragm (from ulcer of the stomach).

Reduplication or division, as the case may be, is a peculiar anomaly of the cardiac sounds, which is observable under both normal and pathological conditions, and may affect both sounds; it is characterized by the existence of a rhythm, which was known even to Bouillaud, and by him described under the name of *bruit de rappel*, on account of its resemblance to the military signal of that name.

We speak of *reduplication* of the sound when additional short pauses intervene between the separate sound-elements; on the contrary, we mean by *division* the transition of one sound into the other without any interval at all. Potain was the first (see bibliographical list) who called attention to the physiological frequency of the phenomenon. He showed that these functional physiological reduplications and divisions are dependent upon the influence which the different phases of respiration exert upon the relations of the intrathoracic pressure, and consequently upon the circulation. Thus, for example, this influence shows itself in the fact that expiration retards the flow of blood in the veins, while inspiration enfeebles the action of the left ventricle, and so diminishes the pressure in the aortic system. If either force preponderates, both auriculo-ventricular valves, or both semi-lunar valves, instead of closing synchronously, will close one after the other, thus giving rise to a reduplicated sound. This accords with the fact that it is possible, by artificially hindering or facilitating the breathing, to alter the relations (as regards time) which the reduplication of the sounds bears to the respiratory phase. The phenomenon, when the symptom of disease, can originate in asynchronism of the closure of the separate valves, perhaps even of the points of the valves, and especially in a more jerky and less energetic contraction of the papillary muscles, arising from nervous disturbances. The quick, transitory manner in which the symptom often occurs, and its disappearance as

soon as the heart's action revives, speak in favor of this explanation. But differences of tension, totally independent of nervous and respiratory influences, can take place in the aorta and pulmonalis, especially in connection with the second cardiac sound, producing asynchronism of the closure of the valves, and thereby a division of the second sound. To Geigel is due the credit of having suggested that the division of the second sound is induced by the inequality of tension in the aorta and pulmonary artery, caused by the unequal quantities of blood they contain. This is often heard in stenosis of the left ostium sinistrum, though (as Guttmann properly observes) not nearly as often as Geigel asserts. In fact, in the last cases, if the patient exert himself somewhat, we hear a diastolic murmur instead of the divided sound. Wherever the transition from a doubled or divided sound to a murmur is very slight, the pathological significance of the symptom is undoubtedly that of a murmur. This is, in all probability, too, the case, when the interval between the two parts of the double sound is particularly long.

The rarest cause which has been adduced for these doubled sounds is an actual asynchronism of the contraction of the two ventricles. This does not appear at all probable from the anatomical arrangements of the muscles ; but still it is possible, and has been witnessed by observers, such as Leyden. In this case we have a *doubled cardiac impulse* as well.

That form of reduplication of the second sound, in which the second of the couplet follows immediately on the second ventricular sound, is entirely different both in its mode of origin and in its diagnostic value. It is caused by the diastolic recoil of the chest after it has been drawn in by the systole ; this latter process, as Friedreich first pointed out, is a symptom of chronic obliterating pericarditis.

All cardiac *murmurs* must be divided into two classes, namely, those that have their origin *inside* the heart (endocardial), and those which arise *on its external surface* (exocardial). The *endocardial* are subdivided into *organic*, or those which are connected with a palpable change in the valvular apparatus, or in the cardiac substance ; and *functional*, or those which take place independently of any perceptible change in

these tissues. The mode of origin is probably the same for all murmurs; they originate by oscillations in the blood itself (the so-called "eddies"). It is doubtful whether unequal vibrations of the valves can also produce murmurs. The old theory, that murmurs are produced on the rough surfaces of the valves and vessels by friction with the blood, is physically untenable since the investigations of F. Neumann, Poiseuille and others on the motion of fluids in closed vessels have proved that the extreme peripheral layer of the fluid merely moistens the walls, and must consequently be at rest. This view, too, is in perfect accordance with a series of well-known pathological facts; as, for instance, that no murmur is to be heard in extensive atheroma of the vascular walls (provided the atheroma is not complicated with alterations in the lumen of the vessels), and further that the maximum intensity of a murmur is not to be heard where we should have to suppose the greatest friction. Thus this frictional origin of the murmurs must be regarded as wholly disproved—while, on the other hand, the oscillations, which take place in the blood, during a transition of the stream through openings of an unequal lumen, produce a friction between the particles of the fluid itself. In the transition from a narrow part a murmur originates immediately in the succeeding part. After Corrigan had referred the origin of the proper cardiac murmurs to movements in the blood, and had initiated an experiment which was to have proved this, Kiwisch, Heynsius, Weber, and later Chauveau, Marey, Thamm and Nollet furnished the experimental proof necessary to establish the oscillation theory on a firm basis. Especially the investigations of Heynsius and Nollet, in opposition to the opinions of Weber, Chauveau and others, have proved with certainty that *the primary vibrations which take place in the blood itself* (the so-called *tourbillons*, which originate during the transition of the stream from a narrower into a broader part of the vessel) are the real causes of the murmurs, and that the influence of tension and lateral pressure on these motions was nothing, while the influence of the *rapidity of the stream* was very great. The fact, which Weber discovered, and Nollet and Thamm confirmed, that, if the stream be only rapid enough, murmurs may be produced even in a glass tube of

uniform diameter, quite disproves Chauveau's theory that the origin of the murmur lay in a *veine fluide* with its molecular oscillations, or, as Paul Niemeyer expresses it, in the compression of a stream through a narrow opening ("Press-strahl"). Clinical experience, too, harmonizes with the physical basis, and lays especial weight upon *the rapidity of the stream.* All murmurs which arise in the heart are intensified by exertion, and weakened when the heart's power decreases. In cases of stenosis of the left ostium venosum, we can sometimes only succeed in hearing the murmur after making the patient exert himself vigorously, and even the loudest murmurs become inaudible towards the end of life, when the activity of the heart has begun to fail. It is possible that the undoubted influence, which change of posture may produce in the audibleness of the endocardial murmurs, may be connected with changes in the rapidity of the stream.

It is with perfect correctness that Sidney Ringer and others have observed that, in some cases, when the patient is in the horizontal posture, a loud murmur is audible, which completely vanishes, or becomes very indistinct, when the vertical posture is assumed. In general, however, it appears to me that the sitting posture is most favorable for hearing these endocardial murmurs. But in dubious cases it is better to let the patient exert himself a little, and then examine him in different postures, before giving one's final opinion on the presence or absence of a murmur.

After the presence of the murmur has been fully established, we must next determine, in order to give it its proper clinical significance, whether it is exo- or endocardial. The following symptoms will serve as data for making this distinction: all endocardial murmurs are exactly simultaneous with one or other of the cardiac sounds, being either systolic, diastolic, or both; the exocardial, on the other hand, are heard after or between both sounds, and hang behind; the endocardial are *of a various character*, blowing, breathing, scratching, or rasping; the exocardial are always rasping, however much the intensity may vary. Exocardial murmurs are generally heard at first over the base of the heart, afterwards at the apex and over its whole extent; endocardial do not increase so much in extent, being more local and

fixed; exocardial, further, if at all intense, can always be felt, which is not the case to such a degree with endocardial murmurs. Exocardial murmurs may also be heard synchronously with the movements of the heart, without there being any affection of the pericardium. Those murmurs which are produced by the rubbing of pleural surfaces upon one another in the vicinity of the heart, generally vary in intensity with the movements of respiration (Skoda).

When it has been decided that the murmur belongs to the endocardial class, the next thing is to learn whether its origin is organic, *i. e.*, the result of anatomical changes, which have produced disturbances in the cardiac functions, or functional and accidental, *i. e.*, independent of coarse, anatomically perceptible changes. The functional murmurs are almost always of the same blowing and sighing character: they occur most frequently over the left ventricle, and are heard as often over the base as over the apex of the heart, but in many cases their greatest intensity is exactly over the ostium of the pulmonary artery. Almost all coincide with the systole, and are dependent upon it. The most important difference, and the one which alone affords perfectly reliable evidence, is, that the organic murmurs are almost always accompanied by corresponding symptoms of cardiac disturbance, that they in fact bring with them regularly more or less extensive alterations in the size of the heart, while no such change is perceptible in connection with functional murmurs. In the meanwhile we must not forget, that on the one hand changes of volume which are actually present, dilatation of the right auricle for instance, can escape detection by percussion, owing to complications such as emphysema of the lung; and that, on the other hand, *temporary changes of volume, which afterwards disappear*—for example, the dilatation we have in strongly developed chlorosis—can occur without any coarse anatomical changes of the vascular apparatus. This shows that it is not sufficient to take cognizance merely of the secondary changes of volume, but that on the contrary we should direct special attention to the intensity of the second vascular sound (in the aorta and pulmonary artery). It should also not be forgotten that, at the apex of the heart, murmurs of an endocardial character may be heard, the origin of

which is to be referred to the lungs (Wintrich's aspiration murmurs).

When all doubts of the organic nature of the murmur have been set aside, the next step, in order to ascertain its more precise signification, is to determine exactly the cardiac phase with which it is synchronous. A skilful practitioner will be at once able to decide, from the rhythm, whether the murmur is systolic or diastolic. The safest method, however, for deciding this is, while auscultating, to lay the finger on the impulse of the heart, or, if this be not palpable, on the carotid artery ; it is clear that the radial artery would not serve this purpose, as it does not beat synchronously with, but a little later than, the cardiac impulse. But we meet with organic murmurs which are audible not during the systole alone nor during the diastole alone. The fact is, all organic murmurs last longer than normal sounds, and although many murmurs, notwithstanding their longer duration, allow us to distinguish both the short interval between the first and second sounds, and the longer interval between the second and following first sound, still we find others which are not sharply confined to one or other of the cardiac phases, but extend into the succeeding interval ; for example, a murmur can be protracted from the diastole to the next systole, so that the first cardiac sound comes in immediate connection with the murmur. This very so-called presystolic murmur has great importance attached to it, since it is a specific symptom for stenosis of the left ostium venosum. It is a matter of choice, if any one likes to imitate Gendrin, and distinguish a peridiastolic as well as a presystolic murmur, and a perisystolic one, when a short murmur extends into the interval between the first and second sounds ; but these distinctions have no claims to practical worth.

Many physicians lay great weight on ascertaining whether a cardiac sound is to be heard as well as the murmur, since in special cases we can from this draw conclusions as to the degree in which the valves are affected. We have already mentioned Gendrin's method of raising the ear slightly from the disc of the stethoscope during auscultation, and thus rendering the sound more and the murmur less audible, and we can recommend it for doubtful cases of this kind. However, the distinction is seldom of any practical importance ; for, as Friedreich has shown,

in most affections of the mitral valve, even when the degeneration is far advanced, we can hear a cardiac sound along with the murmur. (When the aortic valves are insufficient, the intensity of the second sound in the carotids is the simplest and safest aid in diagnosis.)

After we have determined the cardiac phase with which the murmur is synchronous, to localize it still closer we must look for the position of the maximum intensity. From what we have already said of the positions, which were most suitable for auscultating the places where the different sounds originate, it is evident, that, if we hear a murmur in its maximum intensity over one of these positions, the murmur originates here as well. For instance, a murmur with its maximum intensity at the apex is referred to the mitral valve; but if it is most audible on the fifth right sterno-costal symphysis, we refer it to the tricuspid; when at the border of the sternum in the second right intercostal space, it is assigned to the aortic valves; and to the valves of the pulmonary artery, when in the same space on the left side. For the interpretation of the different murmurs we may lay down the general rule, that murmurs which are heard loudest over the position for auscultating the auriculo-ventricular valves, are to be referred to insufficiency of these valves, if systolic, but to stenosis of the same if diastolic or presystolic; when the greatest intensity of a murmur is over an arterial ostium, it is referred, if systolic, to the semi-lunar valves of this ostium, but if it is diastolic in the same place, or in the direction in which the stream of blood flows, it denotes insufficiency of the semi-lunar valves. I have purposely added the words "in the direction in which the stream of blood flows," since murmurs produced by insufficiency of the semi-lunar valves are in most cases heard more distinctly over the sternum than over the arterial ostia. In fact, we should not be content with merely establishing the point of greatest intensity, but we should take into account as well the direction in which the murmur is propagated. Now, all murmurs which arise in the heart are propagated in the direction in which the stream of blood flows. The murmurs of regurgitation arising from insufficiency of the mitral are propagated in the direction of the left auricle and auricular appendix. For this reason the

systolic murmur, in connection with insufficiency of the mitral valve, is often as audible, or even more audible at the level of the pulmonary ostium (on the anterior wall of which the left auricular appendix lies) than at the apex of the heart. The murmurs, on the contrary, which are heard from stenosis of the left venous ostium, follow in the direction of the stream towards the ventricle, and reach their maximum intensity at the apex, sometimes even to the left of and externally to this. As the murmurs may easily be transmitted from one arterial ostium to the other, special attention should be given to their propagation along the courses of the aorta and pulmonary artery. We have already stated that murmurs are chiefly influenced by the rapidity of the stream, and are quite independent of anatomical causes. Hence, it is quite clear that from the intensity we can draw, in general, no conclusions as to the degree of the valvular changes which have led to it.

Auscultatory phenomena, closely related to cardiac diseases, are also to be found on the vessels of the neck and extremities. Under normal conditions two sounds only can be heard in the large vessels near the heart, in the carotids and the subclavian artery, but not in the more remote vessels. The first of these sounds arises from the vibrations of the vascular walls, produced by the alteration of tension between systole and diastole; the second, on the contrary, is transmitted from the semilunar valves. Thus the greater this difference of tension is, the louder we hear the first sound, and that not only on the vessels near the heart, but also on the more peripheral ones. Insufficience of the aortic valves is the disease which gives rise to the greatest difference of tension between systole and diastole, and in such cases the first sound can be heard on the volar arch and dorsal artery of the foot, and other small peripheral arteries, while normally it can only be heard in the abdominal aorta, but not in the crural artery.

Cervical murmurs may arise in veins as well as in arteries, or they may be only transmitted. When the aortic valves are affected it is easy to regard a murmur heard in the carotids as transmitted, and thus systolic if the aortic affection be stenosis, and diastolic if insufficience. Often we perceive a diastolic murmur over the aortic ostium, but are unable to detect it on the

carotids, where we hear in its place the second sound. In such cases there is only partial degeneration of the valves, and the murmur we hear over the ostium conceals the sound, which is in reality still produced; hence the cause of the murmur lies in some changes which do not result in total incapability of closure in the valves. The systolic murmur heard in the carotids during aortic insufficience may be produced in the cervical vessels also by irregular vibrations; for the walls lose in elasticity from the continuous high tension to which they are exposed.

The arterial murmurs are intermittent, or at least louder during the corresponding cardiac phase, and thus differ from the continuous venous murmurs, which are to a great extent independent of the cardiac movements; but, on the other hand, these latter are intensified by respiration, and especially by inspiration, which increases the rapidity of the circulation in the veins, and thus hastens their exhaustion. But even venous murmurs may be somewhat intermittent in character. Chauveau was the first to show that they are either audible only during the diastole, or, if continuous in character, they can be more distinctly heard during the diastole. He attributed this rhythm to the fact that during the diastole there was a diminution in the pressure of the blood within the vein, and therefore an increased rapidity of current. By unskilful auscultation, or by purposely pressing the stethoscope on the arteries, we can produce artificial murmurs; and, as we have already remarked, in the same manner, the slight pressure the omohyoid muscle and its fasciæ exert on the underlying jugular vein in turning the head, suffices to produce murmurs in this vein, which greater compression will cause to disappear. These murmurs are produced by exactly the same factors which we have mentioned in our analysis of the palpable vibration of the veins; and so it is easy to understand that they are not necessarily connected with local heart disease; but that they are rather related to certain general conditions which directly influence the quantity of blood in the veins, their tension, and the rapidity of the current. That venous murmur, however, which is caused by tricuspid insufficiency, and which is therefore in immediate connection with a cardiac phase (the systole),

bears a direct relation to diseases of the heart. Where there is insufficiency of the valves of the veins, without coincident tricuspid insufficiency, this venous murmur can be both heard and felt; it is then, however, pre-systolic. Like all murmurs of the same class, this is heard louder on the right than on the left side, because the interval between the heart and the cervical veins is shorter on the right, and consequently the veins on this side empty quicker. A murmur of regurgitation can be heard, too, in the cervical veins, after an aortic aneurism has opened into the superior vena cava.

DISEASES OF THE ENDOCARDIUM.

Historical Sketch.

LITERATURE: *K. Sprengel*, Versuch einer pragmatischen Geschichte der Arzneikunde. Halle, 1803. — *P. J. Philipp*, Die Kenntniss von den Krankheiten des Herzens im 18. Jahrhundert. Berlin, 1856 — *F. Martini*, Beiträge zur Geschichte der Lehre vom Herzen und den Herzkrankheiten. Inaug. Diss. Berlin, 1869.

IT is self-evident, considering the scanty knowledge the ancients had of the structure and functions of the heart, that various phenomena, such as palpitations and syncope, although attracting their attention and observation, were never attributed to distinct cardiac affections. It was not till the sixteenth century, when post-mortem examinations had laid the general foundation of anatomy, that, after Vesalius' extensive investigations on the normal structure of the heart, and Harvey's brilliant discovery of the mechanism of the circulation, numerous pathological results roused a spirit of more accurate investigation. The progress made in the eighteenth century is astounding. First, Raymond Vieussens, who has enriched our knowledge of the anatomy of the heart, even more than its pathology, describes a case of stenosis of the left ostium, accompanied by insufficience of the mitral valve; and in his review he both gives it its proper significance and acutely explains the secondary hypertrophy of the ventricle, and the quality of the pulse which he had observed during life. He treats a case of aortic insufficiency in the same manner. His contemporary, John Maria Lancisi, drew attention to the dilatation of the right side of the heart, and simultaneous swelling and undulation of the cervical veins which accompany disturbances in the pulmonary circulation. Albertini estimated still more exactly the connection which exists between disturbances of the respiration and circulation, in consequence of

valvular defects. He too made use of and recommended the first physical method, namely, *palpation of the cardiac impulse*, for diagnosis of heart diseases. Morgagni, the inventive and brilliant investigator in pathological anatomy, in four letters of his work, "De Sedibus," devoted to heart diseases, describes the mechanism of the disturbances which valvular defects produce, and even goes so far as to attribute the symptom of cyanosis solely to stagnation of the circulation. His work was greatly lightened by his contemporary, Senac, who first devoted himself to the systematic study of the diseases of the heart. Apart from numerous anatomical and physiological discoveries, which this investigator propagated, he was also the first to bring forward the influence of age as etiological, and mentioned cerebral affections among the secondary symptoms or complications (as the case might be) which follow in the train of heart diseases. Corvisart took a fresh step in advance, by adopting Auenbrugger's newly-discovered method of percussion, as a means of diagnosis in addition to palpation, which had already been practised by Albertini and Senac. By this means, as he assures us, he in general correctly estimated during life the circumference of the heart. Laënnec, too, asserts that his teacher, Corvisart, was the first who recognized the *frémissement cataire* as a symptom of mitral affection. But neither he nor his predecessors were aware of the anatomical foundation of these valvular diseases. It was reserved for Kreisig to find this in the inflammation of the endocardium. He, in fact, has established the significance of all we see in the post-mortem examination with the naked eye. He also found that endocarditis was a frequent complication in the course of scarlatina and rheumatic arthritis. Bouillaud, however, was the first to introduce the term "endocarditis," and must not be denied the merit of having laid more stress than others on the frequency of its occurrence in rheumatic arthritis, and of having recognized the disease during life. This latter he was enabled to accomplish by Laënnec's discovery of auscultation. Though the immortal Laënnec fell into occasional errors in interpreting the symptoms he discovered, still he is the originator of the method which alone enables us to diagnosticate a cardiac affection in the living subject. Among

French and English observers, we should mention especially Andral (in his 4th edition of Laënnec's Treatise), Piorry, Williams, Hope, and, above all, Corrigan, who put forward the oscillation theory, and who was the first to describe the physical signs of insufficiency of the aortic valves. Skoda and his school not only enlarged the limits of the physical signs of disease, and perfected the methods of ascertaining them, but they were also the first to point out what are the general conditions necessary for the production of certain symptoms. Kiwisch, Weber, Heynsius, Marey, Wolff, Landois, Chauveau, and others, have experimentally proved the physical conditions which produce the murmurs, and the different qualities of the pulse. The knowledge of pathological anatomy advanced hand in hand with the progress of diagnosis. We owe an exact insight into the normal and pathological anatomy of the endocardium and endocardial products to Luschka, Rokitansky, and Virchow. Virchow, in his works on embolus, has rendered the pathogenesis of the sequelæ of valvular affections susceptible of a mechanical explanation; and he was also the first to acquaint us, in an accurate manner, with the anatomical changes which lie at the foundation of endocarditis. To Traube is due the credit of having first called attention to the important relations which subsist between the diseases of the heart and those of the kidneys. This investigator has enriched our knowledge of both the physiology and the pathology of the heart by an extensive series of important observations and experiments; and he has also added to our knowledge of the therapeutics of heart diseases, by his monographs on the action of digitalis. Stokes, Bamberger, Friedreich, Duchek, and von Dusch, by their excellent treatises and important investigations into details, have added materially to this department of medical science.

Inflammations of the endocardium, according to their character, end either in ulcers, in thickenings of the membrane, or in villous formations of connective tissue, which in course undergo further changes. Corresponding to this, and with a view to the duration of the process, we shall divide them into

1. Acute ulcerative or diphtheritic endocarditis.

2. Acute and subacute verrucose endocarditis.
3. Chronic sclerotic endocarditis.

Acute Diphtheritic Endocarditis. Ulcerative Endocarditis.

LITERATURE: Besides the well-known text-books by *Bamberger*, *Duchek*, *Friedreich*, and *von Dusch*, consult *W. S. Kirkes*, in Edinb. Med. and Surg. Journal, 1853, Vol. XVIII.; and On ulcerative inflammation of the valves of the heart, etc., in Brit. Med. Journ., 1863.—*Virchow*, Gesammelte Abhandlungen, 1856, and second edit., 1862, from p. 505 on.—*Bekmann*, Ein Fall von capillärer Embolie, in Virchow's Arch. 1857.—*Ogle*, On Ulcerations, etc., Transact. of Pathol. Soc., 1860.—*Westphal*, Endocarditis ulcerosa im Puerperium, Virch. Archiv, 1861.—*Charcot et Vulpian*, Note sur l'endocardite aigue ulcéreuse, etc. Gazette méd. 1862.—*Lancereaux*, Recherches cliniques pour servir à l'histoire de l'endoc. suppurée, Gaz. Méd. 1862.—*Schivardi*, De l'endocardite ulcéreuse. Union méd. 1865.—*Hérard*, Endocardite ulcéreuse. Archiv. génér. 1865.—*Moxon*, Case of ulcer. endoc. of the right heart, in Transact. of Path. Soc., Vols. XXI. and XIX. —*Whipham*, A case of pleurisy with hæmothorax complicated by ulceration of the tricuspid valve. Ibid. Vol. XX.—*Tuckwell*, Endocard. with embolism. Ibid. —*Aufrecht*, in P. Niemeyer's Handbuch der Percussion, 1868, p. 151.—*Bumken*, Die ulceröse Endocarditis. Berlin, 1868.—*Oppolzer's* Lectures, Ueber die ulceröse Form der acuter Endoc. Allgem. Wiener Med. Zeit. 1868, Nos. 14 and 16.—*Rudolph Meier*, Ueber die Endocarditis ulcerosa. Zürich, 1870.—*Virchow*, Ueber Chlorose, etc., insbesondere über puerperale Endocarditis. Berlin, 1872.—*Heiberg*, Ein Fall von Endoc. ulcerosa, in Virch. Archiv, 1873, and Die puerperalen und pyämischen Processe. Leipzig, 1873.—*Lancereaux*, De l'endocardite végetante ulcéreuse et de ses rapports avec l'intoxication palustre. Arch. génér. 1873.—*Eberth*, Ueber diphtheritische Endocarditis. Virch. Arch. Bd. 57.—*Burkart*, Ein Fall von Pilzembolie. Berliner klin. Wochenschr. 1874. No. 13.—*C. Eisenlohr*, Ein Fall von Endocarditis ulcerosa. Berl. klin. Wochenschr. 1874, No. 32.—*Rudolph Meier*, Ein Fall von primärer Endocarditis diphtheritica, in Virchow's Arch. Bd. 62. Heft 2.

It is only in modern times that searching attention has been bestowed on that form of endocarditis which is distinguished no less by its acute, malignant course, than by its peculiar anatomical product. The fact that it is always accompanied by ulcers, distinguishes it indeed from the verrucose or vegetative and the sclerotic forms, which occur more frequently, but still does not fully express its qualities, as there are many benign "losses of substance," which only form slowly and by degrees on

the endocardium; the latter appear to belong more to the fatty consumption we meet with in endoarteritis. The malignant nature which characterizes this affection is not to be attributed simply to the ulcerative process, but to the peculiar fundamental disease which induces the ulceration. Virchow, who was the first to investigate the pathological changes somewhat accurately, and whose description we shall follow, called attention, even in his first communications, to the similarity of this process to that which takes place in diphtheria; and all recent investigations tend more and more decidedly to confirm this view. Hence, it appears more correct to adopt the term "diphtheritic" than "ulcerative" endocarditis.

Anatomical Appearances.—The usual seat of diphtheritic endocarditis is in the left side of the heart. Both the mitral valve (especially the tip of the anterior flap), and also the flaps of the aortic valves, are most frequently affected. We next find it, in order of frequency of occurrence, on the walls of the auricular appendices (especially of the left auricle) and of the ventricles. On the tricuspid and pulmonary valves it occurs only exceptionally, but still this has been repeatedly observed. Whipham, for instance, describes a case (Path. Transact., 1871), where the disease was confined to the tricuspid valve alone; while similar observations have been twice made on the pulmonary valves. As occurs in the course of inflammation in other vessels, so here also the first stage is hyperæmia of the vessels which supply the intermediate layers of connective tissue. There is as little proof for this derived from actual observation as there is for the supposition that an exudation is poured on to the free surface of the endocardium, which is washed away by the circulating blood. The occurrence of parenchymatous changes in the tissue at the very beginning of the disease has been firmly established. The cellular elements absorb material till they enlarge, swell, and are rendered non-transparent by their granular contents. From the very commencement the affected parts of the endocardium appear of a dirty gray color, opaque and dull, but still there is no trace of unevenness visible to the naked eye. Very soon, however, when the proliferation in the connective-tissue nuclei of the elastic reticular fibres grows more active, granulations of

various size are formed. Simultaneously those surfaces which have lost their brightness and smoothness become the seat of fibrous deposits, and these organize themselves into a felt-like mass with the underlying tissue. The tissue itself has grown loose, and can be easily torn away. The small granulations and the fibrinous deposits on these crumble away, the tissue softens, and an ulcer is formed, on and around which the stream of blood is continually depositing fresh fibrin. Actual formation of pus, consequently, seldom takes place, being generally anticipated by the dissolution of the tissue; it is only in rare cases that abscesses have been observed, whether in the connective tissue which unites the two layers of the valvular reduplication, or whether it be in between endo- and myo-cardium. This has been the experience of Lancereaux in particular. The usual result we find is a perforated valvular surface; this is surrounded by excrescences, and is uneven, felted, ulcerated, or even covered by a bleached coagulation. In other cases, which come under observation in an early stage, we see little more than yellowish spots, or a dirty gray patch of a few millimetres in thickness, and around it a swollen and dull endocardium; nor can we remove it from its position without loss of substance. If we accurately examine the dulness occurring in spots, or the more diffuse patch, or the substance which is found on the ulcerated place itself, they will all be found to consist of "the most various-shaped granular particles," as Virchow so happily describes them; and these bear the greatest resemblance to diphtheritic formations. It forms a very finely granulated mass, almost a detritus, which microchemically displays a strong resistance both to acids and to alkalies. These fine granules have often been held for fat globules, but treatment with alcohol or ether shows the groundlessness of this view. In a small but well attested series of cases these substances have been proved with certainty to consist of parasitic organisms of the micrococcus genus (Winge, Heiberg, Burkhart, Eisenlohr). Virchow himself has confirmed this result in the case of Heiberg's specimens. And in this we have further grounds for retaining our view of this pathological product as diphtheritic in the anatomical sense. On the affected parts of

the endocardium, especially on the valves, the ulcerated condition of the membrane, and its mode of origin, can give rise to further changes, both in the valves themselves and also in distant organs. The looser and more easily torn the tissue becomes, so much the easier can a lamella of a valve tear, even before ulceration has set in, and then the lamellæ, which remain uninjured, are stretched and bulged out by the strain of the circulation. This occurs still readier when ulceration has already destroyed one lamella, and in this manner are formed the so-called *acute valvular aneurisms*, convexities varying in size from a pea to a bean, or a walnut; they arise on the auriculo-ventricular valves, and project into the auricles, or on the semi-lunar valves and project into the ventricles. The bottom of one of these aneurismal sacs may be perforated, and the ragged edges of the opening produced may form a source for embolic processes. An aneurism of this kind, especially by depositing large thrombic masses, can build a considerable tumor, and in this way lead to acute stenosis of the ostium. Still the majority of acute insufficiencies are produced by spreading of the ulceration to the base of the flap, which is thus loosened and destroyed, or, if the process spreads in the other direction over the chordæ tendinæ, by dragging these away. When the ulceration is localized in the ventricles instead of on the valves, then the pressure of the blood drives out the cardiac wall; and this, when complicated with myocarditis, may give rise to a so-called *partial cardiac aneurism*. Communications of all kinds, too, are formed according to the situation of the ulceration; thus a communication between the two ventricles has been observed, resulting from ulceration of the septum. In another case we see communication of this kind taking place between the right auricle and the anterior wall of the aorta. If the disease be located in the left side of the heart, and if small particles of the crumbling parts, or—more rarely—large shreds of tissue be swept onward by the current of the blood, and enter the circulation of those organs which possess—as Cohnheim's investigations have shown—"terminal arteries" and valveless veins (as is the case, *e. g.*, in the spleen, kidney, brain and eye), they will give rise in these organs to infarctions, by inducing secondary changes in the cir-

culation. Where the tricuspid valves are diseased, or where thrombi have formed in the right side of the heart, or, finally, where there is coincident thrombosis of peripheral veins, infarctions, or—still oftener—abscesses from embolism, will also be found in the lungs, which possess "functional terminal arteries." Abscesses from embolism may form in all the organs of the body, for, as Cohnheim has shown, the embolus acts in these organs merely the part of an infective exciter of inflammation. It is an extremely interesting fact that organisms, which are now unanimously accepted as parasitic, have been found by Virchow, Bekman, von Recklinghausen and others, in the seats of metastasis, in the afferent vessels of the above-mentioned organs, and that these organisms are similar to those which occur also on the original focus of the embolus. We must here mention a peculiarity of the acute malignant form, which distinguishes it from the subacute verrucose, namely, the acute form but seldom leads to large clots, while in the great majority of cases it produces *multiple*, *capillary* emboli. A peculiar property of these emboli is that they not only cause mechanical obstruction, but they also act as a chemical infectant, and are thus, in themselves, the symptom of a general blood poisoning, or else lead directly to this result. In spite of the certainty which some authors express, that the changes in the skin and mucous membranes, so often occurring in the course of endocarditis, and especially the capillary extravasations which we find almost without exception on the mucous and serous membranes, together with the pustulous exanthemata of less frequent occurrence, depend upon embolical processes, the question is still undecided. Embolic obstruction has as yet been proved in very few cases, and it is quite possible that here, as on other occasions where infection is involved, changes in the walls of the vessels, perhaps only of a functional nature, may render them more liable to rupture. The character of universal infection is also displayed in the coarse parenchymatous alterations in the glandular organs of the abdomen and in the myocardium, and the case is not affected, whether we assume an inflammatory origin for these, or whether we regard them as the expression of deficient nourishment. The spleen

is always found enlarged, often to twice its normal size, even in cases where no infarction has occurred; the liver is sometimes of the normal size, but generally hypertrophied, and only exceptionally atrophied; this last result, however, can seldom, with certainty, be attributed to endocarditis. In the kidneys we meet with stripes of miliary, so-called "abscess formations," and these obstruct the afferent vessels with parasitic organisms. Similar clots, too, occur in the uriniferous tubules and vessels of the glomeruli, even in cases where the bladder and ureters are perfectly intact. Hardly a single case occurs without changes in the epithelium.

The brain still remains to be mentioned in the category of distant organs, which display pathological results that are directly attributable to endocarditis. Extravasations into the meninges have been repeatedly observed, sometimes confined to the surface, and at other times piercing deeper, occasionally into the cerebral substance itself. But embolical obstructions of the larger vessels, and metastatic abscesses do not occur here so frequently in this class of endocarditis.

On the heart itself we find pericarditis and myocarditis (the latter more frequently), either directly produced from the endocarditic centre, or brought about by emboli in the coronary arteries. The changes affecting the lungs are generally the result of emboli from the right side of the heart, and assume the form of metastatic abscesses; this has been several times observed to produce pleuritis, and in one case actually hæmothorax.

In isolated cases, as in the one observed by Herard and Sander, there was no indication whatsoever, either of an abscess or of secondary infarction. About the changes the blood itself undergoes in this severe general disease, nothing is known. Virchow has indeed made one observation, where he found, two days after the death of the patient, that the blood had an acid reaction, and when boiled, precipitated tyrosine and leucine; but then we must by no means forget that these changes were observed post-mortem.

Etiology.

Here we must distinguish between a primary and a secondary form. For the first we know absolutely nothing causal in the sense of a necessary connection between cause and effect. We have only learned empirically some pathological conditions for it, which usually precede the development of diphtheritis, and on account of this we feel bound to assume some allied cause for both. The disease, however, seldom attacks perfectly healthy people. In the great majority of cases it has been observed in the course of acute rheumatism, seemingly unaffected by the number of joints attacked or the degree of pain. The cardiac disease does not usually commence till that of the joints has run on for some days; and after the fever has begun to give way, a severe shivering fit ushers in the endocarditis. The form which occurs during puerperal fever represents a transition stage from the primary to the secondary. Though in some cases we can find no symptoms of disease on the genitalia, yet in others the most undoubted diphtheritic affections have been seen on the mucous membrane of the uterus or vagina. And in these very cases the likeness between the diphtheritic matter found on the genitalia and that which covers the endocardial abscess, is so strong, and parasitic organisms have been detected with such certainty in both cases, that nothing but the most stubborn incredulity could deny a connection, brought about by the blood, between the affection of the genitalia and the endocardial centre. We do not wish, however enticing it may be, to make any conjectures as to the part which parasites play in these cases as the exciting causes of disease; still we must be allowed to consider them as the connecting agent between the puerperal affection of the uterus, etc., and the endocardial process. The same holds good for another series of diseases arising from wounds, where the wounds (whether external, as in Winge's case, or internal [*fausse route*], as Eisenlohr observed) are the primary seat of the disease, and the endocarditis only a secondary affection, or, in other words, where the ulcerative endocarditis is only a part

symptom of the presence of pyo-septhæmia. We should be undoubtedly justified in regarding the colonies of micrococci, which are also to be found here, as the real cause of the endocardial disease, and in looking upon the whole as parasitic, if it were not that on the one hand observations have been recorded, where no thought of such a connection can be entertained, and that on the other hand even cases of puerperal ulcerative endocarditis itself have been observed where no other etiological factor could be brought forward besides the usual rheumatic one due to cold caught during perspiration. Besides this, the verrucose form of endocarditis occurs in rheumatic arthritis, and in puerperal fever, too, oftener than the ulcerative. Thus, though the ground process of the two is the same, yet a disposition to disease appears to exist in the endocardium, which, under circumstances as yet unknown, presents itself in the ulcerative form. As regards the defective development of the vessels, which Virchow has urged, and the existence of which he has only proved in a few cases, it remains to be decided how far it can be regarded as a material ground for a so-called predisposition. In any case, from the fact that old valvular defects can only be regarded as etiological in the third degree, we can imagine how much importance should be attributed to previous vascular changes. Several of the observations already made (and to which I can add one of my own) prove that previously existing old endocardial changes, in particular a thickened and retracted tissue, which so frequently dispose to recrudescent verrucose inflammations, may occasionally form a favorable ground for the development of an acute ulcerous process. It is extremely doubtful whether we may reckon typhoid and acute typhus as etiological. Rudolph Meier has called attention to the fact that in the one case which Griesinger adduced to prove this assumption, and in which endocarditis followed *typhoid*, there is no evidence to prove whether this was the ulcerative form or not; and the female, on whom Vast observed ulcerative endocarditis after *measles*, had previously suffered from rheumatic arthritis.

It is still less justifiable to bring forward acute or chronic diffuse nephritis in any causal relation to diphtheritic endocar-

ditis. Out of the numbers of persons affected with renal disease, who have come under my notice, I have never seen one with this cardiac complication; and even if *one* such case is described, we still have no right to promote such an exception to the etiological paradigm.

Age alone remains to be mentioned among the circumstances whose influence we have empirically learned. In the majority of cases observed the patients were young, under thirty years of age; only a few were over forty. The first case on record, which is described by Senhouse Kirkes, was a boy of fourteen. Sex and occupation have not as yet been observed to exert any influence.

Symptoms.

It would be impossible to draw a tabulated general description applicable to every case of endocarditis, since hardly two cases have been found exactly alike. We can only construct certain types by abstraction from the observations at our disposal, and arrange the different phenomena under these, so as to form a harmonious whole. The types, two in number, are the *typhoid* and the *pyæmic*. The prevailing symptoms in the typhoid form are the general constitutional disturbance and the nervous disturbances, which produce the same impression on the observer as typhoid fever; in the pyæmic form the character of the fever and the occurrence of the various metastases constitute the distinguishing characteristics. However, many cases fall distinctly under neither the one head nor the other. The following is a general outline of the *typhoid* form. After the patient has been complaining for some days of more or less sharp pains in the joints, these give way, but the accompanying fever remains either very high, or, if it has gone down, a severe shivering fit comes on suddenly, and is followed by heat and perspiration. Defined local complaints, which would call the physician's attention to the heart, are seldom made, even though the disease be complicated by pericarditis; the patient only complains of headache, giddiness, general prostration, and sometimes of an oppressive feeling of pressure in the epigastrium.

The temperature generally remains high, but varies too between a remittent and an intermittent character; the pulse is quick, soft, and small; the tongue is dry; the lips are covered with a sooty coating; vomiting comes on, sometimes only at the beginning, other times repeatedly all through the course of the disease; diarrhœa and constipation succeed one another; the abdomen is puffed up, and the spleen swollen. The patient is at first collected, but becomes confused; delirium and somnolence succeed one another, till the latter reaches the stage of perfect coma. Urine and fæces are involuntarily passed; the urine is dark, and contains albumen and sometimes blood. The objective examination of the different organs shows that the spleen is enlarged and painful on pressure. Over the heart we usually hear a loud systolic murmur, occasionally a diastolic one; sometimes it is best heard over the apex, sometimes over the base, but especially in the neighborhood of the aortic ostium. Whether we find an extension of the cardiac dulness to the right or left, or whether there are, as usually occurs, no changes at all in the cardiac volume, all depends upon the situation and area the process has taken up. Complication with pericarditis can produce very considerable changes in the physical symptoms.

The commencement of the *pyæmic form* also announces itself by a severe shivering fit. The fits then recur, either with perfect irregularity, several times in a day, or sometimes with such perfect regularity of paroxysm and apyrexia that the observer would at first imagine he had an intermittent fever before him. A lying-in patient may have left her bed after the puerperium, and feel apparently perfectly well, when the first attack of shivering, followed by considerable depression and general prostration, announces the arrival of endocarditis. In addition to the general disturbance of the system, here strikingly developed, and to the peculiar course of the fever, metastases occur in this form with great frequency, though we are incapable of detecting most of them during life. Amongst the first occur roseolar, petechial, or hemorrhagic spots on the skin, or even a rapidly-developing pustulous exanthema, calculated to attract attention. In other cases we see a jaundice-like coloring of the skin and conjunctiva. Diarrhœa is frequently observed, and blood is

generally mixed with the stool. A swollen spleen and albumen in the urine occur in almost every case. The local phenomena in the cardiac region are similar to those in the typhoid form. In both the patients are generally unconscious before death takes place.

But besides these pretty sharply-defined forms, intermediate ones occur, isolated cases, which give us the impression that we only have a rheumatic fever to deal with. Even the most accurate objective examination can detect no physical symptoms of the local disease. Vague feeling of pain in the joints and muscles, accompanied by a fever, with its temperature curve belonging to no special type, but keeping on the whole pretty high, and with a rather rapid pulse—these, and some corresponding gastric disturbances, are the only signs of the disease, in its course quite as destructive as the others.

When, in order to become better acquainted with the variations, we consider the symptoms one by one, we meet with the following:

The local appearances in the cardiac region, which we should be inclined to rank first, on account of their diagnostic value, are by no means always clearly expressed. Subjective complaints are rarely made; there was no pain when the case was not complicated by pericarditis. Some had a feeling of dread and oppression, others had none. The most frequent cause of trouble was the sensation of palpitations of the heart. The objective examination, too, left a great deal unexplained. It is quite comprehensible that there is no ground for the production of murmurs, and that we do not hear them when the endocardial changes are located on the walls of the ventricles and auricles. But even in cases where the valves are the seat of the disease, there may be no murmur audible, not only before ulceration has actually taken place, but even—as has been ascertained beyond a doubt—during the entire progress of the ulcerative process. In the majority of cases, however, that ambiguous sign, a systolic blowing sound, occurs quite early in the course. Signs of hypertrophy of the heart and stagnation in the pulmonary circulation soon succeed, when the progress of ulceration has produced an acute incapability of closure of the valves, by

loosening off a flap, or by perforation. The duration of the whole process is generally too short to admit of any very manifest development of secondary changes, and it may be laid down as a general rule that we never find an enlarged area of dulness unless chronic valvular disease is present or a fresh pericardial exudation has taken place. Diastolic murmurs, too, are heard when the disease is confined exclusively or in greater part to the aortic valves, while these murmurs are scarcely ever heard when the mitral valve is affected.

Respiratory disturbances are observable in many cases, and are rendered remarkable by the discrepancy between the subjective dyspnœa, and the few changes we can discover in the lungs. Obstruction of the circulation in the pulmonary vessels is generally the cause of the shortness of breath. It is only in exceptional cases that the stagnation is so great as to produce ruptures in the vessels of the bronchial mucous membrane and hæmoptysis. When the tricuspid valves are diseased, or when there is thrombosis of the right side of the heart, or when, finally, there are thrombi of the peripheral veins, the signs of hæmorrhagic infarction, with or without secondary pleuritis, will also be observed in the lungs. When no marked changes are to be detected in the lungs or pleura, we must not forget that the respiratory difficulties can be due to the presence of pericarditis.

The changes in the system in general, especially fever, are far more constant than the cardiac phenomena. Fever, though occurring under different forms, is never absent. Most frequently it assumes somewhat the temperature curve of an irregular intermittent. Especially at the commencement abnormally high temperatures of 105.8° F. (41° C.), and higher, fall in twenty-four hours, or in several days, to or below the normal. This increase of warmth is generally connected with the feeling of a cold shiver, and followed by heat and perspiration. But at the commencement intervals of apyrexia occur, in alternation with high temperatures. But in the succeeding course the character of a febris remittens or subcontinua is generally assumed, though shivering fits often recur; towards the end of the disease these

fits cease altogether. In isolated cases, highly important from their easy confusion with ileo-typhoid, the initial shivering fit is the only one which occurs during the whole course, and the fever assumes a subcontinuous remittent character, with an evening temperature of from 104° to 105.8° F. (40°—41° C.), and a morning one of 102° or 103° F. (38.8°—39.2° C). In one case of this nature I repeatedly found the so-called "inverse type," or a higher temperature in the morning than in the evening.

The *pulse* has always been observed as abnormally frequent, and, especially if the case is complicated by myocarditis, it reaches to extraordinarily high numbers. It is not marked by any further peculiarity, and when sharply defined abnormalities in its fulness or rhythm occur, pericarditis or myocarditis is present as well, and should be regarded as the cause.

Disturbances in the *digestive organs*, as vomiting or diarrhœa, occurred in almost every case, and especially in some observed by Lancereaux, their intensity was so great, and threw everything else so completely into the shade, that at first they gave him the impression of cholera. Vomiting, however, generally occurs only at the beginning, while diarrhœa and meteorismus accompany the whole course; constipation, on the contrary, is seldom observed. In some cases, pathologico-anatomical causes, such as intestinal catarrh, swelling of the follicles, etc., have been detected as the foundation of these functional disturbances; especially when blood has been passed with the fæces, embolical masses have been found in the branches of the mesenteric artery. But frequently we can find no anatomical changes, and the intestinal phenomena, particularly vomiting, must be regarded as symptoms of infection; similar phenomena may be experimentally shown on animals after the introduction of putrid matter; they occur, too, as forerunners of acute exanthemata, and this is especially true of the vomiting. For other disturbances, even for some gastric ones, we must take into account the changes which take place as well in the glandular organs of the abdomen as in the alimentary canal. Of these glands, the liver is the one most frequently affected. Mere epithelial changes, when they do not extend to dissolution, produce no striking result. In one case observed by Schnitzler, marked atrophy of the

parenchyma took place, and was accompanied by icterus. In two other cases where icterus was present, emboli were found in the branches of the hepatic artery. In most cases which were complicated by jaundice, nothing material could be found to account for the symptom. The capillary gall-ducts, indeed, were never examined.

The spleen could generally be recognized during life as enlarged, and was often accessible to palpation. The tension on its serous membrane explains the sensation of pain the patient experienced from percussion, and which often occurred without external cause.

Disturbances in the diuresis are mostly to be recognized as abnormal constituents in the urine. Parenchymatous changes in the epithelium, and the changes in the circulation brought about by the fever, are the principal causes of albuminuria. The occurrence of hæmaturia is very rare, and is only brought about by the infarction of very large clots; metastatic miliary abscesses give us hardly any external symptoms.

The phenomena exhibited in the *nervous system* relate chiefly to the sensorium, and display at first an irritant character, but *depression* very soon succeeds. The symptoms of irritation have never been observed to increase to continuous puerperal mania, except in one case described by Westphal; but this patient had formerly suffered from mental disease; and Rudolph Meier is certainly right in considering this one case as insufficient evidence to establish a causal nexus between ulcerative endocarditis and acute mental disease. It is impossible to say positively how far the deleterious influence of an abnormally high temperature is concerned in the production of the states of irritation and depression in the sensorium. But this much is perfectly certain, that this cause can be assigned only in a very limited number of cases, since most of the nervous disturbances occur right at the beginning of the disease. So in this case, as in typhoid and acute typhus, we must consider the phenomena as dependent on the action of abnormally constituted blood on the nervous centre, until more apparent anatomical changes can be found on which we may lay a material foundation. The cortical substance of the brain has been very seldom examined in these

cases, though occasionally embolic obstructions have been found in the capillaries. In addition to the sensorial disturbances, we often meet with paralysis, generally as hemiplegia, with well-defined central origin. These cases always resulted from coarse lesions in and on the brain, such as blood extravasated into the cerebral membranes, on and between the gyri, and even pressing into the substance of the brain, or else soft patches resulting from embolical stoppages in the arteries of the Sylvian fissure. Where the optical functions were affected, the cause was found to lie in extravasations in the choroid or retina, or in inflammation of the whole eye, resulting from obstruction in the branches of the ophthalmic artery.

Diagnosis.

Diphtheritic endocarditis can seldom be diagnosticated with certainty, and is generally either altogether overlooked, or at most conjectured. This is not surprising, considering how little the complaints of the patient attract attention to the local disease, and how ambiguous both the local and general symptoms are. In fact, there are no local symptoms at all, unless the disease be seated on the valvular apparatus. Even in this case a systolic murmur has no special significance, as it may be of a quite accidental nature. A diastolic murmur would be of more value; but to estimate it properly, we must exclude the existence of a chronic heart disease. As a matter of fact, the mere presence of these auscultatory phenomena is not of so much diagnostic value as the changes in their character. The earlier observers, and especially Charcot and Vulpian, have pointed out the worth of this, showing that according to the degree and progress of the ulceration, at first only a systolic blowing sound is heard, confined to the area of the apex, which next grows weaker here and more audible over the base; afterwards it is complicated with a diastolic blowing sound, and finally exhibits all the signs of perfect insufficience; they showed, in a word, that the auscultatory sounds go through a number of stages. The symptoms of enlarged area of superficial and deep-seated dulness occur when we have acute valvular insufficiency,

and seldom under any other circumstances, except it happen that pericarditis too is present. When such a combination of auscultatory and percussional symptoms takes place under our eyes, the diagnosis of endocarditis in general is an easy matter, and consideration of the general state of the system and the etiology (should we be able to ascertain it) will establish its diphtheritic character. But if, as is usually the case, we find the local appearances indistinctly marked, the general state of the system is more likely than anything else to lead us to confusing it with typhoid fever, irregular intermittent fever, or some other pathological state. Confusion with intermittent fever may be easily avoided, since no genuine apyretic intervals occur in endocarditis, at least only in the very beginning, and later on mere remissions; besides, the shivering fits occur unaccompanied by any decided change of temperature. But ulcerative endocarditis can simulate typhoid much more closely, for the spleen is nearly always enlarged, and a roseolar or petechial exanthema is very often present, and frequently accompanied by meteorismus. However, the striking disproportion in the duration of the disease, and the severity of its symptoms, as well as the absence of the temperature curve so peculiar to typhoid, and the abnormal rate of the pulse, will all suffice at least to establish a well-founded doubt of its typhoid character. But the most important point is the consideration of its etiology, such as rheumatic arthritis, puerperium, chronic valvular disease, pyæmia, and traumatic diseases in general. When one of these etiological factors is present, and the local and general symptoms which we have mentioned occur on its foundation, the probability of the diagnosis is greatly strengthened. It becomes a matter of certainty in those cases where embolical processes, which are easily detected, occur simultaneously (*e. g.*, development of panophthalmitis purulenta, hemiplegia, etc.), and where we can exclude all sources of emboli other than the cardiac valves, and likewise any chronic valve disease.

Duration.

The scantiness of the material which has come under observation renders the calculation of its duration very difficult, and,

besides, the commencement of the disease is often not exactly defined. The cases which occurred after rheumatic arthritis lasted from two to four weeks, while those which came on in the course of pyæmia and during the puerperium ended fatally in a far shorter time, sometimes in from four to six days. Spontaneous cases, and those where the valvular disease is recurrent, last comparatively the longest.

Prognosis.

From an anatomical point of view, recovery is not impossible, especially when the process has not gone beyond its first stages. And even if an ulcer do form, it may heal to a scar, and thus recovery set in, if general infection was not the result or the origin of the endocardial disease. As a matter of fact, however, there is no case of recovery on record, and after the diagnosis has been once firmly established, the prognosis must be regarded as extremely unfavorable.

Treatment.

Though we can easily establish indications, our present means are not capable of meeting them. A genetic treatment is perhaps most admissible in cases of puerperal diphtheritis and traumatic pyæmia in general. Yet here the local treatment is too feeble to ward off the spreading of pyæmia. Besides the regulation of a favorable diet, when possible, our only resources are the lowering of the excited activity of the heart and the reduction of the fever. In regard to the former, the local application of cold in the shape of ice-bags is the most preferable. With this may be connected the internal use of mineral acids, which lower the action of the heart, and have a long-standing repute as antiseptics. To combat the fever, quinine in large doses is indicated. The use of salicylic acid seems especially suited to cases where there is some probability of a parasitic origin of the disease. Digitalis, such a sovereign remedy in all other heart diseases, must be employed here with the greatest caution, since further gastric disturbance and weakening of the

heart's action are greatly to be feared. If this latter occurs, it must be met with such well-known stimulants as camphor, carbonate of ammonia, musk, etc. Friedreich, on account of the relatively successful results he professes to have obtained, recommends the further trial of corrosive sublimate. It seems to me worth while to make further trial of Lender's method of inhaling ozone for pyæmic cases.

Acute and Subacute Verrucose Endocarditis.

In addition to the works mentioned in the preceding paragraph, compare: *F. L. Kreyssig*, Die Krankheiten des Herzens. Vol. II.—*Bouillaud*, Nouvelles recherches sur le rheumatisme aigu en général et spécialement sur la loi de la coincidence de pericardite et de l'endocardite avec cette maladie. Paris, 1836; and Traité clinique des maladies du cœur. Vol. II. pp. 1–150.—*Franz Zehetmayer*, Die Herzkrankheiten. Wien, 1848, p. 232.—*Luschka*, Das Endocardium und die Endocarditis, in Virch. Arch. Bd. IV.; and Ueber zottenförmige Bildungen an den Semilunarklappen der Aorta, Deutsche Klinik, 1856.—*Rühle*, Ueber Herzkrankheiten, in Günsburg's Zeitschrift, 1852.—*Rokitansky*, Ueber das Auswachsen der Bindegewebssubstanzen. Sitzungsber. der Wiener k. Acad. der Wissenschaften. 1854.—*R. Virchow*, Gesammelte Abhandlungen, p. 505, etc.—*Richardson*, The cause of the coagulation of the blood. London, 1858, pp. 371, et seq.—*Möller*, Die Milchsäuretheorie des Rheumatismus. Königsb. Jahrb. 1860.—*G. Reyher*, Zur Frage der Erzeugung der Endocarditis, in Virchow's Archiv, Bd. 21, 1861.—*Rauch*, Ueber den Einfluss der Milchsäure auf das Endocardium. Dorpat, 1860.—*Heschl*, Zur Casuistik und Aetiologie der Endocarditis, in Oesterreichischer Zeitschr. für prakt. Heilkunde, 1862.—*Aug. Olivier*, Nouvelle note sur l'endocardite, etc., in Gazette médicale de Paris, 1862, p. 360, etc.—*L'Epine*, Sur le siège de prédilection de l'endocardite et de l'endopericardite d'après de nombreuses observations. Gaz. méd. de Lyon, 1867. —*Desnos* et *Huchard*, Des complications cardiaques de la variole. Paris, 1871. —*Labadie-Lagrave*, Des complications cardiaque du croup et de la diphthérie. Paris, 1873.—*Réné Blache*, Essai sur les maladies du cœur chez les enfants. Paris, 1869.—*Paul Sperling*, Ueber Embolien bei Endocarditis. Berlin, 1872. Inaug. Dissert.

Pathological Anatomy.

The subacute form of endocarditis, which supplies the transition stage to the chronic, has many points in common with the latter. It occurs far more frequently than the diph-

theritic. In extra-uterine life the subacute form is even more confined than the diphtheritic to the cardiac valvular apparatus of the left side, and to the chordæ tendinæ. Sperling, from the material of the Berlin Pathological Institution, gives a good estimate of the subacute and chronic forms of endocarditis in adults as regards their relative frequency of localization in the right and left sides of the heart. According to this, he found, out of 300 cases of endocarditis, 297 in the left side and 32 in the right; confined to the left side alone 268, and confined to the right alone 3, and simultaneously in both 29. In the fœtus and new-born child the reverse held good. Rauchfuss, in St. Petersburg, found during his investigations 192 cases of fœtal endocarditis in the right side and 15 in the left. Those surfaces of the valve which face the current so as to encounter the greatest mechanical tension and friction, especially the lines of closure of the auriculo-ventricular valves, and the surroundings of the noduli Arantii on the semi-lunar valves, are the favorite seats of disease. Next to these in frequency we see it occur on the chordæ tendinæ, but relatively seldom on the endocardial lining of the ventricles. We find the posterior wall of the left auricle perhaps as frequently affected, according to L'Epine's estimate, more frequently; but I may be allowed to remark that his calculations apply more to opacities and thickenings in general, than specially to verrucose endocarditis. In the adult, too, the tricuspid, and even the flaps of the pulmonary valves, are only affected in very exceptional cases. With a view to express the relative frequency with which the affection attacks the different valves, Sperling tabulated Virchow's material, so that out of 300 cases of endocarditis he found it 255 times on the mitral, 129 on the aortic valves, 29 on the tricuspid, and 3 times on the pulmonary valves. In 157 cases the mitral alone was affected, in 40 the aortic valves alone, and the tricuspid alone in 3 cases, while the valves of the pulmonary artery were not the only affected parts in a single case. The verrucose products, which even Laënnec regarded as purely fibrinous formations, are not the result of an organized exudation on a free surface, but the fruit of inflammatory changes in the parenchyma. In the diphtheritic form we saw how the proliferation of young cells hastened the apti-

tude to tear and general dissolution of the tissue with enormous rapidity. But, in the present case, the most foremost changes are of a progressive and more enduring order, produced by an irritation, which, though not so virulent, lasts very much longer. The connective-tissue elements proliferate to form a jelly-like mucous tissue on the free surface and between the cells. The longer the process lasts, so much the firmer becomes its consistency, and so much the more numerous are the various excrescences it forms. These latter vary, from the hardly perceptible velvet coating on the normally smooth valvular surfaces, to more or less opacity and swelling of the opaque spots, connected with wart-like, papillose, or even knotty and cauliflower-shaped outgrowths in the form of a cock's comb, generally of a red or gray-red color, and always firm at the base, though the point may only have a jelly-like consistency. Fibrin is precipitated from the blood on the raw and projecting surfaces, and the fibrin becomes so entangled in the connective-tissue outgrowth that it is hard to separate the verrucose and thrombotic masses from one another, so much so indeed, that before Rokitansky's and Virchow's investigations almost general ignorance of the histological nature of these products prevailed. The great likeness these small cock's comb papillæ bore to epidermal excrescences and pointed condylomata led Corvisart to regard them as syphilitic. This view, though confirmed in one case (Julia), is incorrect for the majority. These polypous formations sometimes take up a more considerable area, and are sometimes even pedunculated and exhibit a certain degree of motion. It is chiefly in the aortic valves that we find these long tufts, swinging on threadlike stalks, and hanging into the ventricles. Where such pedunculated outgrowths came under my observation and had produced very plain symptoms during life, the cases had all run either a subacute or chronic course, not at all confirming Fuller's supposition that they are always very rapidly formed.

The chordæ tendinæ may undergo the same changes as the valves, so that they come to look exactly like an ear of corn, and form the seat of thrombotic depositions. Whenever these latter occur, whether it be on already formed excrescences, or on parts of the endocardium which are still in the raw stage, or

whether it be as "cardiac polyps," which often accompany endocarditis as coagulations in the auricles or ventricles—in any of these cases there is a possibility of embolical processes taking place, and this possibility is realized in this form of inflammation through the agency of the most varied causes. But the very nature of the emboli, and their manner of occurrence, distinguish the diphtheritic from the verrucose endocarditis. In the diphtheritic the size and number of the destructive metastases are in no way related to the area over which the ulceration has extended, while in the verrucose form metastases never occur except when large quantities of fibrin are deposited, and the clots block up larger arteries; but they are fewer in number, and their action is mechanical and not infectious in most cases. Thus we can more accurately follow all the transformations in an embolus of this kind, such as filling with fat, calcification, etc. According to Sperling's statistics, the kidneys are the organs most liable to infarction, and not, as was generally supposed, the spleen. When the right side of the heart is affected, infarction takes place in the lungs, more particularly in the inferior lobes and especially those of the right lung. The Berlin material exhibited the following relations of frequency: Emboli occurred in one-fourth of the total number of cases. Out of 84 cases of embolus, the kidneys were found 57 times affected, the spleen 39 times, the brain 15 times, the liver and alimentary canal 5 times each, and the skin 14 times.

The verrucose affection in its subacute form is so seldom found alone in the heart, and so generally accompanied by pericarditis, that several observers will only speak of endopericarditis for this reason. The combination with myocarditis is by no means so frequent. The further changes, such as fibrous thickening, cartilagination, calcification, which the valvular tissue and outgrowths undergo during a prolonged course of the disease, fall under the head of chronic endocarditis.

Etiology.

The etiology of the subacute form is in part similar to that of the diphtheritic. Rheumatic arthritis here too takes

the first place, and indeed it combines with the vegetative infinitely oftener than with the ulcerative endocarditis. The uncertainty of the diagnosis, in many cases, explains the great discrepancies between the estimates made by different authors as to the frequency of the combination. Great differences prevail, even on the subject of the cardiac complications in rheumatism; and here the easier course is to make no distinction between endo- and pericarditis. Bouillaud assumes a percentage of 55, Budd of 48, Fuller of 23, and Wunderlich and Lebert of 23. When we consider our present subject, subacute endocarditis, the variance is still greater. Bamberger finds it in 20 per cent., Lebert in 17.1, Wunderlich in 15.7, and Roth in 12.6. According to Bouillaud, on the other hand, in every ten cases of rheumatic fever, there were eight complicated with endocarditis; and Vernay finds even this estimate too low, especially for childhood. From this it may be seen how unfruitful these statistics are at present, and how much they depend on the expansive power of the statistician's conscience as regards diagnosticating the presence of endocarditis. Of one thing we may rest assured, namely, that it is far oftener assumed than really present. The assumption that the severer cases of rheumatism, in which many joints are simultaneously involved, are oftener complicated than the lighter cases, is also not quite safe, though founded on more careful statistics undertaken by Fuller. I am much more inclined to side with Vogel and others, who do not recognize any such distinction between severe and light cases as regards their influence on cardiac complications. Nor can I share the opinion of distinguished physicians for diseases of children (West, Rilliet, Barthez, etc.), who assert that childhood best illustrates the connection between rheumatism and endocarditis, because the majority of cases of rheumatism occurring in childhood is complicated by endocarditis. For I have repeatedly seen cases of acute rheumatic arthritis, even in children, which were not followed by endocarditis; and indeed I consider the disposition to endocardial affections, on the whole, smaller in childhood than after puberty. The maximum frequency of valvular diseases falls between the ages of 20 and 40, or 60 and 70. It is a striking fact, but one which I have

frequently confirmed, that those acute articular affections acquired in the course of gonorrhœa, and which are embraced under the title of "Rheumatismus gonorrhoicus," are seldom or never complicated with endocarditis, while pericarditis has been comparatively often observed in this connection. However, it is not exclusively *articular* rheumatism which lends a favorable site for the production of this heart disease. Thus it may be preceded in children, for example, by the caput obstipum, and in adults by muscular rheumatism. Though in by far the majority of cases the rheumatism, whether articular or muscular, has been first observed, and afterwards the development of the heart disease, yet there are a few well-attested cases on record where the process first localized itself in the endocardium, and the articular affections followed later (Graves, Stokes, Trousseau, Monneret, Gubler, Jaccoud).

Childbirth and *pregnancy*, in general, also stand in close relations to the development of this form of endocarditis, although the connecting link between these two conditions is no more visible than in the case of rheumatism. Ollivier has, however, compiled a series of observations, which render the existence of the connection most highly probable. This period of a woman's life is rendered peculiarly dangerous, if, before her pregnancy, she has acquired heart disease. In fact, what Virchow calls the *recurring* form of endocarditis verrucosa, is far more commonly developed during pregnancy than the primary form.

Old valvular diseases are in general one of the commonest causes of the subacute endocarditis. We must, consequently, distinguish between an *original* and a *recurrent* form. And in this section we have the *original* chiefly in view.

Acute exanthematous diseases in childhood are rather productive sources of endocarditis, especially scarlatina and measles, and the former more frequently than the latter. One might easily be led to think that those rheumatoid affections, which are often observed, especially after the fading of the scarlatina eruption, were the agents to produce the heart disease, and that we have merely a repetition of the rheumatic combination. However, this connection can not be proved, and the

occurrence of these rheumatoid affections is no necessary link, for the heart disease may be quite early developed, even before the exanthema has faded. During an epidemic of scarlatina I saw one child in a family attacked by diphtheritis, and the other by subacute endocarditis. Both complications produced fatal results.

Although endocarditis has been observed in the course of variola, still we are scarcely authorized to speak of any connection between the two. In the severe epidemic which visited Holland during my professional activity there, amongst a large number of small-pox patients I did not once see this complication arise.

According to Labadie-Lagrave's assertions, croup and diphtheritis are also complicated by vegetative endocarditis, and the same author even expresses an opinion (for which I can find no ground in his own observations), that in this very endocardial complication lies a cause, though one which has as yet received little attention, for the fatal issue of those diseases.

Endocarditis has been observed to follow in a few cases of typhoid, but myocarditis succeeds far more frequently.

Chronic or acute inflammation of the kidneys is indeed not very often the cause of endocardial disease, but still there are cases on record. But where an acute nephritis occurred simultaneously with endocarditis, as often happens, I could only regard both as the results of a third force common to both; but I have never observed a renal inflammation as an etiological factor in the heart disease, for they were developed quite simultaneously.

Symptoms.

In many cases the disease exhibits so few general phenomena that anybody who is not in the habit of making a local examination of the organs every day during acute affections is certain to frequently overlook the cardiac complications. The general state of the system which exists previously to the endocardial disease is usually not materially altered by its advent; and the fever connected with the original ma-

lady is not necessarily altered, either in intensity or type. Only when convalescence has already begun in the articular affection, and the fever is on the decrease, then renewed frequency of the pulse and rise of temperature attract attention to the heart, and the patient may sometimes complain of palpitations. But other complaints, such as those of pain, are never made, except in cases where pericarditis or pleuritis are simultaneously developed. The palpitations, however, annoy the patient, sometimes in a continuous form, at other times in fits, and they are accompanied by a suggestive feeling of shortness of breath. The pulse quickens and the temperature rises, though neither with any great intensity. The fever belongs to the intermittent type. The general condition of the patient, without any further striking symptoms, speaks for fever. In the local examination of the heart we find an extended cardiac impulse; percussion proves no changes from the normal conditions in the first stages, and in auscultating we hear murmurs, when one or more valves are affected. In the great majority of cases we can only hear a systolic murmur (with or without the first cardiac sound) in its maximum intensity over the apex, corresponding to the localization of the process on the mitralis. But even in those cases where the aortic valves are the only part affected, and when, too, these valves have been rendered insufficient, the systolic still drowns the diastolic murmur to such a degree that the latter is actually altogether inaudible. Duchek's assertion that even when the disease was localized on the mitral valve, he has always heard a diastolic murmur, is strikingly at variance with the experience of most observers. Quite in the beginning of the disease, too, the second sound in the pulmonary artery is intensified. The verrucose products in themselves give rise to no physical symptoms, but the swelling and thickening of the valves, in connection with this production, are generally considerable enough to injure their vibratory powers, and consequently the most frequent sign is that we hear a systolic murmur along with the proper cardiac sound. A murmur, however, is generally heard in company with other physical signs, as when extensive growths on the edges of the flaps are so considerable as to prevent any further exact closure, or when the tips or flaps

of the valves become adherent, whether with one another or with the surface of the ventricles, or finally when thrombotic formations on the valvular excrescences narrow the ostium—in a word, when *an acute valvular disease is developed.* Also, under these conditions the duration of the process is too short to allow of any striking expression of the secondary symptoms. But the *murmur* is systolic or diastolic, or both, according as insufficience or stenosis preponderates, and is distincter over the apex or base, according as the disease is localized in the mitral or aortic valves. And we may easily perceive that *the second pulmonary artery sound is intensified*, and that the cardiac volume is transversely hypertrophied, when the disease is localized in the mitral. As regards embolism, since we have already discussed its nature in diphtheritic endocarditis, and shall further describe it in connection with the chronic form, where it occurs chiefly in acute recrudescences, we need only mention here that in the verrucose form the infarction does not occur in so many organs at once; but, on the other hand, it is recognized more easily on account of the greater space it takes up in each separate organ. Clots in the spleen are in this case, as in the former one, recognized by the swelling and painfulness of the organ. Infarction often indeed produces no outward symptoms when it takes place in the kidneys; but when the clots are larger than usual, sudden pain is felt in the loins, and hæmaturia results, accompanied by albuminuria, that continues for some time afterwards; but these symptoms do not last long. Emboli in the basilar vessels of the brain, especially in the artery of the Sylvian fissure, are proclaimed by acute hemiplegia, with or without loss of consciousness. Obstructions in the intestinal vessels, which occur occasionally, exhibit themselves in severe abdominal pains like colic, accompanied by the passing of blood. Of the peripheral arteries, those of the lower extremity are the most usually affected, and with the possible result of mortification of the toes or even of a whole limb.

All embolical processes may frequently betray themselves in shivering fits, followed by perspiration.

Diagnosis.

There are few diseases the presence of which is diagnosticated so arbitrarily as that of original acute and subacute endocarditis. Bouillaud and his followers regarded every systolic murmur that occurred in the course of acute rheumatic arthritis as a symptom of endocarditis, while Vernay goes so far as to assert that he never found even the murmur a necessary support in establishing his diagnosis; increased rapidity of pulse, fever, and palpitations, are quite sufficient for him if these symptoms are rapidly developed during the course of acute rheumatic arthritis in patients who before this were healthy. We may grant that the sudden occurrence of such symptoms may be connected with some irritation of the endocardium; but still it must be remembered that verrucose vegetations in themselves produce no symptom at all, and can only be detected when the disturbances have advanced so far as to produce an *acute valvular disease.* Thus, the presence of verrucose endocarditis can only be assumed with perfect certainty either when the physical signs display to our view the development of a valvular disease, or when we hear a systolic murmur alone on the heart in the course of rheumatic fever (or any other of the above-mentioned etiological diseases), and when this is accompanied by visible embolical obstructions in the vessels, which cannot be attributed to any other source, such as thrombi from the walls of the ventricles. The chief difficulty here lies in distinguishing mere accidental murmurs from those which indicate an actual disturbance in the function of the valve, and, further, when we have established the presence of the latter, we must prove that its origin is *acute;* and in this connection we must exclude all recurrent defects, such as accompany a valvular disease of long standing, for the patient may not have been known before to the physician, and besides the valvular disease often runs its course without any striking symptoms. The first difficulty may generally be overcome by proving the existence of other symptoms in addition to the mere murmur, especially an intensified second sound in the pulmonary artery, the localiza-

tion of the murmur by ascertaining the situation of its maximum intensity, or transverse hypertrophy, even though slight, of the cardiac volume. In relation to the slighter degrees of increase in the area of actual dulness, it must be remembered that passive dilatation of the heart can take place during acute fevers, unaccompanied by a valvular defect. The exclusion of the recurrent forms must be founded chiefly on the absence of any symptoms of unusual hypertrophy or dilatation of one or both ventricles; for if these be present, and if we can exclude any obstruction in the peripheral vessels, such as sclerosis of the arteries and shrinking of the kidneys, the existence of a former valvular disease may be assumed with certainty.

Duration and Result.

The duration of acute and subacute endocarditis is in itself relatively short, since it leads to chronic valvular diseases in most cases, and in all others terminates fatally through complications with affections of the cardiac substance or envelop, with myocarditis or pericarditis, or through pleuritis or pneumonia, or finally through embolism in vital organs. I have seen it last for two or three months in cases of the latter character. Instances where the disease has been really cured are not on record, perhaps on account of timidity in diagnosticating its presence. Creditable authorities, indeed, and Fuller, in particular, speak confidently of the absorption of the verrucose products and disappearance of all abnormal symptoms; but they do not give sufficient details to convince others who have not seen it. One should be especially cautious about drawing any conclusions as to the occurrence of recovery, on the ground that the murmur has temporarily disappeared.

Prognosis.

The *prognosis* is favorable in so far as life is comparatively rarely threatened by verrucose endocarditis. But, as regards a complete recovery, it is totally unfavorable, since it is almost certain to result in chronic valvular diseases.

Treatment.

It has been asserted that there are methods of treating acute rheumatic arthritis which prevent its complication with heart diseases. In particular the method of vesication, applying a blister near each affected joint, recommended by Herbert Davies, claims[1] preference on this ground. Though the number of cases Davies, and others after him, have brought forward is not large enough to enable us to form a sound judgment on the success of his method, still it is a striking fact that in the majority of cases treated in this way no endocarditis occurred. For this reason we should most certainly further test Davies' method of treating acute rheumatic arthritis, in its utility in warding off endocarditis, and thus at the same time meet the prophylactory indications of endocarditis, suggested by the rheumatism which is already present. When the symptoms of increased cardiac activity arouse suspicion of the development of endocardial disease, an attempt should be made to moderate the fever by the application of cold, in the shape of poultices or ice-bags. At the commencement, nitre and the tartrates of soda are suited for internal use. Though many assert that the application of blisters only excites the fever and palpitations, I cannot coincide in their opinion, and especially recommend flying blisters on the back. The fever is seldom so intense as to need special treatment; and if it do rise high, quinine, in doses of from 8 to 15 grains a day, suffices.

Though it is impossible to cure any functional disturbance of the valves after it is developed, still by removing all mental and physical harmful influences, and supporting the strength of the patient by suitable diet and drugs (light animal food and iron salts), we may succeed in bringing the disease into its chronic stage, and attain the longest possible duration of an endurable state of health. Thus we must begin using tonics as soon as the fever leaves the patient. Nor must we neglect lukewarm baths, which act on the skin, and thus promote nutrition in

[1] Clinical Lectures and Reports of Lond. Hosp., 1864.

general, and on the whole aid the circulation. In exceptional cases, especially when large clots are formed in the heart, or when extensive infarction takes place in the lungs, disturbances in the circulation so acute and threatening may be developed in endocarditis, that intense cyanosis and dangerous œdema of the lungs require the most vigorous steps to be taken. Moderate bleeding is recommended in such cases. But as soon as the most dangerous symptoms have been banished, we must again have recourse to tonics, and sometimes even to stimulants. English physicians, after Richardson's example, lay great weight on ammonia as a solvent for fibrinous precipitates, and in Germany Gerhard professes to have seen favorable results from the inhalation of carbonate of soda. This latter method is worthy of further trial just in these very cases of recent endocarditis.

Chronic Endocarditis and Valvular Affections.

Contracting and Sclerotic Endocarditis.

Besides the often quoted and universally known works of *Kreyssig*, *Bouillaud*, *Hope*, *Walshe*, *Stokes*, *Forget*, *Skoda* (treatise on auscultation), *Bamberger*, *Friedreich*, *Duchek*, *Dusch*, and *Fuller*, the following special writings are of weight on the subject of endocarditis in general: *Hasse*, Anatomische Beschreibung der Krankheiten der Circulations- und Respirationsorgane. Leipzig, 1861.—*Ecker*, Ueber aneurysmatische Ausdehnung der Herzklappen. Heidelb. med. Annal. 1842.—*Barclay*, Contributions to the statistics of valvular disease of the heart. Med. Chir. Transact., 1848, and Edinb. Med. Journ. 1853.—*Rapp*, Zur Diagnostik der Klappenaffectionen des Herzens. Zeitschr. für ration. Med. 1849.—*Sée*, De la Chorée. Mem. de l'Acad. de Méd. Vol. XV. 1850; and Rapport du Rhumatisme et des maladies du cœur avec les affections nerveuses et convulsives.—*Klinger*, Ueber die physikal. Untersuchungen der Krankheiten der Herzklappen. Würzburg, 1851.—*Traube*, Versuche über die Wirkung der Digitalis. Berliner Charité Annalen, 1851; and Ges. Abhandlungen, Berlin, 1871.—*The same*, Ueber die Wirkung der Digitalis bei Herzkranken. Ges. Abhandlungen, p. 275.—*Rühle*, Ueber Herzkrankheiten in Günsburg's Zeitschr. III.—*Beau*, Considérations génér. etc. in Archives gén. de Méd. 1853.—*Willigk*, Sectionsergebnisse der Prager pathol-anatom. Anstalt. Prager Vierteljahrschrift, 1853 and 1856.—*Virchow*, Gesammelte Abhandlungen. 1856.—*Traube*, Ueber den Zusammenhang zwischen Herz- und Nierenkrankheiten. Berlin, 1856.—

Förster, Uebersicht von 639 in den Jahren 1849–56 verrichteten Sectionen. Schmidt's Jahrb. 1858.—*Buhl*, Ueber Ectasien der Lungencapillaren, in Virch. Arch. 1859.—*Virchow*, Ueber die Natur der constitutionell. syphil. Affectionen, separately printed, 1858.—*Jaksch*, Ueber die spontane Heilung ker Herzkrankheiten. Prager Vierteljahrschrift, 1860.—*Skoda*, Ueber Complication bei Klappenfehlern und deren Therapie. Allgemeine Wiener med. Zeitung, 1860. —*Ogle*, On ulcerations, true and false aneurisms on the valves of the heart. Transact. of Pathol. Soc. of London, 1861.—*Gerhard*, Zur Casuistik der Herzkrankheiten. Würzb. med Zeitschr. 1861.—*Peacock*, On some of the causes and effects of valvular disease of the heart. London, 1865.—*Rosenstein*, Zur Beziehung zwischen Herz- und Nierenkrankheiten. Berliner klin. Wochenschr. 1865.—*L. Martineau*, Des Endocardites. Concours, etc. Paris, 1866.—*Pelvet*, Des Aneurysmes du cœur. Paris, 1867.—*Rindfleisch*, Lehrb. der path. Gewebelehre, 1867–69.—*Fr. Fuchs*, Ueber die mechanischen Bedingungen der Hypertrophie und Dilatation des Herzens. Dissert. Bonn, 1867.—*P. Guttmann*, Ueber die Ursachen der Kurzathmigkeit bei Herzfehlern, etc. Berliner. klin. Wochenschr. 1867.—*Oppolzer's* Vorlesungen über Herzkrankheiten, von Stoffella, 1868.—*Bucquoi*, Leçons cliniques sur les maladies du cœur. Paris, 1869.—*René Blache*, Essai sur les maladies du cœur chez les enfants. Thèse, Paris, 1869.—*Lender*, Zur Behandlung chronischer Herzkranker. Berliner. klin. Wochenschr. 1870.—*Foster*, Digitalis and heart disease. Brit. and Foreign Med. Chir. Review, 1870.—*Fothergill*, On palpitation and on cardiac irregularity. Med. Times, August and December, 1870.—*Salter*, On the hemorrhages of heart disease. Lancet, 1870.— *Cohnheim*, Untersuchungen über die embolischen Processe. Berlin, 1872.—*H. Jaster*, Ueber Aneurysmenbildung der Herzklappen. Dissert. Berlin, 1873.—*Fothergill*, Strain in its relations to the cirulatory organs. Brit. Med. Jour. 1873.—*Handfield Jones*, Cases of heart disease affording evidence respecting the action of digitalis. Med. Times, 1873.—*Ponfick*, Ueber embolische Aneurysmen, etc. Virch. Arch. Bd. 58. 1873.—*Orth*, Zur Kentniss der braunen Induration der Lunge. Virch. Archiv. Bd. 58.—*Penzoldt*, Ueber den hämorrhagischen Infarkt der Lunge bei Herzkranken. Deutsch. Arch. Bd. XII.—*Fothergill*, The mutual relations of the heart and respiratory organs. Med. Times, 1874.—*Michel Peter*, Leçons de clinique méd. Tom I. Paris, 1874.—*Ernst Frommolt*, Ueber das gleichzeitige Vorkommen von Herzklappenfehlern und Lungenschwindsucht. Archiv der Heilkunde, 1875.

The chronic form of inflammation may be localized on any part of the endocardium, and as a matter of fact has been found more frequently than the acute or subacute form on the lining of the ventricles, especially in the region of the apex close under the aortic ostium; but still this chronic form in the majority of cases, at least in extra-uterine life, selects, like the others, the

valvular apparatus for its favorite seat. It occurs with the same relative frequency on the different valves as in the subacute form. But the chronic endocarditis on the semilunar valves is peculiar, from the fact that, at least in advanced life, it is generally connected with atheroma, being in fact a continuation of this. But the essential difference amounts to nothing, since both processes are perfectly analogous, and the chronic endocarditic products resemble those of endarteritis, except in the quality of contraction possessed by the former, and which plays a very important part in destroying the symmetry of the valves in chronic inflammation. The anatomical and histological changes are induced by hyperplasia of the connective tissue, which in the chronic form produces a tough, firm, and fibrous tissue. This tissue is either produced by the metamorphosis of an originally highly cellular, mucous and jelly-like tissue, or it occurs as such from the beginning. Thus, indeed, the whole disease is, in general terms, either *the mere continuation of a process which began acutely or subacutely*, or one which has been *stealthily evolved, escaping from the very beginning the notice of both patient and physician.* The production of the tough connective tissue is confined sometimes more to the borders of the valves, other times more to their insertions, but may often spread over the whole valve, and always has a strong tendency to shrinking. In proportion to the duration of the process the border retracts and shrinks more, the valve becomes stiff and its motion impeded. Its outward appearance varies according as the sclerotic tissue, as such, persists or undergoes further metamorphoses. In the former case it may remain even for some length of time of a whitish yellow, as bright as a tendon, of the consistency of cartilage, but otherwise smooth, though perhaps fringed on its shrunken border with one or more rows of very delicate, incrusted outgrowths. When the deeper layers of the newly-formed tissue undergo fatty degeneration, an atheromatous pap is produced inside them, which gradually presses through into the upper layers, and leaves the free surface of the valve as a superficial or deep ulcer, covered with fibrinous deposits, or impregnated with lime salts. Outgrowths of all kinds, from unevennesses to bulbs, and often calcified, surround the edges

like stalactites. Thus in many cases fatty degeneration and calcification go hand in hand, while in others calcification is the only metamorphosis, and sometimes even advances to ossification. Simultaneously may occur adhesions, both of tip and flap of the valve with one another, and with the wall of the ventricle. The chordæ tendinæ are almost always involved in the process, and, since their structure is exactly similar to that of the endocardium, they undergo the same thickening, fatty degeneration or calcification. And from these tendons the process often spreads to the interstitial tissue of the papillary muscles. Whichever of these processes we have described predominates, whether shrinking, or fatty degeneration, or calcification, the invariable result is the destruction of the functional powers of the valves, or the constriction of the ostium, to which the valve belongs. When the shrunken and contracted valvular borders cannot approach so as to touch one another, or when the shrunken condition of the chordæ tendinæ will not allow the points of the valves to rise to their proper level while the papillary muscles contract, then the communication is no longer completely shut off between the ventricle and auricle during systole, or between artery and ventricle during diastole—in a word, the valves are then *insufficient.* When the points or the flaps in their insertions are too stiff to lie up against the wall of the ventricle or artery (according as it happens to be systole or diastole), or when they have narrowed the space by adhesions to one another, the opening, through which the blood must pass, can no longer open to its normal extent, and the ostium is constricted. Insufficience and stenosis are the constant result of chronic endocarditis, and in fact generally occur together. Indeed, when endocarditis or endarteritis is the cause, insufficience and stenosis perhaps never occur the one without the other. Still the possibility of a stenosis, unaccompanied by any trace of valvular insufficiency, cannot be doubted; while insufficience quite frequently occurs by itself, not only from the breaking off of a valve from its papillary muscle, but also from other causes. Besides, though *valvular* endocarditis is undoubtedly the most frequent, yet it is not the only cause of disturbance in the cardiac functions. Insufficience of the auriculo-ventricular valves, in particular, may be brought

about by other processes, especially by myocarditic changes in the papillary muscles; this may occur quite apart from the suppurative form, when these papillary muscles by fatty or fibrous degeneration have lost the contractile force necessary for tension of the valves. The stenosis may also arise from myocarditis, or from a swollen conus arteriosus. In the first cases, where the endocarditic changes have not passed the first stage, it is not very easy to find post-mortem anatomical proofs of insufficience having existed; for the thickenings and contractions in endocarditis do not necessarily of themselves prevent the valves closing. The points and flaps are so large in proportion to the diameters of the ostia, that a slight shrinking generally produces no insufficiency, and even a more extensive degree can be compensated by expansion of the unaffected part of the valve. It will consequently not be out of place to mention here that the method of pouring in water, commonly used to test how far the valves can close, only gives approximate results; for the difference between the pressure of a small quantity of water and the work done by the ventricles in raising the column of blood, is so enormous, that we can draw no comparison between them as to their effect on the valves. Thus by this method we often may be led into diagnosticating insufficiency of the aortic valves, and perfect closure of the mitral, when neither the one nor the other has existed during life. Aside from clinical phenomena, purely anatomical proofs in doubtful cases should be based more on changes in volume or weight of the whole heart or distinct divisions of it, than on the state of the valves themselves. A post-mortem examination can lead to no conclusion on any temporary insufficiencies which may have been produced, for instance, by imperfect contraction of the papillary muscles. It is also very difficult to form a sound judgment in cases of "*relative insufficience*," where the only anatomical abnormality we can find is that the valve is unable to completely close the opening, owing to the unusual state of dilatation of this latter. Friedreich has proposed a very suitable method for measuring the proportion between the surface of the valve and the circumference of the orifice in such cases. He cuts out the orifice and measures its length; then, assuming the ostium to be a circle, he calculates its diame-

ter by means of the formula $R=\frac{c}{2\pi}$. The measurements of the absolute heights of the valves are then compared with this diameter.

Thus, as we see from the nature of the anatomical causes of insufficience and stenosis, the two nearly always appear in company, and insufficience only occurs without stenosis in those rare cases where it arises from the loss of a valve or its adhesion with the surface of the ventricle. The possibility of an unmixed stenosis must be acknowledged in those severest of all cases, where there is no space left for regurgitation of blood ; but such cases are of the utmost rarity. On the contrary, the case of commonest occurrence is where the two are together, but either the one or the other so preponderates, and, both from anatomical and clinical points of view, throws the weaker so into the shade that, on the principle *a potiori fit nominatio*, we are only entitled to speak of one of the two in our diagnosis.

Causes and Frequency of the Valvular Defects.

The etiology of valvular defects can only be determined directly in the one case of subacute endocarditis origin. Very frequently the development is throughout of a chronic nature, and the actual beginning is so concealed from, and inaccessible to, both the perception of the patient and the observation of the physician, that it is almost or quite impossible to determine the influences which may possibly have acted on its development. We can only make one very general statement, namely, that these defects of the auriculo-ventricular valves are so far different from those of the aortic valves, that the former, if their origin be subacute, may often be referred to the same etiology which was described for subacute endocarditis, *i. e.*, to rheumatic arthritis, pregnancy, and acute exanthematous diseases. Aortic valvular diseases, on the other hand, are incomparably oftener developed in an insidious way, and are so frequently met in the most intimate connection with changes in the walls of the vessels, that one is more inclined to refer their origin to those forces which we know, from experience, to cause

atheroma, namely, to *age*, *alcoholism*, *gout*, and, what has lately been rendered more than probable, chronic nicotine intoxication. The investigations made by Ricord, Lebert, and Virchow have demonstrated beyond all doubt that syphilis also is one of the causes of endocarditis ; but as the changes here are more confined to the parietal part of the endocardium than to the valves, and can scarcely be separated from simultaneously occurring changes in the muscles, they can be better treated under the head of myocarditis. Corvisart's assertion, that syphilitic excrescences, especially condylomata, occur on the valvular endocardium, has met no confirmation from subsequent observers. However, there are isolated cases of syphilitic valvular endocarditis on record, the most noteworthy being one communicated by Leared,[1] where the aortic valves were attacked. In harmony with the influences at work, we find that mitral affections occur more in youth, and aortic more frequently in advanced years. Still this statement must be accepted with considerable reserve. Workmann[2] has observed aortic insufficiency even in a child of four years old, and I myself have an exquisitely marked case in a boy of ten, while stenosis of the aortic ostium from endocarditis has been seen repeatedly (see Blachez). In regard especially to affections of the aortic valves, it remains to point generally to the influence mechanical and traumatic (especially a rupture of a valve flap by falling) causes may have, and also to mention strain due to overexertion—a matter which has received especial attention lately in its etiological importance from Albutt, Myers, and Seitz.

The frequency of valvular defects is of course in direct proportion to the frequency of those circumstances which are etiologically connected with it. But one thing we must remember, namely, that there is by no means any adequate proportion between the occurrence of valvular disease and multiple rheumatic arthritis. For instance, in Holland I am bound to assert that acute multiple articular rheumatism is a relatively seldom occurring form of disease, while valvular disease is just the opposite. This agrees with Hirsch's testimony to the fact that the geographical distribution of heart diseases is represented by no area, dis-

[1] Transac. of Pathol Soc., London, 1868. [2] Ibidem.

tinguished from others by any geographical or climatological peculiarities, and characterized by the frequency or rarity of these diseases in comparison with the numbers in other, differently situated, regions. Still the discrepancies which exist between the statistics of various observers are astoundingly large; Duchek, for instance, estimates their frequency at 2.4 per cent., while Chambers calculates it at 17 per cent. The reason of this lies partly in the sources which have served for compiling the statistics. Duchek made his estimate from the number of patients who came into hospital, and Chambers from the number of cases he found in the dead-house. However, even taking the statistics of mortality as our standard, Chambers' estimate is far too high. Let us take a larger number of cases; for instance, the total number of post-mortem examinations in the Prague and Würzburg pathological institutions amounts to 7,347, and only 677 of them were cases of valvular disease, making a total percentage of eight or nine. Amongst the 7,870 post-mortem examinations Frommolt made in the city hospital, he only found 277 cases of valvular disease, and thus a percentage of 3.5. In this connection it is scarcely necessary to remark on the arbitrariness of statistics based only upon the post-mortem examination, since the limits of anatomical changes due to chronic endocarditis are extremely elastic.

Although calculations made from the number of patients taken into hospital are likewise unsuited to represent the proportional frequency of a disease, yet they come nearer to the truth, incomparably nearer. The books of my public dispensary record during nine years a percentage still lower than that given by Duchek, so that I am inclined to regard one or two per cent. as the approximate proportion.

No definite influence of sex is strikingly perceptible. Still it seems to me, from the literature of the subject, and from my personal experience, that women, especially in their early years, suffer more frequently than men from valvular disease.

The Effects of Valvular Defects in General.

Whenever a valvular defect exists, on whatever valve it may

be, and whether it consists of insufficience or stenosis, it always produces a mechanical effect on the relations of the circulation in general, and thus brings about secondary anatomical changes, both in the heart itself and in distant organs. The mechanical effect produced by all valvular defects is a diminution of the quantity of blood which reaches the aorta, or (as it is termed when all its branches are included) the aortic system, with each ventricular systole. This effect is most directly and strikingly exhibited in those diseases which result in stenosis of the left ostium venosum or aorticum.

In the first case less blood flows into the ventricle during diastole, and in the second less blood is driven out of the ventricle during systole; but the same takes place when the mitral is insufficient, for then a certain quantity of blood flows back with every systole into the auricle, instead of into the aorta. When the aortic valves are insufficient, since the left ventricle is supplied from two quarters with blood, a larger quantity than the normal is pumped into the aorta by each systole, even if the impelling force remain unchanged; notwithstanding this, the aortic system is not so well filled as normally, for the blood which is pumped in does not remain, but regurgitates in great part. Deficiencies in the tricuspid and the valves of the pulmonary artery produce quite as palpable results, though less directly; for both lessen the supply of blood to the pulmonary veins, and thus diminish the quantity which reaches the left ventricle, and consequently the quantity, too, which this ventricle can impel into the aorta. As Traube has very thoroughly demonstrated, the normal medium tension of the aortic system, provided its elasticity is unimpaired, depends on its receiving the normal supply of blood, and this again depends on the equilibrium between inflow and outflow. Consequently any force which lowers the inflow, and thereby also the supply received by the arteries, must directly reduce the tension in the aortic system. The immediate result of a fall in the tension of the aortic system is retardation of the whole circulation; for the circulation is dependent essentially upon the difference of pressure in the arterial and venous systems in so far as everything which raises the pressure in the aortic system, *cæteris paribus*, hastens the circulation. In the given case, of valvular

defects, this difference is not only diminished by the lowering of the arterial tension, but the scale is actually turned in favor of the venous system. For the quantity of blood which the aortic system has lost collects at first indeed in the heart, dilating its chambers, beginning with that which lies immediately before the obstruction to the outflow, or the one which receives an abnormal supply. But when the chambers of the heart have been filled out to a certain extent, then the veins are overfilled, for, owing to their slight elasticity and great powers of expansion, they have a greater capacity than the arteries. And, since the tension rises in proportion as they are more filled, the tension of the whole venous system is raised, but still never so as to counterbalance the fall in that of the arterial system, as the elasticity and capacity of the two are so disproportionate.

Every valvular disease would have to exhibit the same mechanical effect as we have just described, and every patient display symptoms of such a disturbance in the circulation, *if the force of the heart remained exactly what it was at the beginning.* But this latter factor in reality does not remain the same in the majority of cases. We find, on the contrary, that those sections of the heart which lie before the obstruction to the outflow, or those which are subjected to the pressure of an oversupply, are not only dilated, but also hypertrophied. The size of the muscle has increased in direct proportion to the dilatation, or, in other words, the additional resistance is met by additional power, represented by the propelling force of the ventricle. The effect of this additional increase of work depends essentially on the proportion the muscle bears to the capacity, and the hypertrophy to the dilatation. We can easily imagine a balance between the two so perfect as to quite compensate any disturbance in the circulation. Thus the dilated left ventricle may become hypertrophied to such a degree that its increased power enables it to drive all the blood, which has collected in it during the diastole, into the aorta at every systole; and in this way the diminished filling of the aortic system, which would otherwise necessarily result from insufficiency of the aortic valves, is counteracted. In many cases these secondary changes in the heart actually do accomplish this compensation, and for a long time

hinder the occurrence of serious circulatory disturbance. But of course it very rarely happens that the balance is quite perfect. For, even when the compensatory work falls to the left ventricle—chiefly, we may say, in defects of the aortic valves—the correction is only just sufficient for the most ordinary conditions; and in remedying these defects, the action is more precise than in any other case. But even here the respiration is always impeded the moment the smallest extra demands (such as active exercise, walking up a hill, etc.) are made on its exertions. This is still more marked in defects of the auriculo-ventricular orifices, and cases where the compensation is so accurate as to obscure all symptoms of disease, are of the extremest rarity. For ordinary circumstances, however, the correction may be quite sufficient to allow a patient with heart disease to attend to his business for years, provided the business is not of a strenuous nature. But it is quite plain that these relations may assume very various forms. For instance, if the general nutrition of the patient be deficient, no compensatory hypertrophy at all need take place; or, a very common case, the cardiac muscle is hypertrophied, but not in proportion to the extent of dilatation; or, finally, compensation takes place, but is destroyed, either by organic diseases, which throw new obstacles in the way of the circulation, and thus make fresh demands on the activity of the heart (bronchitis, pneumonia, pleuritic exudation), or by direct disease of the heart itself and its envelopes (degeneration, whether fatty or of connective-tissue nature, and pericarditis). In each of these cases there exists a disparity between the resistance and the power; and the effects we originally described, as produced on the circulation, namely, diminished tension in the aortic system, and a higher tension in the venous, with a retarded circulation, all these may be recognized both by anatomical and functional disturbances. The disturbances in compensation are earlier and more markedly apparent in the respiratory apparatus, in those valvular diseases in which the powerful compensatory force, which the left ventricle can supply, is wanting, or is only furnished in an insufficient manner. The influence which this retardation of the stream produces may be perceived in the heart as clots, found sometimes in the valves, and some-

times on the walls; on the latter they collect between the meshes of the trabeculæ carneæ, and more especially in the auricles. We have already shown how much harm these clots may do, when fragments of them, as emboli, block the circulation in the vessels of remote organs. Without entering minutely into the details of how infarctions are formed—a task which Cohnheim has already performed in such a masterly manner—we must once more call attention to the fact that it has been demonstrated that the presence of "terminal arteries" and "valveless veins" (in the spleen, kidneys, brain and eye) is a prerequisite for the reversal of the blood-current in the veins, this being the first result of the engorgement of the district in question; it is only after the reversal of the current has taken place that the walls of the vessels undergo functional changes that lead to hemorrhage—the characteristic feature of infarctions. Thus the clots, which so often occur in chronic endocarditis on the walls of the right ventricle and the auricles, attain a great importance as the genetic origin of the *hemorrhagic pulmonary infarction*, a secondary condition of great weight in valvular diseases. Here we find hemorrhagic knots, and centres of induration, which occur sometimes alone, at other times in groups; are most frequently situated in the periphery, seldomer in the centre; generally in the inferior lobes of the right lung, and not so often in the left; they are cuneiform, of a dark blue or reddish-brown hue; their base points to the pleura, and their apex to the hilus of the lung. Though these are favored by the increased side-pressure in the pulmonary arteries, still there is not the slightest doubt but that they are in great part, though not always (as there are well-established cases, too, of infarction due to stagnation), produced by embolic obstruction of the smaller branches of the pulmonary artery. Cohnheim has shown that in the peripheral portions of the lung the branches of the pulmonary artery, from the manner in which they form anastomoses, are, in a functional sense, terminal arteries, and thus fulfil the conditions which, as he has proved experimentally, are necessary to produce hemorrhage, after closure of an afferent branch. However, from the fact that we find this far oftener in diseases of the mitral than in those of the aortic valve, it is

evident that increase of pressure in the pulmonary is not altogether without its influence on the production of pulmonary infarction.

The symptoms by which pulmonary infarction may be recognized during life are the sudden appearance of dyspnœa (often accompanied by a shivering fit), and along with it a painful cough; this is soon followed by a mucous expectoration tinged with blood, or even by more or less hæmoptysis. If the infarction is peripherally situated and not confined to an extremely small area (or if several large infarctions are coincidently present), it may be detected by physical signs of induration of the tissue, *i. e.*, dulness of the percussion sound and bronchial breathing. Guéneau de Mussy[1] professes to have perceived a peculiar smell in the breath, in these cases, of a garlic character, and which he supposed to be the result of changes in the blood that came into direct contact with the atmosphere; but I have never succeeded in detecting it. The infarction is seldom accompanied by fever, never in my own experience. Even in cases where, in addition to a number of infarctions, there was engorgement of the entire lower lobe, I have failed to discover fever; on the contrary, the temperature was as low as from 98.2° to 98.8° Fahr. Still other observers (Gerhardt and Penzoldt) assert that they have seen the temperature rise. This would produce such an exact likeness to pneumonic infiltration as to render it extremely difficult to distinguish one condition from the other. And this difficulty is greatly increased when, as frequently happens with infarction of the periphery, the case is complicated by pleuritis. The hemorrhagic infarction may heal, and afterwards we can find no post-mortem traces of it beyond a scar. Thus it frequently runs a harmless course, but it is equally certain that it may lead very rapidly to death by gangrenous decomposition. This occurs, for example, when the infarction becomes infected by the embolus. It is this element of infection that the patient has most to fear in an infarction, and not its localized effects. In other cases, again, a fatal termination follows speedily after the occurrence

[1] Gaz. des hôpitaux, 1871, No. 34.

of infarction; but here death does not take place as an immediate result of the infarction, but rather on account of its occurrence toward the end of life, when the heart's action is constantly growing feebler.

All other changes, however, which we find in the respiratory tract of patients suffering from heart disease, must be ascribed to stagnation. At the commencement the only important sign of disturbance beginning in the compensation is congestion, which may be diagnosticated by a dry cough. When the stagnation advances, we seldom fail to find the râles, and mucous or mucopurulent sputa, characteristic of bronchial catarrh. In fact, this bronchitis is often the very first symptom of disturbance in the compensation, and for this reason deserves most particular attention. When the stagnation is very great, as happens especially in stenosis of the left ostium venosum, hæmoptysis, unaccompanied by infarction, is by no means a symptom of rare occurrence. The parenchyma of the lungs, too, undergoes somewhat extensive changes. Then naturally the nutritive functions are disturbed in the walls of the vessels, which are under a continuous high pressure. Dittrich has called attention to the fatty degeneration of the intima coat of the smaller branches of the pulmonary artery. Of equal importance is the dilatation of the capillaries which, as Virchow and Buhl have demonstrated, can reach such a height that they become varicose, and their shoots project far into the lumen of the alveoli. In this way the space is curtailed and dyspnœa is produced by a purely mechanical cause. When this condition is accompanied by slight hemorrhage into the alveoli and the interstitial tissue, the interlobular tissue undergoes hyperplasia, and at the same time we have that peculiar change in the lungs termed *red-brown induration.* The whole substance of the lungs is reddish-yellow, colored by the transformation of the coloring matter of the blood into granular pigment; besides this, they are generally large, tough, and heavy, but disproportionately emphysematous. In a case which Orth observed, in addition to the pigment deposits in the cells of the alveoli and connective tissue, he found in many places numerous capillaries and even larger vessels with a diameter of from 0,063 to 0,075 mm., filled up with the same pigment as that

in the interstitial connective tissue. The formation of pigment in the alveoli is the cause why pigment-filled cells are coughed up with the sputa. When we see them, we may infer the presence of this abnormal condition of the lungs even during life. The cells are characterized by the smallness and plainness of their nuclei; their origin is not quite certain, still Rindfleisch's supposition, that they are wandering cells from the blood, seems to me rather far-fetched; it is much more probable that they are derived from the pulmonary epithelium. When the insufficiency of the heart is so great, that the blood in the over-filled pulmonary vessels stagnates continuously, œdema of the lungs sets in, and this, in the tissue full of pigment, imparts here also a tinge to the sputum, which earns it the name of *brown œdema*. In all these anatomical changes in the lungs we can find quite sufficient grounds for the production of shortness of breath, of which the patient most frequently complains, sometimes before any other symptom, even in the conditions of compensation, could lead to the detection of the disease. However, it is clear, that when the ordinary conditions of circulation do not prevail, we do not at all need any coarse anatomical lesion to explain the dyspnœa, while, on the other hand, additional causes, of more exceptional occurrence, may take part in producing it. The overfilling of the capillaries with blood, and the accompanying retardation in the circulation, are of themselves quite sufficient to disturb the exchange of gases in the lungs; and the result of this being an insufficient oxidation of the blood, would cause a subjective feeling of want of breath. Besides the circulation in this case loses the assistance, which (as Diesterweg has so cleverly demonstrated) it receives from the respiration under normal circumstances; for the respiratory movements act not only as suction, but also as pressure pumps, on the circulation; but in this case the capillaries cannot stretch any more during inspiration. Though all these factors play a prominent part, yet the purely mechanical causes claim a large share of importance. We have already noticed how ectasia of the capillaries may narrow the lumen of the alveoli. In other cases the left lung is compressed by the hypertrophy of the left ventricle. This can reach such a height, that I have occasion-

ally, in making a post-mortem, found the whole anterior border of the left lung quite concealed by the hypertrophied heart. In extremely rare instances the primary bronchus has been known to be compressed by dilatation of the left auricle ; cases of this kind are recorded by King and Friedreich ; the latter has even diagnosticated it during life.

With this constant shortness of breath arising from the above-mentioned causes, we must not confound sudden *fits* of dyspnœa. These are especially frequent in connection with disease of the aortic valves, and are characterized by intense want of air (Lufthunger), though the respiratory movements are unaffected. This dyspnœa is often accompanied by pain, felt in the cardiac region, spreading to the arms (especially the left arm), and arousing a suspicion of angina pectoris. There has not as yet been enough physiological or anatomical proof brought forward to allow the assumption that these fits are connected with irritation of the plexus cardiacus, which lies behind and beneath the arch of the aorta.

The foundation is laid in the respiratory disturbances for the more important changes in the blood, at least for those of the exchange of gases, which are so clearly expressed in the complexion of patients suffering from heart disease. This is the cause especially of the cyanosis, which, when the circulation is disturbed, becomes visible on all those parts which even under normal conditions exhibit a bright coloring ; as the lips, ears, and tongue. The blood-corpuscles, on account of the long time they remain in the capillaries, while the blood flows so slowly through the peripheral vessels, lose far more of their oxygen and take up a more abundant supply of carbonic acid ; and again as they pass through the lungs they take up less oxygen than usual, and get rid of less carbonic acid. Both these processes tend to destroy the bright scarlet color of the blood, and produce a darker, blue-red tinge. It is of course only in congenital defects of the pulmonary artery (especially its contraction), where we have the greatest mechanical disturbance in the exchange of gases, that the most intense cyanosis occurs ; and in this case the whole body is more or less of a dark-blue color, more particularly the peripheral parts, the extremities. Still, cyanosis is never com-

pletely absent even in the mildest cases of disturbed circulation ; the practised eye will often detect the treacherous blue flush in what to all appearance is the fresh, red hue of the lips. The cyanosis always becomes more intense when extra demands, such as physical exertion, are made on the exchange of gases.

When the stagnation has advanced so far as to produce a cyanotic color in the skin, it can be further recognized in the over-filled condition of the peripheral veins. The small veins of the face, which were before scarcely visible, now project in meshes ; and in the neck we can see both the internal and external jugular veins so plainly, that their respiratory movements are nearly always quite perceptible, and sometimes even pulsatory movements. This over-filling generally begins in those vessels in which the circulation has to raise the column of blood, and thus under normal conditions is inclined to go slowly. Consequently, in the lower extremities it is not confined to the mere projection of blue cords, but the vessels, owing to the constant strain, become varicose and contorted, and their fibrous coat grows thick. When coagulation commences in these vessels, painful œdema of the affected parts results ; and the process may extend from the branch far into the trunk. We have seen how the clots on the walls and valves of the heart may be carried off by the circulation, and become wedged into the main trunks, the branches of which supply the lower extremities ; and how they here become the causes of necrosis and of moist or dry gangrene, that begins in the toes and spreads upwards. In the same way the particles from thrombi in the lower extremities get into the right side of the heart, and give rise to pulmonary infarction, which we have already described ; and where they obstruct the trunk or larger branches, they assume a great importance for the whole process, since they may produce sudden death. The more the peripheral veins are filled, the greater is their tension, and, in consequence, the pressure which the walls exert on their contents is increased. And this intensifies the transudation of serum from the smaller veins and capillaries, which takes place under normal conditions. At the same time resorption is rendered more difficult, and the lymph can only be emptied with greater labor, so that there is reason enough for the collection of serum in the meshes of the

subcutaneous tissue. In heart diseases, œdema always commences in the lower extremities; at first it is scarcely noticeable, then it is perceived in the evening about the malleoli and on the dorsal aspect of the feet; but after a night's rest in the horizontal position, it cannot be any more seen in the morning. As the disease advances, the dropsy spreads upwards, first up the lower extremities, then over the sexual apparatus; finally it comes to the superior parts, and, in the very worst cases, it appears even on the face. Of the internal cavities, the peritoneal is the first to suffer from transudation, for local causes are here at work in the affection of the region of the portal vein. Next to this come the pleural cavities, especially the left one, and with it generally the pericardium. The dropsy is not a mere symptom of disease, but it constitutes a new ring in the chain, which literally hems in a patient with heart disease. The pressure which the transuded fluid exerts on the smallest vessels creates new resistance to the circulation, and makes fresh demands on the activity of the ventricles. The skin is disposed to inflammation from being soaked with serum, and when the œdema is very extensive the epidermis is raised in blisters. These burst, and then follows an erysipelatous inflammation of the surrounding skin; and when an inactive diuresis leaves the blood rich in urinary components, deep-seated phlegmonic inflammation in the subcutaneous cellular-tissue gives rise to tedious suppuration, which is generally the commencement of the final stage. Though the hindrance to the circulation is the chief, yet it is not the only cause which produces dropsy. The change in the œdema alone, whilst the mechanical conditions remain unaltered, and the various areas it covers in the same original disease, point to other causes. Thus it is not improbable that a considerable part in the production of the œdema may be played by disturbances in the vascular innervation, and together with this by the changing conditions as regards the participation of the whole propulsive force which proceeds from the vessels. One factor, which comes into account here, is well determined—namely, the loss of albumen and solid components which the blood undergoes.

The changes in the blood are as little confined to the darker

color of the corpuscles and their supply of oxygen as the effects of stagnation are confined to the respiratory organs. The digestion, too, the chief factor in producing and sustaining the solid components of the blood, suffers as the disease advances. When the stagnation spreads to the hepatic veins, the circulation in the liver and in the whole drainage region of the portal vein—the stomach, intestines, and spleen—is retarded, and this gives rise to important anatomical and functional disturbance. At first the liver is swollen and hypertrophied by the abnormal quantity of blood it contains, and especially the left lobe; this enlargement is felt by the patient as a sensation of fulness and pressure in the hepatic region and epigastrium, and may be diagnosticated by palpation and percussion. Corresponding to this is the post-mortem appearance of the organ; when examined in the first stages of the disease, it is large, heavy, containing a very large quantity of blood, and the central lobular veins extremely full. But the cellular elements of the central parts soon undergo atrophy, and when the peripheral parts contain enlarged cells filled with fat, they become clearly marked off from the more central atrophied and bluish-red colored portions, and thus, as a whole, exhibit the well-known appearance of the *nutmeg* liver. As the process goes on, the peripheral parts atrophy as well, and this, together with compression and hyperplasia of the interlobular connective tissue, which results from long-continued venous hyperæmia, render the liver tough and indurated. When actual inflammatory processes take place in the connective-tissue, they may lead to a real cirrhosis; but still we may have objective symptoms of atrophy without cirrhosis. Since perihepatic irritations often accompany the disease, the patients complain of pain in the region of the liver, both of a spontaneous nature, and also on pressure. The formation of gall is always affected in these cases, as may be seen, too, in the alteration of its composition; but real intense icterus only occurs when the outflow of gall is hindered by a catarrh of the mucous membrane of the ductus choledochus. A light jaundice tinge, especially when mixed with slight cyanosis, may be seen on the conjunctiva and face, when the liver begins to be involved in the disturbances, and the pressure of the dilated veins on the gall

canals retards the progress of the secretion, without altogether stopping it.

Of the organs which belong to the territory of the vena portæ, the stomach and alimentary canal are always involved, while the changes in the spleen are not so constant, and, moreover, cannot be recognized by any functional disturbances, since swelling, hyperæmia, and unusual toughness are the only anatomical symptoms of splenial disease. On the other hand, the symptoms in the stomach and alimentary canal are most strongly marked: the appetite is lost, and a feeling of satiety follows quickly on eating, while constipation and diarrhœa succeed one another—in a word, the whole digestive system is disordered. In the cases in which vomiting of blood, or passing of it with the stool, are recorded, the hemorrhage was perfectly independent of the ordinary stagnation, being caused by embolism in the arteria coronaria ventriculi and the mesenteric arteries. There are, as yet, very few cases of this kind on record (Virchow, Bekman, Cohen, Oppolzer, Gerhard, Kussmaul); they generally exhibited severe intestinal hemorrhage, which came on very suddenly, accompanied by sharp pains like colic in the abdomen, and a considerable fall in the temperature; in some cases peritonitis, with or without vomiting, set in. The digestive derangement is a very important factor in the sum of the disease, and exerts a great influence on the course, since it interferes very materially in the formation of blood, and thus adds to the mechanically impeded circulation the further complication of cachexia.

The functional disturbances in the kidneys are an exact reflection of the alterations of the vascular tension. And here we can observe, better than on any other organ, the various stages of decrease of the quantity of blood in the aortic system, and increase of that in the venous system; for the varying conditions of the urinary secretion enable us to see both the serial succession and the development of these circulatory derangements. If, in any unit of time, a smaller quantity of blood flows through the glomeruli, the quantity of the secretion diminishes likewise; according to Ludwig and Goll's experiments, the quantity of water is more affected than that of the solid components. Accordingly the volume of urine passed in twenty-four hours

decreases, when the tension in the aortic system falls in valvular disease, and the solid components are relatively increased—in other words, this urine has a high specific gravity. When the urine cools, the salts of the saturated solution are soon precipitated, and on the bottom of the vessel we see a deposit, consisting chiefly of urates, and rich in urinary coloring matter. If the patient is well fed, we may often see crystals of pure uric acid in the sediment; they are generally cylindrical, and occur in great numbers. The urine becomes albuminous as the tension increases in the venous system, just as H. Meyer and others found when they ligatured the vena cava above the junction of the renal veins. The interval at which albuminuria succeeds the first-mentioned process varies greatly for different cases. Symptoms of venous stagnation may present themselves in other organs long before they can be seen in the kidneys. The quantity of albumen varies with the degree of tension. It is generally accompanied by the appearance of jelly-like sacs in the urine, in the shape of hyaline cylinders. But blood-corpuscles are exceedingly seldom to be found in any number in the urine from a stagnant circulation ; and hæmaturia never occurs unless there be simultaneous inflammation of the interstitial tissue. This inflammation does sometimes occur, although nephritis is entirely unconnected with the paradigm of stagnation in the kidneys. If the kidneys are examined at the commencement of the disease, they will be found enlarged, hyperæmic, and of a tough consistency. In a later stage they are atrophied, tough, and have a cyanotic appearance. The cortical substance appears thickened in section, at first striped with red, and further on of a grayish-yellow color. The cortex and medulla are distinctly bounded; the basilar part of the medullary substance is of a darkish color, while the part turned to the papillæ is quite pale. Under the microscope the glomeruli appear of normal size, the convolutions are generally intact, the capsule slightly thickened and more separated into layers than normally. In the tubules the epithelium is very granular, quite frequently fatty, and sometimes filled with fine pigment. The membrana propria of the uriniferous tubes is thickened, the interstitial tissue is thickened, and very fibrous. The great majority

of cases answer to the description we have just given, so that Traube and those who adopt his view are right in asserting that this passive hyperæmia in the kidneys is a disease, *sui generis*, which cannot be put in the same category with the initial stages of inflammatory derangement of the nutritive process in these organs, or diffuse nephritis. But, besides this, there are a few cases in which advanced anatomical alterations are found in the kidneys, simultaneously with valvular disease of the heart; these are usually regarded as the product of interstitial nephritis, whether it be diffuse fatty degeneration or atrophy. It is only by clinical observation of their development that we can decide what relation exists between the cardiac and renal diseases. We know that, as a matter of fact, valvular heart disease follows in the course of chronic nephritis, and this has already been discussed in the section on subacute endocarditis. But in reality this occurs relatively seldom. On the other hand, I have repeatedly convinced myself, that, from the very beginning, the development of the cardiac and renal diseases ran parallel, which leads me to regard the two as co-effects of a third, and genetically independent of one another. However, there are frequently hemorrhagic infarctions in the kidneys of patients suffering from heart disease, which occur in the shape of wedges, with their base looking towards the cortex, and their apex towards the hilus, being caused by obstruction in the branches of the renal artery; and we must not forget that these too give rise to interstitial inflammation. Consequently, in our consideration of the connection between valvular disease of the heart and inflammatory anatomical changes in the kidneys, we must not forget to reckon infarction as etiological.

Many assert that the renal pelvis and the bladder participate in this stagnation, but I have never seen a case of pyelitis or catarrh of the bladder which was due exclusively to valvular defect in the heart. On the other hand, the effects of an obstructed circulation are much more visible in the sexual organs, at least in the female organs; for irregularities in menstruation occur extremely often in females who have valvular heart disease. Amenorrhœa commences before cachexia assumes a very pronounced form; but in the earlier stages the menstruation is generally very profuse,

and occurs at abnormally short intervals. The shape which the derangement takes is essentially dependent on whether general anæmia is more prominent, or whether the obstruction to the venous outflow preponderates.

The form taken by the symptoms of a deranged circulation in the nervous system, and more especially in the brain, varies with the seat of the valvular defect and its nature. Patients with a stenosis of the aortic orifice very soon exhibit signs of arterial anæmia in the shape of frequent fainting-fits, which occur while all the other functions seem undisturbed and without any apparent cause, and moreover pass away quickly when the horizontal position is assumed. When the auriculo-ventricular valves are affected, the most prominent symptoms are those of venous hyperæmia. The patients complain of headache, dizziness, and a whirring sound in the head; and females, in particular, very often have the feeling of clavus hystericus. Corresponding to these complaints, we sometimes find in the post-mortem examination dull patches in the meninges, and sometimes extensive œdema in the sub-arachnoid, with slight effusion into the ventricles; but these affections probably do not originate during lifetime. Obstructions may be seen in the cerebral vessels, both in the small twigs of the cortex and in the larger branches at the base. Death is often the immediate result of a simultaneous obstruction in several cerebral arteries. Murchison[1] describes a very marked case of a girl, 14 years old, whose mitral valve was affected, and the particles of a fresh fibrinous clot obstructed both the vertebral and the internal carotid at the same time. The symptoms of hemiplegia appear suddenly when the arteries of the Sylvian fissure are obstructed, and this is often followed by softening of the cerebral substance. An embolus so frequently makes its way into the left artery of this Sylvian fissure, that right-sided hemiplegia has been regarded as a means for diagnosticating embolic paralysis. However, *in diagnosticating a central paralysis in patients with heart disease*, from the fact that the patient has *a valvular disease*, or from the attack falling on the right side, we are not entitled to assume with absolute

[1] Transact. of Path. Society of London, 1871.

certainty that the paralysis *originated embolically.* Others, as well as myself, have repeatedly observed ordinary apoplectic spots in similar cases, without any obstruction in the afferent vessels. Nor must we forget that, to produce an embolus, *the affection of the valve must be acute and recent;* and hence, as a result of chronic valvular disease, we must look for it in company with the recurrent form. As a general rule, we may assert that of all valvular diseases those of the aortic valves are the ones most usually accompanied by fatty degeneration of the small cerebral vessels and consequent hemorrhage in the brain. Thrombi and apoplexies, especially the latter, are frequently the cause of death in cases of insufficience of the aortic valves, and they must be taken into account in making the prognosis of a valvular affection, which is otherwise so often well compensated. Saccular aneurisms, which Ponfick has lately found on the vessels of the base of the brain, may be mentioned, besides fatty degeneration, as anatomical causes for sudden death and the disturbances generally proceeding from the brain. The special characteristic of these aneurisms was a wall chiefly composed of a thin layer of connective tissue and thrombotic deposits of various thickness, while a spinous, chalky embolus lay free in the aneurismal cavity, or projected into it with its point. This form of aneurism is most usually found in cases of endocarditis of the aortic valves. As regards their genesis, it is very important to note that the emboli always stop a short distance from the bifurcation of a vessel, and being here sharply pressed against the wall of the artery by the greatly increased pressure of the blood which flows into the side branch that remains pervious, they probably destroy these walls by "*decubital necrosis.*"

Doubt has been thrown on the assumption Nasse made, as to the great frequency with which pronounced psychical derangement, actual "mental diseases," resulted from cardiac disease. The statistics of the post-mortem examinations made in lunatic asylums go against him, for, according to Griesinger's calculations, only 12.5 per cent., out of 602 autopsies made in the Vienna Lunatic Asylum, exhibited valvular disease; and, even in these, the connection between the two affections had not been clinically established for all. Griesinger himself, too, does not consider

mental disease a frequent result of cardiac affections. Still, it cannot be denied that light psychical alterations are peculiar to patients with heart disease, and are often reflected in their features; these are expressed in the shape of oppressed, melancholy temper, feeling of dread, and abnormal excitability. In a case of insufficiency of the mitral which came under my observation, I saw fits of actual confusion and strange behavior, while in the intervals the patient was extremely apathetic and unsympathizing. Of course, such isolated cases do not suffice to prove a genetic connection, but if we consider that in the post-mortem examination we sometimes can demonstrate the presence of thromboses in the vessels of the cortex, and join the two facts together, we obtain what may form a material foundation for psychical disturbance, and a causal nexus is rendered probable, certainly for the cases quoted. The functions of the spinal cord are never disturbed by valvular disease, unless (as some do) we regard St. Vitus' dance, which so often occurs, especially in childhood, along with chronic endocarditis, as a disease of the spinal cord. In fact, the anatomical centre of origin of chorea has not yet been established for certain, and, as far as the investigations hitherto made on the subject go, they point more to the brain than to the spinal cord as the probable seat of the disease; some facts speak for the English observers, Broadbent, Hughlings Jackson, Tuckwell, and others, who assert that it is a capillary embolic affection of the corpus striatum. What, however, chiefly interests us here is the fact, which was first brought forward by Sée, Roger, and other French physicians, that chorea minor is frequently found combined with valvular diseases of the heart and acute rheumatic arthritis. The proportions which Roger assumes are certainly too high, for he asserts that out of 71 cases of *chorea*, 47 were combined with simple endocarditis, and 19 with peri-endocarditis; the only way to account for his assertion is that he diagnosticated endocarditis on the insufficient ground of a systolic murmur over the heart. Considering the anæmic condition of most chorea patients, these murmurs, of course, prove nothing. However, Ogle, too, found fibrinous deposits on the valvular endocardium in 10 out of 16 cases of chorea which ended fatally.

The connection with multiple rheumatic arthritis is not so pronounced; for instance, out of 252 cases of this latter, Steiner only observed 4 cases of chorea. My own observations confirm the frequency with which mitral affections are complicated by chorea, especially in very young patients. On the other hand, I have seen two cases of rheumatic arthritis, in which the chorea was developed before the valvular disease.

What we have said will give an idea of the principal changes produced in the different organs by a derangement of the circulation, which results from valvular disease. We must now describe the symptoms afforded by a physical examination, these being immediately dependent on changes in the valves. But these symptoms can be best treated in connection with the individual peculiarities which are produced by the diseases of the different valves, and figure even in the general sketch. So we shall pass on to the consideration of the diseases of the different valves.

Affections of the Left Auriculo-ventricular Valve and the Left Ostium Venosum.

In addition to all the handbooks, consult: *Briquet*, Mémoire sur la diagnose du retrécissement ventriculo-auriculaire gauche. Archives générales de Méd, 1834.—*Hérard*, Des signes stethoscopiques du retrecissement de l'orifice auriculo-ventriculaire gauche du cœur; ibid. 1853 and 1854.—*Duroziez*, Du rhythme pathognomonique du retrécissement mitral. Arch. génér. 1862.—*Traube*, Eine Bemerkung über das Verhältniss der tuberculösen Pneumonie zu organischen Herzkrankheiten. Berl. med. Centralzeitung, 1864.—*Leyden*, Ein bemerkenswerther Fall von Stenose des Ostium venosum. Virch. Arch. 1864.—*Naunyn*, Ueber den Grund, warum hin und wieder bei Mitralinsufficienz das Systolgeräusch am lautesten in der Gegend der Pulmonalklappen zu hören ist. Berl. klin. Wochenschrift, 1868.—*Geigel*, Der gespaltene Herzton. Würzburger Verhandlungen, 1868.—*Guttmann*, Ueber den gespaltenen diastolischen Herzton. Virch. Arch. 1869.—*Quincke*, Ueber accidentelle Geräusche in der Pulmonalarterie. Berl. klin. Woch. 1870.—*Bucquoi*, Leçons cliniques, etc. Paris, 1870. —*von Bamberger*, Ueber zwei seltene Herzaffectionen, etc. Wiener Wochenschrift, 1872.—*Köster*, Braune Induration mit croupöser Entzündung der Lunge. Virch. Arch. 1873.—*Balfour*, Clinical lectures on diseases of the heart. Edinb. Med. Journal, 1871.—*J. Wickham Legg*, Mitral constriction, and well-marked concentric hypertrophy of the left side of the heart. Transact. of Pathol. Society of London, 1874.

Insufficiency of the Mitral Valve.

Of all the different causes which produce insufficiency of the mitral, by far the rarest are the acute, such as the breaking off of a valve (whether from its basilar insertion or from the papillary muscle by rupture of the chordæ tendinæ) or perforation from ulceration, or finally the development of an aneurism on the valve. In the majority of cases it is produced by the subacute and chronic forms of endocarditis, quite as frequently by the fibrous thickening and contraction of the valve itself as by the shortening of the chordæ tendinæ and their adhesion to one another. The verrucose vegetations, if present in large quantities, may also injure the closing power of the borders of the valve, but in their ordinary extent they never produce this effect. Adhesion of the point of the valve to the ventricular wall brings about the purest form of unmixed insufficience. The same may occur in the course of fever, where it has been occasionally observed, being due to derangement in the innervation of the papillary muscles. It may occur, too, where these muscles are rendered incapable of developing the force necessary to hold back the valve, having lost their contractility from fatty or fibrous degeneration. But in the great majority of cases the insufficience is complicated with more or less stenosis, and so we can only properly speak of the preponderance of the one or the other affection. In a certain sense, we can regard a slight stenosis as compensating the insufficience, since the mechanical operation of the latter is to some extent weakened by the former. The insufficient valve no longer shuts off the left auricle from its ventricle during the systole, and consequently the blood is driven simultaneously into the aorta and the left auricle. The aorta receives proportionately less blood, while the auricle is over-filled, being now supplied from the left ventricle, in addition to its ordinary supply from the pulmonary veins, and is thus subjected to a higher tension. The larger quantity of blood, which reaches the left auricle, dilates it, and the symptoms of increased tension are easily recognized in the hypertrophy of its walls and the opacity of its endocardium. This rise in tension is propagated from the left auricle into the pulmonary veins, and if these too be examined, traces

are found of nutritive derangements in the form of hypertrophied walls, fatty degeneration, and here and there jelly-like proliferations on the intima. The increased tension next extends through the capillaries of the pulmonary artery into the right ventricle, right auricle, and finally into the whole venous system. The general reversal of pressure, the diminished tension in the aortic, and increased tension in the venous system, common to all valvular defects, is so arranged in insufficience of the mitral valve, that the tension is highest in the pulmonary veins and venæ cavæ, and lowest in the aorta. All parts of the heart which lie before the mitral orifice are thus dilated, and are hypertrophied in direct proportion to their normal muscularity. So, for instance, the right ventricle is much more hypertrophied than the left auricle, this latter being more dilated. But the left ventricle, too, is generally hypertrophied, the reason being that it is filled during the diastole under the high pressure which prevails in the pulmonary veins, and must consequently perform its function under a higher pressure.

During the whole course of these successive changes in the substance of the heart, the patients hardly experience any annoyance. They are, indeed, very little troubled, till the compensation is deranged by some pathological process in the cardiac muscle (especially fatty degeneration of the right ventricle), by diffuse bronchitis, or by disease of the pericardium. The patient perceives the vain efforts which the diseased muscle makes to empty its abnormally large contents, in the form of palpitations. These often occur without any special outward cause, but generally commence when larger demands are made on the circulatory organs, as, for instance, during active exercise. Along with this, the patient has a feeling of shortness of breath, since even quick respiration cannot supply the increased demand for oxygen. And, complaining of this annoyance, he generally has recourse for the first time to medical aid. It may happen that the physician some time before has witnessed the subacute commencement of the valvular disease in a febrile attack of rheumatic arthritis; but, even in this case, years may elapse before the patient thinks it necessary to again put himself under treatment. It is frequently the dry cough, produced by congestion

of the bronchial vessels, which brings the patients to consult a doctor. Objective examination then shows, that the cardiac region projects abnormally, and is more arched. This is especially prominent in youthful cases. The cardiac impulse is seen and felt lower down than usual, generally between the sixth and seventh ribs, and either in the mammillary line, or more frequently 2 or 3 cm. external to this. The impulse is more extended, especially to the right, and undulatory movements are visible even in the epigastrium. The resistance of the impulse is increased, and a purring sensation may often be felt in its neighborhood, synchronously with the ventricular systole. Percussion shows that the boundaries of the areas of absolute and relative dulness are extended both to the right and to the left. The area of dulness generally extends as far as the impulse to the left, but often goes even a little beyond this. The outlines of the area of dulness are seldom irregular ; they generally form a triangle, in such a way that the left limb runs abnormally to the left in its lower end, but above it remains within the mammillary line. On the inferior portion of the sternum the sound is dulled, but the border of the right lung is seldom so far displaced as to produce further dulness to the right of the sternum. The inferior part of the right limb of the triangle of dulness coincides with the right border of the sternum, or lies at most 2 or 3 cm. outside it. We cannot lay too much stress on the importance of testing how far the anterior border of the lung moves, and of excluding all other causes for dulness, before diagnosticating an increase in the volume of the heart from an increase in the area of dulness. In auscultating close under the nipple, and a little to the left, we can hear a systolic blowing murmur, which either completely takes the place of the first cardiac sound, and continues to the short pause, or is heard along with, and then only at the commencement of, the first sound. We can often detect the presence of the sound along with the murmur, by raising the ear slightly from the disc of the stethoscope. Though this murmur is propagated over the situations of all the other orifices, still it is very easy to perceive that the greatest intensity is over the apex. Very often we hear the murmur on the left border of the sternum in the second intercostal space, *i. e.*, in the situation of the ostium of the pulmonary

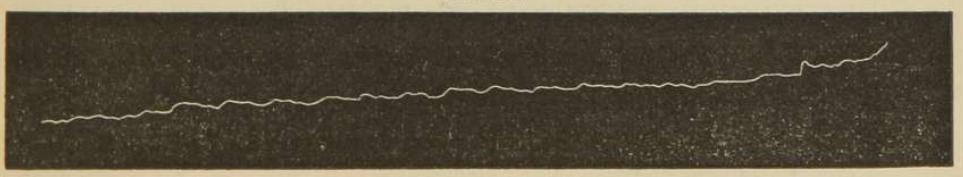
artery, quite as loud as over the apex, and sometimes even more intensely. This cannot be explained by the approach of the pulmonary artery to the thorax wall, brought about by displacement of the left lung, for we can easily convince ourselves by percussion that the vessel is covered by the lung. Nor can it lie in the greater tension of the walls of the pulmonary artery, for though a murmur might be thus produced, yet it is not evident why it should be more intense in this situation; and besides, as far as the pulmonary artery is concerned, the same conditions prevail in stenosis of the left ostium venosum, but still the murmur cannot be heard so plainly. The greatest probability attaches to Naunyn's explanation. He points out that the given situation of maximum intensity in the second left intercostal space does not correspond exactly to the place where the sounds of the pulmonary artery are heard—that is to say, not quite at the edge of the sternum, but about 2″ from it. From investigations made on the dead subject, it appears that this point would coincide with the point of the left auricular appendix which winds round the pulmonary artery and lies in front of it. Since the abnormal current of blood, which in insufficience of the mitral valve gives rise to the murmur, flows towards the auricle, and since the auricular appendix communicates freely with this, we can easily comprehend how the sound should be conducted to the spot mentioned better than towards the apex, and be heard there with greater intensity, more especially in strongly-marked cases where the appendix is long enough to lay its point close against the anterior wall of the thorax.

Beside the systolic murmur, we may hear a diastolic (or presystolic, as the case may be) murmur; but this depends on whether the ostium venosum is simultaneously constricted, and on the degree of stenosis. In most cases, the second sound cannot be heard quite pure at the apex. But as the pulmonary artery is under greater tension, over it the second sound is heard more intensely than normal, while the right ventricle continues to exert more force in the performance of its functions. It is not till the force of the right ventricle has been injured by disease of its muscle, that we lose this important symptom of an intensified second sound in the pulmonary artery. The diagnostic signifi-

cance of this symptom was first pointed out by Skoda. So long as the compensation continues to suffice, the peripheral vessels are quite normally supplied, and the pulse does not necessarily exhibit any abnormalities either in rhythm or in frequency, although at this stage it is generally rather rapid. But as soon as the compensation is disturbed, and the symptoms of stagnation become plain, both on the visible vessels and in the internal organs, then the pulse, too, begins to lose its normal character. It grows rapid, low, soft, unequal, and irregular. The sphygmographic tracing in this affection has nothing characteristic about it. In Marey's work (loc. cit. p. 525) one can observe this great diversity of pulse, for scarce one tracing is exactly similar to another. When Marey remarked, "Le pouls dans l'insuffisance mitrale est toujours irregulier," he probably had the stage of deranged compensation in his mind. Fig. 9, for instance, is a tracing taken in the stage of compensation, and all we see in it is, that it is very low, and strongly dichrotous, but it is almost quite regular. Fig. 10 shows that relative regularity may coexist with waves so exceedingly small as to be scarce traceable.

Fig. 9.

Fig. 10.



The *course* of the disease is such, that the symptoms of stagnation assume a permanent character, and all the more so in proportion to the greater number present. Though at first it is generally possible to reduce the palpitations and dyspnœa by suitable diet and drugs, and temporarily even to remove all œdema, yet, after a long period of alternate relatively good health and total incapacity for motion or work, the patient becomes gradually anasarcous, and exudations begin to fill up the serous cavities and impede the respiration. Bronchitis, of which

more or less is always present, increases the cyanosis, and, whether it be accompanied or not by a pneumonic induration, often developed towards the end of the disease, the usual termination is œdema of the lungs. In a few isolated cases the actual, though distant, cause of death is embolic obstruction in the cerebral vessels and its usual results, or extensive apoplexy. Embolism of the pulmonary vessels does not occur so frequently as in stenosis of the mitral orifice, but, on the whole, it is not rare. Shivering fits, dyspnœa, and bloody sputa are the first symptoms. If the infarction be somewhat extensive, there will be percussional dulness, and bronchial respiration may be heard. I have never seen a single case where fever occurred. Gerhard and Penzold have, however, observed this. Towards the close of the disease we often see thrombosis of the peripheral veins, generally confined to the lower extremities, and one-sided. Clots in one or more large arteries of the lower extremities are fortunately of rare occurrence. These may result, when they do occur, in mortification of the toes, the foot, or even of the whole leg, as in one case which came under my observation.

The *diagnosis* must be based solely upon the physical symptoms we have mentioned, and is in the majority of cases unattended with difficulties. However, the fact that similar physical signs may be produced by other conditions, may sometimes render it quite hard to discover the real state of the case. Hypertrophy of the right ventricle and a louder second sound in the pulmonary artery, may result from any marked derangement in the lesser circulation, quite as often as from an affection of the mitral valve. Besides this, a systolic blowing sound may frequently be heard in cases of emphysema. In cases of this kind the only means of preventing the confusion of the two is, to make a careful examination into the development of the whole disease, and to ascertain if the patient has previously suffered from rheumatic arthritis, whether he has had a cough for a long time, or whether there be any emphysema, etc. It is easy to dispose of those cases of chlorosis, too, which lead us to falsely assume a disease of the mitral valve, on the ground of any temporary dilatation of the heart, in addition to the murmur that is heard over the apex. And besides, in cases of this nature, the second

sound in the pulmonary artery is seldom louder than usual, the murmur over the apex partakes more of the character of an organic murmur, and may usually be heard over the base as well, and the general state of the constitution points unmistakably to the real primary disease. Lastly, there is a class of cases, which, though not of frequent occurrence, still most certainly do sometimes occur. These are spontaneous dilatations of the heart, quite independent of any anatomical change in the valves; they are very misleading, for the relative insufficience of the valves that accompanies them produces a murmur, and at the same time we observe all the symptoms of cardiac hypertrophy. In instances of this kind, it is absolutely impossible to distinguish this from an insufficiency caused by anatomical changes. Seitz's excellent observations, made in Biermer's Clinique, have proved this in the most instructive manner.

The *prognosis* in this valvular disease, though certainly unfavorable as regards total recovery, is still relatively favorable. For the compensatory action, produced here both by the right and by the left ventricles, may often suffice for a long period. There are cases on record of patients who have lived over fifteen years with a valvular disease. Any intervening diseases, however, whether cardiac, pericardiac, or pulmonary, alter the prognosis very much for the worse. Young patients bear the affection better than old people; the danger is very great during pregnancy, and, in cases of women attacked in this state, the issue is often suddenly fatal.

Stenosis of the Ostium Venosum Sinistrum.

Whilst insufficiency of the mitral valve may in some cases, according to its anatomical nature, occur quite by itself, stenosis of the orifice occurs almost unexceptionally accompanied by insufficience of the valve. For the stiffened and hypertrophied valve, which is no longer able to lie flat against the wall of the ventricle during ventricular diastole, has not only a border in greater part shrunken (and thereby alone incapable of closure), but is so altered, too, in its flexibility, that the tension of the papillary tendons no longer suffices to bring the

borders into perfect contact. It is only in the most extreme cases of stenosis, such as those that are brought about by a cohesion between the borders of the valvular flaps, which only leaves a narrow split for the passage of the blood from the auricle into the ventricle, that the contraction of the papillary muscles is still capable of closing the small space. Even in the rare instances where the stenosis has originated acutely in large fibrinous deposits on flaps, or in a coagulation projecting into the orifice from the auricle, there generally is some trace of insufficience of the mitral valve present at the same time. However, the physical signs of stenosis are so prominent in the clinical view of the symptoms, that we are entitled to discuss this as the predominant derangement.

The mechanical action of this stenosis is to hinder the blood, brought from the pulmonary veins into the left auricle, in its passage into the ventricle. The left ventricle consequently receives an abnormally small supply of blood, and must thus propel less into the aorta during the systole. So we see the very same results produced as in insufficience of the valve, only that the stagnation in the pulmonary vessels and venæ cavæ is more intense, since the left ventricle here is not filled under abnormal pressure, as in the former case, and the powerful lever of compensation, formed by the hypertrophy of the left ventricle, never comes into play. The anatomical changes found in the several parts of the heart in the post-mortem examination quite correspond to these relations. The left auricle is dilated and hypertrophied, and in its appendix we frequently find a thrombus ; the walls of the pulmonary veins are thickened and discolored, and covered with white spots ; the capillaries of the pulmonary artery are in a state of ectasis, and in some places varicose ; the right side of the heart is dilated and hypertrophied ;—the left ventricle, on the contrary, is smaller and atrophied. It appears, in fact, so insignificant beside the right ventricle that it might be mistaken for a mere appendage of this one, which now alone enters into the formation of the cardiac apex. The aorta, too, in consequence of this diminution in the size of the left ventricle, is abnormally narrow so that its diameter above the valves may be considerably reduced. This

condition of the left ventricle, though the rule, is still liable to exceptions, and even in cases of extreme stenosis, the left ventricle has been found of *over* normal weight and volume. Friedreich has tried to explain this fact, in the absence of any further complication, by assuming that the increased tension in the venous and capillary systems of the body can produce an additional resistance to the arterial current, great enough to counteract the tendency in the left ventricle to diminish in size. It appears fair to include as well the two other factors of simultaneous insufficiency of the valve, and a contraction of the aortic lumen. If the nutrition be good, the latter must directly tend to hypertrophy even during its development, while the former counteracts atrophy.

A physical examination during life gives the following results: In some cases the apical impulse is visible within the mammillary line, between the fifth and sixth or the sixth and seventh ribs. But in the majority of cases extensive undulations are visible, which pass beyond the normal limits, both to the right and to the left. Owing to the horizontal position assumed by a very much hypertrophied right ventricle, the systolic motion may be seen to extend to the anterior axillary line towards the left, and beyond the right border of the sternum to the right. The resistance of the apical impulse is very weak, and in bad cases it may be quite imperceptible. In palpating, the finger feels a purring vibration over the cardiac apex; this generally precedes the cardiac systole immediately, and is consequently presystolic; but it may be purely diastolic in many cases. Frequently one can perceive a simultaneous systolic vibration. Percussion shows an increase in both absolute and relative dulness as well to the right as to the left. The dilatation of the right auricle, when very considerable, may be recognized by a noticeable dulness to the right of the sternum. Auscultation does not always give similar results. Eddies are produced in the blood, as it flows from the wide auricle during diastole through the narrow orifice into the ventricle, and these eddies give rise to a murmur. Consequently the murmur produced by stenosis can only be heard during the diastolic phase. And, indeed, this harmonizes with a number of cases in which one only hears a

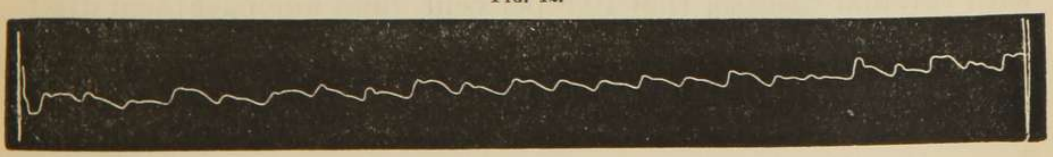
diastolic murmur, or along with this a systolic murmur as well, since there is nearly always more or less insufficience of the valve. The diastolic murmur is blowing or grating, somewhat uneven and protracted, and extends to the succeeding systole. This protraction may be easily understood, if we consider how much longer the diastole must last to permit the same quantity of blood to flow through a constricted opening as would flow through an orifice of the normal size. In many cases the murmur is not properly audible till towards the end of the diastole, when the simultaneous contraction of the auricle increases the force of the current, as at the beginning of the diastole the rate of the current is not great enough to produce this. Consequently it immediately precedes the systole, and Gendrin, who was the first to study it closely, has given it the name of *presystolic*. The first cardiac sound immediately follows on this, and is usually loud and intensified ; sometimes it is pure, but more generally accompanied by a murmur. The majority of cases answer to this description, but occasionally the contraction of the auricles does not suffice to accelerate the current of blood to the degree necessary for the production of this murmur ; and thus it sometimes happens that we hear no murmur, notwithstanding the presence of stenosis.

We may accordingly distinguish three sets of cases in relation to the auscultatory phenomena : first, when we have a purely diastolic murmur ; secondly, a presystolic ; and thirdly, no murmur at all. The murmur proper to the stenosis always reaches its maximum intensity over the apex, or even a little to the left of this, since the current of blood takes that direction. The murmur is often more audible when the patient lies on his left side, instead of on his back ; and even when no murmur can be heard while the patient keeps in a position of rest, one can generally be perceived after active exercise, or when he is put lying on his left side. In addition to the murmur, we have another auscultatory phenomenon peculiar to stenosis. In a few cases (by no means so frequently as Geigel assumes) we may hear over the apex, and very distinctly over the pulmonary artery, a reduplication of the second cardiac sound, which produces a rhythm, called, after Bouillaud's example, the *bruit de*

rappel. Geigel called attention to its frequency of occurrence in stenosis, and explains it by the unequal tension in the aorta and pulmonalis, which may prevent the synchronous closure of the valves of the two vessels, since the aorta with its smaller contents contracts quicker, and thus closes its semi-lunar valves an instant before those of the pulmonary artery. The fact that the second part of the reduplicated sound may be heard much more accentuated over the ostium of the pulmonalis, lends a great deal of probability to the view that it is produced by the valves of this artery. Even where there is no reduplication, the second sound is more intense in the pulmonary artery, when the ostium venosum sinistrum is constricted; this is due to the greater tension of the vessel, and may be heard more plainly than in mitral valve insufficience, since in this case the second aortic sound, on account of the small tension in the aorta, is in itself weaker than normal. When the tension decreases in the pulmonary artery, the intensity of the second sound ceases; this takes place either when the right ventricle's force has been impaired by disease in the performance of its increased work, or when the right side of the heart is so filled by the increased stagnation that the ring of insertion of the tricuspid valve is widened, and the valve is no longer able to close the orifice. Relative insufficience of the kind frequently occurs in cases of mitral stenosis, especially towards the end of life, and may be diagnosticated by the symptoms peculiar to this affection; these we shall enter into more particularly when treating of this form of valvular defect.

FIG. 11.

FIG. 12.



The condition of the pulse is more characteristic in stenosis than in insufficience of the mitral. The frequency and rhythm,

indeed, may be normal, but the peripheral vessels are left so empty that the smallness, emptiness, and softness of the pulse serve as special landmarks. Of the two tracings we give here, the first displays a still perfectly regular pulse, with a slight systolic elevation and a scarcely perceptible elevation from recoil. The second (Fig. 12) shows the most marked irregularity, being taken after the compensation had been destroyed. In this case a purely diastolic murmur was heard at the apex. I cannot agree with Marey's view that the tracing of the pulse exhibits variations in regularity, according as we hear a diastolic, presystolic, or simultaneous systolic murmur; nor can I assent to his assertion, that, "when the mitral constriction is sufficiently marked to give rise to a diastolic murmur, it destroys the irregularity of the pulse" (loc. cit., p. 520).

It is unnecessary to repeat here all the symptoms of stagnation developed with the increase of disturbance in the compensation, both in the peripheral vessels and in the internal organs; they occur here in even greater degree in mitral stenosis than in insufficiency of the mitral valve. The complications in the lungs, the dark-red induration, and hemorrhagic infarction occur more frequently in cases of stenosis than where the insufficience preponderates. Hæmoptysis often occurs without any hemorrhagic infarction. The opinion used to prevail that it would be quite unwarrantable to connect this with the initial stage of phthisis. For observers, even of such large experience as Traube, have never seen caseous pneumonia developed under such conditions, when at the same time the right ventricle was hypertrophied, so that it would appear as though this valvular defect gave immunity from phthisis. However, Frommolt's results contradict this, for he found that out of all the cases in the post-mortem room of the Dresden Krankenhaus, where he met with valvular disease, that 10.8 per cent. (and amongst them diseases of the mitral ostium, and even stenosis of the ostium venosum sinistrum) occurred in company with pulmonary consumption. Thus this questionable immunity requires at least a new investigation.

Icterus, without any catarrh of the gall-ducts, has been observed as a sign of stagnation more frequently in stenosis than

when insufficience predominates. In the cases which have come under my investigation acids of the gall were found in the urine.

The *diagnosis* of this valvular disease is only difficult in cases where no murmur can be heard or where a systolic murmur alone is audible on account of the simultaneous insufficience. Under these conditions it is impossible, and as yet of no practical importance, to avoid the confusion with simple insufficience. But the condition of the pulse alone will excite the suspicions of the observer as to the existence of merely this latter. In the majority of cases the presystolic, and in others the purely diastolic murmur, heard with greatest intensity over the apex of the heart, is an unerring guide, and, if we take into review the results of the percussion, renders our decision easy.

The *prognosis* is more unfavorable in cases where stenosis predominates than where insufficience is predominant, for the compensation is rendered easier by the share which the left ventricle takes in it. Still, the compensation brought about by the right ventricle alone, as experience teaches us, may suffice to sustain life endurably for years, and exceptionally even for more than ten years. But we must not forget that such cases are of an extremely exceptional order.

Diseases of the Aortic Valves and of the Ostium Arteriosum.

Besides the text-books already given, consult: *Corrigan*, Inquiries into a new disease of the heart. Edinb. Journ. 1836.—*Costa Alvarenga*, Mémoire sur l'insuffisance des valvules aortiques, etc. Paris, 1856, and Union Méd. 1863.—*Lambl*, Ueber papillare Excrescenzen an den Semilunarklappen der Aorta. Wien. Wochenschr. 1856.—*Luschka*, Ueber zottenförmige Bildungen an den Semilunarklappen der Aorta. Deutsche Klinik, 1856.—*Maurice*, De la mort subite dans l'insuffisance des valvules sigmoides de l'Aorte. Thèse, Paris, 1860.—*Duroziez*, Du double souffle intermittent comme signe de l'insuffisance aortique. Arch. génér. 1861, and Gaz. hebd. 1865. and Réponse à Mr. Traube, in Gazette hebdomadaire, 1873, No. 7.—*Botkin*, Medic. Klinik in demonstrat. Vorträgen. Berlin, 1867.—*Traube* (*Fräntzel*), Ueber Zwei eigenthümliche Phänomene bei Insufficienz der Aortenklappen. Berl. klin. Wochenschr. 1867.—*Duroziez*, Des Maladies organiques du cœur et de l'Aorte et du double souffle crural de l'origine saturnine. Gaz. des Hôp. 1867.—*Traube* (*Fräntzel*), Zwei Fälle von

Stenose des Ostium der Aorta. Berl. klin. Wochenschr. 1867.—*Marey*, Note sur un nouveau signe de l'insuffisance aortique. Gaz. Méd. de Paris, 1868.—*Leared*, Aortic valve disease apparently caused by syphilis. Transact. of Path. Soc. 1868.—*Guéneau de Mussy*, Leçon clinique, etc. Gaz. des Hôp. 1871.—*O. Becker*, Ueber Retinalarterienpuls bei Insufficienz der Aortenklappen. Monatschr. für Augenheilk. 1870.—*Michel Peter*, L'insuffisance aortique, etc. Union méd. 1871.—*Riegel*, Ueber den Doppelton u. s. w. Deutsches Archiv, 1871.—*Hoffmann*, Der Duroziez'sche Doppelton u. s. w. Berl. klin. Wochenschr. 1872.—*Robert King*, Extreme aortic stenosis. Path. Trans. 1873.—*Foster*, Clinical lecture on rupture of the aortic valves from accident. Med. Times, 1873, December.—*Michel Peter*, Leçons de clinique médic. T. I. 1874.

Insufficiency of the Aortic Valves.

Slight anatomical changes frequently occur in the aortic valves without causing any disturbance in the mechanism of their closure. To this class belong those jelly-like thickenings, the soft-tufted proliferations of the endocardial connective tissue around the nodulus Arantii, or atrophy of the same towards the border, causing the so-called "fenestrations." Even these latter produce no derangement, as they are generally mere slits, and lie between the free border and the line of closure. But when several of these become confluent and form one large opening, they too may produce insufficience. Another cause which may produce this incapability to close, are the wart-like and polypous excrescences, if situated along *the line of closure*, and when they fringe the whole border of the flap, partly as chalk and partly as fat, or when this border exhibits irregular gaps and cuts between the warts, as frequently happens. On the other hand, chalky deposits in the basal parts, even when of some extent, can exist without any injurious results. But the insufficience is generally produced by fibrous thickenings of one or two (seldom of all three) flaps, accompanied by considerable contraction of the borders, and consequent diminution of the height of the flap. We may further mention as anatomical causes of rarer occurrence: the formation of an aneurism on a flap of the valve, with or without perforation, the breaking off of a flap from its insertion and adherence of one to the wall of the aorta. Though in all the changes we have mentioned there generally occurs a

lateral coalescence between the two flaps, still insufficience is in most cases the only result, either because the union is not extensive enough to produce stenosis, or, as is usually the case, because the septum brought about by coalescence soon atrophies, and later on only exists as a small border between the two flaps united now to one. The close relation of the large flap of the mitral to the aortic valves generally entails an affection of the mitral, when endocarditis attacks the aortic valve. Still the endocarditic process may occur exclusively confined to the semi-lunar valves, and the mitral be entirely free. But, as a fact, endocarditis is by no means so frequently the origin as disease which commences in the walls of the aorta, for these are but seldom quite free from atheroma, of which we generally find most just above the valves, and from here it spreads downwards on to the valves. Many observers still question the possibility of a relative insufficiency of the semi-lunar valves, independent of any anatomical changes in the valve, and resulting purely from too great expansion of the initial part of the aorta, and consequent over-dilatation of the orifice. I myself have never seen any confirmatory cases, and must accordingly leave it an open question.

The mechanical effect of this insufficience is a regurgitation of blood during the diastole of the ventricle, proportionate to the degree in which the valve remains open, and thus the left ventricle is supplied not only from the auricle, but also from the aorta. This produces dilatation of the ventricle (especially its portio aortica) so great that the longitudinal sulcus no longer corresponds to the actual line of separation of both ventricles, since the septum bulges very abnormally into the right ventricle. The ostium venosum sinistrum, too, is in consequence always dilated, and the flaps of the mitral are stretched and lengthened. The increase of force, necessary to propel the larger quantity of blood, supplied from both sides, into the aortic system, must correspond to the dilatation of the ventricle. And though in general the dilatation appears more considerable than the increase in volume, yet in most cases the hypertrophy bears a very favorable comparison with the dilatation—that is to say, it is well developed. On opening the pericardium, we see that the left ventricle occupies a very large space, and not only is it the sole factor forming

the apex, but this apex projects, too, lower than the end of the right ventricle. On further observation we see that the thickness of the wall of the left ventricle has increased more than anything else, and that it often measures more than from 2 to 3 cm. and even at the apex 1 or 1½ cm. Often, indeed, the volume of the wall is not absolutely greater, but still, in comparison to the breadth of the ventricle, it looks hypertrophied. The papillary muscles are not proportionately enlarged with the other parts of the left ventricle—that is, they are not round and hypertrophied, but generally, as Traube first pointed out, lengthened and flattened, corresponding to the considerable strain to which they are exposed during diastole. It happens exceptionally that the right side of the heart is simultaneously a good deal hypertrophied, though no cause for it can be found in the lungs. And in two cases I have seen the process so far advanced, that the right ventricle actually formed the whole of the apex by itself.

The physical signs exhibited by this class of patients arise chiefly from hypertrophy of the left ventricle. The cardiac impulse is visible outside the mammillary line, as far as or even to the left of the anterior axillary line, lower down than usual, generally between the sixth and seventh ribs, but often too between the seventh and eighth. The impulse is broad, raising sometimes an area of 3″ (in breadth) with some force; in other cases we see no defined impulse, but only a systolic elevation which raises the whole thorax. Further, we observe considerable palpitations in the jugulum, vigorous pulsation of the carotids, and in many cases too a pulsation on the right border of the sternum at the level of the second intercostal space, and corresponding to the position of the aorta ascendens. In palpating with the finger, we experience considerable resistance from the cardiac impulse; over and along the sternum, especially about the third sternal cartilage on the right, we may often feel a diastolic vibration, which, indeed, may be also felt at the apex, though very much less plainly. The percussion shows a considerable increase in the area of dulness, both relative and absolute, especially in its long diagonal. It generally begins high up, on the third and sometimes even on the second rib, extending to the

sixth or seventh rib, crossing the mammillary line as high up as the nipple, and passing downwards and outwards—sometimes two inches outwards. To the right also the boundary of dulness generally passes over the right border of the sternum. Botkin records cases in which the dulness has extended a little beyond the situation of the impulse, and similar cases have occasionally come under my own notice.

In case the arch of the aorta is dilated, as so often happens, we find a dulness from 1 to 2 cm. broad running from right to left in an oblique direction over the manubrium sterni.

In auscultating the heart we hear a weak first sound over the apex, and a diastolic murmur, the intensity of which increases as the ear approaches the base, and becomes most distinct about the third right sterno-costal articulation. The murmur is especially marked over the sternum, and particularly along the left border; it is propagated along the course of the aorta, and is loudest in the left interscapular space. Its character is almost invariably a whirring, rushing one. In the situation where we generally auscultate the aorta, the first sound is either short and definite, or is accompanied by a murmur, which may have its origin in the simultaneous stenosis, though it more frequently arises from the passage of the blood into the enlarged aorta ascendens. In nearly every case a systolic murmur is audible on the carotid arteries, and at the same time we may feel a vibration. The gentlest pressure of the finger on the vessel intensifies the vibration greatly. We may look for the origin of the systolic carotid murmur in the vibrations of the arterial walls. For, since the vessels which lie so near the heart can empty their contents during the diastole into the heart as well as into the capillaries, their tension falls very low, and then is raised very high during the next cardiac systole, through the expansion from an immense wave. And in this transition from a low to a high tension vibrations are easily produced. Though this explanation is generally received, and appears the most probable, yet after Talma's experiments the murmur may be regarded as a murmur in the blood, produced by eddying movements of the fluid particles, arising from the sudden increase in pressure during the systole. The physical conditions are in reality here the same as

in Talma's order of experiment. On the other hand, I maintain that when, as in other cases, a systolic sound is heard, it can be produced only by the vibrations of the vascular walls, since, according to our present knowledge, only a murmur and no sound can be produced by these eddies. During diastole there is either no perceptible sound at all over the carotids, because no sound capable of propagation arises from the aortic valves, or else we hear a murmur propagated from the aortic ostium. Even when one part of the semi-lunar valve is still capable of vibration, the sound produced by these vibrations is concealed at the ostium itself by the murmur; yet still it may happen that we can perceive a second sound in the carotids, though over the very ostium of the aorta nothing can be heard but a diastolic murmur.

Quite as considerable changes of tension take place in the peripheral vessels during systole and diastole as in the carotids, only differing in degree; they are also perceptible both to the ear and to the touch. In the first place, the radial pulse assumes a quick character from the greater facility the blood has for flowing centripetally. The wave is high, and the finger of the observer experiences a considerable stroke from it; but the very moment after the expansion of the vessel the systole follows extremely rapidly. It is this short duration of the diastole which gives such an exquisite impression of rapidity—of the celerity, which Corrigan first described, and in whose honor the French called it *pouls du Corrigan*. The sphygmographic tracing exhibits for aortic insufficience perhaps the most characteristic curves of all heart diseases, but it is most certainly not specific. The sharp line of ascension indicates the rapid and forcible dilatation of the wall of the vessel, since the lever of the instrument is raised with force, and falls at a very acute angle. The anachrotous formation of small hooks in that part of the tracing which corresponds to the diastole of the vessel should be no more regarded as a result of some defect in the instrument than the sharp point of the summit. Besides, the tracings are not at all constant. They vary with the degree of hypertrophy and the strength of the left ventricle, and, further, they vary according as the aortic insufficiency is complicated with much atheroma of the

arteries, or produced by the endocarditic process alone. Further variations may be caused by a combination with stenosis of the orifice, or finally by a complication of mitral insufficience, which so often occurs from disease of the aortic flap. I intentionally print two sphygmographic tracings, which vary from the usual curve given by Marey. Marey's (Fig. 13) occurs most frequently, but they all have the sharp line of ascension in common.

Fig. 13.

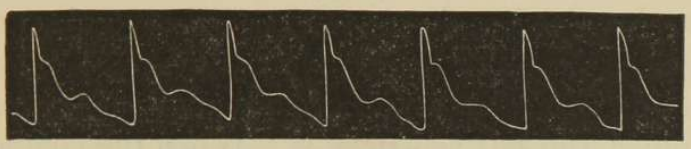
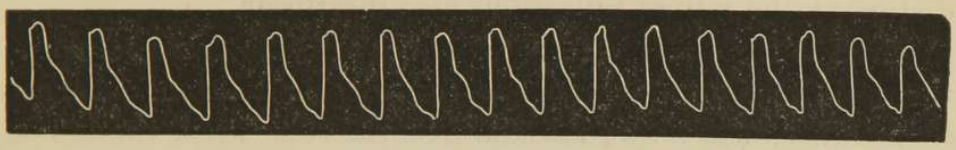
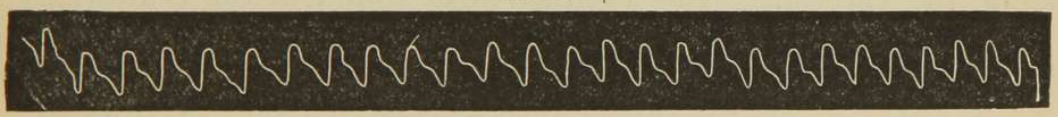


Fig. 14.



Aortic insufficiency with slight stenosis of the ostium and atheroma; especially characteristic are the steep ascending line, the marked formation of a hook or plateau, and the slight expression of the elevation for recoil.

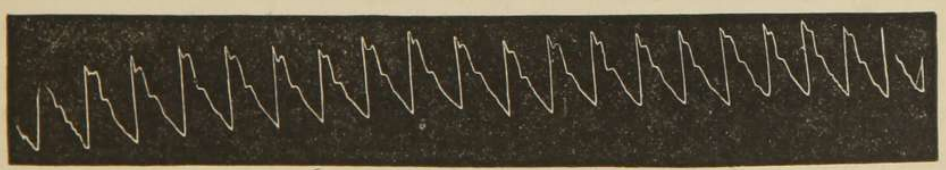
Fig. 15.



Insufficiency of the aortic valves, accompanied by insufficiency of the mitral, and after the compensation has been deranged. Caused by endocarditis, as proved by the post-mortem examination.

The tracing in Fig. 16 shows how little characteristic the "formation of hooklets" (Häckchenbildung) or the sharp summit of the ascending line is for this definite valvular disease as such;

Fig. 16.



for here the former is very much more strongly expressed, though there was no valvular defect at all, and nothing but hypertrophy of the left ventricle in consequence of renal disease and sclerosis of the aortic system.

Also the great force with which the hypertrophied ventricle propels the abnormal quantity of blood into the aorta, may be recognized not only by the height of the pulse, but also by the fact that pulsation becomes visible in the smaller vessels, where before it was not even palpable. In the very capillaries, where pulsation is usually only seen under the most favorable circumstances, it can be seen when the aortic valves are insufficient, as Quincke has demonstrated. Under these conditions, too, the *spontaneous pulse* has been seen by different observers in the branches of the retinal artery (comp. Becker). But this only occurs in highly-developed cases, and is more easily seen the more hypertrophied the left ventricle is. When the compensation is disturbed, the retinal pulsations also disappear.

Even in the small vessels audible vibrations are produced in the rapid transition from a low to a higher tension. One can, for instance, hear a systolic sound on the volar arch, though only in extreme cases, while, even when the defect of the valve is but slight, a systolic sound may be heard on the large arterial trunks, especially the cruralis, the tension of which is greater than that of the carotid. This production of sound in the cruralis is, indeed, no specific symptom of the particular disease under consideration, since it can occur as well under other conditions, when the retreat of the blood during cardiac diastole is facilitated, as, for instance, when the tone of the vessels is low in fever or chlorosis; still it is one of the most constant symptoms. There is another symptom to which special attention has been drawn by Duroziez, and which occurs on the cruralis. It too is not pathognomonic, but occurs most frequently in aortic insufficiency. Duroziez found that if one pressed, whether with the stethoscope itself, or with the finger above and below the stethoscope, on the crural artery, he would hear, not as usual, a simple systolic murmur, but a reduplicated murmur, both a systolic and a diastolic one. The latter is owing to the eddies which are produced in the blood during the diastole of the heart by the regurgitation of the centripetal wave through the artificially constricted lumen. Though this fact is very valuable and perfectly established through repeated observation, yet it only adds one to the number of symptoms of insufficience of the aortic valves.

But Traube has made us acquainted with a new symptom, which enables us to *estimate the degree of insufficiency of closure in the valve.* He demonstrated that when the insufficience was very great, a *reduplicated sound*, a systolic and a diastolic, can be heard on the cruralis without any artificial pressure. He explains the production of the sound from the fact that membranes begin vibrating audibly during a transition from a maximum to a minimum tension. These conditions are best fulfilled by the crural artery, since its tension during the systole of the heart is abnormally great, and its slackness during diastole is very considerable, owing to the facility the blood has of flowing both into the capillaries and back towards the heart. However, we must here presuppose that the function of the left ventricle is undisturbed, and the vascular elasticity unimpaired. Otherwise this reduplicated sound may be absent, as in a case of atheroma which came under my observation.

We comparatively seldom hear a patient make any subjective complaints so long as the hypertrophied muscle of the left ventricle can perform its work and propel its increased supply of blood with sufficient force into the arteries. As insufficiency of the aortic valves produces no injurious reaction in the pulmonary circulation till a very advanced stage, patients suffering from this disease are for a long time free even from shortness of breath, which is seldom quite absent when the mitral valve is insufficient even in the compensation stage, and almost never absent after exertion. But in the hypertrophy itself lies a source for evils, which too begin to work. The constant expansion and persistent pressure to which the vessels are exposed serve to produce a condition of congestion perceived by the tendency to hemorrhage. It is more especially in the brain, where the compensation of circulatory derangements is greatly hindered by the unyielding auxiliary substance around the vessels, that fluctuations and agitations often assume a dangerous character, and annoy patients under the form of a beating headache, for the disturbed nutrition of the vessels (both etiologically connected with the valvular defect, and also being the result of a persistently high pressure) diminishes their power of resistance so much that death often results from hemorrhage into the brain. In this case both

factors combine, both the atheromatous process and the fatty degeneration of the small cerebral vessels, along with hypertrophy of the left ventricle. But there is a further symptom which annoys the patients—namely, *a pain occurring spasmodically*, situated beneath the sternum, of a constricting nature, and increased by exertion. It is often accompanied by an unspeakable feeling of terror, and is probably due to the simultaneous atheromatous process in the arch of the aorta and the irritation propagated from here to the cardiac plexus. Michel Peter records a case in which anatomical changes were found in the ganglia of this plexus after death, which goes to support his explanation of the symptom as we have just stated it.

When the propelling force of the circulatory apparatus is impaired by disease of the hypertrophied cardiac and vascular muscles, all the signs of over-filling and stagnation appear in the venous system alongside of arterial anæmia. These signs have been already frequently mentioned. The patients become short of breath and dropsical ; the peripheral cervical veins are expanded and over-filled. Most worthy of mention among the symptoms of disturbed circulation in the internal organs are the dizzy fits, which express venous hyperæmia in the brain, and albuminuria, indicating renal cyanosis. In this connection we must not omit to mention the inflammatory processes in the nutrition of the kidneys, which are one of the most frequent complications of aortic insufficiency, even at an early period, before there is any stagnation whatsoever. It is erroneous to look for the origin of albuminuria, as some have done, in active congestion, since mere increase of pressure in the aortic system never produces albumen in the urine. The anatomical changes in the kidneys in cases of this kind point to interstitial nephritis, and must be regarded as a complication of the cardiac disease. Occasionally there occurs a striking paleness of the face, which we must connect with the simultaneous albuminuria.

The *diagnosis* of aortic insufficiency can only be founded on the physical signs, the most definite of which are the diastolic murmur, with maximum intensity over the sterno-costal articulation of the third right rib and on the sternum, and, connected with this, dilatation and hypertrophy of the left ventricle. For

the other symptoms, as, for instance, the sounds in the arteries, especially in the cruralis, are not specific, and are not produced exclusively by the derangements in the circulation resulting from valvular disease, and consequently occur as well under other conditions, or may entirely cease when the compensation is disturbed, as in the case of the otherwise very characteristic pulse. In cases where doubts arise as to the signification of an audible diastolic murmur, especially where the mitral valve is involved, whether it is stenosis of the left ostium venosum or insufficience of the aortic valves (in the stage of disturbed compensation), apart from the diagnostic value of ascertaining the situation of maximum intensity of the diastolic murmur, great advantage may be derived from Duroziez's symptom of the "double souffle intermittent" in the cruralis, which occurs only in this one form of disease. Traube's symptom of the reduplicated sound in the crural artery enables us to estimate the degree of insufficience when this is considerable and while the compensation remains undisturbed. As an insufficience, capable of producing this reduplicated sound, excludes any simultaneous stenosis of the ostium, the symptom obtains a new importance.

The *prognosis*, as regards the length of life and activity of the patient, is more favorable in aortic insufficiency than in any other valvular disease. He may enjoy years of undisturbedly good health, as long as the increased force of the left ventricle suffices to prevent any stagnation of the circulation. It has been observed that disturbances of this course set in earlier, when the patients are young and the process originates in endocarditis, than in old people with atheroma. As soon, however, as conditions appear which render the compensation imperfect, the reaction on the venous system takes place rapidly, and the whole cyclus of symptoms of stagnation leads far more quickly to a fatal termination than is the case in uncomplicated mitral disease. Special caution must be taken in the prognosis, when the patient exhibits signs of a disarranged intracranial circulation, in the form of dizzy fits, or, more unfavorable still, fainting-fits. The fear of the occurrence of apoplexy cannot be regarded as a mere bugbear, in spite of weighty protests against it, and it should be well taken into consideration in forming a

prognosis. It is to be apprehended more especially in cases which originate in an atheromatous process. Even when death does not occur from apoplexy, yet it is more frequently of sudden occurrence in this than in the other valvular diseases.

Stenosis of the Ostium Aorticum.

The anatomical changes in the valves, producing stenosis of the orifice, consist chiefly of fibrous thickenings or calcifications of one or more flaps (sometimes all over and sometimes only at their base), and also lateral adhesions of the free borders. We generally find both changes occurring more or less together. When they are highly developed, the orifice is so constricted that only a narrow triangular split remains open, whereas in the normal condition it is large enough to allow the thumb to pass through it. In general, however, the stenosis of the ostium aorticum does not reach such an advanced stage as in the left ostium venosum. Just as in this latter case, on account of its anatomical cause, the insufficience was accompanied by stenosis, a similar relation is exhibited by the arterial ostium. It is only in the extremest cases of constriction, when the rigid flaps remain invariably expanded alike in systole and diastole of the ventricle, almost touching one another, that scarcely any blood can regurgitate into the ventricle during the diastole; in such cases alone do we see exclusively stenosis. The mechanical effect, whether this be produced by alteration of the valves, or by myocarditic changes in the pars aortica (a case of the very rarest occurrence), is always that the left ventricle has a difficulty thrown in its way which embarrasses its propulsion of blood into the aorta. If the power of the ventricle for the same unit of time remain the same, it cannot propel all the blood brought to it from the pulmonary veins through the constricted orifice. Consequently less blood must get into the aorta, and a positive quantity corresponding to this minus must accumulate in the left ventricle. This ventricle is dilated, and its muscle increases in proportion to its greater tension, so that its higher propulsive force overcomes the disturbance in the circulation. Accordingly,

the secondary changes we find in the heart itself are dilatation and hypertrophy of the left ventricle. The excentric hypertrophy, namely, that in direct proportion to the dilatation, is the more pronounced the greater the tension undergone by the ventricle. It is consequently greater if the stenosis be accompanied by insufficience of the valve than without this ; whereas, even in the most extreme cases of pure stenosis, the dilatation is thrown into the shade by hypertrophy, as is plainly demonstrated by Bamberger's measurements. Since death seldom occurs before the compensation has been destroyed by disease of the cardiac muscle or otherwise, we generally find the result of this in the shape of a dilated left auricle and right side of the heart. For, as soon as the blood accumulated in the left ventricle can no longer be propelled forwards by the stronger exertions of this hypertrophied muscle, the already abnormally high pressure in the pulmonary venous system must be propagated through the pulmonary branches into the right side of the heart, and thus compel this to take part in the compensation. The peripheral arteries and the aorta itself are found to have abnormally small lumina, in proportion to the small quantity of blood which flowed through them in the same unit of time.

The chief physical signs which the patients exhibit during life are increase of the volume of the heart, and especially dilatation and hypertrophy of the left ventricle ; but these are subject to variation, more particularly according to the degree of simultaneous insufficience of the valves. When stenosis greatly preponderates (the case now under our consideration), the area of cardiac dulness, as ascertained by percussion, is found to be moderately enlarged in its long diameter, below, and to the left. On the right the boundaries of dulness are generally normal. The apical impulse is quite different from what we see in insufficience of the aortic valves ; it does not raise a large area and make it vibrate ; on the contrary, it is often weak, and may frequently be even quite imperceptible. Traube has explained this striking fact by pointing out that one of the factors in the cardiac impulse, that is, the force of recoil or the power which drives the heart in the direction opposite to the systolic outflow, is impeded in stenosis, since the size of the aortic opening is

smaller, and the resistance to the outflow greater, than under normal conditions.

In auscultation we generally hear a systolic murmur and a weak diastolic sound over the apex of the heart. When the valves are at the same time insufficient, we may hear a diastolic murmur as well. This systolic murmur is of a sawing, whistling character, occasionally musical; it grows louder as one nears the articulation of the third left costal cartilage with the sternum, and reaches its maximum intensity to the right of the sternum at the sterno-costal articulation of the third right rib; but it can generally be heard so loud all over the heart that it is difficult to assign its situation of maximum intensity. The second diastolic sound is quite absent, or only slightly audible. The same may be found in the case of the carotids, where too we hear a loud systolic murmur and no second sound.

The state of the pulse is of as much importance as the results of auscultation, for it is distinguished by two characteristics peculiar to this valvular disease. Though the lumen of the radial artery remains normal, the pulse is strikingly low, while the tension is normal or even slightly increased. Besides its *smallness* and *hardness*, the pulse is remarkable for its slowness. The explanation of all this is very simple; the wave is low, because even the hypertrophied left ventricle can only propel the normal quantity of blood through the narrow orifice of the aorta by prolonging its systole; and thus the wave is made longer. Its slowness is due to the smaller number of contractions in the same unit of time, and this again is due to the abnormal length of each systole. That the systole is much longer, may be seen from such a sphygmographic tracing, as the one we give by

Fig. 17.

Marey, taken from a left ventricle still capable of work. Here the abnormal length is shown in the rounded top, almost forming a plateau, and in the long descending line. However, these tracings vary with the force of the left ventricle and the degree

of complication by insufficiency of the valve; so it cannot be regarded as specific for all cases. As soon as the working power of the left ventricle begins to be incapable of overcoming the resistance caused by the stenosis, the pulse grows smaller and strikingly soft.

In this case, too, when compensation is deranged, the symptoms of stagnation are developed in the different organs. In the *lungs* the altered condition of the circulation causes the patients to complain of dyspnœa, which occurs in fits, sometimes accompanied by appearances of catarrh, sometimes in the form of purely so-called stenocarditic attacks. But the stagnation here often gives rise to genuine hæmoptysis, corresponding to which apoplectic centres or hemorrhagic infarctions are found in the post-mortem examination of the lungs. Anæmia in the brain produces fits of faintness and dizziness, which generally occur suddenly. It may happen, too, that the nervous derangements exhibit quite a peculiar picture. In apparently good health, for instance, all the joints relax, so that the patient gives one the impression of a person sleeping calmly in a sitting posture; he retains consciousness, but has no command of motor power. In other cases, genuine epileptic fits may be observed.

There are no special difficulties attached to the formation of a *diagnosis*, when all the physical signs are present, and when attention is paid as well to the condition of the pulse with its smallness and hardness, as to the auscultatory symptoms. The systolic murmur, indeed, with its greatest intensity over the third right sterno-costal articulation, can originate in disease of the ascending aorta as well as in mere roughnesses on the flaps of the valves. However, the simultaneous absence, or at least striking weakness of the second sound, and the accompaniment of a small, hard, slow pulse, when present, insure against any such confusion. But when the left ventricle's force is impaired, the characteristic hardness of the pulse may completely disappear, leaving only its smallness. In this case we should remember Forget's accurate remark: "La petitesse du pouls est un signe banal, qui n'a de valeur que lorsqu'il est joint à la dureté, laquelle indique la force de contraction du ventricule gauche." For the auscultatory sign of the systolic murmur over the aortic ostium, with

merely a small pulse, even with perceptible hypertrophy of the left ventricle, does not justify us in assuming the presence of stenosis. The absence of the apical impulse materially supports the diagnosis, when the other signs are present, but only after the exclusion of all other forces which could render the impulse invisible.

The *prognosis* as to the duration of the affection is in so far favorable as the disease may last a long time without impairment of the general health and activity, until the compensation begins to fail. But, when the stenosis is extreme, the symptoms of disturbed compensation appear far sooner than in a case where insufficiency of the valves predominates, and, compared with this latter, the prognosis of stenosis is not nearly so favorable. But, if we compare the prospects of stenosis of the ostium aorticum with those of stenosis of the ostium venosum sinistrum, the prognosis of the former is far more favorable as regards the duration of life.

Valvular Diseases of the Right Side of the Heart.

Allan Burns, On some of the most frequent and important heart diseases; chapter on pulsation in the epigastric region.—*Friedrich Cramer*, Die Krankheiten des Herzens. Cassel, 1837. Anmerkung über die Venenpulsation, p. 50.—*Knabe* (*Traube*), De venarum intumescentia atque pulsatione. Diss. Berol. 1853.—*Ch. Bernard*, Quelques remarques sur les lesions valvulaires des cavités droites du cœur. Arch. génér de méd. 1856.—*Roth*, Fälle von Insufficienz der Tricuspidalklappe. Baier'sches Aertzl. Intelligenzblatt. 1858.—*P. Guttmann*, De insufficientia valvulæ tricuspidalis.—Berol. 1858.—*Kolisko*, Fall von Insufficienz der Tricuspidalklappe. Zeitschr. der Wiener Aerzte, 1859.—*Bamberger*, Beobachtungen über den Venenpuls. Würzb. med. Zeitschr. 1863.—*Geigel*, Ueber den Venenpuls, ibid. 1863.—*The same*, Weitere Beobachtungen über Tricuspidalinsufficienz und Venenpuls, ibid. 1865.—*Haldane*, Case of disease of tricuspid valve. Edinb. Med. Journal, 1864.—*M. Seidel*, Ueber Pulsation der Vena cava inferior. Deutsche Klin. 1865.—*Friedreich*, Ueber den Venenpuls, im Deutsch. Arch. f. klin. Med. 1866.—*Bouger*, De l'insuffisance de la valvule tricuspide. Thèse, Paris, 1866.—*Mahot*, Des battements du foie dans l'insuffisance tricuspide. Paris, 1869.

Insufficiency of the Tricuspid Valve.

The anatomical changes which produce incapability of clos-

ure in the tricuspid valve, just as in the mitral, originate almost solely in endocarditic processes. Very rarely, indeed, are cases seen of myocarditis involving the septum and forming abscesses, which burst into the ventricle, and thus give rise to the rupture of a flap from its insertion. In such cases insufficience exclusively is produced ; but in cases, too, where the endocarditic changes lead simultaneously even to a slight adhesion of the flaps, the accompanying stenosis of the orifice is extremely trifling. Disease of this valve nearly always begins during fœtal life, and seldom occurs as an independent affection in adults—in fact, out of a hundred cases of cardiac valvular disease, scarce two occur in the tricuspid ; and even in these few cases the tricuspid is generally not the only affected part, being nearly always accompanied by disease of another valve, usually of the mitral. But the form of relative insufficience, described by Gendrin, occurs more frequently than that which is due to shrinking and thickening of the borders of the valves. Hindrances in the lesser circulation, whether from affection of the lungs themselves or from stenosis of the left ostium venosum, produce an accumulation of blood in the right ventricle. This dilates the ventricle very considerably, so that the ring of insertion of the tricuspid becomes larger, so much so that the flaps, though very slightly, if at all, anatomically altered, are rendered insufficient for the closure of the orifice. All arguments which have been brought against the possibility of this relative insufficiency are founded mainly on the fact that the flaps of the tricuspid are so long that one of them would suffice to close the normal ostium. But these arguments will not stand testing, and have been refuted by different investigators, most thoroughly by Friedreich, whose measurements have put it beyond doubt that even the height of the tricuspid flaps may be too small for the enlarged circumference of the ostium. Besides, clinical appearances prove the presence of a relative insufficiency with the greatest certainty.

The mechanical result of this valvular defect consists of regurgitation of blood into the right auricle during every ventricular systole. The auricle is consequently dilated and hypertrophied, too, in proportion to its slender muscle. The tension in the venæ cavæ must rise considerably, whilst that of the pulmonary

artery and aortic system falls. Just as we saw the left ventricle dilated and somewhat hypertrophied in insufficience of the mitral valve, where the filling took place under high pressure, so here we see the right ventricle dilated and hypertrophied, since it, too, is filled during diastole under extra pressure. This change in the ventricle is the only one directly connected with the affection of the tricuspid, but not the only change we may observe. Other changes may take place in the left ventricle, chiefly the result of mitral affections which are present at the same time, and these are naturally not without their influence on the degree of dilatation and hypertrophy of the right ventricle.

The physical symptoms, in the majority of cases, indicate a complicated affection of the heart, and we must accordingly in each case separate the signs of tricuspid disease from those of others simultaneously present. Inspection and palpation always indicate an extension of the cardiac impulse to the right. In percussing we find the area of cardiac dulness enlarged chiefly in width; when the right lung is not at the same time hypertrophied, a dulness is most strikingly audible on the right of the sternum between the fourth and second ribs, corresponding to the dilated auricle. Alterations of the figures of absolute and relative dulness to the left only occur when changes are at work at the same time in the valvular apparatus of the left ventricle, resulting in the development of changes of volume of the left ventricle. In auscultation we may hear a blowing murmur over the lower segment of the sternum, synchronous with the cardiac systole, and reaching its point of maximum intensity on the right border of the sternum, between the fourth and fifth ribs. This is to be regarded as a symptom of an affection of the tricuspid. On account of the mitral affection which accompanies nearly every case, we hear a systolic or diastolic murmur over the cardiac apex, according as insufficience or stenosis of the bicuspid preponderates. The second sound is weak over the right side of the heart, where we hear it as transmitted from the pulmonary artery. On this artery itself, too, both sounds are weak, on account of the emptiness which prevails in it during this valvular disease. Still the symptom may be

modified in another direction by the presence of a mitral affection. The aortic system receives less blood than normally, in consequence of the empty condition of the pulmonary artery, and so the radial pulse feels very low. But no further variations can be detected in it. On the other hand, on the visible veins of the neck, and, first of all, on the bulb of the internal jugular vein, then along the whole vein itself, and later also on the external jugular, we can see and feel a pulsation quite plainly in the majority of cases. Even before the cervical veins begin to pulsate we may observe the phenomenon in the liver. Here its occurrence is attended with less difficulty than in the veins of the neck, since the hepatic veins possess no valvular apparatus which could hinder the regurgitation of blood. For, as we have already explained in the general introduction, the venous pulse in the neck is never visible and palpable until the valves of the veins are incapable of closure, in addition to insufficience of the tricuspid valve. When there is no insufficiency of the venous valves, the pulsation only occurs in the bulb of the vein, and not along its whole course in the neck; while in the liver it is perceptible at an earlier stage, on account of the absence of the above condition. When the pulse is confined to the bulb, and the valves are unimpaired, a sound is produced by the vibrations from their closure. This "sound of the jugular valves" was first remarked by Bamberger, and is of great diagnostic value for those cases of tricuspid insufficiency where we cannot perceive any pulsation on the venous bulb. The venous pulse in both the neck and the liver may appear and disappear successively in the course of the disease. All forces which tend to diminish the stagnation in the venæ cavæ, or lessen the quantity of blood in the body, or reduce the propulsive force of the right ventricle, may make the venous pulse *in the neck* disappear; on the other hand, quite local influences, as, for instance, an accumulation of serum in the peritoneum and the change in the liver's position produced by this, may render the hepatic pulsation for a short time invisible, but it returns immediately after puncture. Under favorable conditions the vena cava inferior, as Geigel has demonstrated, may be seen and felt as a pulsating vessel even in the right mesogastric space. All further

details regarding the venous pulse need no repetition here, as we entered so fully into them in the general introduction.

Functional disturbances occur here at an earlier stage than in the left ventricle, for the compensatory action of hypertrophy in the right auricle, owing to its slight muscularity, is very trifling, and the increase of volume of the right ventricle has generally to do duty for the mitral affection. Consequently all the symptoms of stagnation, such as dropsy and cyanosis, appear early in the course, and are never absent. The pulmonary circulation, too, suffers some derangement both at the commencement, since the pulmonary artery is not normally supplied, and also later, when there are numerous coagula formed in the right auricle and ventricle, giving rise to stagnation and infarction.

The *diagnosis* is rendered specially difficult by the simultaneous presence of mitral disease. It is particularly important not to be deceived by murmurs transmitted from the mitral orifice. In this respect an accurate discrimination of the timbre of the murmur is of great service. The diagnosis may be established with certainty when we see a plain and vigorous pulsation in the cervical veins or in the region of the liver, in conjunction with a systolic murmur between the sternal insertions of the fourth and fifth ribs, and a weak second sound over the pulmonary artery.

The *prognosis* is unfavorable on the mere grounds we have mentioned, even if the tricuspid affection were completely uncomplicated; and it is all the worse, as this valvular disease seldom develops before others are already in action.

Stenosis of the Ostium Venosum Dextrum.

Stenosis of the right venous ostium occurs still more rarely than insufficiency of the tricuspid valve, and when it does occur it dates from fœtal life, and, one may say, always is accompanied by some other cardiac affection, especially one of the left venous orifice. The anatomical change which produces the constriction, too, is similar to that of the left side which we have already described. In examining the heart, we find as the result a considerable dilatation of the right auricle, and hypertrophy of the

right ventricle generally, as the venous ostium of the left side is usually constricted at the same time, but the left ventricle is usually atrophied. Still, we must not forget that the condition of the left ventricle is also dependent upon simultaneously occurring disease of other valves, and may, accordingly, in some cases be found hypertrophied. This was the case in an instance observed by Forget, where the aortic ostium was constricted. He found the left ventricle considerably dilated and hypertrophied.

Since a stenosis *completely confined* to the right ostium *is not recorded*, the physical signs correspond to the complications. Percussion proves an extension of the areas of absolute and relative dulness in the transverse diameter of the heart, indicating chiefly hypertrophy of the right auricle. In auscultating we actually hear a number of murmurs, from which we must isolate those that point to stenosis. It is consequently merely a rule derived from theory which states that a diastolic or presystolic murmur is heard on the right border of the sternum, about the fourth rib. In a case where the ostium was only wide enough to admit the point of the little finger, Hope could perceive no murmur at all. This will cause less astonishment when we consider that the same has been observed of the mitral orifice; both may be explained on the ground of weakness of the circulation. Theory would also require a strikingly weak second sound in the pulmonary artery, owing to its small contents. Yet the presence of other valvular diseases may render this symptom, too, illusory. The appearances on the cervical veins, notwithstanding the tricuspid insufficience always connected with stenosis, are not necessarily so prominently expressed as in a case of the valvular disease alone, where the force of the hypertrophied right ventricle comes into play. And it is probably an error on Kreyssig's part to state that both the cervical veins and liver pulsate *most* strikingly in stenosis. Others state that the symptoms are wholly absent; but this is most distinctly disproved by a case which Burns records. It is absurd to mention any compensation that may take place in this disease, for this must be derived solely from the right auricle, the muscle of which, even when hypertrophied, is insufficient. All symptoms

of venous stagnation, such as dropsy, cyanosis, etc., are accordingly quickly developed, and soon lead to a fatal result.

The *diagnosis* would be easily established if the constriction of the right venous ostium were the only valvular disease we had to deal with. But the isolation of the symptoms from those of the other diseased valves renders the task very difficult. Theoretically, we should lay most weight on a diastolic or presystolic murmur, which reaches its maximum intensity over the fourth sterno-costal articulation, and which can be distinguished by its timbre as one that has not been propagated from the left ostium. Besides this, as Hope's observation shows, there may be no murmur in stenosis at all, and this deficiency is no proof against the presence of the disease. The great scarcity of actual observations does not as yet allow of an exhaustive treatment of the diagnostic possibilities.

The *prognosis* is even more unfavorable than that of preponderating insufficience.

Diseases of the Valves and of the Ostium of the Pulmonary Artery.

In addition to the handbooks we have given, consult: *Norman Chevers*, Recherches sur les maladies de l'artère pulmonaire. Archives génér. de méd. 1847 (extract from the London Med. Gaz. 1846).—*Benedict*, Fall von Insufficienz der Valv. semi-lunar. Arter. pulmonal. Wiener Wochenschr. 1854, No. 35.—*Frerichs*, Insufficientia valv. Arter. pulmon. etc. Wiener Wochenschr. 1853.—*Stanhope Templeman Speer*, Case of cyanosis with extreme contraction of the orifice of the pulmonary artery. Med. Times, 1855.—*H. Meyer*, Ueber angeborene Enge der Lungenarterienbahn. Virch. Arch. 1857.—*Whitley*, Cases of diseases of the pulmonary artery and its valves. Guy's Hosp. Rep. 1858.—*Kolisko*, Fall von Insufficienz der Pulmonalarterienklappen. Zeitschrift der Wiener Aerzte, 1859.—*v. Dusch*, In Verhandlungen des naturhistor. Vereins zu Heidelberg, 1859.—*v. Wahl*, Acute Endocarditis der Pulmonalklappen. Petersb. Med. Zeitschr. 1861.—*Klob*, Beiträge zur Pathologie der Pulmonalarterienklappen. Zeitschr. der Wiener Aerzte, 1861.—*H. Lebert*, Ueber den Einfluss der Stenose des Conus arteriosus des Ostium pulmon. auf Entstehung von Tuberkulose (the older records of cases may, too, be found here). Berl. klin. Woch. 1867.—*Kappeler*, Stenose der Arteria pulmonalis. Arch. f. Heilk. 1863.—*Mannkopf*, Ueber Stenose des Ostium arteriosum der rechten Herzkammer. Berl. Charité-Annalen. 1863.—*H. J. Halbertsma*, De afwijking van het tusschenschot, etc. Ned. Tijdschr. voor Geneesk. 1862.—*Brondgeest*, Over pathol. veranderingen

der Arteria pulmonal. enz. Nederl. Archief. voor Geneesk. en Naturk. 1864. —*Karl Stölker*, Ueber angeborene Stenose der Arteria pulmonalis, Bern. 1864. —*Kussmaul*, Ueber angeborene Enge und Verschluss der Lungenarterie. Zeitschr. f. rat. Med. 1865.—*van Veen* (*Rosenstein*), Over Stenose van het Ostium der Arteria pulmonalis. Gron. 1870.—*Roeber*, Ein Fall von Insufficienz der Pulmonalklappe. Berl. klin. Woch. 1870.—*H. Meyer*, Mittheilungen aus den pathol.-anatom. Demonstrationen von Buhl. Baier'sches Med. Intell.-Blatt, 1870.—*Const. Paul*, Du rétrécissement de l'artère pulmonaire contractée après la naissance, etc. Union médic. 1871. Nos. 97–112.—*Chr. Fenger*, Stenose of ostium pulmonale, etc., aus Nord. Medisk. Archiv (Refer. in Virchow und Hirsch. Jahresber. 1874).

Insufficiency of the Pulmonary Valves.

Whilst insufficience of the right venous ostium is almost unexceptionally found to be accompanied by disease of some other valve, insufficience of the pulmonary valves occurs more independently; it may also be acquired after birth, though exceedingly rarely, and thus it does not originate solely during fœtal life. Both the endocarditic and the atheromatous processes give rise to the anatomical changes in the valves, consisting mainly of shrinking and thickening of the free border. Wahl has observed cases in which the valvular flap was almost completely destroyed by acute endocarditis. In the small number of cases as yet recorded we find one where the flap of the valve was ruptured at its insertion, after perforation of the septum by myocarditis, and thus insufficience was produced; and in two cases occurred the rare anomaly of a superfluous flap, which was either atrophied or totally annihilated (Kolisko, Klob). Under the last-mentioned conditions the case is one of pure insufficience, whereas in all other cases there is generally more or less stenosis at the same time. The same mechanical results of the valvular disease prevail for the right ventricle as for the left in insufficience of the aortic valves. Accordingly, where the defect has lasted long enough to allow the development of the secondary symptoms, dilatation and hypertrophy of the right ventricle and auricle are observed. As the consequence of this, and harmonizing with the conditions in aortic insufficiency, we find the pulmonary artery dilated, both trunk and branches, and along with this we see signs of deranged nutrition on the walls of this

vessel. When the aortic valves are at the same time affected, results of this are found in the *left ventricle;* whereas it is otherwise always atrophied, as may easily be imagined from the small quantity of blood it receives. At the post-mortem examination less blood than usual is found in the lungs, even when the mitral valve as well is insufficient, as in one case recorded by Klob. But this does not justify Kolisko in excluding the possibility of a temporary hyperæmia during life. The quantity of blood is in reality dependent on the degree of stenosis accompanying the insufficiency, and also on the secondary hypertrophy of the right ventricle. Both the lobular patches of pneumonia, which are repeatedly found, and also the hemorrhagic infarction, testify to increased pressure existing in the pulmonary vessels at the time of compensation. As physical signs we may recognize: enlargement of the volume of the heart, found by percussion, chiefly towards the right; in auscultating, a diastolic murmur, which reaches its maximum intensity on the left border of the sternum between the second and third ribs. This murmur is very loud, and can be heard also over the inferior part of the sternum, as it is propagated towards the right ventricle in the direction of the current which produces it. On the other hand, it cannot be propagated along the course of the large cervical vessels, and is never heard over them. A second sound is sometimes heard as well as the diastolic murmur, but this depends on the degree of capability of vibration which a part of the valvular flap may retain. Instead of the first sound, we hear a systolic murmur, which may originate in the transition of the current into the dilated trunk of the vessel, and, consequently, cannot always be regarded as a sign of the presence of stenosis, though it may arise from this in some cases. The subjective complaints of the patient relate chiefly to impeded respiration and palpitations of the heart, according to the over-filling or emptiness of the pulmonary vessels at the time. Neither of them, however, is severe; on the contrary, the course of cases on record shows that hypertrophy of the right ventricle is capable of compensation for a comparatively long time. The non-closure of the foramen ovale, which we often find in company with this valvular disease, contributes favor-

ably here, in as far as it allows blood to flow from the already overfilled right auricle into the left. By this means stagnation in the venæ cavæ is avoided, and, besides, it helps to fill the left ventricle. It is not before the compensation begins to fail, slowly and gradually, as we have observed, that very marked cyanosis, swollen cervical veins, and dropsy, etc., set in.

The *prognosis* is unfavorable as regards the final result. However, if we may draw conclusions from the exceedingly limited number of cases on record, the length of a patient's life, who is suffering from insufficience of the pulmonary valves, may be comparatively long.

Stenosis of the Ostium of the Pulmonary Artery.

An alteration of the ring of the valve, and of its flaps, by the endocarditic or atheromatous process, is acquired so very seldom after birth, that the whole literature only contains a few isolated cases (Whitley, Speer, Mannkopf), to which I can add my own, described by van Veen in his dissertation. Such an alteration can, like the process on the aortic valves, produce so marked constriction of the orifice, as to figure as the only, or at least very preponderant, derangement. All the other observations, even those regarding this valvular disease as one acquired after birth, were concerned with a combination of stenosis and a very considerable degree of insufficience (Frerichs, Benedict, etc.). When the stenosis is acquired after birth, it is chiefly produced by adhesion of the free borders of the valvular flaps, with or without simultaneous atheromatous thickening of their bases, and can reach such a point that it is impossible to pass a thin catheter or, as in Speer's case, a quill through the opening. In the case which came under my own observation, the atheromatous thickening was completely confined to the base of the valvular flap, while the trunk and branches of the pulmonary artery were quite free from it. Besides this, I only found a small opening in the position of the foramen ovale, two millimetres in diameter; the septum ventriculorum was perfectly closed, likewise the ductus Botalli. Though the patient did not come under my observation till he was thirty-three years

old, and had, from early childhood, exhibited signs of dyspnœa and cyanosis, still, the fact that all the fœtal passages had closed, justifies me in asserting that the disease was acquired after birth. The small opening of the foramen ovale may be regarded as within normal limits. Since a comparatively old age (up to thirty years and more) may be reached by a patient with *congenital* stenosis of the pulmonary ostium, which occurs incomparably oftener than the *acquired*, the question to decide, whether in each special case we are dealing with a congenital or an acquired cardiac affection, is not always an easy one, especially when we have not been previously acquainted with the patient. The most distinguishing characteristics of the two are as follows: the congenital stenosis always originates in a number of anatomical changes, chiefly *in the trunk of the artery*, which is itself constricted in consequence of constriction of the conus arteriosus by myocarditic formations; the congenital, too, may be accompanied by an affection of the valves, whereas a stricture confined to the latter alone, induced by fœtal endarteritis, is very rare; acquired stenosis nearly always depends on endocarditic or atheromatous changes in the valves or in the valvular ring alone. Further, in the congenital form we always find a number of anatomical anomalies of the heart and blood-vessels, especially an unclosed foramen ovale (in sixty-four out of eighty-two cases, according to Stölker's statistics), and a deficiency of the septum ventriculorum. This latter is always displaced to the left, and exhibits one opening of more or less importance, or else is not at all closed above. This deficiency in the septum is correctly regarded by H. Meyer as a result of the stenosis, and not the cause of it, as Heine asserts; nor can the two be regarded as co-ordinate, according to Halbertsma's view. Still it must not be denied that some cases confirm Halbertsma in regarding the defect of the septum, and the anomaly of the pulmonary artery, as co-effects of the original hindrance. The origin of the aorta from both ventricles, or even from the right alone, has often been observed in connection with a septum both deficient and displaced to the left. The ductus Botalli is found sometimes open and sometimes closed. According to Stölker's statistics, in sixty-nine cases, where this point was accurately examined, the ductus Botalli

was thirty-eight times closed and thirty-one times open. Its remaining open is easily explained by the over-supply of blood in the aorta, consequent on constriction of the pulmonalis, as the lungs must now continue to be supplied by this fœtal channel. In the cases where the duct was closed, the closure must have taken place at a stage in fœtal life when the lumen of the pulmonary artery was still large enough to transmit a current sufficient for the lungs. When the ductus Botalli is closed, or when this channel is no longer quite capable of transmitting enough blood after respiration has commenced, we see the arteriæ bronchiales, branches from the aorta, and sometimes also the arteriæ œsophageæ and coronariæ anteriores, dilate to form a collateral circulation, which supplies the lungs by anastomoses between these arteries and the pulmonalis. But whether the constriction be congenital or acquired, the right ventricle, auricle and auricular appendix are found almost unexceptionally dilated and hypertrophied, and the whole heart is more globular than usual, and lies transversely. The substance of the right ventricle may increase to such a degree, that it equals, and sometimes exceeds, the volume of the left. In the case which came under my observation, the wall of the left ventricle measured 1.5 cm. at the base, and that of the right 1.1 cm. The left ventricle is always relatively small, though absolutely it may be of the normal size. The condition of the pulmonary artery beyond the stricture is variable, but, as a general rule, we may say that it is narrow in the congenital, but normal, or even dilated, in the acquired form. In the majority of cases, the branches beyond the bifurcation are dilated. The lungs are generally anæmic, and frequently contain caseous products or tubercles. The physical signs of acquired stenosis of the pulmonary artery are: an extension of the area of dulness to the right, extending beyond the right border of the sternum. The dulness may also extend to the left, owing to the more horizontal position of the heart. In auscultating we hear a systolic blowing sound, which reaches its maximum intensity on the left border of the sternum in the second intercostal space, and spreads considerably to the left, but never to the right or into the large cervical vessels. The second sound in the pulmonary

artery is very weak, and may be quite inaudible sometimes. A systolic vibration may often be felt, though not invariably, in the situation of maximum intensity for the audible murmur. When at the same time the valves are insufficient, we hear a diastolic as well as the systolic murmur. The subjective condition of the patient is usually undisturbed as long as his activity is not specially taxed. But with active physical exercise, or the occurrence of any acute disease, whether of the heart itself, or of the respiratory passages, especially diffuse bronchitis, severe dyspnœa is produced, and more or less intense cyanosis follows; on the other hand, the dropsical swellings do not occur till a later stage. These latter, and stagnation in the glandular organs of the abdomen, do not set in till the compensation of the hypertrophied right ventricle has been destroyed by degeneration of the cardiac muscle or by other forms.

The physical signs in congenital stenosis of the pulmonary artery, as far as accurate descriptions go, and judging from three cases which have come under my own notice, seem to be the same as in the acquired disease. Only the development of hypertrophy of the right ventricle in early youth, while the thorax is still elastic, leaves a more marked impression on the chest, and the cardiac region is more arched. The cardiac impulse is weak and diffuse. The thinness of the child's chest, too, and its smaller extent, explain the fact that the intensity of the audible murmur and palpable vibration is far greater and further extended over the thorax in children than in adults. It is consequently extremely difficult to localize the murmur, for we can hear it over the whole cardiac region, and can even follow it into the cervical vessels. From some observations that have been made, it seems probable that stenosis of the pulmonary artery may occur without any abnormal murmur at all. To explain such cases as these, we must assume that the intensity of the circulation was not great enough to produce a murmur. In the majority of cases of children (according to Stölker, thirty-two out of fifty-seven, immediately after birth), cyanosis was quite noticeable from the very beginning, and especially during crying, not only in the face, but also on the hands and feet. But even where the cyanosis is not visible, or is absent owing to the

quiet life led by the patient, yet it is generally quite striking after physical exertion. However, there are exceptional cases, in which either the first years of life run on without any derangement, and cyanosis does not occur plainly till the patient has grown somewhat, or else the subjective symptoms of a cardiac disease do not appear till the twentieth or thirtieth year. In such cases the collateral circulation probably sufficed, in conjunction with the unclosed fœtal channels, to compensate the derangement till further development of the lungs demanded a larger supply of blood, or new endocarditic processes rendered the existing compensation insufficient. These latter may occur at any time, and so, too, relatively late. The absence or presence of cyanosis is always dependent upon the degree of stagnation in the veins and capillaries. When the compensation is sufficient, there may be no cyanosis at all, but it sets in at once when the compensation is defective; and when it is gradually deranged, cyanosis slowly develops. The low temperature which is objectively perceptible on the hands and feet of these patients, and is always remarked by them, is connected with stagnation and insufficient oxidation of the blood. Even when there are no other symptoms, the little patients long for warmth, and cannot endure a cold wind very well. Small children sometimes have paroxysms of asthmatic breathing. In slightly older cases the characteristic symptoms of caseous pneumonia have often been observed proceeding from the lungs, and especially after hæmoptysis and hectic fever. Sometimes children have crying fits, accompanied, too, by convulsions. In a case I had of a one-year-old child, these convulsions grew to actual epileptic attacks. The crying fits sometimes seem to result from painful paroxysms of coughing, but occur, too, independently of these. Children who exhibit these symptoms die very soon. We may also see signs of a deranged cerebral circulation, especially attacks of dizziness and fainting, in children who have outlived the first few years. Besides this, in most cases of congenital stenosis, though not in all, we have an effect produced on the general constitution. Bodily development is in many ways backward, the bones are soft, the muscles weak, the subcutaneous fat insignificant, and the whole appearance of the patient generally does not correspond to his age.

But cases occur, as one of a boy twelve years old, who lately came under my care, where the cyanosis, setting in with physical exertion, alone points to a heart disease, while otherwise the general constitution is quite strong.

When the physician has known the patient before he acquired the stenosis of the pulmonary orifice, and if the physical symptoms already described develop under his eyes, then the *diagnosis* of the disease is not difficult. Under such conditions, there are only two cases in which there is any great difficulty in recognizing the disease. It may be accompanied, as I have seen it, by emphysema, especially in the right lung, and thus the lung may cover the heart so much that we can no longer ascertain its hypertrophy by the extension of the dulness to the right. In a case of this kind, besides the audible systolic murmur, which one might be tempted to regard as accidental, special weight should be laid on the weakness, or absence, of the second sound in the pulmonary artery. This sound can indeed become weak from fatty degeneration of the once hypertrophied right ventricle, even without the presence of emphysema ; but in this case we have other signs of stagnation expressing the derangement of compensation, especially symptoms of dropsy, which are the very last to come in this acquired stenosis. A second difficulty lies in the combination of insufficience with stenosis. Under these conditions the diastolic murmur may be very loud, and either conceal the systolic, so that this can no longer be heard at all, or, if it is heard, it is more likely to be attributed to vibrations of the wall of the vessel itself than to stenosis. This latter confusion, however, has no practical importance. But of course no one would found a diagnosis on the presence alone of a systolic murmur in the neighborhood of the ostium of the pulmonary artery, since accidental murmurs, and others caused by compression, often occur here. When the murmur is accompanied by a palpable vibration, it gains greater importance. Confusion with stenosis of the aortic ostium is prevented by the extension of the murmur to the left, and by its not being propagated into the cervical vessels, and also by the perfectly normal condition of the pulse.

The diagnosis of congenital stenosis of the pulmonary artery is greatly strengthened if, in addition to finding the physical

signs, we ascertain from the history of the case that more or less cyanosis was observed at the birth of the patient, or was developed immediately afterwards. But the absence of cyanosis does not at all exclude the congenital nature of the disease. Still the conclusion, as to its being congenital or not, can never be more than a probability, especially where there is no cyanosis ; all the more so, as in some cases doubt may still exist after the post-mortem examination, even when the foramen ovale is found unclosed.

The *prognosis* is so far unfavorable that old age is never reached by any case of this stenosis, whether congenital or acquired. The majority of congenital cases end in an early death. According to Stölker's statistics, 42 out of 99 cases died before their tenth year, and only 15 per cent. survived the age of twenty. Tuberculosis and caseous processes in the lungs figure prominently among the causes of death in the cases which lived to the age of twenty. Stölker calculates this to occur in 14 per cent.; Lebert's estimate is still higher.

Combination of Various Valvular and Ostial Affections.

In describing the individual valvular and ostial affections, we explained how the anatomical changes were generally of such a nature that on one and the same valve insufficience and stenosis of the ostium went hand in hand. Insufficience of the valves can, indeed, occur alone, but, as a matter of fact, it is only isolated in the rarest instances ; while stenosis is always accompanied by insufficience, except in the worst developed cases, where, strictly speaking, the insufficience is so small, in comparison to the stenosis, that it is not taken into account. It is consequently superfluous to go again into the details of a combination of insufficience and stenosis, and it will be enough to recall the fact that the aortic valves differ from the auriculo-ventricular, inasmuch as both stenosis and insufficience occur in an isolated condition oftener on the former than on the latter. Further, we should not forget that on an auriculo-ventricular valve, especially on the mitral, one form may gradually overbalance the other during the course of the disease. For instance, a case of mitral insufficience may

be complicated by stenosis of the orifice, and finally this stenosis may take such possession of the foreground that the main physical symptoms belong more to it than to the insufficience.

The simultaneous combination of affections arising on different valves and orifices, is of special importance, both as regards the modifying influence it exerts on the secondary changes in the heart and the difficulties it adds to the diagnosis. Its occurrence is by no means rare (in about 10 per cent.), and it may arise at the very beginning of the disease, as when a number of valves together are attacked during acute rheumatism. But the more usual case is the gradual spread of the disease from one to another during the course of some already existing valvular or ostial affection. I do not coincide in the view, held by Gerhard and others, that the mechanical condition of tension produced by the affection of one valve tends to injure the other valves; for were this so, we should see anatomical changes in the tricuspid valve much oftener, apart from relative insufficience, when the mitral has been previously attacked. In cases where, from the beginning, the same harmful influences have not operated simultaneously on different valves, it is simpler to look for the cause of the anatomical extension of the disease in the contiguity of the parts, particularly as it occurs most frequently on the mitral and aortic valves, where the transition is rendered so easy by the aortic flap of the former, that we can only wonder this combination does not occur still oftener.

The modifying influence of a combination of valvular diseases is most plainly exerted on the heart itself by a simultaneous insufficience of the aortic valves and stenosis of the mitral orifice, or stenosis of the aortic ostium and insufficience of the mitral valve. In the first case, especially if the disease began on the aortic valves, part of the influence of the stenosis of the mitral orifice is exerted on the left side of the heart, the left ventricle being usually found hypertrophied instead of atrophied. But the degree of dilatation and hypertrophy cannot be so great as when the blood flows unhindered from the left auricle. On the other hand, the secondary dilatation and hypertrophy of the left ventricle may be very considerable in the second case, where there is at the same time stenosis of the aortic valves and insufficience

of the mitral, for under these conditions the two diseases tend to produce the same effect on this ventricle. The mechanical results of this combination are the most unfortunate, for both hinder the supply of the aortic system. Besides, though the defects of the aortic valves seldom produce any indirect effect on the right side of the heart, still, when combined with mitral deficiency, they may produce a highly beneficial change in the right ventricle. Stenosis of both orifices of the left side work in the same self-correcting manner when they arise at the same time, the results being concentric atrophy of the left ventricle and dilatation and great hypertrophy of the right side of the heart. My own experience, as regards the condition of the left ventricle in a case of insufficiency of the aortic and mitral valves combined, does not agree with that of Friedreich and Dusch. These authorities assert that the greatest excentric hypertrophy of the left ventricle is reached in this combination; whereas I have only observed very great dilatation, without hypertrophy, of the left ventricle, and on the contrary, the right ventricle was so very much dilated and hypertrophied that the apex of the heart even was formed by it. The degree of perfection and the time of development of each factor of the combination exert the main influence on the final result of the whole.

Of the simultaneous defects of the valves on both sides of the heart, the most frequently occurring combination is that of stenosis of the mitral orifice and relative insufficience of the tricuspid valve. Since the latter is evidently a result of the former, it is indeed perhaps unfair to call it a combination. The relative insufficiency, while it lasts, diminishes the tension in the pulmonary artery caused by the stenosis of the left venous ostium, and thus performs a certain amount of compensatory action.

Constriction of the ostium venosum dextrum occurring simultaneously with stenosis of the pulmonary artery, and insufficiency of its semi-lunar valves, is a combination of the rarest occurrence, there being only one case on record. The action of the latter affection modifies the change of volume of the right ventricle.

Wherever a combination of valvular diseases exists the physical symptoms are correspondingly manifold, and from the

signs peculiar to each factor we may construct a scheme of the whole. Thus it is self-evident that, if for instance a systolic blowing sound with maximum intensity over the apex be heard in insufficience of the mitral valve, and if in insufficiency of the aortic valves we perceive a diastolic murmur, with its maximum intensity over the ostium of the aorta and on the sternum, we may expect, when the two affections are combined, to hear both a systolic and a diastolic murmur, with their maximum intensity each in its respective place. In the same way, for every combination we can construct a series of auscultatory and percussional signs, which should theoretically be heard. But in reality, things are not so. The combinations are extremely difficult of recognition, far more so than theory would lead one to expect. The reason of this lies in the easy transmission of the murmurs from one ostium to another, thus throwing the whole difficulty of isolating the different affections on the discrimination of the timbre. But even this differentiation deserts us, when, as in a combination of aortic and mitral insufficience, we might be led to expect a pregnant distinction from the whirring character of the one and the blowing character of the other. We must consequently direct all our efforts to find the situation of each murmur's maximum intensity when many are present, and also to distinguish the timbre peculiar to each. If we can succeed in this, it is possible to make a diagnosis of several valvular affections together. This diagnosis is not alone scientifically interesting and indispensably necessary for the comprehension of the separate factors of the case, but it must also be brought well into account in forming the prognosis. For, as is plain from what has been already set forth, the effect of a second on an already existing valvular disease may, according to the nature of the second, be either very injurious, or at least temporarily favorable.

Treatment of Valvular Diseases.

It is useless to talk of prophylaxis for valvular disease in most cases, for the endocarditic as well as the atheromatous processes

frequently run such an imperceptibly slow course, that we seldom know of their existence till the circulation begins to be impaired by a disturbance in the function of the cardiac valves and diminution of the vascular elasticity. However, in as far as in youth and middle age rheumatic fever is the undoubted cause of endocarditis, and as for later years we may reckon, among the causes of the atheromatous process, both abuse of alcoholic drinks and immoderate smoking, the avoidance of all harmful influences which have a tendency towards rheumatism or atheroma may be considered at the same time of great prophylactic importance for valvular disease. Special care should be shown in the hygienic regulations made for individuals, in whose family heart disease has often been observed, since statistics do not allow us to quite throw aside the doctrine of hereditary predisposition to affections of the heart and vessels. Under such conditions a physician should be consulted before a young person chooses his profession. But it is only possible in a few cases to carry out this kind of prophylaxis, as it seldom happens that the physician has opportunity of becoming intimately acquainted for any length of time with the patient and his private affairs. We generally have to face the *fait accompli* of a fully-developed heart disease. In fact, medical advice is seldom taken till the signs of slight derangement of compensation first attract the attention of the patient himself. For, since nature more or less compensates any derangement of the central circulatory apparatus, by a parallel development of dilatation and hypertrophy in the corresponding ventricle, the patient himself remains unaware (at least under favorable conditions) of any symptoms of his disease. However, quite casual matters, such as overexertion in taking a very long wlak, or in climbing a mountain, the necessity of performing some arduous work, a slight cold with succeeding catarrh of the respiratory tract, psychical emotion, whether of joy or sorrow, any of these, by means of palpitation and dyspnœa thereby developed, may arouse the attention of the patient and physician at a stage when the compensation is still perfectly sufficient for all the circumstances of every-day life. The physician must pursue very different courses, according as he is called in at a stage where the compensation is sufficient for

ordinary daily life, or when his aid is not sought till signs have set in of severe derangement of the compensation. The first and most fundamental rule which holds good here and in all other chronic diseases, though one that is often neglected, in spite of its self-evident simplicity is, not to make the case worse by the use of drugs. As far as the patient's social position and calling will allow, general hygienic measures should be taken to maintain as long as possible the natural correction exerted by the development of hypertrophy. The indication we have is, *to advance the development of compensation when already in existence, to maintain it as long as possible, and to moderate over-compensation.* Accordingly, to promote and preserve the compensation, the diet of the patient should be nourishing but not stimulating. Animal and farinaceous food may be taken according to individual taste. Those vegetables should be avoided which develop much gas, and thus interfere with respiration. Coffee and tea should only be taken when extremely weak, the form known as *family coffee;* spirituous liquors, especially punch, grog, etc., and much smoking, should be equally disallowed. On the other hand, a light bitter beer and a little good wine are to be recommended as aiding the digestion. But the best means for promoting a good digestion are fresh air and exercise. This latter, however, must be taken cautiously, and all severe exertion, such as mountain climbing, etc., should be strictly avoided; but it is just as injurious to condemn a patient with heart disease to perfect quiet. Where means allow, we should try to procure a few hours daily in the open air, during the winter months as well, and people who can afford it should be sent to some sanitarium like Pau, Pisa, or Mentone. On account of the important part the vessels of the skin play in moderating and regulating the circulation in the internal organs, we should not undervalue the importance of a regular culture of the skin. Lukewarm, plain, or saline baths, with a cold rub-down afterwards, act favorably, by attracting the blood into the peripheral vessels, and by producing a general vigor of the functions through the influence they exert on the sensory cutaneous nerves. Some time ago great alarm prevailed about over-excitement of vascular activity by too hot baths in heart disease;

but the extremely favorable results obtained by Beneke in Nauheim in the use of saline baths containing carbonic acid, at temperatures from 88° to 93° F., obtained even in cases of severe derangement of compensation and recorded most accurately, have completely set aside this alarm. Even at the stage where compensation is still almost sufficient, we may modify and combat the unusual irritability of the heart, displayed in the palpitations which occur with the least exertion or even while the patient is at rest. Psychical as well as physical stimulants must be as much as possible removed. Olympian calm, when its attainment is possible, should be warmly recommended as the mental dietary of a heart-disease patient. The patient's attention should never be attracted to his disease either by those about him or by the physician. There are a number of drugs specially suited to allay the pathological irritability of the cardiac muscle, which may be regarded as the first sign of exhaustion in the struggle against the mechanical difficulties. At the head of these stands the application of cold. I have repeatedly seen good results obtained from the old method of allowing patients, troubled with palpitations, but without any symptoms of bad derangement of the compensation, to carry a few hours a day a metal or gutta-percha vessel (adapted to the cardiac region and with the concave surface against the ribs), filled with cold water or ice. On the other hand, I cannot speak for the use of lasting irritants on the skin (blisters, issues, etc.), so much recommended after the English practice; indeed I consider them injurious. Of the narcotic agents, preparations of hydrocyanic acid, such as cherry-laurel water or bitter almond-water, and tincture of belladonna, act favorably in combination with valerian. But the sovereign remedy, *which may be used in every stage of valvular disease with merely variations in the doses*, is digitalis. Experience teaches us that a combination of digitalis and iron preparations is specially serviceable in combating abnormal irritation of the heart, and that there need be no apprehension whatever of stimulating the heart's action by moderate use of the iron. I usually prescribe a mixture of two parts each of the tinctures of digitalis and valerian, and three parts of ethereal tincture of acetate

of iron.[1] Of this I give twenty-five drops three times daily. If no special counter-indications are exhibited, the patient takes this for fourteen days regularly; but after this it should be given up, except in attacks of palpitations, and then it may be replaced by steel drops alone. For the same object, and especially for longer continuous use, Botkin recommends nitrate of silver alone or combined with digitalis. He administers it for five or six weeks in pills of one-sixtieth of a grain three times a day, and, as the activity of the heart increases, he raises the dose every three or four days by one pill of the given composition, till he reaches one-sixth of a grain per diem; then he diminishes the dose rapidly, but does not leave off the medicine at once. If the irritability be very great, he adds one grain of digitalis to the dose of nitrate of silver. I have had no experience myself of this treatment, but did not wish to pass over the advice of such a tried physician without mention. But as long as it is at all possible, we should hesitate to use any drugs whatsoever, except tonics. Further, we should not forget in using digitalis that, on the one hand, its long-continued use acts injuriously on the digestive organs, and that, on the other hand, the medicine is chiefly indicated when it can *make the action of the heart more regular by retarding it*, and when it can *raise the decreasing tension in the aortic system.*

Slight stagnation, which occurs as the first symptom of a slower circulation in the glandular organs of the abdomen, the trifling swelling of the liver, and its accompanying gastric catarrh, all these require to be counteracted by the use of bitters and gentle aperients, in the shape of saline mineral waters. Favorable results are obtained with rhubarb, in its different preparations, with quassia, gentian, and the waters of Homburg and Kissingen, and especially with the hot springs of Soden. They remove this complaint temporarily, so that the strength of the patient improves with renewed appetite. When dropsy occurs, the case is far more difficult to treat. The first warning we get of this, is a swelling around the ankles, which merely makes the patient complain that his boots squeeze him in the evening and feel too

[1] Solution of acetate of iron, 9 parts; alcohol, 2 parts; acetic ether, 1 part.—*German. Ph.*

tight; but this generally disappears with just a little rest. Rest, too, and a horizontal position, are efficacious in more serious cases as well, for the swelling does not increase if they are observed; but to banish it completely, we must use diuretics and stimulants for the intestinal secretion. At the head of these stands acetate of potash, when the pulse is still vigorous and the cardiac action regular. If the digestion is good, I generally combine it with iron (acetate of potash, two drachms; ethereal tincture of acetate of iron,[1] forty-five minims; raspberry syrup, five drachms; water, to make six ounces), which has the further advantage of increasing the intestinal activity in addition to its diuretic power. Squill may be reckoned among the tried diuretics, and may be administered as vinegar of squill, with carbonate of potash added to saturation, thus combining the action of both drugs. This medicine, however, cannot be employed if there is any intestinal catarrh, or if it produces any tendency to vomiting, as sometimes happens even in cases quite free from catarrh. In such cases we can recommend the vegetable diuretics, such as lovage root, rest-harrow root, and juniper berries. I have never seen any great success attained by the use of the diaphoretic methods through internal medicines in dropsy from stagnation. But when the pulse is low, the action of the heart irregular, and the urine scanty, though containing no foreign elements—in a word, in the condition which Beau has described as "asystole," and which now goes under the name of the "stage of disturbed compensation," the diuretic value of digitalis, in small doses, particularly when combined with quinine, is inestimable. At this stage, as Traube's brilliant and thorough researches have taught, there is no better tonic for the cardiac muscle than digitalis, in small doses. After administering nine grains of digitalis, in infusion with twelve of muriate of quinine, I have seen considerable dropsy, with a daily secretion of from five to ten fluid ounces of urine, disappear in a short time, while the urine rose to sixty-eight fluid ounces and more. While the intestinal canal remains intact, we may try to combat the dropsy by the use of drastics, and for this purpose special credit is given to gamboge and

[1] See foot-note on preceding page.

scammony. It is, however, better to avoid these methods, with the object of removing every possible abnormal irritation from organs which of themselves are extremely liable to secondary affections. When the œdema steadily increases, in spite of all internal remedies, the only remaining course to get rid of the fluid is scarification. A number of pretty deep incisions a few lines long allow the serum to flow off freely; then we have only to avoid all erysipelatous inflammation of the skin by keeping the wounds perfectly clean, and this is best effected by bandages steeped in chlorine and chamomile waters.

I have seen as great, though not as lasting, success in promotion of cardiac and renal activity, from the use of the *pneumatic* method, lately added to the medicinal repertoire. The observations which I have made on the results of Waldenburg's apparatus fully confirm the inventor's expectations, founded on theoretical grounds. The compressed air, as it is inspired by means of this apparatus, raises the tension of the aortic system. Though Drosdorff and Botschetchkaroff [1] found in their physiological experiments on dogs that the arterial pressure fell as soon as the animal inspired compressed air, I can, on the contrary, adduce cases of patients whose pulse could scarcely be felt, and most certainly not counted, but after inspiration of compressed air, it grew so full and strong that it could be quite easily counted; and, further, the diuresis, which had been extremely scanty, rose considerably, though no disturbing influence, in the way of drugs or change of diet, had been employed. On this latter symptom I lay peculiar stress, as it is quite independent of any subjective influence. Haenisch's [2] researches, too, clearly show that, under the influence of compressed air, the systolic elevation of the ascending limb of the sphygmographic tracing rises higher, and the elevation of recoil on the descending limb is less pronounced. He further shows that the symptoms of a deranged compensation were met by this agent, and corrected. Though I consider myself justified in placing the influence of compressed air parallel to digitalis in small doses for heart disease, yet it must be admitted, that, as

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[1] Med. Centralblatt, 1875, No. 5.

[2] Deutsches Archiv. Bd. XIV. Heft 5 and 6.

far as a lasting effect is concerned, my own experience convinces me that digitalis, when its continuous use is well borne, is undoubtedly superior, and this applies also to its regulatory and retarding action on the pulse. On the other hand, the pneumatic method must be considered a valuable substitute for digitalis in all cases where this latter cannot be administered on account of gastric complications and generally where it does not agree with the patient. Respiration of compressed air is indicated, as is digitalis, in small doses, in *mitral affections*, especially stenosis of the *ostium venosum sinistrum*, and in stenosis of the aortic ostium, when the compensation becomes deranged. In insufficience of the aortic valve, digitalis may be considered as a means of moderating over-compensation, and must then be given in larger doses. I am not able to judge the effect of breathing rarefied air, from personal experience. But on theoretical grounds, it would be indicated in those cases of heart disease where we wish to raise the tension in the lesser circulation.

If we succeed (whether by the pneumatic method, or by the use of digitalis) in regulating the pressure relations in the arterial and venous systems, we have overcome in the best possible way the worst subjective troubles, especially the palpitations, the feeling of terror, and the dyspnœa. But all these afflictions are, in a whole series of cases, more directly dependent on nervous influences, and often occur in paroxysms, even though the general conditions of the circulation remain unaltered. Pain, too, frequently accompanies these. In such cases a very favorable effect is produced by the subcutaneous injection of morphia. I have seen patients who could not lie in bed, and who were consequently much reduced by sleeplessness, living quite comfortably for months with this treatment. But, on the contrary, the greatest caution should be taken in administering chloral to patients with heart disease. It is impossible to form any estimate previously as to the individual powers of sustaining the depressing influence of chloral on the excito-motor ganglion system of the heart. The evidence as yet has not gone to support Liebreich's opinion, that croton-chloral produces better results in heart disease than chloral itself.

The secondary affections of the organs and functional disturb-

ances must be treated according to the general rules for these conditions, whether the trouble be in the brain (apoplexy, embolism), in the bronchi and lungs (bronchitis, hæmoptysis, pneumonia, hemorrhagic infarction, threatening pulmonary œdema), in the pleura, or in the kidneys. These are only modified by the primary cause, the valvular disease, in so far that we must always take into consideration that most are produced by the weak condition of the heart. We must specially emphasize the necessity of prompt medical assistance in the treatment of fainting and dizzy fits, so often exhibited by patients with stenosis of the aortic ostium. Such cases arouse alike the anxiety of the physician and that of the patient's friends. The prompt application of volatile stimulants (ether, Hoffmann's anodyne, wine) is urgently necessary, and the head should be placed low, while the body lies horizontal. It is not improbable that nitrite of amyl, administered cautiously (five drops for inhalation), would produce a favorable result. On the other hand, the venous stases of the brain, which exercise a certain influence on the mental action, in defects of the auriculo-ventricular valves, or the active congestion which occurs in insufficience of the aortic valves, must be combated by fontanelles, sinapisms, or dry-cupping on the neck, or else by small local bleedings over the mastoid process. In treating hæmoptysis, arising from an affection of the left venous ostium, we should not forget that when the bleeding is not sufficient to compel active measures, it relieves the lesser circulation of a superfluous load, and thus produces a beneficial effect on the general condition of the patient and his dyspnœa.

CHANGES IN THE POSITION OF THE HEART

AND

DISEASES OF THE HEART SUBSTANCE.

SCHROETTER.

CHANGES IN THE POSITION OF THE HEART.

The manuals and handbooks of *Laënnec*, *Andral*, *Cruveilhier*, *Rokitansky*, *Förster*, *Bock*, *Bamberger*, *Lebert*, *Trousseau*, *Duchek*, *Klebs*, *Dusch*, *Jaccoud* and others.—*Gilbert Breschet*, Mém. sur l'ectopie du cœur. Paris, 1826.—*Karl Ewald Hasse*, Anatom. Beschreibung der Krankheiten der Circulations- und Respirationsorgane. Leipzig, 1841.—*J. Engel*, Darstellung der Leichenuntersuchungen und deren Bedeutung. Wien, 1854.—*William Stokes*, The diseases of the heart and aorta.—*Da Costa*, Effect of respiration on the size of the heart. Americ. Journ. of Medical Science, Oct. 1859.—*Bamberger*, Ueber die Lage des Herzens beim Lungenemphysem. Würzburger med. Zeitschr. I., 419, 1860.—*W. Kobelt*, Ueber Form und Dimension der Herzdämpfung. Inaugural-Dissertat. Giessen, 1863.—*Engel*, Ueber einige pathol. anatom. Verhältnisse des Herzens. Wiener med. Wochenschrift, 44, 45, 46, 1863.—*W. C. Maclean*, Report of a case of lateral transportation of the heart and liver in a soldier. Lancet, 8. Aug. 1863. —*Gerhardt*, Ueber einige Formen der Herzdämpfung. Prag. Vierteljahrschr. IV. S. 113, 1864.—*E. Rindfleisch*, Ueber eine eigenthümliche Drehung des Flüssigkeitsstromes im elastischen Rohre, über die Lageveränderung des Herzens und den Situs viscerum perversus. Hermann's med. Centralblatt, No. 21, S. 323, 324, 1864.—*Greenhow*, Displacement of the heart to the right side, consequent upon disease in the right lung. Transact. Path. Soc. XIX. p. 159, 1869.—*Schrötter*, Beitrag zur Kenntniss der Lageveränderungen des Herzens. Oesterr. med. Jahrb. XX. S. 189, 1870.—*E. Rindfleisch*, Lehrbuch der patholog. Gewebelehre. 3. Auflage. Leipzig. Seite 198, 1873.

The changes in the position of the heart form no independent disease, but are only the indication of pathological alterations, less frequently in the heart itself than in adjacent organs. But since the determination of the position of the heart is of great importance in coming to accurate conclusions as to the condition, not only of the thoracic viscera, but frequently of the entire system, it seems quite proper to devote an independent chapter to this subject.

If we reject congenital anomalies, such as transverse position

of the heart, abnormal origin of vessels, etc., the changes in position may be classified as follows :

1. Turning of the organ on its axes.
2. Change in position, according to the laws of gravitation.
3. Pressure of the heart to right or left—upward or downward.
4. Traction on the heart producing same effects.

Before beginning, it seems best to describe the normal position of the heart.

Normal Position of the Heart.

As the heart rests on the anterior portion of the central tendon of the diaphragm, the position of the organ is such that its long axis forms an angle of 60° with that of the body, and the portion which is lowest and furthest removed to the left is the apex of the organ. Inasmuch as the apex is the part whose location can best be established, it is of great importance in determining the position of the heart ; if we succeed in exactly locating its beat, we can give the position of the whole heart with considerable accuracy.

In by far the greatest number of healthy individuals, the apex is found in the fifth intercostal space, about a thumb's breadth inside the left nipple. Not unfrequently it lies in the fourth intercostal space, and then usually close to the nipple, occasionally in the sixth ; but so rarely does this occur with the healthy organ, that under these circumstances we are justified in suspecting pathological changes ; but we must remember that in little children the apex is very frequently found in the fourth intercostal space, and in old people in the sixth. To this extent age has an influence on the position of the heart, namely, in so far as it affects the position of the diaphragm. In childhood this depends, doubtless, on the more active contractile power of the lung. Beside these, we must remember that in the gradual development of the system the aorta grows longer, and so the heart comes to lie lower in the thorax. Towards the right, the heart extends about three fingers' breadths beyond the median line, and the parts found here are the right auricle and a small por-

tion of the right ventricle, in front, and, deeper in, the right half of the left auricle as well. The anterior surface is largely covered in by the edges of the lungs, so that only a small portion of the right ventricle, which generally makes up the greater part of the anterior surface, lies in contact with the chest-wall.

As regards inspiration, we must remember two points:

First.—The heart descends with the descending diaphragm, and frequently to the extent of a whole intercostal space.

Secondly.—The lung presses further and further forward over the heart, and in deepest inspirations it expands until arrested by the reflection of the pleura costalis on to the lung, on the one hand, or, on the other, by the connection of the fibrous pericardium with the chest-wall, by means of the so called ligamenta sterno-cardiaca superius and inferius.

In the most extreme expansion of the lung there remains uncovered only a small triangular portion of the anterior surface of the heart, which lies at the lower end of the sternum, extending from the median line towards the left.

For aid in physical diagnosis we must consider, that if we make a longitudinal section (as Luschka has done) through the median line of a frozen body, we find in the right half the right auricle, the right half of the left auricle, a small portion of the right ventricle, and only a very small portion of the septum ventriculorum; while in the left half of the body we find the left half of the left auricle, the whole left ventricle, by far the greatest portion of the right ventricle, with the greater part of the septum ventriculorum. As regards the upper limit of the heart, the highest portion is the top of the left auricle, which reaches as far as a horizontal line, which, if produced forward, touches the lower border of the second costal cartilage.

Turning of the Heart on its Axes.

Turning of the heart on its long axis generally occurs in such a manner that the right auricle and ventricle come to look more directly forwards, and the left cavities backwards. Such changes are quite rare, especially when existing alone, and usually are occasioned by simultaneous dislocations of the heart, arising

from antecedent pressure, or from traction upon the organ by the contraction of adhesions in the pleura, or more rarely by contracting bands in the lung itself.

Bamberger relates a case of pneumo-thorax on the left side, in which the heart was not only pushed over to the right, as is usual, but had so revolved on its long axis that the greater portion of the left ventricle looked forward ; this revolution was so considerable that torsion of both the great vessels could be easily recognized. It is hard to picture to one's self how this came to pass, inasmuch as there were no contracting bands found here, and it may be that, with an original tendency of the heart in that direction, a weak spot in its suspension gave occasion to the displacement and torsion.

Revolutions on an antero-posterior horizontal axis occur especially in cases of aneurism of the ascending aorta, when extension of the aneurism downwards presses the base of the heart down, and the apex rises. This same axis-revolution occurs in other cases of pressure, which will be mentioned hereafter; but, in comparison with the changes in position which the heart undergoes at the same time, it is of minor importance.

Changes in the Heart's Position dependent upon Gravitation.

In spite of opposing statements, it is now settled beyond all doubt that, in changes of position of the body, the heart follows the laws of gravitation.

The displacement towards the left, where the impulse is distinctly to be felt, is by far the most noticeable ; that towards the right is only slight. Doubtless, when the body is bent forwards, the heart lies in contact with the anterior chest-wall over a greater extent, as is learned by careful percussion. Adhesions which the pericardium alone or the heart and pericardium have formed with the neighboring parts will limit the mobility of the heart as decidedly as stronger indurations and immobility in adjoining organs which cannot yield to the pressure of the heart. A considerably hypertrophied and dilated heart, as well as one suffering from new-growths, will by its weight press the diaphragm downwards ; this displacement is, however, by no means

so great as occurs occasionally from the same cause in pericardial effusion; if this is so great as to be found, not only at the base, but also on both sides of the heart, the depression of the arch of the diaphragm will be still more marked.

What position does the heart take in vesicular emphysema of the lungs? It follows the law of gravitation here, for, as it rests upon the diaphragm, when this is depressed, in consequence of extreme expansion of the lung, the heart descends as well; the question is, what position does it assume in this case.

Upon this point there are two opposing views. The older, maintained especially by Skoda, holds that the heart presses downwards with its apex, and the organ, as a whole, is brought nearer to the median line. Bamberger, on the other hand, claimed that, as the base was pressed down, the heart assumed a more horizontal position. The grounds upon which Skoda based his view were: the recognition of the impulse of the heart, not at its proper site, but nearer the median line, even in the scrobiculum cordis (often with such distinctness that you can feel a pointed object thrusting itself between the finger-tips applied to the chest); also, in part, the harmony of this view with the results of autopsies; and, finally, the probability that, in the depression of the heart, the apex, which is the most movable portion of the organ, would be affected rather than the base, which is the least movable part. Bamberger, on the other hand, held that that which we feel beating in the scrobiculum cordis is not the apex, but a portion of the wall of the right ventricle, inasmuch as, by seeking carefully, we can find the apex beat in its normal place, and, moreover, in autopsies the heart has been found lying horizontally. Concerning this last point, Prof. Klob has given us some valuable information, by showing that we were not authorized in thinking that the heart necessarily had the same position in life as in the cadaver. The rigor mortis, the distention of the intestines with gas, and other causes, must naturally alter the position of the heart after death, and it is only by fixing the heart *in situ* with needles passed through the chest-wall, as soon as possible after death, that we can be sure of its position during life.

I have followed up this subject with some care, and have

arrived at the following conclusion. In the more advanced stages of emphysema the apex lies considerably further to the right than normal, but certainly that which we feel giving the impulse in the scrobiculum cordis is not always the apex, as this frequently lies directly behind the point of junction of the left costal cartilage with the sternum, but is actually a portion of the wall of the right ventricle.

In the recumbent position we cannot, after the most careful search, find the apex beat at its normal place; but if we cause the patient to turn towards the left, we can very frequently find the apex between the left edge of the sternum and its normal site, more or less near to the latter, and in this way we learn that the heart, displaced by disease towards the median line, is now by a change in position caused to fall towards its normal place.

Now, if we observe an extension of the heart dulness beyond the right edge of the sternum, while it is but slight towards the left, this is occasioned by a change in the position of the heart, and not by hypertrophy of the right ventricle, for hypertrophy of this ventricle to any great extent is by no means the rule in all cases of emphysema of the lungs; nor is it always found in post-mortem examination; but in this case the epigastric pulsation may have been very forcible, yet, on the other hand, you will find that in many cases of advanced hypertrophy of the right ventricle no epigastric pulsation is observed. Accordingly, though I accept it as a rule that the apex presses downwards towards the median line, I cannot deny that several cases of extensive emphysema have been observed by me where the dulness over the heart was limited in extent (by encroachment of the lung), but the apex could be distinctly made out by percussion at its normal place, or even further to the left. These cases would seem to support Bamberger's view, but they are only exceptions, and seem to depend upon some peculiarity of the individual; for if originally, in a given individual, the heart has a more horizontal position than normal, where the diaphragm is depressed, it cannot press downwards with its apex, but with its base, and thus the heart comes to hold a more horizontal position still. In this manner it seems to me we must explain the relation of the parts in emphysema of the lungs.

Change of Position by Pressure.

The heart is exposed to the greatest pressure in cases of pleuritic exudation and pneumo-thorax, especially of the left side. In cases of displacement towards the left, as the heart rests upon the arch of the diaphragm (and this latter in these cases is always pressed downwards), the apex beat may be seen in the sixth or even the seventh intercostal space in the axillary line. In cases of pressure towards the right we may easily make a mistake as to the cause of the change in position. We must remember that the heart will be pressed over to the right in the same relative position as in health, and accordingly cannot assume a position such as we should find in congenital transverse position of the heart. Hence, the apex comes to lie behind the sternum, the xiphoid process, or that cartilaginous plate formed by the union of the costal cartilages of some of the lower ribs; its impulse is, therefore, not felt, and a portion of the right ventricle, lying against the thoracic wall, simulates, by its pulsation, the apex beat.

This was most beautifully marked in the person of a student of medicine with intense pneumo-thorax of the left side. Here the impulse of the right ventricle in the fourth intercostal space, about two inches beyond the right edge of the sternum, was so distinct that you would be led to doubt the truth of the above rule; but in this case the usual relative positions of the organs existed; for, after following the example of Hope and Gendrin, I had fixed the heart *in situ* by piercing it with needles from without immediately after death, I was able to show at the autopsy how the heart lay just as I have stated above, with its base furthest over to the right, and the apex behind the right edge of the sternum.

In pleuritic effusion on the right side we occasionally find a peculiar form of displacement of the heart. If the quantity of fluid is very great, the right lobe of the liver is pressed downwards and the left tilted up, and the heart by this means assumes a horizontal position, or may even be pressed upwards the breadth of the two intercostal spaces.

Pneumonia of itself cannot cause pressure on the heart, for the shrinkage of healthy portions of the lung counterbalances the swelling of the parts affected by the inflammatory process. Occasionally, however, the heart assumes an abnormal position

in consequence of a change in the position of the diaphragm; for if the lung of one side is hepatized throughout, its weight is so increased thereby that it presses upon the diaphragm, which descends, and with it the heart also.

I was able distinctly to recognize this change in the case of a young man of 19 years of age with pneumonia on the right side, where the hepatization was so intense that it was mistaken for a pleuritic exudation,—and to confirm my diagnosis by an autopsy. Smith reports a similar case in the "Dublin Journal of Medical Sciences." Vol. XIX. p. 122.

In a pneumonia of this kind upon the left side the changes in position of the heart must be more considerable, but I do not recall any such case.

Diseases of the mediastinum in advanced stages influence the position of the heart—such are abscesses, neoplasms, and especially carcinoma; but these are comparatively rare. Aneurisms of the great vessels, and particularly of the aorta, are of much more importance. If an aneurism has developed in the course of the ascending aorta with dilatation of the vessel and bulging towards the right, generally the heart lies in a horizontal position, and, in advanced stages, suffers pressure downwards and towards the left. Skoda has also seen cases when the heart had partially revolved on its antero-posterior horizontal axis, where the base was pressed downwards and the apex upwards.

When the aneurism develops with bulging towards the left, or in the concave portion of the arch of the aorta, the heart will be pushed downwards and at the same time more towards the right. The same is true in cases of aneurism of the pulmonary artery.

Curvatures of the spine, when considerable, likewise occasion alterations in the position of the heart. This is particularly true in kypho-scoliosis.

From the influence which the position of the diaphragm exercises upon that of the heart, it naturally follows that certain *abdominal diseases*, with the change they occasion in the diaphragm, affect the heart's position as well. General enlargement of the abdominal cavity, as occurs in ascites, will not alter the position of the heart so long as the abdominal walls yield to the pressure; but when this limit is passed, the diaphragm will be impeded in its descent, will be pressed upwards, and with it the

heart will be pushed upwards and towards the left; often in such cases the apex beat may be found in the third intercostal space. Abdominal tumors will only cause pressure on the diaphragm, and thus affect the heart, when they are so firmly attached that they can only increase in an upward direction.

Diaphragmatic hernia is usually congenital, and, in consequence of the non-viability of the children, does not come particularly under our observation. Occasional instances, however, may be found in the literature of the subject:

Weiland in Bouillaud.—In a case of diaphragmatic hernia, the left half of the thorax was found filled with coils of intestine as high as the second rib, and the heart was thereby pressed over towards the median line; the child lived seven years.

Skoda recalls an autopsy on a man who died at the age of forty of tuberculosis, where he found the heart pressed over to the right by the stomach and portions of the transverse colon which lay in the left thoracic cavity.

In "Cruveilhier's Pathological Anatomy" we find the case of a woman of seventy-five years of age in whom the heart was found lying entirely in the right side, being pressed over by a diaphragmatic hernia.

Sennert (Practica P. II., P. 2, chap. 15, p. 703) relates the case of a student who died suddenly eight months after receiving a dagger wound in the breast, which had entirely healed, and it was found on examination that the diaphragm had been perforated and the stomach lay in the left thoracic cavity, pushing the heart over towards the right side.

Changes in Position occasioned by Traction.

The changes in position which the heart undergoes, in consequence of traction by neighboring organs, are very interesting. As regards the lung, we must remember that in consequence of its innate power of contraction, and continuous attempts to accomplish it, in all cases where one-half of the thorax is filled with pathological products, the heart is drawn over to the healthy side. This holds especially in pleuritic exudations, partial emphysema, and pneumo-thorax; but in these diseases only to a moderate extent. It is different, however, when, as a result of contracting induration, there is a diminution in the size of the lung. In these cases the change of position in the heart is often very great.

I have had under observation for the last seven years a young girl, now eighteen years of age, in whom, as a result of chronic pneumonia, following measles, with

subsequent contraction of the lung and bronchiectasis, the heart is drawn so far over to the left and upward, that the apex beat is found in the axillary line at the third intercostal space. Several years since I described the position of the thoracic viscera in a man in whom, doubtless from the same cause, the heart was found in the right axillary region. In this case, if we wish to explain it thoroughly, we must assume that behind the contraction of lung as a cause there lay a congenital *dextrocardia.*

A contracting pleuritic exudation occasions the same changes in the position of the heart.

Tumors of the abdominal cavity, if they have reached a considerable weight, and are attached to the diaphragm, may pull it downwards, and with it the heart. It is particularly tumors of the liver, and more rarely of the spleen, which are able thus to overcome the contractile power of the lung.

Diminution in the size of the liver and spleen, and indeed of the abdominal viscera, generally occasions no change in the position of the diaphragm and the heart. for in these cases the abdominal walls alone sink in.

Pathology.

Course of the Disease.

The change in the position of the heart as such, as a rule, occasions no functional disturbance. The more extreme displacements, however, may, from interfering with the normal contractile power of the heart, or insufficient filling of its cavities, give rise to a tendency to stasis and its results. This will depend chiefly upon the condition of the heart as regards its nutrition and functional power. So likewise the circulation will suffer when the great vessels have been twisted or bent. In such cases murmurs may be developed over the great vessels which simulate lesions of the valves.

In a case of very extensive pleuritic exudation, in which the heart was pressed very far over to the left and downwards, in listening over the heart, I heard a loud systolic murmur. The autopsy revealed no alteration whatever in the valves, and I believe the murmur was occasioned only by the stretching of the aorta.

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[1] Jahrbuch der k. k. Gesellschaft der Aertze, in Wien. V. u. VI. Heft. 1870.

Forster states that interference with the circulation will give rise to hypertrophy of the heart. Frequently special nerve symptoms are associated with it.

In a patient in whom the heart had been pushed over into the right thoracic cavity, as the result of a wound, for years after, the heart still remaining dislocated, any excess in eating would bring on vomiting, pain, and a sense of tension or tearing in the right breast. In this case, however, we must remember that the pleura had been wounded, and doubtless the ramifications of the vagus came to lie in abnormal positions. Besides this, there was a very singular disorder of sensation. Application of cold in the region of the right breast would occasion attacks of suffocation; dipping the right arm in cold water, besides giving rise to peculiar feelings in the right breast, would cause cramp-like contractions of the right arm, drawing it over towards the breast.

Palpitation of the heart, which is frequently observed in connection with curvature of the spine, depends not only upon change in the position of the heart, but also upon simultaneous interference with the circulation in other ways.

Diagnosis.

If we can feel the impulse of the heart distinctly, the diagnosis of a dislocation will present no particular difficulty, and only at the first glance could we mistake it for an aneurism. The next point is to find the cause of the dislocation. This also, as a rule, will not be hard to discover, except when the signs are obscure and apparently contradictory, as, for instance, in the case mentioned above of diaphragmatic hernia observed by Stokes, in double pneumo-thorax, and others. If the heart's impulse is not distinctly to be felt, then the case is quite different. Here we must try if changing the patient's position to right or left may not make it perceptible. Occasionally we may find it by putting the patient in a stooping position; let him stand and bend forward, resting his hands on the edge of a bed or a chair. If these means do not accomplish the purpose, then we must fall back upon the fact that we cannot find it at its normal place, and to that add the results of percussion and auscultation.

In regard to percussion, the place and shape of the dulness over the heart are different from that which we find under the

head of hypertrophy, and its boundaries can only be made out when the heart is surrounded by air-containing organs. There occur occasionally very peculiar combinations; for instance, in a case of double pneumo-thorax I could make out absolutely no heart dulness anywhere. In the case of the young girl mentioned above, with dislocation towards the left, over the normal heart region, from the left sternal border over to the mammillary line, the percussion note is perfectly clear, and it is only at the latter line that there begins an irregular dulness, which extends over to the axillary line. Resting upon the evidence of auscultation, we must believe that the apex lies where we hear the heart sounds with greatest distinctness, and starting from here as one end of the long axis, we locate the base by the accentuation of the second sound in the pulmonary artery. It is clear enough, from what has been said above, that there are often many difficulties to overcome, even with this assistance.

In complicated cases we shall have to call in every aid, such as the history of the case, etc., to assist us in forming an accurate diagnosis.

Prognosis and Treatment.

Both these will largely depend on the underlying cause of the displacement, inasmuch as the heart itself rarely suffers. Some heart symptoms will, however, be considered further on, in accordance with principles to be there laid down.

DISEASES OF THE HEART SUBSTANCE.

Hypertrophy and Dilatation of the Heart.

In addition to the recognized works on diseases of the heart, consult: *Clendinning*, London Med. Gaz. 1838.—*Bizot*, Recherches sur le cœur et le système artériel chez l'homme. (Mémoires de la soc. med. d'obs. de Paris, 1838).—*Vernois*, Dimensions du cœur chez l'enfant nouveau-né. Paris, 1840.—*Peacock*, Monthly Journ. Sept. Oct. Nov. 1854.—*Forget*, Herzkrankheiten, übersetzt von Wolf. Giessen, 1855.—*Traube*, Ueber d. Zusammenh. zwischen Herz- und Nierenkrankheiten. Berlin, 1856.—*Bamberger*, Ueber die Beziehungen zwischen Morb. Brightii u. Herzkrankheiten. Virch. Arch. Bd. XI. 1857.—*Rosenstein*, Beitrag zum Zusammenhange zwischen Herz- u. Nierenkrankheiten. Virch. Arch. Bd. XII. 1857.—*C. Gerhardt*, Untersuchungen über die Herzdämpfung u. s. w. Archiv f. phys. Heilkunde, 1858.—*van der Byl*, Med. Times and Gazette. May, 1858.—*Filaudeau*, Des causes de l'hypertr. ducœur. Thèse, Paris, 1858.—*Larcher*, Arch. génér. de Méd. Mars, 1859.—*Traube*, Fall von Nierenschrumpfung mit Hypertr. d. l. Ventr. Deutsche Klinik, No. 49, 1859.—*Gerhardt*, Der Stand des Tübingen, 1860.—*Wilh. Baur*, Ueber reine Hypertr. des Herzens ohne Klappen-Diaphragma. fehler. Diss. Inaug. Giessen, 1860.—*Liebermeister*, Deutsche Klinik, 1860.—*Campana*, Considerations nouvelles sur l'origine de l'hypertr. et de la dilatation du cœur. Gaz. des hôpit., No. 61.—*Duchek*, Ueber Hypertr. des Herzens. Med. Jahrb. 1, 1861.—*Geigel*, Ergebnisse aus 84 Sectionen. Würzburg. med. Zeitschr. II. 4. 1861.—*Valentin*, De l'acetate de plomb dans les hypertr. commençantes du cœur. L'union méd. No. 110, 1861.—*Albert Eulenburg*, Ueber den Einfluss der Herzhypertr. und Erkr. der Nierenarterien auf das Zustandekommen von Hæmorrhagia cerebr. Virch. Arch. Bd. XXIV. 329, 1862.—*Erichsen*, Zusammenhang von Herz- und Nierenkrankheiten. Petersburg. med. Zeitschr. III. 1862.—*Ackermann*, Die Wirkungen des Brechweinsteins auf d. r. Herz Virch. Arch. Bd. XXV. 1862.—*Scheiber*, Zur Lehre vom Herzstosse. Virch. Arch. Bd. XXIV. 1862.—*Stark*, Vergrösserung des Herzens bei Chlorosis. Arch. der Heilk. 4. Jahrg. S 47, 1863.—*Förster*, Ueber den Zusammenhang von Herz- und Nierenkrankheiten. Würzburg. med. Zeitschr. Bd. IV. 1863.—*Skoda*, Allg. Wien. med. Zeit. 1863 u. 1864.—*Tüngel*, Einige Fälle von Hypertr. des. l. Ventr. in Folge von Nierenschrumpfung. Klin. Mitth. Hamb. 1863.—*Roth*, Zum Zusammenhange zwischen Herz- und Nierenkrankheit. Würzburg. med. Zeitschr. V. 1864.—*Hamernik*, Die Grundzüge der Physiologie und Path. des.

Herzbeutels. Prag, 1864.—*Löwer*, Nierenschrumpfung mit Hypert. u. Dilat. des l. Ventr. Berl. klin. Wochenschr. 1864. No. 37.—*Gerhardt*, Ueber einige Formen der Herzdämpfung. Prager Vierteljahrschrift. Bd. IV. 1864.—*F. A. Zenker*, Ueber die Veränderungen der willkürl. Muskeln im Typh. abdom. Leipzig, 1864.—*Polotebnow*, Berl. klin. Wochenschr. 1865. No. 35.—*Gouraud*, De l'influence pathogénique des maladies pulmonaires sur le cœur droit. Thèse, Paris, 1865.—*Rosenstein*, Zur Beziehung von Herz- und Nierenkrankheiten. Berliner klin. Wochenschr. 1865. No. 4.—*Gordon*, Case of hypertr. of the heart from renal disease. Dublin Journ. of Medic. 1866.—*Liagre*, Hypertr. du cœur. Hydropericarditis. Presse méd. de Belge, No. 27, 1866.—*Da Costa*, Med. Memoir of the U. S. Sanitary Commission, 1867.—*Maclean*, Brit. Med. Journ. Febr. 1867.—*Thurn*, Wien. med. Wochenschr. 1868. No. 45.—*Crocq*, Presse méd. belge, 21. Jahrg. No. 3. Séance de la Soc. anatom. Bruxell. (Hypertr. d. r. Ventr). 1869.—*Wilson Fox*, Cyanosis, hypertr. of the heart, chiefly affecting the right ventr. Transact. Path. Soc. XIX. p. 104. 1869.—*Skoda*, Bemerkungen über die Hypertr. des Herz. Klin. Vortrag. Allg. Wiener med. Zeit. Nos. 28, 29, 31, 1870.—*Oscar Weitling*, Ueber d. Hyp. des l. Ventr. nach Nierenschrumpfung. Diss. Berl. 1870.—*Bruzelius u. Blix*, Hygiea, 1870.—*Axel Key*, Nordisk med. Arkiv. Bd. I. 1870.—*Myers*, London, 1870, Concerning strain of the heart. —*Thompson*, St. Georg. Hosp. Report. Vol. V. 1870.—*Da Costa*, The American Journ. of the Med. Scienc. for Jan. 1871.—*Thomas Clifford Albutt*, St. George's Hospit. Report. Vol. V. 1870.—*Black*, Lancet, 1872, Aug. 24.—*Treadwell*, Bost. Med. and Surg. Journ. 1872.—*Moinet*, A treatise of the causes of heart disease. Edinb. 1872.—*Traube*, Berlin. klin. Woch. 1871. No. 29, 1872. 18 u. 19.—*Seitz*, Zur Lehre der Ueberanstrengung des Herz. Arch. für kl. Medic. Bd. XI. u. XII. 1873–1874.—*O. Fräntzel*, Ueber d. Entsteh. von Hypertr. und Dilatat. der Herzventr. durch Kriegsstrap. Virch. Arch. Bd. 57. S. 215.—*C. Handfield Jones*, Cases of heart disease affording evidence respecting the action of digitalis. Med. Times and Gazette, Oct. 18, 25, 1873.—*Hayden*, Hypertr. and granul. degeneration of the heart, acute pericarditis, calcareous transformation of the aortic valves and atheroma of the aorta. Med. Press and Circ. Apr. 2, 1873

History.

Under the name of heart disease the ancients comprehended all possible diseases of that organ, and very frequently it signified hypertrophy and dilatation. In later times, also, they confounded under the general name, induration of the heart, not only the diseases under consideration, but inflammatory disorders and neoplasms. Senac is the first to make any accurate and definite reference to the condition of the walls of the heart and dilatation of its cavities; he describes, also, the bulging of

the thoracic wall, occasioned by enlargement of the heart. It is interesting to observe that H. F. Albertini (who was born at Bologna as early as 1672) called attention to the fact that the left ventricle was especially prone to hypertrophy, and the right to dilatation.

We first find the basis for a thorough comprehension of hypertrophy and dilatation mentioned by Lancisi.[1] He stated the belief that these conditions were frequently the occasion of sudden death, and described a series of symptoms, such as palpitation, difficulty in respiration, etc., which accompanied them. He recognized also, with wonderful clearness, that the cause of hypertrophy lay in various hindrances to the greater and lesser circulations. He had no knowledge, however, of the fact that inflammation might be the occasion of dilatation. He indicated the turgescence of the veins of the neck as a characteristic of hypertrophy of the right ventricle, although he was not able to distinguish between secondary symptoms and the primary disorder. Corvisart retained the name, aneurism of the heart, which he seems to have found in Auenbrugger, and distinguished between active and passive aneurism. He described more thoroughly the mechanical causes of the disease, and is generally considered as the founder of this theory. Laënnec and Bertin speak of hypertrophy of the heart, and make the distinction between it and dilatation. Bertin also describes accurately concentric hypertrophy. It is astonishing that Laënnec does not go more fully into the causes of hypertrophy. The literature of that period is filled with the controversies of different writers (Burns, Hope, Williams, and others) concerning the causes of hypertrophy. Bizot,[2] by means of his accurate measurements, first laid the foundation for rational comparisons of size and volume. Since the days of Rokitansky and Skoda, both conditions have held a more secondary position. But now again, quite recently, through the labors of Peacock, Thurn, Myers, and Seitz, great stress has been laid upon the independent development of the disease.

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[1] De motu cordis et aneurismatibus. Romæ, 1728.

[2] Memoire de la Soc. med. d'observ. 1836.

Introduction.

By hypertrophy of the heart we understand an increase in the mass of the muscular tissue, usually associated with an increase in volume; by dilatation we understand an expansion of the cavities of the organ. It seems best to consider the two conditions together, for they are so frequently associated that a separate consideration would involve continual repetitions. These particular forms of heart disease are made especially interesting at the present time, from the fact that so many writers in all directions are endeavoring to show that many cases, not only of hypertrophy, but of dilatation as well, deserve to be considered primary rather than secondary disorders.

Both conditions may affect the whole heart, but are usually confined to some one portion; in accordance with this fact, we distinguish between general and partial hypertrophies and dilatations, where the diseased condition affects one section of the heart, or perhaps is limited to a single portion of that section, as, for example, hypertrophy of a single papillary muscle, dilatation of the conus arteriosus in the right ventricle, etc.

Under the head of hypertrophy we distinguish the following:

1. *Simple hypertrophy.*—Increase of the heart in volume, with normal cavities.

2. *Concentric hypertrophy.*—Thickening of the walls, with diminution in the size of the cavities.

3. *Excentric hypertrophy.*—Increase in muscular tissue, with enlargement of cavities.

Dilatation of the heart may likewise be embraced under three heads.

1. *Simple dilatation.*—Enlargement of the cavities, with normal thickness of the walls.

2. *Active dilatation.*—Enlargement of the cavities of the heart, with increase in thickness of the walls; but since a dilatation of the cavities cannot be accompanied by a normal thickness of the walls except they are hypertrophied, the condition is similar to *simple dilatation*, which also is the same thing as an *excentric hypertrophy.* Lastly,

3. *Passive dilatation.*—Enlargement of the cavities, with thinning of their walls.

Further on we will show how, among the various forms of disease, only excentric hypertrophy and passive dilatation particularly interest us. The first is by far the most frequently observed combination of dilatation of the cavities with hypertrophy of the walls. The existence of concentric hypertrophy is widely denied; and, in truth, proof of its existence is no easy matter, for, in a given case of thickened walls with diminished cavities, the distinction between a heart simply found at its maximum of contraction, and one where there is an actual increase in muscular tissue, is very difficult and requires great experience. Nevertheless, we must maintain that there is such a disease, even though it occurs rarely, inasmuch as during life we can recognize changes in the pulse, which may be perfectly well explained on the hypothesis of a concentric hypertrophy.

By *spurious hypertrophy*, we understand increase in the interstitial connective tissue, of which we shall speak later.

Etiology.

Hypertrophy always occurs wherever a portion of the heart has been called on to perform work beyond its normal capacity, either to overcome mechanical obstacles or in consequence of increased innervation.

Left ventricle.—This is in general more inclined to hypertrophy than the right, for since it is originally the more powerful, it will be easier for it to meet any obstacle with increased energy than for the feebler right ventricle, which will sooner yield to such opposition, and hence dilate. In consequence of the innate power in the heart to regulate its own action, all those conditions which interfere in a mechanical way with the emptying of the left ventricle, must lead to an increased activity on its part, provided the heart substance is healthy. Such conditions are:

1. A narrowing of the aortic opening. It is clear that the narrower the opening is, the greater will be the obstruction to the column of blood; the same will be the case,

2. When the aorta is contracted in its ascending portion, or throughout its whole course, or only at the isthmus, where it sometimes becomes totally obliterated, close to the ductus arteriosus Botalli; these various conditions are almost always congenital; the first allusion to such a hypertrophy in congenital narrowing of the ascending aorta is made by J. F. Meckel, in Virchow.[1]

3. In this connection belongs the diminution in calibre throughout the whole arterial system, as it is found, according to Rokitansky, most frequently in the female sex, and associated with slight development of the sexual organs.

4. The cutting-off of whole portions or regions of the capillary system ought to occasion hypertrophy of the left ventricle, and although this is found to be the case for the capillary system supplied by the right ventricle, no such state of affairs is developed in the capillary system belonging to the left heart. For example, after amputation of the thigh, when the capillary system is immensely reduced, there is, nevertheless, no hypertrophy of the left ventricle.

Under this head we must consider the hypertrophy which, as is well known, is a very common accompaniment of Bright's disease. Traube has suggested the very clever theory that it arises from the cutting-off of a certain portion of the capillary system in the kidney, in consequence of which the blood-pressure in the arterial system must increase considerably, and which is further augmented by interference with the escape of fluid from the renal arteries and consequent tendency to stasis in these vessels. Numerous as the contributions are in support of this theory, we have an equal number of proofs that it is untenable. Amongst these we may mention: *a*, The observation that we find cardiac hypertrophy already developed in that stage of the disease where as yet there is no granular atrophy (and only this condition will occasion capillary atrophy); if Dusch thinks to explain this by saying that at this stage of the disease there is amyloid degener-

[1] Virchow, Ueber die Chlorose, Berlin, 1872. S. 10. In the collection at Halle, there is a colossal heart, which in the cadaver occupied nearly all the left half of the thorax, and a very narrow aorta, the cross-section of which is half as small again as that of the pulmonary artery.

ation, in which no hypertrophy occurs as an accompaniment, we would reply that we frequently find absence of hypertrophy where there is even no amyloid degeneration; *b*, That after the extirpation of one kidney (Rosenstein [1]), and the removal thereby of a whole capillary region, there followed no increase of pressure in the aortic system. Beckman,[2] on the other hand, produced hypertrophy of the heart by ligation of the ureter in a dog. From this and from two similar cases, communicated by Roth,[3] it would seem to follow that not merely atrophy of the capillaries, but other mechanical obstructions in the renal circulation are capable of producing hypertrophy of the left ventricle. On the other hand, *c*, if this were true, the occurrence of hypertrophy of the left ventricle would be so frequent as to be nearly a constant condition, which we find from statistics is by no means the case. Accordingly it is most probable that we must seek for the true cause of hypertrophy of the heart in other conditions which are found at the same time, such as disease of the heart substance, an improper quality of the blood itself, and hence depraved nutrition, etc.

5. If I only now allude to sclerosis of great arterial tracts as a cause of hypertrophy of the left ventricle, it is because it has a double influence, and two causes act in unison: *a*, The arteries, over a greater or lesser extent, are diminished in calibre, and hence the opposition to the flow of blood into a given region must increase; and, *b*, by the diminution of elasticity in the arteries an important factor in the forward movement of the blood is lost, and accordingly must be replaced by increased activity in the heart.[4]

6. In the same way that contraction of the aorta and other arteries leads to an increased development of the left ventricle, so the same result may follow on dilatation of these vessels likewise from two causes: *a*, If the dilatation is considerable, the

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[1] Virchow's Archiv. Vol. LIII.

[2] Beiträge zur. Exper. Pathol. Vol. IX. 1858.

[3] Würzburg med. Zeitschr. Vol. V. 1867.

[4] Polotebnow (Berl. klin. Wochenschr. No. 35, 1867) relates four cases of hypertrophy of the left (and right) ventricle, which was accompanied by sclerosis in the aorta, in the arteries of the upper, and particularly of the lower extremities, etc.

left ventricle must make a powerful exertion in order to force the current of blood through that which is retarded in the dilated portions. Krause [1] describes a case where there was considerable diffuse dilatation of both carotids and of the abdominal aorta, with very noticeable hypertrophy of the left ventricle; *b*, as such portions of the arterial system usually have lost their elasticity, there is the same failure alluded to above, in the force which drives the volume of blood forward.

7. Insufficiency of the aortic valve is doubtless the most frequent cause of hypertrophy of the left ventricle. If the valves do not close, then at each systole of the left ventricle it must expel not only the blood which has flowed into it from the left auricle during the diastole, but also, in addition, that which has regurgitated from the aorta in the same time. In brief, it must with each systole send forward a larger quantity of blood than normal, and, in order to meet this demand, must suffer hypertrophy.

8. Here we must consider what influence a general increase in the quantity of blood in the system must have on the left ventricle. Theoretically, an increase in the quantity of blood should increase the labor of the left ventricle; but such a condition never lasts continuously for any great length of time, and frequent repetitions of it seem to be followed by no such consequences. As instances, I can only recall those people who are in the habit of drinking great quantities of water. As opposed to other nations, I consider the Germans great water-drinkers, but in Germany not only do we not find more hypertrophy than in other countries, but on the whole much less.

In all these cases we are speaking of an excentric hypertrophy, sometimes with a preponderance of dilatation of the cavities, and sometimes with preponderance of hypertrophy of the muscular tissue.

9. Pathological changes in the texture of the heart, such as myocarditis, endo- and pericarditis, and fatty degeneration must frequently be the cause of this disease of the left ventricle. In the last form doubtless the order of succession is the reverse, viz.:

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[1] Berl. klin. Wochenschrift. No. 11. 1873.

the hypertrophied heart at last takes on fatty degeneration. In myocarditis the formation of indurated tissue especially leads to hypertrophy of the muscular tissue in its vicinity. If hypertrophy of the left ventricle has followed upon adhesions of the heart and pericardium, it must have arisen, on the one hand, from an accompanying disease of the heart tissue, and, on the other, from an increased difficulty in movement caused by the adhesions, and the consequent increase in energy which is thereby called forth.

Hitherto we have only considered hypertrophy as a secondary disease. What can we say of idiopathic hypertrophy? With the great advances which have been made in pathological anatomy, autopsies, where no cause can be found throughout the system for an hypertrophy of the heart, will become more and more rare, and only such observations as have been made in quite recent times will demand our consideration.[1] We might term those cases idiopathic which arise from the use of certain poisons —as, for instance, from the excessive use of alcohol, coffee and tobacco, also those cases which follow upon great mental excitement. Lancisi and Corvisart maintained this view with regard to the latter class of cases, and traced them back to palpitation occasioned by perturbation of mind; their view is also generally accepted, and explained by the theory that there is increased irritation proceeding from the centres of innervation, and in this way there arises excessive action of the organ, with the usual results. Whether even in these cases there are not other disturbances, occasioned by the above-mentioned noxious influences which work with them, must remain undetermined.

We cannot reckon amongst idiopathic hypertrophies those which occur in pregnant women (Larcher, Ducrest), or following upon excessive bodily exertions, such as ascending mountains, or the fatigues of a campaign (Myers, Moinet, Thurn, Fraentzel), or after repeated attacks of epilepsy, for in all these the

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[1] Bruzelius and Blix, Hygeia, 1870.—A young man of eighteen years had suffered since his ninth year from attacks of palpitation which came on suddenly, and lasted ten or twelve days, and in one of these he died. On examination, no cause could be found for the hypertrophy of the heart. (It had not suffered fatty degeneration.)

ultimate cause of the hypertrophy must be sought in disturbances of the circulation.

First, as regards the views of Larcher, who claimed that in the pregnant woman the arterial tract throughout the body had become elongated to supply the fœtus, and accordingly increased power was required, these views received no support from his measurements, the results of which showed that they fell within the normal limit. The others may be explained on the supposition, that in consequence of violent bodily exertions there arise frequent stases and congestions in the vessels, which are compressed by excessive contraction of the muscles; and when the heart is able to meet the new demands, there follows hypertrophy; and where it fails, dilatation (of this latter, more hereafter).

Now if we accept all the contributions on this subject as founded in truth, we shall yet be forced to the conclusion that there was a condition of the heart substance peculiar to the individual, which existed prior to the operation of the above causes, for, otherwise, how very frequent the observation of such disorders would be; and indeed some of the authors above mentioned are astonished that such conditions have not been observed by them more frequently or in all campaigns. Although Traube has proved that in extensive muscular contraction there is increased pressure in the arterial system, yet in the periods of rest which intervene there must be an entire compensation, so that no evil results follow.

No satisfactory explanation has yet been offered of the cause of hypertrophy in that complex set of symptoms which has been styled *Basedow's disease.*

Right Ventricle. — After the thorough consideration of this subject in connection with the left ventricle, we need now only to occupy ourselves particularly with an explanation of the causes of hypertrophy in the *right ventricle.* These are :

1. Stenosis at the origin of the pulmonary artery.

2. Narrowing of the calibre of the pulmonary artery by pressure from without, as by aneurisms of the aorta,[1] tumors, etc.

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[1] I have before me a preparation which shows an excessively hypertrophied right ventricle taken from a patient fifty-six years of age, in whom I diagnosticated, during

3. The cutting-off of capillary regions in the lung develops it in the most striking manner. Here not only does the hypertrophy develop very gradually, but it reaches its highest development in these cases. In this class belong compression of the capillaries of the lung by pleuritic exudation (pneumo-thorax, as a rule, does not last long enough as such to cause it); tumors of any kind; curvatures of the spine; rarefaction of the lung, and capillary compression in vesicular emphysema; obliteration of these capillaries by permanent infiltration, and by induration with or without bronchiectasis.

4. Clots in extensive portions of the pulmonary artery.

5. Over-distention of the system of the pulmonary artery, occasioned by incomplete evacuation of the pulmonary capillaries into the pulmonary veins, always leads to hypertrophy of the right ventricle, in consequence of the increased opposition to each new stream of blood thrown forward by that ventricle; these over-distentions are most intense in those cases where there is disease at the left auriculo-ventricular opening.

6. The hypertrophy which accompanies insufficiency of the semi-lunar valves of the pulmonary artery may be explained in the same manner as was the hypertrophy of the left ventricle, depending on a similar lesion in the aorta.

7. Atheromatous changes in the pulmonary artery will cause hypertrophy of the right ventricle, in the same two ways in which, as we explained above, it occasioned left hypertrophy where the same process was going on in the aorta (p. 193), and upon which Klob[1] has laid great stress.

8. As the same author has personally informed me, dilatation of the pulmonary arterial system, though it rarely occurs, will occasion hypertrophy of the right ventricle. Klob found, at an autopsy which he had recently made upon the body of a man forty years of age, a very considerable hypertrophy of this

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life, an insufficiency and stenosis of the pulmonary artery, and the autopsy showed the existence of these lesions, but depending upon a large aneurism of the ascending aorta (the size of the two fists) which crowded and thoroughly compressed the pulmonary artery.

[1] Wochenblatt d. Ges. der Aerzte, 1865.

ventricle, occasioned by a notable dilatation, which was generally distributed through the system of the pulmonary artery.

Hypertrophy of the auricles is much more rare than dilatation of the same cavities, and is usually combined with the latter in such a manner that the dilatation is in excess, *i. e.*, it is an excentric hypertrophy. This occurs in all those cases where the same pathological condition exists in an advanced stage in the ventricle of the same side, and it also occurs independently in stenosis of the right and left auriculo-ventricular openings respectively.

Hypertrophy of the **whole heart** is found where there is a combination of several of the causes which served to develop hypertrophy in single portions of the organ. These are especially:

1. Simultaneous disease at several of the openings.
2. Complications of valvular disease, with other disturbances in the circulation, such as aneurisms, or extensive sclerosis of the arteries, or with diseases of the lung, and the like.
3. Communication between the two ventricles.
4. General disease of the heart substance; *e. g.*, following pericarditis or myocarditis and fatty degeneration; in this class we may reckon chronic Bright's disease.

Here we may best take up the question how it happens that, with an insufficiency of the mitral valves and subsequent hypertrophy of the right heart, there occasionally follows an hypertrophy of the left heart as well, when this latter would seem to find no opposition to a complete emptying of its cavity by systole, and accordingly would have no cause to become hypertrophied. Friedreich explains it by saying that here, as in other important diseases, such as mitral stenosis, emphysema of the lungs, etc., there follow disturbances in the capillary circulation throughout the whole body, occasioned by dropsy, and these must be met by compensatory hypertrophy of the left ventricle. This explanation might hold when dropsy is present, but this is by no means always the case. The other mechanical theories are untenable, and especially that which claims that the current of blood must come with increased force from the auricle into the ventricle, which accordingly will dilate, and subsequently must,

in compensation, become hypertrophied. There remains to us, then, only the following view, viz., that from the harmony of action in the two ventricles, and the unbroken continuity of the muscular fibres from one-half of the heart over on to the other, an affection of the muscular tissue of one side must have communicated itself to the other.

Hypertrophy of the heart is by no means a rare affection, for its presence at autopsies varies in frequency from 12 per cent., according to Willigk,[1] to 18.4 per cent., according to van der Byl.[2] As regards the sex, the statistics are so variable that we cannot make any approximate ratio between the two. There seems, however, to be no special preponderance of either sex. This, of itself, is of importance in considering the etiology of the disease; for, if hypertrophy were so frequently occasioned by excessive physical exertions, then, without doubt, the preponderance would be immensely on the side of the male sex. In childhood the disease is very rare, and, when found, is usually congenital. It occurs especially in middle life and advanced age, and in the latter is without doubt a natural sequence of diseased arteries.

Dilatation of the Heart.

This is always a secondary disease, either occasioned:

1. By increased pressure within a cavity whose walls have not increased in thickness; or by

2. Disease of the heart substance.

At the close of this section we will speak of the possible occurrence of this disease idiopathically.

We can best clear up the first point by citing two examples of the disease, after considering the special effects of the causes of excentric hypertrophy. In the left ventricle, dilatation is most beautifully developed in cases of insufficiency of the aortic valve. In consequence of the cavity being filled from two sides at once at each diastole (viz., by entrance of blood from the left auricle, plus that which regurgitates from the aorta), the walls of the

[1] Prag. Vierteljahrschrift. Vol. XLIV.

[2] Transactions of the Pathological Society of London, Vol. IX., 1861.

ventricle must submit to an increase in pressure for which they are not adapted and to which they will gradually yield. Soon, however, by virtue of the compensatory power, innate in the heart, the condition changes into one of excentric hypertrophy, and now it generally depends on the nature and quality of the heart tissue which shall prevail, hypertrophy or dilatation. When the capillary circulation of the lung is over-distended, the right ventricle cannot discharge its contents with the usual facility into the pulmonary artery, and this stasis, together with the pressure of the blood which normally enters the chamber from the right auricle at the same time, will develop dilatation, and just so much sooner on this side than on the other as its muscular tissue is feebler and more ready to yield to increased pressure. Here, also, as we have described above, the condition soon changes into one of excentric hypertrophy.

From what precedes, it is clear that the auricle on the same side as the ventricle affected, and which is subjected to the same influences, will undergo extreme dilatation. Enormous dilatations of the left auricle are found in cases where there is stenosis of the left auriculo-ventricular orifice. I have before me a similar dilatation of the right auricle, but even here associated with partial increase in its muscular substance, in a case of extreme stenosis at the orifice of the pulmonary artery with insufficiency of its valves.

If the heart has lost its normal contractile power, through disease of its muscular tissue, then it will yield to the normal blood pressure, and the greater the advance the disease has made the greater will be the dilatation. First amongst the causes of this tissue change is myocarditis, whether idiopathic or following upon pericarditis ; second is fatty degeneration of the heart substance. After certain febrile affections, especially pyæmia, severe puerperal diseases, typhus, variola, scarlet fever, etc., the action of certain poisons, particularly of phosphorus (Munck and Leyden),[1] there ensues, on account of the breaking down of the muscular fibres of the heart into a molecular detritus, and the subsequent relaxation of the muscular

[1] Berl. klin. Wochenschrift, 1864, Nos. 49 and 50.

tissue, an acute dilatation of the heart. Doubtless the changes which occur in chlorosis may be explained in this manner. Of late years it has been held, and especially by Stark,[1] that in severe cases of this disease we can make out an enlargement of the heart, which again gradually disappears with the return of health. Here also we must take up the question of an idiopathic dilatation of the heart without any special disease of its muscular tissue. Various authors, Forget, Peacock, Maclean, Thurn, Thompson, Seitz, Black, Treadwell, Fraenckel, have made the assertion that in the over-exertion incident to the fatigues of a campaign, and similar laborious occupations, the heart suffers and undergoes a dilatation of its cavities which may be followed by expansion of its orifices and subsequent insufficiency of the valves. The subject is most exhaustively treated in a very carefully-written work by Seitz ; yet even this does not by any means convert me to a belief in idiopathic dilatation. It would not be strange if it should sometimes happen that the heart was not in a condition to meet great additional demands upon its strength, and it should gradually succumb to the task, and suffer dilatation and all the subsequent ills ; but if this could take place without any disease of the heart substance, how frequent an occurrence it would be, for do not the mass of mankind live by severe physical exertion? We can only believe, then, that certain hearts, *i. e.*, abnormal ones, are not able to endure severe exertion. Wherein the disease lies, whether in some tissue change which we cannot recognize, or, what is quite probable, in disturbances in the innervation, we cannot at present determine. From the distinguished names cited as authorities by Seitz, we cannot doubt that the autopsies were conducted with the greatest care. But we must not forget that appearances quite similar to those quoted here are very frequently found in autopsies on drinkers. Now, porters and men of similar occupations, concerning whom he treats, are very frequently hard drinkers.

The same statement holds true for the view presented by Peacock, according to whom, the laborers in the Cornwall

[1] Arch. f. Heilk. Vol. I., 1860.

mines, from their constantly-repeated ascent of ladders, the disturbances thus occasioned in the circulation, and the demands for over-exertion of the heart, suffer from dilatation of its cavities, and even subsequent mitral insufficiency. But nature has so ordained, that when a healthy muscle is called on for extra exertion, it shall develop hypertrophy, and not atrophy, and, accordingly, a hollow muscle will not dilate. The hypertrophy of the right ventricle shows in the most beautiful way how the heart is ready to meet an increased demand upon it. If the views of Seitz and Peacock were correct, how very frequently must dilatation of the right heart occur. Fraentzel confesses that his observations were made only in the campaign of 1870–1871, and that he had made no similar ones in previous campaigns, and even here his entire number was only nineteen cases; finally, he allows that there must have been a certain deficiency in the resistance of the heart tissues to make the existence of dilatation with the hypertrophy possible.

Pathology.

Pathological Anatomy.

It requires considerable skill and experience to recognize slight cases, either of hypertrophy or dilatation, and not to fall into the error, on the one hand, of mistaking a strongly-contracted heart for one that is hypertrophied, or, on the other, a heart relaxed and temporarily distended with blood for one which is dilated. It seems desirable to give the normal measurements and weight of the heart, in order to have a starting-point for the comparison of normal with diseased conditions of the organ. According to Bizot, they are as follows:

	MALES.	METRES.	FEMALES.
Length of the heart	0.097	"	0.092
Breadth " "	0.107	"	0.099
Thickness " "	0.038	"	0.031
Length of the left ventricle	0.066	"	0.072
Breadth " " "	0.119	"	0.104
Length of the right ventricle	0.084	"	0.075
Breadth " " "	0.188	"	0.172

	MALES.	METRES.	FEMALES.
Thickness of the Walls of the Left Ventricle—			
At the base	0.010	"	0.009
At the middle	0.011	"	0.010
Near the apex	0.008	"	0.007
Thickness of the Septum Ventriculorum—			
At the middle	0.011	"	0.009
Thickness of the Walls of the Right Ventricle—			
Base	0.004	"	0.003
At the middle	0.003	"	0.002
Near the apex	0.002	"	0.002
Breadth of the Auriculo-Ventricular Orifices—			
Left ventricle	0.100	"	0.091
Right ventricle	0.122	"	0.106
Breadth of the Origin of the Aorta (above the valves)	0.069	"	0.063
Breadth of the Origin of the Pulmonary Artery	0.071	"	0.066

In children under eight years of age the left ventricle is relatively larger than in adult life (Gerhardt); this difference seems to depend upon a contraction of the aorta still remaining, which was originally formed at the point of emptying of the ductus Botalli. According to Bizot, the whole heart of children under eight years is relatively large. In new-born children the walls of the right ventricle are as thick as those of the left; the true relation between the two is established later. According to Bizot, all the dimensions of the heart increase up to the most advanced age, but most actively before the twenty-ninth year. The weight of the heart, according to Engel,[1] for both ventricles without the auricles, is 220.6 grammes, according to Clendinning,[2] for the whole heart, 300 gr. in males and 270 gr. in females.

We have already said that the hypertrophy might affect the whole heart, or only one of its divisions, or even only an integral portion of that (*e. g.*, only the papillary muscles, the conus arteriosus, the septum, etc.). We also mentioned the classification, both of hypertrophies and dilatations, and laid stress on the statement that concentric hypertrophy was of rare occurrence, simple hypertrophy not common, and the most frequent was the excentric form, sometimes in the ventricles and auricles as well. When all portions of the heart are affected at the same time, it often reaches an enormous size (cor taurinum).

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[1] Concerning some Pathologico-Anatomical Changes in the Heart. Wien. med. Wochenschr. 1863-64.

[2] Med. Chir. Transactions. II. Series III. 1838.

The thickness of the wall of the left ventricle may increase to 4 cm., that of the right to 2 cm., that of the left auricle to 0.7 cm., and that of the right to 0.5 cm. (Rokitansky). The greatest weight, according to Rokitansky, was 1120, and according to Hope 1250 grammes. When the left ventricle is hypertrophied, the shape of the heart is altered by its becoming elongated. If, at the same time, there is a dilatation of its cavity, then the septum will be pushed over and will invade the right ventricular cavity. In hypertrophy of the right ventricle, the heart becomes broader and its apex is rounded off. This variation in the character of the change in shape, according as one or the other ventricle is affected, may be readily explained on the recoil-theory to which Scheiber [1] has already called attention. The enlarged heart is wont to assume a more horizontal position, and when the left hypertrophy preponderates, it extends towards the left and downwards; the diaphragm, upon which it rests to a greater extent than normal, may likewise be pressed downwards.

The texture of the organ is strikingly hard; the tissue creaks and gapes open on section, and this is particularly striking in the right ventricle. The color in simple hypertrophy is dark reddish-brown. Later on in the disease, when fatty degeneration has occurred in some portions, we find the heart substance sprinkled over with clearer, yellowish spots.

Dilatation, with thinning of the walls, occurs most frequently in the *auricles*, when the distention, especially of the right, may be enormous, and terminates in complete disappearance of the muscular tissue, the endo- and pericardium coming into contact. If the borders of the orifices are not otherwise diseased, they are oftener enlarged at the same time, and the valves as well as the trabeculæ are thinned; the tissue is pale, flabby, and easily torn, and on section the walls immediately collapse.

The general view is, that the increase in mass of the muscular tissue is produced by an increased thickness of the primitive bundles, although it has not often been reduced to figures. Hepp,[2] who puts the normal thickness of the primitive fibres at

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[1] Virchow's Archiv. Vol. XXIV. [2] Dissertation. Zürich, 1853.

0.007 mm., found them to measure 0.03 mm. in an hypertrophied left ventricle.

Friedreich found 0.025 mm. to be the mean of ten measurements of the hypertrophied left ventricle in a drinker. According to Rindfleisch, the increase in volume takes place by splitting of the muscle cells. He formed his opinion on his finding some of these cells, containing, instead of one, two and even more nuclei, which, according to Weissmann,[1] indicates that splitting of the fibres in the long axis will follow. Zielonko,[2] who carried out some studies concerning the nature of hypertrophy, found, in his experiments on frogs, on which he had partially ligated the aorta, causing a certain contraction of its calibre, that there followed an actual increase in mass of the muscles, which was not occasioned by enlargement of all the cells, but more particularly by increased growth and multiplication of free nuclei, which might serve as material for new cell formation; this agrees with the statement of Zehetmayer on the same point, who found numerous cell nuclei. In hypertrophy of the heart in man, no true indication of it could be drawn from the size of the muscular cells (*i. e.*, their dimensions bore no fixed relation to it), but, on the other hand, in atrophy of the heart the decrease in its volume was accompanied by a corresponding decrease in the diameter of the cells. It is an interesting fact, according to R. Lee,[3] confirmed also by Cloetta,[4] that in hypertrophy of the heart there takes place also a thickening of the nerves; it is not certain, however, whether this arises from increase in their primitive fibres, or from a greater development of the connective tissue forming their sheath.

Symptoms.

Since hypertrophy and dilatation are usually secondary and not primary diseases, in the statement of symptoms, we must

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[1] Reichardt's Archiv. 1861.

[2] Virchow's Arch. Vol. LXII. First Part. 1874.

[3] Memoir on the Ganglia and Nerves of the Heart. London, 1851.

[4] Virch. Archiv. Vol. V.

carefully distinguish between those which belong to the disease in question and those which may be referred to the original disease. As regards inspection, we frequently find it stated (Gerhardt, Friedreich, Bamberger—Niemeyer and Dusch, particularly in young subjects) that both general hypertrophy and that of the left ventricle cause an increased bulging over the heart, as if it were occasioned by the increased pulsation of the heart against the thoracic wall. I must decidedly object to this view ; I have seen no case of either of these diseases alone where such a bulging had ensued. As Skoda has already stated, this occurs only where these diseases are complicated with pericarditis, in which, as in pleuritis, there is a softened, spongy condition of the textures forming the chest-wall ; and on this account, and particularly, also, from the increased traction of the inspiratory muscles, we have an abnormal bulging over the heart.

The character of the impulse is one of the most striking symptoms of hypertrophy, especially in that of the whole heart and in that of the left ventricle. Very often we find it pushed downwards and further over to the left than normal, in the sixth, seventh, or even eighth intercostal space. Not unfrequently we may observe the whole change in position of the heart at each systole ; while at the time of the greatest bulging we may observe retraction of the intercostal space lying over the apex of the heart, and also at the scrobiculum cordis. Displacement of the apex further to the left only indicates, as a rule, hypertrophy of the *right* ventricle, while dislocation to the left and downwards at the same time indicates usually hypertrophy of the *left* ventricle. The character of the heart's impulse is very varied; the so-called heaving impulse is characteristic only of a left ventricular hypertrophy, in which very often, not only over the apex but also above it, the hand laid upon the chest will be raised with considerable force at each impulse. This phenomenon will be most distinctly observed by placing a stethoscope against the wall over the apex, and seeing how it will be thrown forwards. Frequently the whole left side of the chest will be pushed over to the left with each systole, so that there ensues an actual heaving of the chest-wall. In other cases the impulse is simply

increased, and communicates a jarring blow to the head resting upon the chest-wall. Only in concentric hypertrophy, or at times of profound rest, or in diseases of the heart substance, does the impulse become weak.

In hypertrophy of the right ventricle the jarring impulse is most evident between the apex and the lower portion of the sternum, and just here is strikingly distinct. This may be perfectly well explained, as I have suggested above, on the recoil theory, for if the right ventricle has become hypertrophied, it is just here that the rebound must take place, while in hypertrophy of the left ventricle the recoil from the aorta is directed towards the apex.

In percussion of the heart, I hold firmly to the point, that we must ascertain accurately the place where the sound is absolutely dull, *i. e.*, the extent over which the heart is immediately in contact with the chest-wall. In a normal condition of the parts this space is quite small, and has usually a triangular form. When you have found the apex beat (usually in the fifth intercostal space) inside the left nipple, and percuss from this point, passing somewhat obliquely upwards and from left to right, following the line of the insertions of the diaphragm, you will find that the dulness begins either directly over the apex or a little further toward the median line (since the apex is very frequently covered by the edge of the lung), and extends over to the middle of the sternum. Now, if you make percussion from the clavicle downwards in the parasternal line, the dulness corresponding to the base of the heart begins at the lower border of the third or upper border of the fourth rib; if, from this point, we draw a line to the apex, or, better, to the point just inside where the dulness began, we then shall have found the second side of the triangle, reaching usually to the middle of the sternum, and indicating the left border of the heart; the third side is found by connecting the termini of these two lines in the median line of the body. Occasionally we find on careful percussion that that side of the triangle, indicating the left border of the heart, is not a straight line, but is either somewhat curved with its convexity towards the left, or is a more or less broken line with a shorter portion

running nearly straight from right to left, and a larger extending obliquely downwards. The whole forms no longer a triangular, but rather a more trapezoidal figure.

When there is enlargement of the heart, we must remember that as the borders of the lung are pressed back by it, unless pathological changes prevent, it will lie in direct contact with the chest-wall over a greater extent than normal. But since it is clear that the lung, which does not cover the heart to an equal extent in all men, is also not always retracted with equal force, we can see that the shape of the heart dulness will not always be the same in equal enlargements of the heart in different individuals. We can also say that an increase in the dulness over the heart is found to be an indication of enlargement, and only when there is very considerable increase in volume of one or the other ventricle can this fact be recognized by percussion. It follows then that in hypertrophy of the left ventricle the dulness is extended in the direction of the long axis. To recognize the enlargement of the right ventricle by the known pathological increase in breadth of the heart, is not an easy matter. If the heart lay nearly vertically, as it is represented in many books, then its breadth would be indicated by that line which we find, percussing from right to left at the level of the base, as given above. But since the heart lies more horizontally, this line does not correspond to its breadth, but is a secant of its long axis; the actual breadth is represented by a line extending from the point near the base in the left parasternal line where dulness begins, obliquely across the sternum to the point to which we find that dulness reaches in percussing towards the right from the apex across the sternum.

We occasionally find dulness beginning in the sixth or seventh intercostal space, from 2 to 5 cm. outside the nipple line, and reaching from 2 to 4 and even more cm. beyond the right edge of the sternum; and at the base we sometimes encounter dulness at the level of the lower border of the third or upper border of the fourth rib, beginning at the right edge of the sternum, or even further to the right, and reaching a distance of from 5 to 8 cm. towards the left. I have pictured rather a large extent of dulness, as is not unfrequently found in enlargement of the

whole heart, and as I have this very day observed it in the person of a patient in my service, in whom, besides this, I could only make out the existence of mitral insufficiency.

Auscultation.—Theoretically, we should expect to find that both sounds of the heart would be intensified, because the increased energy of the heart's action will cause more intense vibration in the media which convey the sound. Clinically, however, as regards intensity of the sounds, we certainly find no fixed relation to the hypertrophy. It is true that in hypertrophy of the right ventricle, the second sound in the pulmonary artery is almost always intensified, and in that of the left ventricle there is frequently accentuation of the second sound in the aorta. This depends on the increased elasticity which is called forth in the wall of the vessel under consideration, for as the current of blood is driven with greater force into the artery, which is thereby more violently distended, it will endeavor (with all the greater intensity) to restore the equilibrium, and hence the blood in recoil will strike with greater force upon the upper surface of the closing semi-lunar valve. But now we must remember that even in normal conditions the heart sounds are so variable in intensity, that if we have not been familiar with them before in a given individual, we cannot come to any accurate conclusions as to a possible change in sound. The second sound in the aorta is very often not intensified, for the reason that its coats have partially lost their elasticity, which need not astonish us, when we consider that this form of cardiac disease is frequently associated with atheroma, aneurisms, etc. Baur, in his eighteen cases of idiopathic hypertrophy, in which he noticed that the sounds were increased and heard over a greater space, has stated that he heard them with unusual distinctness over the posterior surface of the thorax. I consider this a purely accidental occurrence, for the same thing is noticed with many normal hearts.

Occasionally we find on auscultation over the apex, particularly with the unaided ear, a peculiar clink with the first sound, called by Laënnec "cliquetis métallique." Bouillaud, who described it under the name " tintement métallique," was fully persuaded that it was only occasioned by a peculiar jar-

ring of the chest-wall, which was set in vibration by the impulse. Many points serve to establish the accuracy of this view: for example, we observe the sound when the heart's action is increased in a given individual; but in the same person when the action is quiet and feeble, we do not hear it at all. And also the clink is more often observed in people who have thin ribs—hence better adapted to vibration—than in those with thick ribs and narrow intercostal spaces.

The remaining symptoms of a pure hypertrophy are derived from a consideration of the influence which it must have upon the circulation. In all cases of simple, and especially of excentric hypertrophy, the effect will be to quicken the circulation. The hypertrophied left ventricle drives the blood, and where there is dilatation at the same time, a greater quantity of blood, with increased force into the arterial circulation, which soon becomes distended with blood; nevertheless the shorter circulation will not be wanting in blood, for the increased *vis a tergo* drives it more energetically through the capillaries into the veins, and it must easily escape from the pulmonary veins into the left heart, so that the result of all will be an acceleration of the whole circulation.

An hypertrophied right ventricle will soon occasion over-distention in the system of the pulmonary artery, while the left ventricle will impel a less quantity of blood; but for this very reason the blood will soon flow all the more easily from the pulmonary veins into the left ventricle, and accordingly a more rapid circulation will be the result. Finally, if both ventricles are hypertrophied, there will first be an accumulation of blood in the arterial system, and a corresponding diminution of the contents of the veins. But from this reason, since the blood flows with increased *vis a tergo* more easily into the capillaries, and also easily escapes from them, there will result an acceleration in the circulation.

The *pulse* sometimes acquires such a peculiarity that it becomes characteristic of the disease. In excentric hypertrophy the powerful left ventricle drives a relatively large stream of blood into the arterial circulation. So the distention of the artery is sudden and very considerable, and since the increased

impulse of the heart is conducted far from its source, it is perceptible also in the small arteries. In consequence of the powerful jarring of the arterial wall, listening over it we may frequently hear a humming sound, or, if the vibrations are repeated with sufficient exactitude, we may hear a note, this particularly over the brachial and femoral arteries.

Just here we may speak of concentric hypertrophy. Does such a disease actually exist? or is it only observed at autopsies? and is it, then, simply a strongly-contracted heart and no hypertrophy?

Skoda, Bamberger, Rokitansky, and others believe there is such a disease, and in fact the proof of it, rare as it is, may be drawn from clinical observation. We find in a patient a small, slowly-filling pulse, which we cannot ascribe to stenosis of the aorta, for the other symptoms are wanting. At the autopsy, the cause of this symptom becomes clear. We find the left ventricle concentrically hypertrophied, and often very considerably so; the cavity of the heart being so small, only a small quantity of blood can be forced into the arteries. Why does this take place with diminished force? It would appear, apart from disease of the muscular tissue, that the muscle is so thickened that contraction is interfered with.

Since the phenomena of hypertrophy of the left ventricle and of the whole heart are nearly the same (or vary only in degree), we will speak of them under one head. They are the symptoms of increased vital activity and greater lateral pressure in the arterial vessels. Often enough we see such patients, who have the appearance of perfect health, and in fact are perfectly well. Occasionally we notice in them a more brilliant coloring of the cheeks and stronger pulsations in the carotids. Often, however, they are annoyed by determination of blood to the head, headache, dizziness, roaring in the ears, specks before the eyes, and palpitation; this last may be the most prominent of all the symptoms. From peculiarities in the innervation of the heart, there is often a lack of correspondence between the feeling of the patient and the objective intensity of the heart's impulse. We notice, for instance, in a patient a very vigorous impulse, and yet find, on questioning him, that he is not at all disturbed by it.

In other cases, the visible movement in the vicinity of the heart is very inconsiderable, and yet the patient complains of palpitation. Another time palpitation comes on only after excitement, or great physical exertion, or the use of spirituous liquors, and occasionally in a very troublesome manner, especially on lying down at night, when not only palpitation but strong pulsation in the carotids may torment the patient considerably.

Where there is great hypertrophy, the patients complain of a feeling of fulness, weight and pressure in the region of the heart, but of no actual pain. Shortness of breath is usually first observed after attacks of palpitation.

Since hypertrophy of the right ventricle hardly ever occurs as an idiopathic disease, we are not able to separate its characteristic symptoms from those which depend upon its cause, viz., stasis in the system of the pulmonary artery, etc.

Dilatation of the left ventricle, as we have already mentioned, is much more rare than that of the right. The impulse of the heart is, as a rule, weaker than normal, sometimes of normal intensity, and occasionally even, surpassing that, is agitated and excited: it is broader, and, corresponding to the enlargement of the heart, may be felt lower down and to the left. It would appear that the insufficiency of the heart's contractions is the cause of the irregularity and unusual rapidity of their succession. Percussion gives us dulness of that peculiar shape which we have described above. On auscultation we hear the sounds either with their normal intensity or considerably duller, a change which we can easily explain. When the dilated heart is no longer able to contract fully, a portion of the blood remains in its cavity, and the arteries are lacking in blood, while the venous system is overfilled. If the dilatation becomes still greater, there follows a stasis of blood in the capillaries as well, since they cannot discharge their contents into the already overloaded veins; following this, the left ventricle is not fully emptied, and *slowing* of the circulation is the result. Accordingly, such patients are, as a rule, either paler than usual or else easily become cyanosed or even dropsical, and then, from the efforts which the heart makes to meet the new demands upon it, they suffer from palpitation. *Dyspnœa* is caused by the

irregularity in the circulation, as also by the distention of the capillaries of the lung and the pulmonary veins, and the *cough* depends upon the same cause. The attacks of fainting which sometimes occur are caused by anæmia of the brain.

Dilatation of the *right ventricle* in its advanced stages is usually associated with *dilatation* of the *right auricle*. Not nearly so often as is generally supposed, however, does the extension of dulness towards the right beyond the edge of the sternum indicate the existence of this lesion; but there is another symptom, which frequently does call our attention to it. The over-distention of the auricle occasioned by the turgescence of the venous system calls forth unusual contractions, and these are manifested by pulsation or undulation in the veins of the neck.

Among the subjective symptoms we should mention the *dyspnœa* which is so frequently observed, caused by the slowing of the circulation and the more venous condition of the blood.

The *left* auricle is so hidden out of reach, that when it is dilated there are no signs which can be recognized by percussion.

Diagnosis.

The diagnosis of secondary hypertrophy (following upon diseases of the valves, etc.), as a rule, is not difficult; but it is quite otherwise when the disease is idiopathic; in this case all the signs and symptoms should be maturely considered and weighed. I would call attention here to the fact, that we so often find the diagnosis of hypertrophy given where there is merely an increase in the heart's impulse. Now, this is quite wrong, for with each passing excitement this symptom may recur.

To establish the diagnosis of *simple and excentric hypertrophy*, the following signs will serve, after carefully considering the various precautions which I have mentioned under the head of symptoms: an increased impulse of the heart, either heaving in character or jarring the chest-wall, more or less displaced towards the left and downwards, enlargement of the heart

in its long axis, sometimes an intensification of the sounds, especially of the second sound in the aorta, occasionally "tintement métallique," increased pulsation of the carotids, murmurs in the larger arteries and also in the small vessels far removed from the heart, and a pulse perceptibly larger in volume under the finger.

In hypertrophy of the right ventricle, the heart's impulse is not increased, except occasionally near the lower portion of the sternum; the apex beats farther to the left, but not lower down. The area of dulness is increased in breadth, and the second sound in the pulmonary artery is accentuated.

In total (general) *hypertrophy* we find a combination of symptoms corresponding to the hypertrophy of (the different) ventricles. It is seldom, however, that we can succeed in declaring with accuracy which half of the heart is the more enlarged.

The subjective symptoms do not give us much aid in diagnosis, for all of them are frequently observed in other diseases of the heart in which palpitation occurs.

Finally, in all cases in which the impulse of the heart can be made out simply beyond, or else beyond and below its normal position, and this has not arisen from pressure or traction on the organ, we may assume that hypertrophy exists.

As regards *differential diagnosis*, it is possible to mistake it for (1) *exaggerated action of the heart*—an examination of the size of the organ will quickly set us right here; for (2) *pericardial effusion*—here the diagnosis will be established by the change in the shape of the area of dulness, lack of friction sounds, frequently by variation in the character and place of the heart's impulse on changing the patient's position, finally by the feebleness of the heart sounds; for (3) *aneurism of the aorta or pulmonary artery*. If the characteristic indications of these diseases are wanting, the diagnosis can only be made out by considering the shape of the area of dulness and the secondary symptoms; for (4) *mediastinal tumors*—these also must be recognized by the place and shape of the region where dulness is found, by the results of auscultation, and perhaps by the presence of enlarged lymphatic glands; for (5) *encapsulated pleuritic exudations*. These, when single or double (*i. e.*, on one or

both sides), may resemble, in a wonderful manner, a greatly enlarged heart, by the similarity in the shape of the area of dulness. Here, however, when we change the position of the patient, there will be no corresponding change in the position of the heart, and also the dulness will remain the same with inspiration and expiration, which is not true in hypertrophy, unless other conditions interfere to prevent the free movements of the lung.

Great extension of dulness over the region of the heart may occur without any enlargement of the organ, when it is pressed forwards by tumors situated behind it, or by antero-posterior curvature (kypho-scoliosis) of the spine; so, likewise, if during the absorption of pericardial exudation the pleura costalis has grown fast to that of the mediastinum, and thus the lung is prevented from pushing forwards over the heart. In these cases we form our diagnosis by the location of the apex beat and lack of mobility in the heart when the patient's position is altered.

Moreover, hypertrophy might be overlooked when the heart is covered over by the lung in emphysema, or when the lung has grown fast to the pleura costalis, so that it cannot retreat before the hypertrophied heart; here the place of the apex beat compared with the dulness must be our guide. Finally, in all cases, the recognition of some cause for the disease will be of great importance in establishing a diagnosis of hypertrophy.

The diagnosis of *dilatation* is just as difficult as that of hypertrophy is, as a rule, easy and sure.

By percussion we make out the same shape of the heart dulness which we find in hypertrophy. The character of the impulse accompanying this increased dulness will assist us in the diagnosis, for it is generally feeble, certainly not heaving, which, however, would not of itself make the diagnosis sure, for this feeble impulse may be due to other causes, such as diseases of the heart substance, pericardial exudations, etc. The feeble impulse will be accompanied by feebleness of the heart sounds and of the pulse. If we should find murmurs being developed, we should make a mistake if we concluded that they were due to relative insufficiency of the valves caused by dilatation, and then employ them to strengthen the accuracy of our diagnosis. Under such circumstances, a consideration of the sequelæ of the

dilatation may help our diagnosis. As we have already stated, we have no means of diagnosticating dilatations of the left auricle. Those of the right auricle are more easily made out, and yet it is very easy to mistake an extension of dulness from the second to the fifth rib on the right of the sternum for other diseases, especially aneurisms. A lack of pulsation does not necessarily signify that no aneurism is there, neither does its presence always indicate the existence of the disease, as is seen in the case of Stokes.[1]

Here the dulness, which was found on the right of the sternum from the second to the fifth rib, and over which systolic pulsation was seen, was occasioned by the dilatation of the right auricle, and the pulsation which occurred with systole doubtless was due to insufficiency of the tricuspid valve.

Pulsation of the veins of the neck can be relied on as an indication of dilatation of the right auricle.

Complications and Sequelæ.

As the disease is usually secondary and not primary, we need not allude to other complications than those which we have already mentioned under *etiology;* one more will be cited in treating of the course of the disease.

The sequelæ are sometimes occasioned by traction on the neighboring parts from the increased weight and volume of the organ, but first of all they are observed in the blood-vessels. Long-continued hypertrophy of the left ventricle to a considerable extent will finally occasion such distention of the vessels that they lose their normal tone, and then a very considerable interference with the circulation follows.

The abundant supply of blood to individual organs causes disturbances in their nutrition, and the increased blood-pressure occasions ruptures of the vessels. Bruberger[2] and Geigel[3] relate cases where very noticeable contractions, equally dis-

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[1] L. c., p. 226.

[2] Berlin. klin. Wochenschrift. No. 50. 1870.

[3] Results of 84 Autopsies. Würzburg. med. Zeitschr. II. 1861.

tributed throughout the aorta, narrowing the calibre of the vessel, and which were accompanied by a considerable hypertrophy of the left ventricle, ultimately produced ruptures in the ascending aorta, and writers have at all times associated cerebral apoplexy with hypertrophy of the heart. It would seem, however, as if this only gives the "coup de grace," and the real primary cause is the existence of diseased vessels of the brain. In *hypertrophy of the right ventricle* it is far less probable that the attacks of hæmoptysis are produced by the increased force of that part of the heart than by the stasis of blood which is the result of the primary disease.

In long-continued hypertrophy of the left ventricle there almost always ensues a fatty degeneration of the heart-substance, and its results.

The first result of a dilatation of one portion of the heart is a failure in the discharge of its contents, and from this follows stasis of blood, with dropsy and cyanosis.

Rupture of the heart does not occur unless its muscular tissue is at the same time invaded by disease.

Course. Termination.

Hypertrophy may exist for many years, and the individual still continue to have relatively good health, but in the end it surely leads if not to a so-called catastrophe through some of its sequels, at all events by fatty degeneration and subsequent dilatation to disturbances in the circulation, which are of themselves full of danger to the patient. What we might say under this head concerning dilatation, may be easily inferred from what has been given above. Dilatation of the right ventricle, from the weakness which it causes in the contractions of the heart, may finally lead to thrombosis in the larger branches of the pulmonary artery, and so to a fatal termination.

Prognosis.

It is only when hypertrophy of a portion of the heart takes place as a compensation for some interference with the circula-

tion, that this can be considered as a favorable occurrence, and it will remain such only so long as it does not develop more force than is required to maintain a normal circulation, and the usual sequelæ are not developed. Even though idiopathic hypertrophy may be borne for years without injury to the patient, yet ultimately the degeneration of its muscular tissue is seen to follow with its usual damage to the circulation.

The prognosis of a *dilatation* of a portion of the heart is, under all circumstances, unfavorable. Only lately have we learned to appreciate the danger of acute dilatation during the course of febrile diseases, such as pneumonia, typhus, etc., and a few chronic diseases, *e. g.*, chlorosis.

Treatment.

It is chiefly in cases of idiopathic hypertrophy that the question of treatment would have to be considered. Energetic measures, such as bleeding, starving, the use of iodine and mercury, have long since lost all their repute, and all we can do is to ward off injurious influences, and attack the symptoms Of these the most important is the overaction of the heart, whether observed by us, or only subjectively felt by the patient. The indication, then, will be to establish and maintain a proper action of the muscle. This can best be done by causing the patient to avoid severe physical exertion, to abandon the use of exciting drinks, especially strong coffee, tea, or wine, and also the use of strong tobacco; moreover, the bodily functions must be maintained in proper condition, and, if necessary, mild cathartics, etc., may be employed.

Among the remedies, there are two in particular which are especially useful. The first is the persistent and faithful use of *cold*. This simple remedy is fitted most wonderfully to quiet the overactivity of the heart, and is best applied by the temporary or continuous use of ice-bags laid over the chest. The second is *digitalis*, which most decidedly diminishes the frequency of the heart's action, and, since it usually is only required temporarily, is quite in place here. *Tincture of veratrum viride* is a much more uncertain remedy, and, from the sudden and un-

accountable attacks of collapse which sometimes follow its use, is highly objectionable. Tartar emetic, likewise, certainly has the power to diminish the frequency of the heart's contractions, and, as Ackermann[1] has shown, to diminish the arterial pressure; but its action cannot always be depended upon in every case, and, moreover, its use is attended by very disagreeable effects. Together with the use of digitalis, quinine in large doses is often of very great value.

For the treatment of secondary dilatation we must refer to that of the primary disorder; and amongst the appropriate remedies, together with rest, we shall find here also that the use of cold and digitalis are most efficacious in so diminishing the frequency of the heart's action that its labors are lessened. For the acute dilatation which occurs in the course of inflammatory diseases, our object will be simply to maintain the energy of the heart's contractions only through a short period of time; and for this purpose stimulants will be of great assistance, and amongst these I consider tea of special value.

Atrophy of the Heart.

See the literature of heart disease in general, especially *Zehetmayer* and *Bouillaud*. *Albers*, Die Atrophie des Herzens. Casper's Wochenschrift, 1836.—*Hasse*, Anatomische Beschreibung der Krankheiten der Respirations- und Circulationsorgane, 1841.—*Virchow*, Virch. Archiv. Vol. XI. 1851.—*P. Finn*, Ueber die Atrophie des Herzens. Centralblatt für med. Wissenschaften, No. 36, 1868.

Introduction.

By atrophy of the heart we mean only the shrinkage or disappearance of muscular tissue, and not the destruction of the heart substance in the course of other special degenerative processes, of which we shall speak under those special heads.

Accordingly, the amyloid degeneration of Virchow, and the atrophy by sclerosis of Friedreich are here thrown out, even although it must be allowed that a complete separation of these

[1] Virch. Archiv. Vol. XXV.

processes is scarcely possible. It is purely on theoretical grounds that the division is made into—

1. Simple atrophy, with normal size of the cavities and thinning of the walls.

2. Concentric atrophy, with diminution of the cavities, and thinning of the walls; and,

3. Excentric atrophy, with dilatation of the cavities and thinning of the walls; for, granted that simple atrophy may exist for a short time, it must soon pass into simple dilatation, of which we have already spoken.

History.

From the importance which was attached to the heart in olden times, we cannot wonder if, in opposition to the opinion held to-day, its atrophy was held to be the cause of other and serious changes in the whole organism.

Friedreich is in favor of the distinction between excentric atrophy and simple dilatation. But this support is purely arbitrary, and it will be found to be impossible to distinguish between these two conditions; and accordingly we shall occupy ourselves here only with concentric atrophy.

We first find a somewhat elaborate treatise on atrophy in the eighth chapter of Senac's work on the heart; he calls attention to the point that we must not consider every diminution in the volume of the heart to be atrophy. Laënnec considered it a secondary disease, and Bouillaud made the division into simple, excentric, and concentric atrophy, which is very generally retained at the present day.

Etiology.

Atrophy of the heart most frequently occurs—

1. As a congenital diminutiveness of the organ associated with deficient development of the whole vascular system and the genital organs.

2. After long and tedious diseases, in the course of which the

quantity of blood in the body has been considerably diminished. Here it should be considered as a special form of marasmus.

The chief diseases under this head are: Tuberculosis, in which, according to Engel (loc. cit.), the heart loses more than a quarter of its weight in men of from twenty to thirty years of age, and the left ventricle more than the right; diabetes, cancerous cachexia, prolonged purulent discharges from diseased bone, severe and exhausting forms of typhus, dysentery, etc. Here also should be included the cases of atrophy which have been observed after serious attacks of chlorosis (Copland).

3. Where there is interference with expansion of the heart by pressure from without. Here pericardial effusion should be especially mentioned. But we surely should remember that an accompanying disease of the heart substance is often associated with the pressure as a factor in the production of the atrophy. Considerable deposits of fat upon the heart and large mediastinal tumors may have the same effect.

4. It is sometimes occasioned by atheromatous disease of the coronary arteries.

5. Partial atrophy occurs in any portion of the heart where there is a less demand for functional activity than normal.

Thus we usually find an atrophy of the left ventricle where there is insufficiency and stenosis of the mitral valve; for at each systole it is more readily emptied, and with each diastole it receives less blood than in a normal condition. Under hypertrophy we have already spoken of the opposite condition of affairs.

Pathology.

Pathological Anatomy.

The disease may affect the whole organ, or only a single portion, or even only parts of this latter, as the papillary muscles, etc. The subdivisions based on the relations borne by the thickness of the wall to the dimensions of the chamber have been already mentioned in the introduction.

The heart is somewhat flabby,—in some forms, however, firmer in consistency than normal,—brownish-red in color, the subperi-

cardial deposit of fat wanting; the pericardium, which often is cloudy, may be wrinkled, or even œdematous in spots. The coronary arteries, in adapting themselves to the diminished volume of the heart, have become tortuous. Brown atrophy is most frequently found as the result of senile marasmus, in tuberculosis, and cancerous cachexia. On microscopical examination, the muscular fibres are seen to be not only thinned, but also otherwise altered; in the *brown atrophy* there is found a yellow and brown pigment deposited in the muscular fibres, probably derived from the blood-coloring material; under the head of fatty degeneration, we shall speak of the *yellow atrophy*, and of that by sclerosis (Friedreich), as well as of the *amyloid degeneration.*

Symptoms. Diagnosis.

Since there are no symptoms specially characteristic of this disease, it will be impossible to establish a diagnosis with certainty.

A weak, trembling, or even irregular impulse, and feeble heart sounds may occur equally well in a normal heart or in one otherwise diseased. If the impulse is inside its normal position, and at the same time the apex beat is distinct, then a diminished area of dulness over the heart may be referred to atrophy; but if these signs are absent, it might equally well be occasioned by other conditions, in particular by emphysema, which, however, is usually easily recognized. When, as occasionally happens, there is also a considerable effusion in the pericardial sac at the same time, the pericardial dulness will not fall below its normal dimensions, and will thus interfere with the diagnosis. Among the subjective symptoms, palpitation is the most frequent, and seems to indicate the attempt of the atrophied heart to meet the demands upon it. It would not be right to refer the cold extremities from which many patients suffer, or the attacks of dizziness and fainting, to atrophy of the heart. Accordingly, we can only suspect the existence of this condition after some of the diseases we have mentioned, when there is at the same time diminution of the dulness over the heart, feebleness of the impulse, of the pulse, and of the heart sounds.

Course, Termination, Prognosis, and Treatment.

We cannot be at all sure that the atrophy of the heart exercises a great influence on the course of the diseases which have given rise to it; yet it will at all events increase the progress of destruction of tissue already going on.

From the secondary nature of the disease but little can be said about the treatment; it would seem to consist in attacking the primary disorder, urging the avoidance of every over-exertion of the heart, and quieting individual symptoms.

Inflammation of the Heart Substance, Abscess and Aneurism Formation.

Myocarditis.

The works of *Laënnec*, *Andral*, *Bouillaud*, *Rokitansky*, *Lebert*, *Förster*, *Bamberger*, *Trousseau*, *Duchek*, *Stokes*, *Friedreich*, *Dusch*, and others.—*Skoda* und *Klob*, Fälle von ausgebreiteter Schwielenbildung im Herzen. Wiener medic. Wochenschrift, 1856.—*Skrzeczka*, Eigenthümliche cavernöse Entartung d. Muskelsubstanz d. Herzens. Virch. Arch. XI. 181. 1857.—*Virchow*, Ueber die Natur der const. syphil. Affectionen—dessen Archiv. 1858.—*Berthold*, Merkwürdiger Fall eines von der rechten Vorkammer ausgehenden Herzaneurysmas. Teplitz, 1859.—*Dr. R. Demme*, Beiträge zur Anatomie und Diagnostik der Myocarditis. Schweiz. Zeitschr. f. Heilk. I. 79 u. 461.—*E. Wagner*, Fall einer eigenthümlichen Myocarditis. Arch. d. Heilk. No. 1, 1861. *William Jenner*, On congestion of the heart and its local consequences. Med. Chirurg. Transact. XLIII. 1860.—*H. Stein*, Untersuchungen über Myocarditis. Gekr. Preisschr. München, J. J. Leutner, 1861.—*Jasinsky*, Zur Casuistik der Herzaneurysmen. Wien. Medic. Halle 2 (Jhb. CXII. S. 180), 1861.—*C. Fomman*, Parenchymatöse Myocarditis mit lethalem Verlaufe bei Grippe. Prager Vierteljahrschr. I. Bd. 1862.—*P. Da-Venezia*, Sulla vera cardite o miocardite, illustrata con due esemp. Giorn. Venet. di scienz. med. Gennajo, 1862.—*C. A. Wunderlich* und *E. Wagner*, Acute Entzündung des linken Herzvorhofes. Arch. d. Heilk. Heft. 3, 1864.—*Griesinger*, Aneurysma der Ventrikelscheidewand. Archiv der Heilkunde, 1864. — *Jaccoud*, Sur un cas d'aneurysme ventriculo-aortique. Union medic. 1866.—*Virchow*, Die Gummositäten des Herzens. II. 441, 1863–1867 —*Klob*, Zwei Fälle von Myocarditis. Wiener Medic. Wochenblatt, 1865. —*Godineau*, De la pericardite, de l'endo- et myocardite, Presse med. No. 30, 1866.—*Klob*, Zur pathol. Anatomie der Myocarditis. Wien. med. Wochenschrift, No. 14, 1866.—*E. Wagner*, Das Syphilom des Herzens und der Gefässe im

Speciellen. Arch. d. Heilk. No. 24, 1866.—*Oppolzer*, Ueber einige Formen von partieller Myocarditis. Wien. med. Zeitung. No. 1, S. 4, 1867.—*H. Bernheim*, De la myocardie aiguë. Thèse. Strassburg, 4, 1867—*M. Roth*, Ein Fall von Herzabscess. Virch. Arch. Bd. 38, S. 572, 1867.—*E. Kock*, Anevrisme partiel de la point du cœur. Presse med. XIX. No. 9, p. 69, 1867.—*Spencer Watson*, Small partial aneurism at the apex of the left ventricle. Rupture. Med. Times, July, p. 32, 1867.—*N. Pelvet*, Des anevrismes du cœur. Thèse pour le doctorat. Paris, 1868.—*Buckingham*, Boston Med. and Surg. Journ. 1868.—*Morgan*, Gummy tumors in the wall of the left ventricle. Med. Press. and Circular, November 18, 1868.—*Gfroerer*, Phlebitis des linken Armes; Myocarditis. Memor. No. 4, 1869.—*T. L. Walford*, Aneurism of the heart. Brit. Med. Journ. July 3, 1869.—*Henry Arnott*, Aneurism of the left ventricle of the heart with partially ossified walls, winding round the root of the aorta. Transact. of the Path. Soc. XIX. p. 149, 1868.—*Moxon*, Case of abscess of the heart, bursting into the left ventricle. Trans. of Path. Soc. XX. p. 113, 1869.—*Wm. R. Sanders*, Case of heart disease. Partial fibroid degeneration, the result of myocarditis. Edinb. Med. Journ. Feb. 1869.—*R. Fowler*, Fibroid (probably syphilitic) degeneration of the heart. Transact. of Path. Soc. XIX. p. 108, 1868.—*Desnos et Huchard*, Des complications cardiaques dans la variole, et notemment de la myocardite varioleuse. Union méd. 1870.—*P. H. Pye Smith*, Suppuration of the heart. Transact. of Path. Soc. XXI. p. 94, 1870. —*B. Peacock*, True aneurism of the apex of the left ventricle. Transac. of Path. Soc. XXI. p. 118, 1870.—*Simon*, Zur Enstehung des Herzaneurysma. Berl. klin. Wochenschr. No. 45, 1872.—*Edwards Crisp*, Abscess in the left wall of the heart in pyæmia. Transact. of Pathol. Soc. XXIII. No. 12, 1872.

Introduction.

The name carditis, by which in general we formerly understood any inflammation of the heart, without considering individual portions of it, has now been abandoned, and the name myocarditis has been generally accepted, as indicating inflammation of the heart substance, as distinguished from endo- and pericarditis.

We recognize an acute and a chronic form, general and partial, and anatomically, parenchymatous and interstitial inflammation, which last again may be purulent or fibrous in character.

The special seat of this disease in extra-uterine life is the left ventricle, both sides of the heart being attacked, but the left more severely. In intra-uterine life it is just the reverse, for here the inflammation of the heart substance is by far more fre-

quent in the right ventricle, and is often the cause of congenital cardiac diseases. In the auricles this affection is very rare, perhaps somewhat more frequent in the left than in the right. In general myocarditis, however, all portions of the heart may be more or less affected.

History.

The possibility of an inflammation of the heart substance was not recognized by the old physicians; it was first in the Middle Ages that independent observations and reports of such disorders gradually made their appearance. Although we find already in the works of Benivenius, a Florentine physician (1529), the account of an induration in the heart, and in Rota (1555), Massa (1559), Fernelius (1656), narrations of ulcers in the heart, yet Morgagni must be considered the first who observed and wrote in detail on the subject, and he took up inflammations, indurations, ulcers, and rupture of the heart. Senac (1749) pointed out the base of the heart as the most frequent seat of the development of abscesses and ulcerations, and mentioned particularly the existence of diseases of the neighboring organs, and in special of the pericardium as one of the chief causes of these pathological changes. Corvisart (1806), who had the opportunity of making a series of further observations, speaks always of carditis, the existence of which alone he cannot comprehend, but always associated with inflammation of the peri- and endocardium; from him also we have the account of active and passive aneurism. Kreyssig, who proposed the term carditis polyposa, held that among all the tissues of the heart the muscular substance proper was least frequently attacked by inflammation, but suggested that possibly we frequently overlooked the disease; he gave also a series of rules for treatment. Laënnec studied the subject of myocarditis very closely, and readily allowed the existence of the partial form of this disease, but expressed himself very cautiously concerning the general form, and as regards both of them he declared that the diagnosis could only be imperfectly made. Andral devised a very complicated subdivision of the various forms of inflammatory trouble occurring in the heart, without notably advancing the subject.

Hope made the attempt to distinguish between the symptoms of softening, following upon chronic inflammation of the heart substance, and those of a pericarditis. Bouillaud suggests only that myocarditis may possibly exist without endo- and pericarditis. Sobenheim[1] describes fully and plainly the symptoms of myocarditis collected from observations made by various other writers. Hamernik (1844) gave us the first microscopical report of an inflammation of the papillary muscles, and this was soon followed by another microscopical observation of Dubini.[2] Latham (1846) and Craigie[3] described and strongly insisted on the occurrence of a purulent form of inflammation in the myocardium, and the latter describes it very beautifully. According to Dietrich (1852), myocarditis occurs more frequently in the septum and near the base of the heart than at the apex; he described also the true stenosis of the heart as a result of myocarditis in the right ventricle. Stokes believes that inflammation of the muscular substance, without endo- and pericarditis, is a very rare disease, and that its pathological anatomy and diagnosis are still very obscure. Skoda insists on the impossibility of making a diagnosis, as also does Loebel. Rokitansky gives a complete description of its results in relation to aneurism of the heart. Substantially new points of view in regard to certain changes in the heart substance have been gained by the publication of Virchow's views concerning parenchymatous inflammation.

Acute Myocarditis.

Etiology.

Primary disease of the heart substance is very rare. There are only a few cases described where no possible cause could be ascertained. Injuries (such as a fall or blow upon the chest) are comparatively so frequent, while myocarditis is so rare, that it is open to doubt whether some predisposition did not exist before the injury, or possibly some other cause for the disease

[1] Praktische Diagnostik der inneren Krankheiten u. s. w. Berlin, 1837.

[2] Gazetta di Milano, 1844.

[3] Edinburgh Journal, 1848.

were not present. Amongst the diseased conditions likely to produce myocarditis, we should particularly mention those generally termed rheumatic. The secondary form appears most frequently after endo- and pericarditis, and either as a parenchymatous or as an interstitial inflammation; more rarely as a sequel to disease of neighboring organs, as simple pneumonias and pleurisies.

In the course of ulcerative diseases of the lung, dislodged thrombi may pass through the pulmonary veins into the left heart, and from here reach the coronary arteries, where they form emboli, and set up a circumscribed purulent myocarditis.

In the course of acute articular rheumatism, and the acute infective diseases—amongst these especially typhus—myocarditis will be observed, especially in the parenchymatous form, considerably diffused, and more rarely in the interstitial and purulent form; whereas in pyæmia, glanders, and puerperal processes, the latter more frequently appears.

The occurrence of an acute affection (of the heart) in syphilis (Demme) certainly must be very rare.

According to the compilations by Loebel,[1] Dittrich,[2] and Stein (l. c.), the disease is more common among men than among women.

The data concerning the age of patients affected with myocarditis do not altogether agree, but it would appear that it is most frequent between the twentieth and fortieth years. In the introduction we have already spoken of its occurrence in intrauterine life.

Pathology.

General Description of the Disease.

From the rarity of an idiopathic form of the disease, and from the circumstance that many of the symptoms of myocarditis accompany other diseases of the heart, or of the respiratory organs, or, indeed, of organs further removed, such as the brain, it is

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[1] Dissert. de aneurism. cord. part. Vind. 1840.

[2] Ueber Herzmuskelentzündung. Prag. Vierteljahrsch. XXVII. 1852.

very difficult, or we might better say scarcely possible, to give a delineation of the disease which shall be characteristic. This statement certainly holds as regards the possibility of distinguishing between the parenchymatous and the interstitial form of inflammation.

Often enough the course of the disease is entirely latent; occasionally it is obscured by evidences of peri- or endocarditis existing at the same time, and may indeed run its course with exactly the same symptoms as pericarditis. In other cases, especially in childhood, the disease may simulate an acute affection of the brain. Very frequently the first evidence of its existence is a rupture of the heart, or an attack of apoplexy (embolic). Only a few cases have a certain similarity, and from these we might adduce the following:

A man, previously in good health, begins to complain of great weakness and lassitude, of pain in the region of the heart, or of a feeling of oppression, which may suddenly increase to the greatest agony. Soon, with this, are associated attacks of dyspnœa and irregular action of the heart; then, fainting or delirium, or finally lethargy; occasionally symptoms also of a different character appear, which will be described below.

Exactly the same description may serve for pericarditis, and particularly for endocarditis ulcerosa.

Pathological Anatomy.

The description by Virchow of a form of inflammation, known as parenchymatous, first let in some light on this disease. When the process is extensive, there are often striking changes in the appearance of the heart, even externally. The pericardium is cloudy, sprinkled over with ecchymotic spots, covered here and there with a thin layer of exudation; the subpericardial fat has disappeared; the heart substance, at first dark in color, or bright red, especially in cases of a hemorrhagic character, is swollen, glistens as if with fat, for it is studded throughout with more or less numerous spots of albuminous exudation (Stein), and is accordingly friable. If the affected spot lies nearer to the cavity of the heart, then the endocardium manifests

its share in the disease by cloudiness and injection of the vessels.

In microscopical examination we observe various stages in the process ; the muscular fibre is swollen, paler, its transverse striæ less distinct or entirely wanting ; in other spots the contents are already cloudy and filled with granular matter. In those places where the changes have advanced still further, the muscular fibres are seen to be broken into fragments, and we observe among them occasional blood corpuscles, collections of pigment matter, and oil globules, which after the addition of acetic acid become more distinct from the clearing up of the granular contents of the bundles of muscular fibre. In still higher grades the oil globules are so abundant (*i. e.*, the fatty degeneration has so far advanced) that they have arranged themselves in rows, parallel to the primitive fibres. Last of all we find, when the process is farthest advanced, a brownish pulpy spot of softening.

This form of inflammation may affect either single small spots in the myocardium or extend over larger regions. Moreover, in the circumscribed form of the disease, there are particular portions of the heart which show a certain predisposition to the disorder. Thus, in the left ventricle the apex will specially suffer ; at the base, the posterior wall is the spot affected, and usually first the aortic valves, next the septum, and more especially the base of it, then the papillary muscles ; and in the right ventricle, the trabeculæ carneæ.

If an endo- or pericarditis extends into the subjacent myocardium, this latter will be found more or less extensively involved in the pathological processes described above ; this is especially the case in the severe forms.

Interstitial Inflammation, in its acute course, is identical with the purulent. Between the breaking down muscular fibres in the intervening areolar tissue, we find pus following more or less extensively the course of this tissue, as a so-called diffuse purulent inflammation, or the pus may be collected in spots of greater or less size, which in great numbers are often disseminated through the heart-substance, from the size of the head of a pin to that of a bean, and occasionally lead to the formation of

large excavations, constituting abscess of the heart, of which we shall speak more particularly later.

It is not yet settled from which of the histological elements the pus is formed; according to O. Weber,[1] it is formed from the nuclei of muscle cells; according to Demme (loc. cit.) it is derived from the nuclei of the cells in the arterial walls.

In those cases where the disease is a sequel of pyæmia, the numerous small accumulations certainly may have purulent contents, contrary to the view of Rindfleisch, who assumes that they are filled with vibriones. I saw in a girl, twenty-two years of age, pyæmia following upon a paronychia of the left hand, in whom, besides the peripheral muscles and the diaphragm, the heart substance also was found studded throughout with numberless little spots, from the size of a millet-seed to that of a hemp-seed, whose contents were shown to be entirely purulent.[2]

Ordinarily, in cases which occur by metastasis, after emboli, we find a commencing formation of pus in the middle of a hemorrhagic spot.

Both forms of inflammation may occur together. Thus, between bundles of muscular fibre, pathologically changed by parenchymatous inflammation, the interstitial connective tissue will be infiltrated with pus. By the growth of the interstitial connective tissue, both forms may lead to a fibroid degeneration of the heart substance, concerning which we shall speak under the head of chronic myocarditis.

Another termination is in a slow form of fatty degeneration, of which we shall speak at length under that head.

Abscess of the Heart.—Even though a spot of softening, following upon a parenchymatous inflammation, when superficially situated, may lead to an ulcer, yet the true abscess of the heart has its origin in a suppurative myocarditis.

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[1] Virch. Archiv. Bd. XV. Zur Entwickelungsgeschichte des Eiters.

[2] On the other hand, E. Wagner (Archiv. für Heilkunde. Bd. II.), in examining microscopically numerous roundish or ovoid spots, from the size of a millet-seed to that of a coffee-bean, bluish-red in color, with semi-fluid contents, which were situated in the substance of the left ventricle, in a man seventy-four years of age, found a completely homogeneous mucoid substance, which was filled with a net-work of connective tissue corpuscles of medium size. Again, Klob (Bericht der Rudolfsstiftung, 1868) found pus in abscesses of the heart in a young girl of thirteen years.

Where there is a considerable accumulation of pus, a collection of this kind may occasionally be recognized externally as a soft sac, with flabby walls, over which the pericardium, or, if situated deeper, near one of the cavities, the endocardium may be seen ecchymosed, suffused with blood, and bulging forward. Such a collection of pus may either empty outward into the pericardial sac, followed by suppurative pericarditis, or, after the purulent destruction of the endocardium, or more frequently, after tearing through it and the remaining muscular fibres overlying it, it may discharge itself into the cavity of the heart. If, in this latter case, the blood pushes its way into the cavity so formed, it may lead to a more extensive tearing of the muscular fibres which bound it, and then to a rupture of the heart, tearing-off of one of the papillary muscles, or, finally, to the formation of a so-called acute aneurism, which again may itself occasion a complete perforation of the heart.

If the abscess has formed in the upper portion of the septum ventriculorum, occasionally it may here have a special importance, and this depends upon whereabouts it breaks through. This may take place either into one ventricular cavity or simultaneously into both, whereby a temporary, or if it heals without closing the sinus, a permanent communication between the ventricles will be established; or else it leads to the undermining and tearing-off of a leaf of the semi-lunar valve, or in the same manner, after perforation into the right ventricle, to the same accident with the pulmonary valves, or one point of the tricuspid; finally, the pus may open for itself a way upward, and thus give rise to a perforation into the right auricle, or indeed to a communication between the latter and the left ventricle.

The sequelæ of an abscess may be of various kinds: 1, by perforation outward it may cause purulent pericarditis; 2, by perforation inwards, it may cause emboli (usually in the course of the greater circulation and with the character of a malignant thrombus), and metastatic inflammatory processes in the spleen, kidney, brain, etc., or, by perforation into the right ventricle, similar processes in the lung; 3, it may terminate in a temporary or permanent healing by absorption of the contents and formation of a scar, either thin and slight or firm and fibroid in

character, with considerable growth of interstitial connective tissue; or else it ends in encapsulation of the purulent accumulation by connective tissue, with thickening, and finally calcification of its contents.[1]

Aside from perforation and abscess, the results of myocarditis are easily observed in the muscular tissue of the heart. The change in its consistency must lead to a dilatation, which will be more or less considerable according to the extent to which its substance has been invaded by the inflammatory process, and the shape of the heart will thereby be changed in the manner we have spoken of above under the head of dilatation. An apparent thickening of the wall of the heart is not caused by hypertrophy, but only by an inflammatory swelling of the heart substance.

Blood Clots.—The diminished functional activity of the heart, and the more sluggish movement of the blood arising therefrom, as also the formation of aneurism, just alluded to, will further occasion a tendency to the formation of clots in the heart, which will be still more increased from the fact that over the spots where myocarditis has existed we find irregularities on the surface of the endocardium. Then follow thrombi and globular vegetations in the interior of the heart, of which we shall speak later.

Partial myocarditis, situated on the trabeculæ or papillary muscles, may also lead to functional disturbances, and, when the latter are affected, to insufficiency of the corresponding valve, etc.

The pathological changes in other organs are occasioned by the stasis of blood: congestion of the lung, bronchial catarrh, later, œdema of the lungs, hemorrhagic infarctions, more or less extensive œdema of the meninges and brain, great hyperæmia and swelling of the liver and spleen, and in the latter often hemorrhagic infarctions.

Not unfrequently, in cases of parenchymatous myocarditis, the

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[1] Roth (Virch. Archiv. Bd. 38) found in the body of a man, twenty-three years old, who had suffered from mitral stenosis, the cavity of a large abscess, which might contain an ounce of pus, whose walls were lined with thickened purulent matter, and occasional calcareous fragments.

same affection is found in the kidney (Buhl,[1] Stein [l. c.], Demme [l. c.]). But since the same disease, viz., a parenchymatous degeneration, is found occasionally in the liver cells, it may be often very difficult to decide into which of these organs the primary disease has been transplanted, and in which it has originated.

The usually thin condition of the dark blood may be the cause of the ecchymotic spots which we observe on the serous and mucous membranes, and also, associated with venous stasis, of the dropsy which affects the subcutaneous areolar tissue and many of the cavities of the body.

In cases of secondary myocarditis, we naturally find in other organs pathological changes of various kinds, which are due to the primary disorder.

Symptoms.

We must remember that the varied symptoms which we observe clinically depend upon a diminution of the functional capacity of the heart, and a consequent weakening of blood-pressure in the aortic system, over-distention of the pulmonary circulation, and of the veins of the body.

Physical signs.—The impulse of the heart is usually weaker than natural, occasionally normal, and only rarely increased. If dilatation has already taken place, just in correspondence with the amount of expansion will the apex beat vary from its normal position.

By *percussion*, in advanced stages of this process, when it is extensive, we find an increased dulness over the heart, corresponding to the amount of dilatation, in the manner we have explained before.

Auscultation may yield only negative results, *i. e.*, the sounds may be normal, but occasionally very striking changes may be perceived. Amongst these, above all, we may observe a weakening of the heart sounds, especially the first, or even complete absence of that sound, which may be readily explained by the diminished mechanical force of the ventricle and the incomplete

[1] Zeitschrift f. ration. Med. No. VIII. 32.

tension of the papillary muscles. Occasionally murmurs are developed, and of various kinds. A systolic murmur in the left ventricle in partial myocarditis may undoubtedly be caused by incapacity of the papillary muscle, in other cases by the sudden development of insufficiency in the valvular apparatus from the tearing-off of one leaf of a valve. Again, in other cases we may notice an unusually loud blowing murmur after perforation of the septum ventriculorum.

Finally, in those cases where we find clots upon the endocardium, or on the ragged walls of an abscess, or only irregularities on the endocardial surface, we must suppose that the murmur was caused by friction against these, for there is no condition present to explain its occurrence on the theory of the compression of a stream of blood; at all events, we may in this case also assume a profoundly diseased condition of the papillary muscle to be the cause, although we may not be able to prove it.

By various writers, and especially by Stein, we have had our attention called to the fact, that when myocarditis supervenes upon a faulty condition of the valves, the murmurs which were present become weaker, and indeed may disappear altogether. This may be explained by the diminution of power in the heart, as Stein assumed the disappearance of a diastolic murmur at the apex to be caused by a fatty degeneration of the muscular tissue of the left auricle, which theory was established by a microscopical examination. We must remember, however, that heart murmurs may disappear under other circumstances, so that this phenomenon is not characteristic of myocarditis.

Just at the present time I have the opportunity of observing a patient whose case bears directly on this point; for the last seven years I have been accustomed to show him to my hearers as a distinctly-marked classic case of mitral insufficiency and stenosis. On exhibiting him again, a year since, I was greatly astonished, and hardly could believe the testimony of my ears, on finding the murmurs absent and replaced by dull heart sounds. Since that time the patient's condition has certainly grown no worse, but rather better, so that here the theory of a fatty degeneration cannot be entertained.

The *pulse* is always frequent, often irregular, small or unequal, naturally so, since the force of the contractions of the heart is variable.

The accounts of the amount of febrile action accompanying this disease are so varied, that we cannot beforehand lay down any fixed laws concerning it. From my own observations, the bodily temperature is only moderately increased. When metastases occur, which, moreover, are frequently announced by a chill, the temperature may rise very considerably.

Wunderlich and Wagner[1] observed a continued high temperature, sometimes reaching 107° F. (41.6° C.), in a rare case of myocarditis of the left auricle, in a girl eighteen years of age, previously entirely healthy, whose attack began with a chill.

The surface of the body is usually pale, occasionally also cyanotic, which may be explained by the changes in the lung and also by the venous stasis, which is particularly evident in the veins of the neck. Other changes in the skin, such as purpuric spots and pustules, are occasioned, doubtless, by secondary pathological processes arising from emboli in the vessels.

The brain symptoms are very interesting; these are dizziness, headache, delirium, fainting turns, convulsions, and lethargy, and in the myocarditis of childhood, especially, these are the most prominent symptoms. They may be fully explained by the interference with the afflux of blood to the central organs of the nervous system, and especially by the insufficient renewal of the circulating fluid.

In the *respiratory organs* we observe bronchitis, more or less considerable, an increase in this trouble even to œdema of the lungs, expectoration of a bloody and purulent character, and hydrothorax. The attacks of labored respiration, which may sometimes increase to a most intense gasping for breath, are not caused by these changes alone, but by those in the heart substance as well.

The symptoms of disorder of the digestive apparatus are very inconstant. The vomiting, which is observed chiefly in childhood, has its cause, apparently, not in the stomach, but in the brain.

Icterus we should expect to find only in the later stages of the disease, and then probably it may be referred to the pyæmic condition.

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[1] Archiv f. Heilkunde. V. Jahrg. 1864.

The blood-stases which we have mentioned lead to a more or less extensive *dropsy*.

As the kidneys are almost always sympathetically affected, we have, as a result, change in the character of the urine. The scanty urine contains albumen, frequently also tube casts and blood corpuscles, varying with the extent to which the kidney is involved.

The patients complain of great weakness and heaviness, often of a decided pain in the region of the heart, or at least of a feeling of tightness in the chest, which, at times, is spasmodically increased, and also of shortness of breath.

Diagnosis.

In consideration of the fact that myocarditis is most frequently an accompaniment or sequel of peri- or endocarditis, or is developed in the course of some other severe diseases, and, moreover, that the character of the idiopathic form, as above described, is so unusually varied, it is evident that it will be almost impossible to make a diagnosis.

The diagnosis of idiopathic myocarditis may possibly be made out by a careful observation of the sickness in all its various phases, after excluding other diseases, and by the most careful attention to the course of the disease. For this purpose certain symptoms are of special importance, such as the moderate amount of fever which attends the commencement of the disease, while there is at the same time great languor, pain in the region of the heart or tightness in the chest, great feeling of anxiety, shortness of breath, a very frequent pulse, usually small and very irregular, with an accompanying feebleness in the impulse, the development of bronchial catarrh with lung symptoms and gradually increasing in severity, and finally anasarca and urine characteristic of Bright's disease.[1]

If, moreover, we find the signs of a sudden development of insufficiency in some of the valves, particularly the mitral, or were

[1] We ought to lay special weight upon this symptom, if the views of Stein concerning the relation between parenchymatous myocarditis and Bright's disease are confirmed.

two different sets of valves to become involved in succession (*e. g.*, insufficiency of the aortic valves to be followed by insufficiency of the pulmonary or the tricuspid), from the frequency with which myocarditis attacks the septum, the probability of our making an accurate diagnosis would be increased, as it likewise would by the sudden appearance, after the above-mentioned group of symptoms, of apoplexy (by embolism) or of emboli in other organs. Here we can scarcely avoid confounding it with endocarditis, and the distinction will be very difficult and almost impossible.

In regard to the diagnosis of inflammation of the heart substance when it is complicated with pericarditis, we should lay special stress upon the fact that we now know very well that many of the symptoms, which have been held hitherto to be characteristic of the latter disease, are due to the myocarditis. For example, if in a case of pericarditis, with only a small quantity of fluid exudation, the heart's impulse, hitherto forcible, has now become weak, we can hardly err in assuming the existence of a myocarditis complicating the original disorder.

Course, Duration, Complications, and Termination.

The course is usually very rapid, from three to eight days. Cases of shorter duration, where the whole process was run through within a few hours (Simonet,[1] Oppolzer[2]) and those of long duration (Demme, loc. cit., 43 days) certainly may be considered rare. Amongst the sequelæ the sudden development of valvular insufficiency has already been mentioned. Occasionally, after the healing of an abscess in the immediate vicinity of a valve, or in one of the papillary muscles, we may have a distortion of the valvular apparatus to which it pertains, causing a lasting insufficiency. The trabeculæ carneæ may, in the same manner, remain as thin tendinous strings, which are sometimes stretched across the cavity of the left ventricle. I have before me a preparation of the heart of a patient in whom I diagnosti-

[1] In Friedreich, p. 148.

[2] Wiener allgemeine med. Zeitung. 1866. No. 50.

cated the above condition from the high-pitched, whistling, systolic murmur which I recognized during life.

Further on we shall speak of fibroid growths and acute aneurism of the heart. We have already alluded to the deposit of emboli in various organs, and also to the pericarditis, and, more rarely, the endocarditis after the bursting of abscesses. Hypertrophy and dilatation of the ventricles were considered under the appropriate heads.

A fatal termination occurs rarely by general collapse or simple paralysis of the heart, more frequently through complications, as œdema of the lungs, emboli in vital organs, especially in the brain, occasionally very suddenly by rupture of the heart.

Prognosis.

This follows easily upon what we have already said; undoubtedly both in parenchymatous and in purulent inflammation we may have a relative cure, and such hearts at the autopsy show partial spots of fatty degeneration, and also more or less branching fibroid tissue, which evidently has existed for a long time. But even in these most favorable cases the heart will be in no condition to meet any unusual demands upon it. In consideration, therefore, of the acuteness and rapidity of the disease, and the great number of complications and sequelæ which may arise, the prognosis must be unfavorable.

Treatment.

The treatment is most uncertain and difficult. The chief indication would naturally be, as in inflammation of any other organ, to put the patient in a most complete state of rest, and energetically to employ all such remedies as may tend to that result (such as cold, digitalis, etc.). But such measures would here be dangerous, for they would only tend to increase the great source of danger in this disease, viz., weakness of the heart. But since, as shown above, it is quite possible that our diagnosis is incorrect, it is evident that we can only treat symptoms as they arise. The moderate employment of cold, entire rest from

physical exertion of any kind, in order not to increase the labor of the heart, together with narcotics to quiet severe pain or restlessness, will be found to be the most useful. When the weakness is excessive, besides quinine and caffeine, we must resort to stimulants, such as wine and tea.

Chronic Myocarditis.

Etiology.

Chronic myocarditis, in so far as it is not a sequel of the acute form, consists in a growth of the connective tissue between the muscular fibres, with, at the same time, a more or less considerable atrophy of these latter. It is intimately allied to cirrhosis of the liver, kidney, and lung.

Common as it is to find myocarditis, certainly in its lesser degrees, excited and developed by peri- and endocarditis, it is equally rare to find the idiopathic form of the disease ; and yet we must not forget the development, we sometimes see, of fibroid tissues, which may be very extensively distributed throughout the body, and for which we can find no cause.

In intra-uterine life, however, it is by no means very rare, and the right ventricle particularly is the portion affected, leading to serious congenital deficiencies. In adults it seems to be brought about by the same causes which give rise to endo- and pericarditis, and, above all, by the rheumatic tendency. Many cases are found in the literature of the subject which were of traumatic origin, *e. g.*, from blows or falls, or some fragment or foreign body remaining behind after an injury. Moreover, at the present time we cannot doubt that this disease is developed as a sequel of syphilis. As regards sex, this affection should be found more frequently in men than in women. In the matter of age, excluding fœtal life, we find that mature life, and especially after thirty years, is the period which furnishes us with most of our observations, and here almost exclusively developed in the left ventricle.

Pathology.

Description of the Disease.

In many respects this will be the same as in acute myocarditis, or else it will simulate other chronic affections of the heart. In regard to its results, the nature of the disease is such that it will necessarily injure the functional capacity of the heart by insufficient contraction, and so lead to diminished blood-pressure in the aorta and stasis in the venous system. Accordingly we meet with all those symptoms which we have described under acute myocarditis, and which will be mentioned in treating of fatty degeneration.

Pathological Anatomy.

The process consists in a growth of the interstitial connective tissue, by means of which, between the bundles of muscular fibre there are developed, now single delicate, whitish bands, and now larger and firmer bundles, now far-reaching branches of fibroid tissue, and now indurations, which finally entirely replace the muscular tissue in a given region, and which lie close under the endocardium on the one hand, or the pericardium on the other. The pathological process may have its seat in the wall of the heart, in the left ventricle at the apex, particularly, but less frequently in the septum and the base of the heart, thus differing from the acute form ; in the right ventricle, however, it is less rare to find it just at this place, with the relations we have above described. In the interior of the organ it occurs on the papillary muscles, and usually close to their upper or free extremity, and especially in the left ventricle, and leads to those changes which we have described above.

We have already spoken of the shape of these indurations in general. We may only allude to the fact that within these formations single portions of muscular tissue are sometimes enclosed, while in other cases it has entirely vanished. This is particularly the case with those indurations which are situated in the wall of the heart, involving a large section of the

ventricle. The process is of special importance when it surrounds a whole ventricular wall in a circular form, as it occurs chiefly in the conus arteriosus, and usually in that of the right ventricle. It develops here the only form of the disease which, analogous to other scars, leads to contraction, and so gives rise to a narrowing of the affected portion, which is the true *stenosis of the heart* (Dittrich). In all other places the indurated portion yields to the pressure of the blood, and the result is a dilatation of the affected part, forming the partial chronic aneurism of the heart, of which we shall speak more at length hereafter.

We must, however, allude to the fact that, while it is generally believed that indurations of the heart arise in the manner we have above described, Rindfleisch (loc. cit.) considers it to be an extension of chronic endocarditis into the subendocardial and interstitial connective tissue. He arrives at this conclusion chiefly from the fact that we frequently find the appearance of the endocardium changed (actually a notable portion overlying the induration is thickened), and the myocardium not wholly destroyed, and in such cases it is always the outer layers and not the inner which are intact. Luken[1] has in part adopted this view; according to him, we meet with these indurations ordinarily under the endo- or pericardium, and only very rarely in the middle layers of the heart substance.

Syphilitic myocarditis demands special consideration; it may occur in two forms—

1. In the form just described, of simple fibrous myocarditis, which only, from its occurring simultaneously with constitutional syphilis, may be attributed to the latter as a cause; and

2. In the form of special pathological changes, namely, the gummy tumors imbedded in the heart substance. Ricord[2] was the first to call attention to this disease, and Virchow has established its existence beyond a doubt. They are yellowish-white, cheesy collections, appearing either as roundish tumors or in branching masses, which remind one of the same disease in the liver.

[1] Die pathol. Neubildungen des Myocard. Henle's Zeitschrift für ration. Med. XXIII. Bd., 3. R.

[2] Clinique iconographique. Plate 29.

They are situated in the intermuscular connective tissue, and like the other form cause atrophy of the muscular fibres; they project, usually, beyond the greatly thickened endo- or pericardium into the cavity of the heart, or else outward into the pericardial sac. They consist of firm, fibrous bands of connective tissue externally, and within of a mass of cells with a single nucleus, which easily undergo fatty degeneration. Sometimes many such tumors are found in a single heart.

Diagnosis.

It would only be possible to diagnosticate chronic myocarditis when we could with certainty exclude all other diseases of the heart, which cause the same or similar symptoms, and especially fatty degeneration; this we could scarcely succeed in doing. Naturally there will be no means of distinguishing between the insufficiency of a valve occasioned by fibroid induration of a papillary muscle, and that caused by deformity of a portion of the valve itself. If the symptoms which we believe to be characteristic were to be developed as a sequel of constitutional syphilis, we might possibly make the diagnosis of its probable existence.

Course, Complications, Termination, Prognosis, and Treatment.

Quite frequently the disease runs a course which is entirely latent, especially when only a limited portion of the heart is thus degenerated. Occasionly the patients exhibit for years together the above-mentioned symptoms of a diminution of the functional capacity of the heart.

The hypertrophy which takes place in the rest of the heart, and which serves to compensate for the faulty action of the diseased portion, may in no small degree enable the patient to bear the disease so well as we find it to be borne in some cases. After a long sickness, the end may come either directly through feebleness of the heart, the compensatory hypertrophy having gradually given place to a passive dilatation, or by secondary diseases, especially in the lung, or finally by rupture of the heart.

The *prognosis* must always be unfavorable.

The *treatment*, from a theoretical point of view, can only confine itself to lightening as far as possible the demands upon the heart. Accordingly, the patient must avoid all severe physical exertions and excitement of all kinds. For the rest, our treatment must be directed only to the alleviation of symptoms.

Partial Aneurism of the Heart.

In opposition to Corvisart's nomenclature, aneurysma cordis (activum, simplex or passivum), under which were comprehended the different forms of dilatation of a single cavity of the heart, we now give the name aneurysma cordis partiale to a partial dilatation of a cavity, and under this name we recognize two different forms—the acute and the chronic.

Acute, Partial Aneurism of the Heart.

This occurs in two ways. If the endocardium, in consequence of an inflammatory process, has become yielding, the result is, that under the pressure of the blood it is torn, and the blood makes its way through the rent thus formed, and gradually undermines the heart substance; or else the endocardium, overlying a spot affected by myocarditis, is torn across, or destroyed by purulent inflammation; in this manner a cavity is formed into which the blood forces its way, and thereby again contributes to its enlargement. In both cases there is thus formed in the wall of the heart a spot which is more yielding than the surrounding parts, and which gradually bulges outward more and more, and so leads to the formation of a partial aneurism. Although the cavity is filled with fibrinous coagula, yet the walls never acquire thereby a sufficient power of resistance, and after continuous destruction of the heart substance it ultimately ends in rupture.

The seat of this aneurism is almost exclusively the left ventricle, the anterior wall of this, especially close to the apex, and the upper portion of the septum ventriculorum, starting from which, a loosening of the leaf of the aortic valve which is nearest

to it, very readily takes place. While Dittrich supposed that the walls of this cavity may undergo fibrous induration, and thus the duration of the aneurism may be prolonged, Rokitansky has recorded no observation of such a process. Duchek, however, has seen two such cases.

The *symptoms* cannot generally be distinguished from those of myocarditis.

On account of the unusually rapid course of the disease, and the almost constant occurrence of rupture, the *prognosis* is absolutely unfavorable, and all the more so from the fact that before the fatal accident occurs, the patient may suffer from embolism, produced by detached fragments of clots or of the disorganized heart substance.

Chronic Aneurism of the Heart.

Aside from its possible occurrence as a sequel of acute aneurism of the heart, which is generally doubted, this form follows upon an induration (fibroid) of the heart substance. Contrary to the behavior of connective-tissue growths in other parts of the body, indurated tissue in the heart is capable of no contraction, and is not in a condition to offer resistance to the pressure of the blood. The more extensive this process is, the less will be the power of resistance, and particularly when it embraces not only the heart substance, but at the same time the endo- and pericardium as well.

The extension of the aneurismal cavity into the substance of the heart may be very gradual, and thus its shape may be compared to that of a fusiform aneurism in the arteries, or, as also occurs in the arteries, the aneurismal sac may be connected with the cavity of the organ only by a narrow opening.

Here again it is particularly the left ventricle and the apex of the same, where, aside from the septum, this form of disease occurs. Pelvet (loc. cit.), in a collection of eighty-seven cases of aneurism of the heart, found the apex to be the seat in eighty-five, and, in the literature of the subject, only three cases are found where the right heart was affected. What importance the pars membranacea of the septum has in the development of this

disease, Rokitansky [1] has lately shown by citing six cases which bear upon this point. In two of them the aneurismal sacs were broken through, all projected from the left into the cavity of the right ventricle (Rokitansky knows only one case where the converse was true, and this occurred in intra-uterine life), and all had occurred after endocarditis which could be recognized. In the literature numerous cases are found where the disease was developed in the auricles, and then the whole wall was involved. There can be no doubt that syphilitic myocarditis may give rise to the formation of chronic aneurism in the heart.

The size of the aneurism is very varied. It may attain the dimensions of a man's fist.[2] The larger the sac, the thinner, as a rule, are the walls; and in these we not infrequently find places where calcification has taken place, or else the whole sac may have the appearance of a bony case or box. The sac is filled with various layers of fibrinous coagula of older and later formation.

Usually only one such aneurism is present, but in exceptional cases several may be found (even as many as four), and then they lie close against each other, separated only by a low wall of partition. According to Löbel and Thurnam [3] the male sex seems to show a greater predisposition to this disease than the female.

As regards age, it is usually those of advanced age who are the subjects of the disease, and its occurrence among young persons must be considered as one of the rarities.

The rest of the substance of the heart may remain in its normal condition, or may become hypertrophied, which certainly would be a more favorable condition.

The *symptoms* are in no way to be distinguished from those

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[1] Die Defekte der Scheidewände des Herzens. Wien. Braumüller. 1875.

[2] The largest is that described by Berthold (Tiplitz, 1859). It occupied the whole right auricle, distending it to the size of a man's head, was in contact with the chest wall throughout, and had absorbed a large portion of the sternum between the levels of the second and fourth ribs, and likewise the portion of rib adjoining was only covered by the skin, which itself was so destroyed by the pressure that the blood trickled through in some places.

[3] On Aneurism of the Heart, with Cases. Med. Chir. Transact. 1838. XXI. 2 Ser. No. 3.

of chronic myocarditis. Skoda has observed a bulging of the intercostal space overlying the seat of the aneurism. Theoretically we can easily see that this should be so, but up to the present time no similar case has been recorded. Where the aneurism is of considerable size and lies in contact with the wall of the chest, there may be a corresponding dulness in the percussion note, which cannot be distinguished, however, from that caused by an enlarged heart, or, as in the case of Berthold, by an aortic aneurism. It is very improbable that even in the sessile aneurism any murmurs are produced; and should any such be heard, they must have their origin in other alterations in the heart.

From what we have already said, it will be seen that the question of *diagnosis* can hardly be considered. As regards *prognosis*, experience shows us that when there is sufficient thickness of the walls, the aneurism may exist a long time, and that sessile aneurisms may possibly terminate in cure by the deposit of fibrinous coagula in their interior. As a rule, however, they end in rupture at a period more or less remote, and which we cannot determine. Moreover, the fibrinous coagula may give rise to emboli in various organs of the body.

Fat Metamorphosis, Fatty Heart, Colloid Degeneration.

See the literature of heart diseases in general.—*v. Plazer*, Zur Casuistik der Fett degeneration des Herzfleisches. Aus der med. Klinik des Prof. Rigler in Graz. Spitals-Zeitung. No. 21. 1860.—*E. Wagner*, Fall von eigenthümlicher oder primärer Erweichung des Herzfleisches. Arch. d. Heilkunde, I. 2. 1860.—*Levin*, Studien über Phosphorvergiftung. Virchow's Arch. Bd. XXI. 1861.—*Page*, Hæmatopericardium and complete *fatty degeneration* of the heart, sudden death. Lancet, 1863.—*Fritz, Ranvier et Verliac*, De la Stéatose dans l'empoissonement par le phosphore. Archiv gén. de Méd. Juillet, 1863.—Prof. *E. Wagner*, Die Fettmetamorphose des Herzfleisches in Beziehung zu deren ursächlichen Krankheiten. Verhandlung der med. Gesellschaft zu Leipzig, 1864.—*Sick*, Ueber acute Fettdegeneration innerer Organe bei Pocken. Würtemb. med. Corresp.-Blatt, 1865.—*Piotrowsky*, De la dégénescence graisseuse du cœur. Thèse de Paris, 1865.—*Leyden* und *Munk*, Die acute Phosphorvergiftung. Virch. Arch. Bd. XXXIV. 1865.—*Saikowsky*, Ueber Fettmetamorphose der Organe nach innerlichen Gebrauch von Arsenik, Antimon und Phosphorpräparaten. Virch. Arch. Bd. XXXIV. 1865.—*Klebs*, Zur pathol. Anatom. der Phosphorvergiftung.

Virchow's Arch. Bd. XXXV. 1865.—*Senftleben*, Ueber die Erscheinungen und den anatom. Befund bei Phosphorvergiftung. Virchow's Archiv. Bd. XXXVI. 1866.—*Nothnagel*, Die fettige Degeneration der Organe bei Aether- und Chloroformvergiftung. Berliner klin. Wochenschrift, 1866.—*Blachez*, De la Stéatose. Thèse de concours. Paris, 1866.—*Kastan*, De degeneratione cordis adiposa. Dissertat. Berolini. 1867.—*Krylow*, Ueber fettige Degeneration der Herzmuskulatur. Virchow's Arch. Bd. XLIV. Heft 4, 1868.—*Blachez*, Gaz. des hôp. 10. Soc. de med. de Paris, 1868.—*Russdorf*, Deutsche Klinik, 1868.—*J. F. Payne*, Two cases of sudden death from affection of the heart examined in the post-mortem theatre. Brit. Med. Journ. Feb. 5. 1870.—*Clarke*, A case of angina pectoris with advanced fatty degeneration of the heart. St. Georg. Hosp. Reports, IV. 1869.—*Luithlen*, Beitrag zur Casuistik der Fettdegeneration der Herzmuskulatur. Betz' Memorab. No. 11.—*Goodwin Ralph*, Fatty degeneration of the heart. Angina pectoris. Philad. Med. and Surg. Rep. Jan. 28, 1871.—*S. O. Wasastjerna*, Fall af fettjärta. Finska. läk. sallsk. handl. Bd. XIII. S. 51. 1871.—*Traube*, Berliner klinische Wochenschrift. 1871. No. 26.—*H. Curschmann*, Zur Lehre vom Fettherz. Virch. Arch. 1873.—*Ponfick*, Ueber Fettherz. Berlin. klin. Wochenschrift, Nos. 1 u. 2, 1873.

Introduction.

Under the title, *fatty heart*, are comprised several conditions quite different from each other—

1. Sometimes there is an increase in the accumulated subpericardial fat at the apex, in the ventricular furrows along the course of the vessels, and at the base around the junction of the auricles and ventricles, and at the origin of both the great vessels. This condition of itself is unimportant and only attains any importance when, by the great accumulation of fat, the muscular substance of the heart has fallen away.

2. The true *fatty degeneration*, where the muscular fibres are themselves changed into fat, the real *fatty heart*. But even this condition will have no great weight classically, unless it attacks large portions of the heart; in this class belongs the yellow atrophy which is a very extensive fat metamorphosis of the heart substance.

3. The result of parenchymatous myocarditis. It will be very difficult and often almost impossible to decide whether in a given case we have to deal with this form, or with the above-mentioned idiopathic form of the disease.

History.

The ancients were familiar with the increase in the normal fat about the heart. Hints concerning fatty degeneration of the muscular tissue are found in Lancisi and Morgagni, but Laënnec was the first to distinguish accurately between true fatty degeneration and fatty heart from external deposit of fat, which by pressure or by interference with nutrition causes an atrophy of the muscular substance.

In later times great credit has been gained in Germany by Rokitansky, and in England, where there has always been a predilection for investigating this subject, by Williams, Paget, Ormerod, Quain, and Stokes, in advancing our knowledge of this form of disease.

Finally, quite recently, scientific men have endeavored to show, some of them, that the importance of this pathological condition has been exaggerated (Duchek), and others that it has been underrated and not sufficiently examined ; of the latter party we should mention especially E. Wagner, who has given the subject a most careful investigation.

Etiology.

The deposit of newly-formed fat upon the heart is very frequently found, and especially in drinkers, together with a simultaneous deposit of fat throughout the whole system, and in this fact do we find the explanation of it. It is quite different, however, when fat is found only around the heart in individuals already deteriorated by various other diseases. Here the disease is purely local, and only in such cases could the theory of an occurrence of metastasis of fat find any support.

If we wish to bring the causes of fatty degeneration under one single head, we may say that, in general, we find it wherever, as a result of a disturbance in the interchange of elements in the body, there is an insufficient supply of oxygen. Such conditions are very frequently present in the body :

1. Associated with a series of *general disturbances of nutri-*

tion, as, after various severe chronic diseases, protracted suppuration, great loss of blood, in the course of tubercular and cancerous cachexia, long-continued and severe forms of syphilis, but particularly in profound anæmia (Ponfick,[1] Biermer,[2] Gusserow[3]).

Biermer relates several cases of patients of middle age in whom he met with fatty degeneration of the heart as a sequel of anæmia. Gusserow found a fatty degeneration of the heart substance as the only recognizable pathological condition in the bodies of five patients, who suffered from most profound anæmia during pregnancy, and in all of whom a fatal end occurred before arriving at term. Ponfick, who, considering the alteration which takes place in the blood, has declared the existence of a special anæmic form of fatty degeneration of the heart, characterizes it by diminution in the quantity of blood, great poverty in fibrine, and a considerable decrease in the number of red corpuscles; it is this condition of the blood which renders it so liable to cause dropsical effusions.

The experiments of Tschudnowsky[4] and Perl,[5] by venesection of dogs, gave a fatty degeneration of the heart as the result. Perl made small venesections at short intervals and also larger ones at longer intervals. In six cases out of seven in the last series, there was found at the autopsy a distinct and well-marked fatty metamorphosis furthest advanced in the papillary muscles of the left side, next in those of the right side, after this followed in succession the walls of the left ventricle, then those of the left auricle and the right ventricle, and, least of all, those of the right auricle.

Although it had in none of them reached the advanced stage in which we find it in man, yet the perverted nutrition of the heart substance was the only discoverable cause of death. In the cases of the first series a perfectly normal condition of the heart was found throughout.

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[1] Berlin. klin. Wochenschr. 1873. Nos. 1 and 2.

[2] Correspondenzblatt für schweizer. Aerzte. 1872. No. 1.

[3] Archiv für Gynäkolog. II. Bd. 1871.

[4] Botkin's Archiv. 2. Bd. 1866–9.

[5] Virchow's Archiv. LIX. Bd. 1 Heft.

An *acute fatty degeneration* occurs after severe puerperal processes, typhus and typhoid fever, the exanthemata, and acute yellow atrophy of the liver. Among the acute infective diseases the heart is most frequently and seriously involved in remittent fever (Ponfick, l. c.). There is no reason here why we should not consider the disease of the heart as an acute parenchymatous inflammation, resulting, without doubt, from the general disturbance of nutrition.

Under the influence of *certain poisonous substances* there arises a very considerable fatty degeneration of the heart. Among these we may specially mention poisoning with phosphorus, with mineral and vegetable acids, and chronic alcohol intoxication. Since in this form the disease is always associated with similar changes in the liver, the kidneys, the gastric follicles in the stomach, as well as in other muscles in the body, it is evident that the cause must be a general one, and hence existing in the blood. It is highly probable that a portion of those cases of fatty degeneration of the heart which accompany Bright's disease should be placed in this category, for certain constituents of the urine which act as poisons remain in the blood and exercise a deleterious influence on nutrition.

Wherever there is a fatty degeneration, not only of the heart, but of most of the muscles of the body as well, it is evident there must be some general disturbance of nutrition, for which, however, it is often difficult to discover the true cause.

It is wrong to consider fatty degeneration of the heart a physiological retrograde metamorphosis attendant upon old age, though it is true that this disease occurs frequently in old people; for this, however, we may find abundant explanation in the insufficient supply of oxygen caused by simultaneous disease in other organs.

2. There is among the causes a series of *disturbances of nutrition which act locally upon the heart.* Here *pericarditis* deserves especial mention, as a sequel of which fatty degeneration of the heart is found with unusual frequency. Virchow[1] thinks they must belong to that form of pericarditis which runs

[1] Archiv. XIII. Bd.

its course with high fever, and with an unusually accelerated action of the heart, even early in the disease. Wagner (l. c.) out of 2,000 autopsies, gives 35 cases of severe pericarditis, and in 17 of these he found, on microscopical examination, a moderate fatty degeneration of the heart substance; according to my own observations, I should place the proportion much higher.

Very often a fatty degeneration follows upon valvular lesions. In 2,000 autopsies, Wagner found 75 valvular lesions, and of these 28 were the subjects of fatty degeneration, either moderate or severe.

It is possible that the cause of this may be sought in the increased labor of the heart, which in general must be considered a cause for the development of fatty degeneration. It seems more probable to me, however, that the downward impulse is given by other disturbances of nutrition, doubtless of a local nature.

Disturbances of the circulation in the aortic system, diseases of its coats, and contraction of its calibre, lead to partial fatty degeneration of the left ventricle; and so likewise do disturbances in the pulmonary circulation, especially those caused by extensive emphysema, pleuritic exudation, chronic tuberculosis, and curvature of the spine, lead to partial fatty degeneration of the right ventricle. It may occur in both together as a consecutive change after antecedent hypertrophy.[1]

In fatty degeneration of the heart great stress has hitherto been laid upon the condition of the coronary arteries, and many excellent observations have been recorded of cases where, with a more or less complete obliteration, endarteritis, atheromatous process, and fatty degeneration of the inner coat of these vessels, there was a fatty metamorphosis of the heart. Since the changes under consideration are more frequently found in the left coronary artery, or at least more distinctly marked there, this may possibly explain in part the more frequent disease of the

[1] Traube (Berl. klin. Wochenschr. 1871. No. 26), amongst other explanations, offers this: That in consequence of the dilatation, there is a decrease by compression in the size of the capillaries which pass through the distended walls, by means of which there is a diminution in the number of red corpuscles which pass by a given point in the muscular fibres in a given time.

left ventricle[1] As regards the etiology of fatty heart, however, we must remember that this condition of the coronary artery is by no means so common an accompaniment of the disease as is generally supposed.

3. Finally, I believe that we must learn to consider a great number of the cases of fatty degeneration as only the result of a *chronic parenchymatous myocarditis*. This view finds support from the fact that the disease often exists simultaneously with the remains of other inflammatory processes in the heart, degenerations of the valves, thickening of the endocardium, adhesions of the heart to the pericardium, etc.

The male sex seems to have a greater predisposition to this form of the disease than the female ; but in the anæmic form the female sex preponderates (Ponfick).

Pathology.

General Description of the Disease.

We can only speak of a general description of the disease in that particular form which we are accustomed to call the chronic, idiopathic, the form which the English physicians most frequently describe ; it is the description of a chronic weakness of the heart. In very anæmic patients this will vary to some extent by the increased prominence of anæmic symptoms. In other cases it assumes the character of an undefined affection of the heart, and this is especially the case when the disease is confined to single portions, at all events when limited to the papillary muscles. Again, the disease may run its course without any signs, and our first knowledge of its existence is acquired by the rupture of the heart, for which its inroads have paved the way.

Pathological Anatomy.

In the first form the fat is at first found collected in the above-mentioned portions of the subpericardial connective tissue,

[1] Clarke (St. George's Hospital Reports. IV. Canstatt, 1870) describes a case of advanced fatty degeneration of the heart in which there was found an almost complete obliteration of the left coronary artery by atheromatous disease.

but often embracing the whole heart in such a manner that it is completely enveloped in a capsule of fat. The fat is not only collected on the surface of the organ, but it pushes its way in masses of greater or less dimensions, following the course of the fibres of connective tissue in between the muscular bundles of the heart substance.

Thus it may cause a considerable atrophy of the heart substance, which is seen only in thin pale strips and layers.

In the second form the process takes place in the primitive bundles of muscular fibres themselves. Their contents become cloudy, the transverse striæ disappear; at first we find single drops of fat, later they are formed in rows, and finally we see them in such quantity that the whole sarcolemma is filled with them, and the drops run together and form large globules. The heart substance is thereby strikingly changed in appearance, becomes pale and yellowish in color, and its texture is flabby and easily torn.

This condition, which occurs with far greater frequency in the left ventricle, is found in small scattered spots in the wall of the heart, the trabeculæ carneæ, the papillary muscles, or the septum, or is spread over larger regions following the course of the endo- or pericardium, or, finally, invades the greater part of that section of the heart which is affected, more rarely the right ventricle and the auricles, but occasionally the whole heart at once.

The disease may be either acute (and here usually the diffuse form prevails) or chronic, and doubtless most of the cases of partial degeneration belong under this latter head.

A case of cavernous degeneration of the muscular tissue of the heart, of very great interest, is described by Skrzecka.[1] On cutting through the walls of the heart of a man twenty-one years of age, who died suddenly after a fall, he found an appearance resembling a bath-sponge, caused by cavities, from the size of a pin's head to that of a small bean, lying close against each other, and separated by a muscular tissue of a yellowish brown color. The process affected especially the left ventricle and the septum ventriculorum. The muscular tissue was everywhere fatty. Skrzecka believes the holes to have been formed by the destruction by fatty degeneration of scattered portions of the tissue, as opposed to

[1] Virch. Archiv. Bd. XI.

Virchow,[1] who twice observed a similar condition in a new-born male child and in an eight months' fœtus, and thinks in all the cases the cause was a cavernous myoma.

The assertion of Weber,[2] that hearts affected with fatty degeneration contain no more fat which may be extracted by ether than normal hearts, is contradicted by Boettcher[3] and Valentiner.[4] The former found in the normal heart from 8.9 to 12.9 per cent. and in fatty heart tissue from 10 to 16.7 per cent. Valentiner found in the normal heart from 1.8 to 2.3 per cent., and in the heart tissue of the left ventricle of seven drinkers, which otherwise did not appear to be affected by fatty degeneration, he found from 2.3 to 2.9 per cent. of fat.

In conclusion, I will mention two other forms of degeneration of the heart tissue.

In the condition to which Friedreich has given the name "*sklerosirende Atrophie*," the heart substance is firmer, shows on section a waxy appearance, the primitive bundles have partially lost their transverse striæ, or so far that they have the appearance of colorless cylinders.

The *amyloid degeneration*, which Virchow has observed, differs from the form we have just described only by its reaction with iodine.

Symptoms.

These vary very much according to the extent of the process and the rapidity with which the condition is developed. Even though later and more accurate observations have shown that a partial fatty degeneration of the heart is very frequently observed in autopsies where it was not recognized during life, this was not the fault of the observers, but only shows that the process ran its course without giving rise to symptoms. E. Wagner has called special attention to the fact that the few remaining healthy muscular fibres are able to sustain the heart in the performance of its functions, and especially is this the case under the influence of increased innervation. Very fre-

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[1] Gesamm. Abhandl. 1856.

[2] Virch. Archiv. Bd. XII. 817.

[3] Virch. Archiv. Bd. XII.

[4] Archiv für wissensch. Heilkunde. 1869. Bd. V.

quently the disease ran its course unperceived, at most giving occasion now and then to unimportant disturbances before the occurrence of rupture of the heart. Where the disease is distinctly formed and runs generally a chronic course, it gives rise to a series of symptoms which we will now describe.

Physical signs.—When the process is extensive, the impulse is weakened, and the pulse correspondingly small. Not unfrequently a very considerable diminution in the frequency of the contractions of the heart was observed, it being reduced to between thirty and forty beats or even to fifteen in the minute (Stokes). This diminished frequency is, however, not characteristic; for, on the one hand, it is found also in other diseases, and, on the other, even in fatty degeneration we often find it normal in frequency or even accelerated. *An increased area of dulness over the heart* is observed in all those cases where fatty degeneration has attacked a heart already changed in its dimensions.

There may be also a dilatation of the cavities as a sequel of the fatty degeneration, and thus a corresponding increase in dulness may arise.

Auscultation may either give us perfectly normal sounds or perhaps weaker and duller than natural. Allusion is often made to a systolic murmur, which is believed to be occasioned by insufficient contraction of the papillary muscles, caused by their fatty degeneration. Though we may grant the possibility of such a thing theoretically, yet I think that fatty degeneration of the papillary muscles is one of the rarest of the causes of mitral insufficiency.

The first result of diminished power of contraction in the heart will be a condition of stasis, and, in fatty degeneration of the left as well as the right ventricle, this will be made manifest in the system of the superior and inferior venæ cavæ, by swelling and pulsation of the veins of the neck, cyanosis, and slight dropsy; the skin is not always cyanotic, however, but of a dirty yellowish color, and pale, especially in those cases which are developed with the symptoms of anæmia.

Not unfrequently the patients complain of a feeling of distress and constriction in the chest, and often of pain extending down the arms, thus simulating angina pectoris. As regards the pecu-

liar form of respiration which Stokes has described as characteristic of this disease, and which has been named after him "Stokes' Respiratory Phenomenon," we know now that this singular and distinctly marked kind of respiration occurs in many diseases. I have seen it many times most remarkably exhibited in Bright's disease, without any pathological changes which could be recognized in the heart.

On the part of the *brain*, we have a series of symptoms which are described under the title of pseudo-apoplectic, and which probably depend rather upon a condition of anæmia than on one of hyperæmia. They are manifested partly by slight attacks of dizziness, partly by fainting turns, or even, finally, by actual coma. Ordinarily they have no serious results, except that the patient may show signs of loss of memory for some little time after recovery, and they very rarely have a fatal termination.

The relations of hemorrhage and fatty metamorphosis of the heart to each other have not yet been fully explained. In this disease hemorrhages certainly occur without our being able to recognize any fatty degeneration in the vessels. But more frequently it would appear that repeated hemorrhages are at last followed by fatty degeneration of the heart.

Williams has called attention to the appearance of an arcus senilis in the cornea where fatty degeneration of the heart exists. Even if it be true that fatty metamorphosis in the eye often occurs simultaneously with that in the heart, yet this is by no means a constant accompaniment.

Acute fatty metamorphosis of the heart is obscured in its symptoms by those of the primary disease, and its existence is not made manifest.

Diagnosis.

Inasmuch as all the symptoms occur in other diseases of the heart as well, it will be very difficult to make an accurate diagnosis, and we can only do so when we can exclude these other diseases; this is particularly true of chronic myocarditis and fibroid degeneration of the heart. First of all, we must make up our mind that the deposit of epicardial fat, which leads to atrophy

of the muscular tissue, cannot be distinguished from true fatty degeneration.

In diagnosis, special stress should be laid upon the following points. The disease occurs especially in advanced age, in corpulent people. If we observe the symptoms already described of weakness of the heart, feeble impulse and pulse, dilatation of the cavities, and attacks of fainting, and other diseases can be excluded, we may suspect the probable existence of a fatty heart. The presence of arcus senilis under such circumstances may give us a confirmation of our diagnosis, but of itself the sign is not of critical importance. According to the statements of Stein, the simultaneous existence of Bright's disease would favor the diagnosis of parenchymatous myocarditis, which also attacks cachectic rather than corpulent people. In that acute form of fatty degeneration of the heart which occurs as a sequel of profound anæmia, the diagnosis will be easier from the existence of this latter disease; but the difficult question here will be whether the anæmia or the fatty heart is to be considered the more prominent affection.

The question of diagnosis is different, however, when the fatty degeneration has led to the formation of valvular lesions, or diseases in the respiratory organs. For, if it appears that the signs of compensatory action gradually become feebler, if the interference with the circulation increases, or at all events the existing murmurs lose their intensity, and cyanosis is steadily developed, then we may, with considerable certainty, declare the existence of this form of degeneration of the heart substance.

Course, Duration, Termination.

Partial fatty degeneration may, as we have learned from so many autopsies, be of little significance as regards the condition of the whole system. Acute fatty degeneration, and that which arises from poisons, usually cause a rapidly fatal termination. Chronic deposit of fat and fatty degeneration of the heart substance may, under favorable circumstances, exist for many years, since fortunately a small portion of the muscular fibres,

which yet remain sound, suffice to maintain the functional capacity of the organ, until finally the circulation in vital organs is not sufficiently kept up, or, the process still extending, with gradual failing of the patient, and more and more frequent attacks of fainting and dyspnœa, death occurs by paralysis of the heart or rupture, of which we shall speak more at length further on.

Prognosis.

This will be unfavorable in all forms of the disease except the partial degeneration, and even in this it will be very bad if the process has attacked a papillary muscle. In the acute form, from the severity of the whole process, we could hardly anticipate other than an unfavorable termination, and only in the toxic form could we imagine that, if the extent of the poisonous influence has been moderate, possibly restoration might follow.

The prognosis of the chronic form will also be unfavorable, for we should get too deep in the doctrine of final causes if, for example, we should argue as follows: Hypertrophy of the left ventricle serves as a compensation for the insufficiency of the aortic valves. Now if the hypertrophy exceeds the necessary limits, might not a moderate amount of fatty degeneration of the muscular tissue serve to compensate for this excessive hypertrophy? What influence could we then find to keep this fatty metamorphosis within bounds? Quite the opposite of this; we must always consider it as a retrograde and necessarily deleterious process.

Treatment.

In individuals who have a tendency to excessive formation of fat, we must endeavor to put a stop to the accumulation, to prevent its deposit in great quantity about the heart. This will be best accomplished by a careful regulation of the habits and method of life, and a selection of appropriate food, avoiding, as far as possible, the use of articles containing starch, fats, and sugar, and also beer.

Where a considerable deposit of fat already exists, diluent salts

will be particularly indicated, especially the alkaline carbonates, sulphates of soda and magnesia, and mineral waters which contain these salts. In the anæmic form our medication will be particularly directed to increasing the quantity and quality of the blood.

In the other forms, the treatment must necessarily be purely symptomatic, designed, above all, to strengthen the system in general, and make the functional activity of the heart sufficient for its demands.

The remedies necessary here are those which are well known, and which we have frequently enumerated. So also, the separate prominent and dangerous symptoms, such as fainting and distress for breath, will demand their appropriate remedies. The treatment described by Stokes, and which was discovered by a patient who suffered from this disease, viz., bending the head downward between the knees, has since that time been generally employed in such attacks, at least all over England, and with good results.

Spontaneous Rupture of the Heart.

The works of *Morgagni*, *Cruveilhier*, *Rokitansky*, *Förster*, *Bamberger*, *Duchek*, *Friedreich*, *Dusch*, and others.

Ollivier, Dict. de Med. 2. edit. tom. VIII. Paris, 1834.—*Chrastina*, Beitrag zur Cardiorhexis. Oesterr. Zeitschrift für praktische Heilkunde, 1857.—*Elléaume*, Essai sur les ruptures du cœur. Thèse, Paris, 1857.—*Berthold*, Merkwürdiger Fall eines von der rechten Vorkammer ausgehenden Herzaneurysmas. Teplitz, 1859.—*Giusseppe Orsolato*, Sulle rotture spontanee del cuore. Annali universali di Medic. Milano Gennajo, 1860.—*Hamilton*, Rupture of the heart. The Lancet, 28. Jan. 1860.—*Malmsten*, Fall von Ruptura cordis, Hygiea, 21. Bd. p. 619. 1861.—*Arlidge*, Case of rupture of the heart. Beale's Archives of Med. No. IX.—*Neuffer*, Plötzlicher Tod an Herzzerreissung. Würtemberg. Corresp.-Blatt XXXI. 24. 1861.—*Edgar Lowe*, Un cas de rupture du cœur. Gaz. hebd. No. 39. 1862.—*Malahard*, De la perforation interventriculaire du cœur. Thèse, Strasbourg, 1863.—*Böttger*, Ueber spontane Rupturen des Herzens. Arch der Heilkunke, IV. 502. 1863—*Soulier*, Sur an cas de rupture du cœur. Gaz. med de Paris, No. 9. 1863.—*Müller zu Calw*, Ruptura cordis. Würtemberg. Corresp.-Bl. XXXIV. 28. 1864.—*Höring*, Ruptur des Septum cordis. Würtemberger Corresp.-Bl. 10. Bd. I. 1864—*Cocheteux*, Ramollissement rouge du cœur, rupture, mort instantanée. Gaz. des hôp. No. 71. 1864.—

Larcher, Perforation du ventricule gauche du cœur. Union Med. No. 49. 1864. —*Dunlop*, On rupture of the heart. Edin. Med. Journ. M. 1866, p. 998.—*Thos. J. Yarrow*, Rupture of heart. Americ. Journ. Med. Sci. p. 421. 1867.—*Cuq, François Marie Paul*, De la rupture des parois du cœur. Thèse de Strassbourg. 1867.—*E. Tachard*, Apoplexie interstitielle du cœur. Rupture de cet organe. Mort. Autopsie. Gaz. des hôp. No. 104. p. 411. 1867.—*Turel*, Rupture progressive du cœur (ventricule gauche). Gaz. med. de Lyon. No. 4, p. 87. 1867.—*Shattuck*, Ruptured heart. Boston Med. and Surg. Journ. May 9. p. 284. 1867. *Moxon*, Rupture in the anterior wall of the left ventricle. Transact. of the Pathol. Soc. XVII. p. 70. 1866.—*J. S. Ramskill*, ibidem, p. 49, Fatty heart—death by rupture of the heart. 1866.—*W. B. Lewis*, Fatty heart and subsequent rupture. New York Med. Record, II. No. 42, p. 412. 1867.—*Magnan et Bouchereau*, Infarctes multiples avec ramollissement dans plusieurs organes, rupture de la paroi antérieure du ventricle gauche. Gaz. med. de Paris, No. 12. 1868.—*Lionville*, Rupture externe du cœur. Gaz. med. de Paris, No. 50. 1868.—*Buckingham*, Boston Med. and Surg. Journ. 1868.—*Little*, Spontaneous rupture of the heart. Dublin Quart. Journ. Nov. 1868.—*Nobiling*, Spontane Ruptur des Herzens.—Bayer. ärztl. Intellig.-Blatt. No. 24. 1869.—*George May*, Case of rupture of the heart. Brit. Med. Journ. July 3, 1869.—*Hooper*, Rupture of the heart. Transact. of the Path. Soc. of London. XIX. p. 186. 1868.—*Leon Marcq*, Mort rapide par rupture spontanée du cœur. Presse méd. belge, No. 12. 1869. *Biffi*, Arch. ital. delle mal. nervose. 1869.—*J. de Bary*, Fall von Herzruptur. Arch. f. klin. Med. VII. S. 152. 1870.—*Gregoric*, Ueber einen Fall von Herzruptur. Memorabil. No. 1870.—*Dickinson*, Rupture of the chordæ tendineæ of mitral valve. Transact. of Pathol Soc. XX. p. 150. 1869.—*Ch. Kelly*, Rupture of the chordæ tendineæ of the mitral valve. Ibid. p. 153. 1869.—*Lund*, Ruptura cordis. Norsk Magaz. for Laegevidsk. Bd. 23. Forhandl. p. 103. 1870. *Lund og Jacob Heiberg*, Ruptura cordis. Ibid. Bd. 24. Forh. p. 28. 1870.—*Sherman*, New York. Med. Rec. 1871.—*Meyer*, St. Louis Med. and Surg. Journ. 1871. —*Reginald Thompson*, On distrain of the heart. St. Georg. Hospit. Rep. V. p. 119. 1870.—*Alfred Wiltshire*, A case of spontaneous rupture of the heart in two places. Transact. of Pathol. Soc. XXI. p. 97. 1870.—*M. Védié*, Rupture du cœur. Gaz. des hôpit. No. 37. 1871.—*Barth*, De la rupture spontanée du cœur. Arch. général de méd., mars, 1871. p. 548.—*Laboulbène et Labarraque*, De ruptures prétendues spontanées du cœur. Gaz. med. de Paris, No. 35. 1872.—*Lowe*, Case of rupture of the heart. Lancet, Octob. 1872.—*Wynn Westcott*, Rupture of the heart. Brit. Med. Journ. May, 1872.—*Hughes*, Case of rupture of the heart. Lancet, July 13, 1872.—*N. P. Dandridge*, Aneurism and rupture of the heart. Philadelph. Med. and Surg. Rep. Vol. XXVIII. May 3, 1873.—*C. Mettenheimer*, Verkalkung im Herzfleisch. Memorabil. No. 4. 1873.—*R. Amory*, Rupture of the right auricle of the heart from sudden compression of the thorax. Boston Med. and Surg. Journ. p. 577. 1873.—*Le Pier Arist.*, Etudes sur quelques cas de ruptures dites spontanées du cœur. Paris, 1873.—*J. Higham Hill*, Case of dissecting rupture of the heart. Brit. Med. Journ. Apr. 12, 1873.

Introduction.

Under the head of wounds of the heart, we shall speak of that form of rupture which follows upon severe shock, a fall, jars, blows with spent balls, and the like. Here we shall speak only of spontaneous ruptures.

History.

We are indebted to Harvey for the first observation of a rupture of the heart. Morgagni wrote at length concerning it, and gave expression to the opinion that it could only occur where the muscular tissue was diseased. It is singular that he himself came to his end by this very fatal accident.

Our first knowledge of a rupture of the chordæ tendineæ is gained from Corvisart. In later days, by a great number of observations, the cause of this always rare occurrence has been considerably cleared up.

Etiology.

We can now lay down the rule with certainty, that spontaneous rupture does not occur in a heart whose muscular texture is sound, and in all those cases where the opposite was maintained the investigation could not have been made with sufficient care and accuracy. There is always a diminution in the cohesion of the muscular elements occasioned by pathological changes. Increased pressure within the heart will not suffice by itself to cause rupture, but when associated with a disease of the muscular tissue, it certainly may do so. Among such diseases fatty degeneration and breaking down of muscular tissue should be specially mentioned. According to Quain,[1] out of sixty-two cases of rupture this disease was found twenty-five times. According to Barth,[2] in twenty-four cases it was found nineteen times.

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[1] On Fatty Diseases of the Heart. London, 1851.

[2] Archives Gén. de Médecine. Feb. et Mars. 1871.

The next most frequent cause is *the pathological condition found in acute and chronic myocarditis*, and here it may occur either during the original inflammatory process, or later when it has resulted in abscess or fibroid induration. When abscess is formed there may be a breaking through of the inner muscular layer, and the force of the blood current streaming in will cause a rupture of the outer wall as well, or the abscess may open either inward or outward and the remaining muscular wall may be too feeble to sustain the blood pressure, and finally give way. A fibroid induration may rupture as such or only after prolonged resistance and the final formation of a partial aneurism of the heart. In nineteen cases of aneurism of the heart Monneret[1] found three ruptures.

Disease of the coronary arteries is not unfrequently a cause of rupture. One of these, enlarged by aneurismal dilatation or become brittle by atheromatous degeneration, will sometimes be the starting-point of a gap in the muscular tissue, which continues until ultimately perforation is established. An embolus, impacted in the coronary artery, may set up softening in its neighborhood, which shall ultimately lead to rupture of the heart wall.

New growths, *gummy tumors*, and *echinococci* may occasion rupture by the degeneration which they cause in the muscular tissue in their neighborhood.

Theoretically, we can easily imagine that narrowing of the calibre of one of the great arteries at its origin or further on in its course, either congenital or caused by pressure from without (by aneurism, tumors, and the like), must lead to a very great increase in blood pressure in the corresponding ventricle. But this ventricle is so constructed that it can endure this increased pressure for a long time, and finally by hypertrophy may be rendered still further competent for its work. Rupture is so rare under circumstances like these, that if it occur we must assume a simultaneous disease of the heart substance to be the chief cause of its occurrence.

In addition to the causes above mentioned, there is usually

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[1] Compend. Vol. II.

some accidental circumstance which is the immediate cause of rupture; such are anger, fright, a full meal, severe bodily exertions—among these an epileptic attack (Tissot), straining at stool (George II., king of England, died in this manner [Nicholls][1]), a soldier by straining during copulation (Morgagni). Sometimes the rupture takes place when the body is entirely at rest, and many cases are related where this occurred during sleep. According to Devergue (in the essay by Elléaume), in every forty cases of sudden deaths there is one by rupture of the heart. According to Aran, in two hundred and two cases of sudden death, following upon pathological changes in the circulatory apparatus, thirty-three occurred by rupture of the heart.

As regards sex, it is very much more common among men than among women; in the collection of cases by Elléaume, for example, out of sixty-one, thirty-seven were males and twenty-four females. Only in the collection of Barth are the females in excess; out of twenty-four cases, seventeen were females and seven males.

As regards the time of life, the occurrence of this accident is immensely more frequent in old age—that is, after the sixtieth year.

Pathology.

Pathological Anatomy.

As regards the seat of the rupture, this is generally considered to be most frequently in the left ventricle, and close to the apex on the anterior wall, or extending towards the septum. Next in order of frequency comes the right ventricle, then the right auricle, and, most rarely, the left auricle. In the collection of Elléaume (loc. cit.), in fifty-five cases this occurred—

43 times in the left ventricle.
7 " " " right ventricle.
3 " " " right auricle.
2 " " " left auricle.

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[1] Philos. Trans., Vol. 52, p. 256.

According to the collection by Ollivier, the rupture occurred in forty-seven cases—

34 times in the left ventricle.
8 " " " right ventricle.
3 " " " right auricle.
2 " " " left auricle.

Concerning the frequency of its occurrence in the papillary muscles, no accurate figures are given.

Usually there is only one rent to be found, but cases are related where two and more, and even as many as five distinct ruptures, were recognized in a single ventricle.

The shape of a rupture is simply that of a fissure, which ordinarily follows the course of the muscular fibres, and is usually nicked and irregular on its edges, and as the direction of the muscular fibres varies in the different layers, it often has a tortuous course in passing through to the cavity of the heart, so that the inner opening does not correspond exactly with the outer either in position or in shape. Moreover, one or the other of the openings of the canal, or its whole tract (which also may be forked and branching), is filled with blood coagula. As it is asserted by some authors that the outer opening is the larger, and by others just the reverse, evidently both forms occur.

The fissure does not always go through the entire wall, but may extend more or less outward into the muscular tissue, and thus we have incomplete ruptures.

The size of the opening is very various ; it may reach a half centimetre or at most one or two centimetres. Cases are recorded, however, where the fissure extended from the apex to the base of the heart (Becker,[1] Beer,[2] Dubreuil,[3] Worbe [4]).

In the vicinity of the fissure, the heart substance is found to be in that condition which led to the rupture, and containing more or less extravasated blood. In the pericardial sac we find an effusion of blood, more or less considerable according to the size of the opening. Doubtless the rupture occurs at the moment

[1] Med. Zeitschr. d. Ver. f. Heilk. in Preussen, No. 44, 1841.

[2] Oest. med. Wochenschr. No. 26. 1842.

[3] Journ. de Montpellier, 1842.

[4] In Günsburg.

of systole, and at its commencement, when the pressure within the heart is at its greatest. This at least we must assume theoretically.

Certainly the rupture often takes place all at once and very suddenly, but there can be no doubt that the breaking through may occur gradually, and at more or less remote periods of time, since the pathological change as well as the cohesion of muscular fibres in the several layers is varied; this follows naturally from the protracted course of the disease.

Symptoms.

The symptoms are varied, according to the length of time which the rupture takes for its accomplishment. Often death occurs so quickly that the patient does not utter a sound, or sometimes gives a short cry, and sometimes indicates that the catastrophe which has overtaken him is occasioned by something wrong in the breast or the heart. They fall to the ground pallid, breaking out in a cold sweat, with a pulse gradually failing in power, soon lose consciousness, and after a few slight convulsions die. Again, on the other hand, many cases are recorded where this fatal condition lasted hours and days—even eleven days (Barth, loc. cit.). In such cases the rupture may begin with pain or a feeling of oppression in the chest, causing great anxiety to the patient, the pain extending across the breast and down the left arm. Nausea, vomiting, fainting turns, or stupor may follow, and death may occur after this has lasted some time, or the patient may gradually come to himself, and then exhibit the symptoms of an affection of the heart, ordinarily with feeble impulse and pulse, and more rarely with an excited action of the organ. Such attacks as these may be repeated with more and more frequency, increasing anxiety of the patient, and greater weakness, until finally in one of these, or else suddenly, he passes away.

This protracted death doubtless occurs from the fact that, on the one hand, the breaking down of the various layers takes place gradually, and, on the other, that coagula may temporarily obstruct the opening.

The whole character of this occurrence will be changed when the rupture follows upon an already existing disease of the heart.

With regard to the signs on the part of the heart itself, in addition to what has already been said concerning the impulse and the pulse, we need only mention that effusion of blood into the pericardial sac may lead to increased dulness on percussion.

The question, how does death occur? we can only answer, as we do in wounds of the heart: sometimes by the suddenly-developed anæmia of the brain, sometimes by interference with the contractions of the heart by extravasated blood (in the pericardium), and in other cases doubtless by nervous shock.

Diagnosis.

The symptoms which accompany the fatal termination by rupture differ in nothing from those seen in other forms of sudden death. In the case of protracted death we could only make out the diagnosis when, besides the symptoms of internal hemorrhage already described, the increased dulness over the heart from effused blood could be made out by percussion. How rarely will this be the case?

The diagnosis of rupture of single portions within the heart will be quite as difficult as the ordinary form.

Prognosis.

If we consider that in many cases, from the resistance offered by the muscular fibres yet intact, the rupture may be long delayed, and that, moreover, the fissure is frequently found to be filled with clots, we must at least grant the possibility of a cure.

Yet the observations of Rostan,[1] who believed that the scars found upon the heart are due to ruptures which have healed, are not by any means all in accord; this we can easily explain

[1] Mémoires sur les ruptures du cœur. Mémoir. nouvelles de méd. 1870, Juillet.

when we reflect on the etiology of rupture. Heart tissues suffering from fatty degeneration will show little tendency to healing.

Treatment.

From what we have already said, it will be seen that there is no opportunity to lay down any satisfactory plan of treatment, aside from the fact that the physician is rarely able to reach the patient in time to see the catastrophe, and that only a purely symptomatic treatment can be employed in those cases where the end is protracted.

Here our treatment must vary between giving the patient the most perfect rest, on the one hand, and sustaining and stimulating the heart's action, on the other. Accordingly cold, digitalis, and morphine will be proper for the one, and tea, wine, or other stimulants for the other purpose.

Wounds of the Heart, Traumatic Rupture, Foreign Bodies.

See the literature of the diseases of the heart in general.—*Cruveilhier*, Traité d'anatomie pathol. gén. I. p. 201 (Needle in the Heart), 1852.—*Hernoux*, Gaz. hebdom. No. 3, 1857.—*Flügel*, Abreissung des Herzens durch Druck auf die Brust. Aerztlich. Intelligenzblatt, No. 26, 1859.—*Tüfferd*, L'union medic. No. 40. Penetrating wound of the heart by stabbing with a knife. 1860.—*Mühlig*, Gaz. med. d'Orient. IV. Sept. 1860.—*G. B. Balch*, Case of gunshot wound in which a leaden bullet remained twenty years in the walls of the heart. Amer. Jour. of Med. Science. Gaz. hebdom. No. 32, Juli, 1861.—*Brugnoli*, Presse med. belge. No. 31, 1862.—*Rupprecht*, Geheilte Stichwunde d. Herzens. Spitalztg. No. 12, 1862.—*Podraczki*, Allgem. med. Zeitung, No. 16, 1863.—*Thompson*, Med. Times and Gaz. Nov. 1863.—Haematopericardium, the result of laceration of the apex of the heart from injury. Lancet, 1863.—*Kussmaul*, Wanderung eines verschluckten Dornes ins Herz und ein freies Concrement im Herzbeutel. Würzburg. med. Zeitschrift. V. 1, 1864.—*Morel-Lavallée*, Gaz. med. de Paris, Nos. 46, 48, 51, 53, 1864.—*Maschka*, Prag. med. Wochenschrift, No. 2, 1864.—*Schalle*, Ueber Herzwunden. Leipzig, 1864.—*Simon*, Zwei Fälle von Nadelstichverletzung des Herzens. Vierteljahrschrift f. gerichtl. Med. III. S. 287, 1865.—*Peacock*, On some of the causes and effects of valvular diseases of the heart. London, 1865, page 34.—*Jul. Poullet*, Recherches sur les caillots du cœur. Thèse, Montpellier et Paris, 8, 1866.—*Spencer Smith*, Punctured wound of the heart. Lancet, Jan. 26, 1867, p. 115.

—*Buchanan*, Case of lodgment of a needle in right bronchus with perforation of heart. Trans. of the Path. Soc. XVII. p. 87, 1866. —*Falko*, Traumat. Ruptur des Herzens. Russ. Arch. f. gerichtl. Med. 1867.—*Dr. Georg Fischer*, Ueber Wunden des Herzens und des Herzbeutels. Archiv für klinische Chirurgie, IX. Bd. 1867–1868.— *W. Monro*, Extensive injury to the heart in a dog by a pistol-ball, death not immediate. Edinb. Med. Jour. July, p. 66, 1869.—*Frederick Wright*, Wound of the heart by a needle, death. Brit. Med. Jour. No. 13, 1869.—*Serafino Biffi*, Caso di infissione di un ago nel curore di un maniaco e sua dimora per ventidue messinelle cavita cardiache sinistre. Arch. ital. delle mal. nervose Fasc. 5, 1869.—*D. R. Ambrose*, A pin encysted in the walls of the heart. New York Med. Rec. April 15, 1870.—*A. Reifer*, Stichwunde des Herzens. Wien. med. Presse, No. 39, 1871.—*Thomas Whipham*, The heart, left lung, and portions of the costal cartilages of a man who shot himself in Hyde Park. Trans. of Path. Soc. XXI. p. 92, 1870 (apex lacerated and left ventricle laid open).—*B. J. Sherman*, Rupture of the heart from external pressure. New York Med. Rec. Oct. 2, 1871.—*Desquin*, Observation de blessure du cœur par un coup de poignard. Annal. de la Soc. de Med. d'Anvers, 1872.—*Callender*.—Berliner klinische Wochenschrift, 1873, No. 12.—*Steudner*. ibidem, No. 7, 1874.

History.

Of the wounds in the breast, the wounds in the heart have always excited the greatest interest, not only among physicians, but among the laity as well; nevertheless, our accurate knowledge upon the subject has been gained in late years, for in olden times, when they were considered absolutely fatal, very little attention was given to them in a therapeutic point of view. Celsus, who also held the same view, which also was the prevailing one until the Middle Ages, was the first to give us an accurate account of the symptoms attending these wounds. In Jacob Hollerius, at the beginning of the sixteenth century, we first find the assertion, that a wound of the heart was not necessarily fatal. Accurate information is given by N. Muler. He gives (1627) the description of a wound of a single portion of the heart—the right ventricle. The first reliable account of the healing of a wound in the heart is given by J. Wolf (1642); this occurred by formation of a cicatrix. The attempt to recognize a wound in the heart, and in which portion of the heart it had occurred, is found a hundred years earlier in Fallopius, who

stated that the blood proceeding from the right ventricle would be dark, and that from the left bright red. Senac (1794), who had carefully studied the subject, called special attention to the relative danger attending the wounds in the various portions of the heart. In Morgagni (1761) we find a theory of the cause of death, which even at the present day must be generally considered as, in the main, the true one—namely, compression of the heart by the effused blood. In the eighteenth century we find evidences of earnest endeavors to promote the healing of such wounds, although Benjamin Bell (1783) did not believe that any such healing had ever been witnessed. A. Richter (1786) considered it dangerous to employ a sound in the exploration of these wounds, and laid stress upon the assistance rendered by bleeding and rest in forming a thrombus which should plug the wound. At the close of the last and beginning of the present century, Larrey and Dupuytren, assisted by a great amount of material, had also studied the subject carefully, and laid down many rules for treatment. In later times, and especially quite recently, medical literature is rich in cases accurately described, and particularly such as had a favorable issue in healing, in France, as well as in England and Germany. The greatest service, however, in collecting everything pertaining to this subject has been rendered by G. Fischer, in Hanover, who, in a truly classical treatise, has given us a most complete review of the literature down to the year 1868, and from this rich mine a great part of the data here employed have been drawn.

Etiology.

Most of the wounds of the heart are caused by direct injury, and in the collection by Fischer we find, out of 452 cases there were 44 punctured wounds, 260 punctured and incised wounds, 72 wounds by projectiles, and, finally, 76 contused wounds and ruptures.

The various ways in which wounds of the heart have occurred are classified as follows:

Punctured Wounds.		*Incised and Punctured Wounds.*		*Wounds by Projectiles.*	
Needles	28	Knife	93	Ball	58
Shoemaker's awl	4	Rapier	3	Small shot	10
Stiletto	3	Sword	33	Ramrod	1
Shaving-knife	2	Sabre	7	Stone	1
File	2	Bayonet	9	Water	1
Iron pin	1	Dagger	9	Wooden peg	1
Fishbone	1	Lance	1		
Toothpick	1	Sickle	1		
Splinter of wood	1	Incision (?)	54		
Thorn	1	Uncertain	48		
		Teeth, bone	2		
	44		260		72

Thus it appears that needles, knives, and balls have been the most frequent cause of wounds of the heart.

Contusions.

a. With an open wound: 1, a log of wood; 1, stone.—2.

b. Traumatic carditis and pericarditis from blows, jars, and falls.—5.

Rupture.

By being run over	23
Wheels of machinery	2
Fall from a great height (house, tree)	13
Fall from a slight elevation (ladder, wagon, horse)	7
By being buried under falling earth	6
Blow with a hoof or the foot	4
Being knocked down by a tree (falling?)	2
Blow	3
Simple shock	1
Uncertain	8

In this class, being run over is seen to be the most frequent injury. Murder is the most frequent cause, suicide the next, and accident the least common. Strangely enough, most of these wounds have occurred to persons in civil life, and not at a time of war. Larrey and Dupuytren have recorded the first seven and the second eleven cases, all drawn from civil life; this is all the more striking when we consider the great amount of material which Larrey had access to in time of war. The statistics of the last American civil war give 4,759 wounds of the thorax; of these 2,302 were penetrating wounds, and, strangely enough, among them all only four wounds of the heart. According to statistics, adults of the male sex form the greater proportion of those thus injured.

Much more rare than all these are those cases produced by penetration of the heart from within by a needle, bone, thorn (Kussmaul), or the like, making its way through from the œsophagus.

Among the *foreign bodies* which have penetrated the heart (Fischer has collected 41 cases), there is the greatest variety: needles, bullets, a paper-cutter driven in by a shot (Deguise), a fragment of the rib driven in by the same means, hairs from the breast, portions of clothing (Gent), fragments from a bursting gun, an ivory tooth-pick (Barbier), part of a file (Roux), a splinter of wood, and the blade of a knife. We have already said that some of these foreign bodies, especially needles, fragments of bone, and fish-bones, have made their way through from the œsophagus and stomach.

Pathology.

General Description of the Disease.

This is varied, according as we have to deal with a form of injury which works quickly and violently, *e. g.*, a dagger-wound or a gun-shot wound, or with a form produced by something making its way through from the stomach or œsophagus, such as a needle or a bone. In this latter case, the symptoms produced by the heart may have been preceded by an illness of greater or less duration, or it may be that some pointed object has been swallowed, and then the heart trouble may appear instantly (*e. g.*, a juggler swallowed a sword which perforated the œsophagus and pericardium).[1] Usually even slight wounds of the heart are followed immediately by alarming symptoms, such as fainting, great pallor, great feeling of distress, and hemorrhage. We find, however, quite a number of cases in the literature of the subject, where the patient had no idea of the severity of his injury, and continued to make considerable physical exertion after being wounded. Thus a child of ten years[2] went a distance of five hundred paces to his home after a splinter of wood,

[1] Lancet, 1860. V. II.

[2] Thomas Davis. London Med. Gaz. 1834, July, p. 344.

three inches long, had been driven into the breast on the right side, between the third and fourth ribs, and yet at the autopsy (death did not occur till thirty-seven days later) the splinter of wood was found sticking in the right ventricle between the columnæ carneæ.

So also a man (Ambroise Paré[1]) who had received a rapier wound under the left nipple in a duel, after making several thrusts at his enemy, who then turned and fled, followed his adversary two hundred paces, and suddenly fell dead. The wound in the heart was large enough to admit the finger.

A man (Gerard[2]) who had been struck with a kitchen-knife in the region of the heart, in a quarrel with the wife of a neighbor, ran forty or fifty paces to his home, when he fell dead. The physicians claimed that such a thing was impossible; but the cross-examination of witnesses established the truth of the assertion.

A great number of analogous cases, especially in animals who have been wounded, are recorded, and the most interesting is one related by L. Schröck.[3] A large stag had been wounded in the heart by the Electoral Princess Dorothea, of Brandenburg (1685), at a distance of one hundred paces; he ran quite a distance, and fell, after receiving two other balls in the back of the head; remained lying as if dead, but after three-quarters of an hour revived, broke away again, and was not overtaken by the hounds till he had gone three or four thousand paces, when he again received a ball in the back. The first ball had passed from behind through the right ventricle, grazed the left, and emerged under the right auricle. The canal of the wound was large enough to allow the finger to pass easily through it.

Aside from cases like these, and those where death followed instantaneously, the character of the disease will be that of the most prominent symptoms, and they will be sometimes those occasioned by hemorrhage, sometimes those due to severe nervous shock (great anxiety, a chill, vomiting), or, finally, those of great dyspnœa.

Pathological Anatomy.

First of all, we must mention here those rare cases (Fischer

[1] Opera chirurogica. Cap. XXX. Frankfort a. M. 1594.

[2] Essai sur la léthalité des plaies pénétr. du cœur. Thèse. Strasbourg, 1858.

[3] Ephemer. nat. cur. B. IV. ann. V. Norinberg, 1687.

gives six such) where, through the effect of great violence, the heart was completely torn off. In a man[1] who was crushed by a falling tree, the heart, torn off below the valves, was thrown out a distance of ten paces; it was ruptured in several places near the apex. The following case is yet more wonderful, for the injury was not direct. A beam in falling struck a boat, and by the jar a man was thrown into the water, though the beam did not touch him, and although immediately taken out of the water, he was found to be dead. The autopsy showed the pericardium to be ruptured and containing much blood, and the heart was found torn off at the origin of the aorta; its muscular tissue was healthy.

The 452 cases of injury in the large collection of Fischer, whose statistics are not only very extensive, but most conscientiously made, are arranged in the following manner, as regards the *locality* of the injury:

		Per Cent.
Right ventricle	123	27.2
Left ventricle	101	22.1
Both ventricles	26	5.7
Right auricle	28	6.2
Left auricle	13	2.8
Apex and base	19	..
Septum ventriculorum	7	1.5
Uncertain	57	..
Pericardium	51	11.3
Whole heart	16	3.5
Coronary artery	2	..
Right heart	4	..
Left heart	5	..

From this we learn that, as is generally believed, the wounds of the right ventricle are the most frequent. The difference between the two ventricles in this respect, according to this collection, is not so great as appears in other authors. Doubtless the much more exposed position of the right ventricle is the cause of its being more easily wounded. From these statistics we learn, moreover, that wounds in the ventricle are six times more frequent than those in the auricle.

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[1] *De Berghes*, Casper's Wochenschr. für die ges. Heilk. Berlin, 1844.

As regards the depth of the wound, out of three hundred and fifty-one cases, three hundred and nineteen were penetrating and thirty-two not.

The *pericardium* is not necessarily always wounded when the heart is, although this is generally the case. We have accounts of three bullet wounds where the thorax and heart were wounded and the pericardium escaped (Borellus, Holmes, and Heydenreich); moreover, we have read of cases where the heart was ruptured without injury of the pericardium and no external wound was found (Hufeland, Frin, Ward). Of the seventy-six ruptures and contusions (Fischer) there are five cases of rupture of the heart without injury of the pericardium.

The *form and direction* of the wound will generally be determined by the character of the object which caused it; it may be so changed by the contraction of muscular fibres in the different layers that a wound, which on the surface runs parallel with the fibres, and hence is more or less inclined to close, in the deeper layers will be seen to be tortuous and gaping.

The obliquity of the wound is important, for the more oblique it is the more likely will it be to close like a valve. In very small wounds, occasioned by a needle or awl, it may be very difficult to recognize external and internal openings; generally all punctured and incised wounds are found to have portions of coagula clinging to them and the edges of the wound are ecchymosed. Bullet wounds cause a loss of substance, with ragged ecchymosed edges, which are covered with clots, or we may find a canal, which passes entirely through the heart, of the diameter of the projectile, or sometimes the whole portion of the heart which is wounded is broken down into a blackish, bloody pulp. In incised and punctured as well as bullet wounds it often happens that not only the walls of the heart are injured, but also portions within the organ; thus the papillary muscles, leaflets of the valves, or the septum, may be incised, or cut through, or torn off. Finally, we may occasionally recognize the foreign body still lying in the wound.

If the patient survives the injury some time, or if the wound heals, we shall find it in one of the various stages of restoration, the foreign body lying bathed in pus or, in the latter case, the

formation of a cicatrix. Cicatrices like these often show long afterwards the shape of the object which caused the wound, but sometimes, in consequence of the myocarditis which is set up during the healing process, there in an extensive fibroid induration, which is not confined to the immediate limits of the wound. This may lead on its part to the development of an aneurism of the heart, or to the formation of a permanent abnormal communication between the various cavities and the like.

Possibly there may be various tendinous spots remaining of traumatic origin, corresponding to the site of the wound (Friedreich).

In ruptures also there may be no injury of the pericardium accompanying that of the heart. These may affect the inner portions of the heart, such as tearing of a papillary muscle or of the septum with the establishment of an abnormal communication between cavities, or they may be found on the external or internal surface of the organ. These latter are the more frequent, and they may be either longitudinal or transverse, may pass entirely through the ventricular wall or even encircle the whole heart. The rents are of various shapes, irregularly notched, and filled with coagula.

In the whole rich collection of cases there is no authenticated case of the cicatrization of a ruptured heart.

Besides the entrance of foreign bodies from without, we have already mentioned the fact that they sometimes find their way from within into the heart from the neighboring organs, the œsophagus and stomach. There are even cases on record where foreign bodies, after boring through the walls of the great vessels and the vena cava, have penetrated the heart. Entering in this way, they may be completely embedded in the wall of the heart, partially project into its cavity, or lie free in the same, and become firmly fastened *in situ* by a sheath of fibrine or connective tissue, or finally may pass entirely through the heart.

Symptoms.

Presupposing that the wound is not immediately fatal (when death occurs thus, there is hemorrhage, pallor of the whole surface

of the body, fainting, and convulsions in a very short time), there is a whole series of more or less striking symptoms, among which hemorrhage is the most prominent.

There are on record a number of wounds occasioned by the most various instruments as well as by firearms, where no external hemorrhage occurred, and this in wounds which penetrated as well as in those which did not. This depends doubtless upon the fact that the heart with its wound has changed its place, or a coagulum has immediately plugged it, or the foreign body itself occludes it, or the like. On the other hand, a very considerable hemorrhage may take place when the heart itself has not been injured, but only an intercostal artery or the internal mammary. A non-penetrating wound of the heart may also cause considerable hemorrhage by the injury of a coronary artery. When the wound is large, especially when a gunshot wound, there may be very great external hemorrhage, and it is believed this will be greater when the left ventricle is injured than when the right has been wounded; but this certainly is not always true. The internal hemorrhage may lead to hæmopericardium, and also to extensive effusions of blood in the thoracic cavity. The color of the blood doubtless depends upon which portion of the heart is injured; but very often, from admixture of blood from other parts which are wounded, the original color is changed. Sometimes air may escape with the blood from the wound.

We have already related cases where the patient, even after severe injuries, retained his strength for some little time, and was able to make certain exertions. But usually there is a sudden attack of fainting, which may last but a few minutes, or even as long as four hours (Carnochan[1]). The case related by Durande[2] is quite unique, where the fainting attack passed into a semblance of death, which lasted five days. The penetrating wounds of the heart, pericardium, and lungs had healed meantime; but the poor soldier died at the end of fifteen days, from gangrene of the lower extremities, which had been frozen. The fainting which occurs with pallor of the whole surface, shivering, and often

[1] Edinburgh Med. Journ., 1855.

[2] Mémoires sur l'abus de l'ensévelissement des morts. Strassbourg, 1780.

involuntary passages, since it is frequently observed immediately upon the receipt of the injury, cannot be the result of hemorrhage, but must be caused by the shock to the nervous system. This is indicated, moreover, by various accompanying symptoms; thus many times the patient complains of a feeling of unspeakable anxiety and great weakness, which certainly are often associated with a great loss of blood, the former especially with great internal hemorrhage, but which cannot be caused by this alone. Many patients are attacked, immediately after the injury, with trembling, and later with convulsions. Whether the difficulty in swallowing and the feeling of suffocation, which are sometimes experienced, are occasioned by the wound of the heart, or, which is more probable, by the direct injury of the nerves, we cannot say with certainty. If we consider, however, that these symptoms are chiefly noticed where there is a complicated wound, we shall see that the latter view would appear to be strengthened.

The vomiting, likewise, which we sometimes see immediately after the injury, should be considered as a purely nervous symptom. The feeling of oppression and the difficult respiration appear, moreover, to depend upon the amount of the pericardial effusion which sometimes occurs—that is, upon the extent of the interference with the function of the heart.

Pain is a very inconstant symptom in wounds of the heart. Cases enough are recorded where the patient himself at first paid little attention to very serious injuries; contact with the heart by objects passed through the wound in the thorax often caused no pain at all, which also was observed in the experiments with acupuncture. Harvey considered the heart wanting in sensibility; his observations were made on the young Duke of Montgomery, in whom the heart was visible after the healing of a severe wound in the breast. Other patients, where the heart was touched with a sound, had an unpleasant feeling of cold, or fainted; and others, again, aroused from a faint when so touched. At all events, the observations made up to the present time, upon the sensibility of the various parts of the heart, do not agree with those of Budge and Goltz upon the heart of the frog.

The impulse of the heart and the pulse are also very variable

in their nature, and exhibit no special characters peculiar to wounds in the various regions of the heart. As a rule, the impulse and pulse will be feeble and small in proportion to the amount of the loss of blood externally or to the effusion of blood into the pericardial sac and consequent compression of the heart. Occasionally, however, a strong and excited action of the heart has been noticed throughout the whole course of the disease.

The changes observed on percussion would naturally bear no relation to the injury, but by them we may occasionally ascertain a change in the position of the heart, or make out a collection of blood or air in the pericardial sac.

The results of auscultation may be very various. The occurrence of murmurs in the heart, occasioned only by the wound, and of a hissing or blowing character, is well authenticated. In some cases (Jobert) they seem to be caused by the escape of blood from the penetrating wound of the heart (?), in others by the accompanying injury of some of the inner portions, as the papillary muscles and portions of valves, or by perforation of the septum. Later, murmurs may be caused by the endo- and pericarditis which are developed.

Diagnosis.

When we have to deal with a wound of the thorax in the region of the heart, the question is whether the heart is injured, and if so, what portion of it. This question will also naturally arise with wounds in the neighborhood of the heart, when, from the surrounding circumstances, we have reason to suspect such injury. An external hemorrhage, if not dependent upon the wound of the thorax, may proceed from one of the great vessels or from the heart itself. Here the position of the patient at the time of receiving the wound, the direction of the instrument, its length and the depth to which it penetrated, will be of the greatest importance. An effusion of blood into the pericardial sac will occur in an injury at the origin of the great vessels, as well as in one of the heart itself, and accordingly will of itself give us no information whether or not the wound has merely injured the

heart substance or penetrated to its cavity ; if murmurs are suddenly developed, the latter will be probable. It cannot be denied that exploration with a sound or with the finger may, under some circumstances, assist in the diagnosis. Larrey and Podrazky felt with the sound the resistance of the heart, and observed its vibrations simultaneously with the movements of that organ. But, theoretically, we should suppose, and experiment has proved, that it is undesirable to make such exploration merely for the sake of diagnosis, for how easily the coagulum, which is of such importance to the healing of the wound, may be displaced either directly by the sound or by the over-action of the heart which it causes, and then a fresh hemorrhage be set up, etc.

It has already been mentioned that the color of the blood could not be relied on for accuracy in diagnosis, on account of the admixture which may take place from various sources.

The diagnosis of a wound of the heart by an object entering from within, could hardly be possible, except where the symptoms could be followed from the beginning with great distinctness ; when, for example, after swallowing a foreign body, pericarditis follows, and later other symptoms of cardiac trouble, then possibly such a succession might establish the diagnosis. The diagnosis of a traumatic rupture will usually be established by the character of the injury, the general severity of the symptoms, and the rapid course of the sickness, and that occasioned by the penetration of a foreign body will be assumed when we can find the absence of some portion of the instrument which caused the wound, such as the fact that a blade of the knife is broken off, or the like.

Complications and Termination.

Neighboring organs may be injured at the same time in the most various ways, according to the nature of the object producing the injury, the force with which it acted, as well as the direction from which it came, according also to the position of the organ, and finally to the position of the patient's body at the time the injury was received. In wounds which are received

from without, the thoracic wall and pericardium are involved as well as the heart, both of which, and especially the latter, essentially modify the nature of the disease. Later on, myocarditis and endocarditis are added to the wound of the heart. Among the other diseases associated with it, and often indeed produced directly by the same injury, are those of the pleura, the lungs, the diaphragm, and the intestines. All these complications may themselves again cause changes in other organs still.

Wounds of the heart produced by objects acting from within will be complicated by injury of the œsophagus or stomach, and by the pathological changes which preceded it. In ruptures likewise many of the above-named complications occur; but as these are so rapidly fatal, the complications are of little importance.

From the statistics of Fischer we learn that the frequency with which *immediate* death occurs may be numerically expressed as follows: in

Ruptures, 36.0 per cent.
Gunshot wounds, 26.0 per cent.
Incised and punctured wounds, 20.0 per cent.
Punctured wounds, 9.2 per cent.

In all, it is seen that twice as many cases terminated fatally at some later time as did so immediately, and that life lasted longer in wounds of the right ventricle than in those of the left, and in those of the right auricle than in those of the left. According to Fischer, out of 401 wounds of the heart 50 healed, and of these fifty the fact was established by a subsequent autopsy in thirty-three; and as regards the location of the injury, the frequency of the occurrence of a cure was in the following order: apex and septum (both non-penetrating), right ventricle, left ventricle. Injury of the auricle is apparently always fatal. So also there is no recorded case of the healing of a rupture, but, on the other hand, according to Velpeau, even a wound which penetrates the entire thickness of the ventricular wall may heal by cicatrization. The direction of the wound will be of the very greatest importance as regards a cure—the more oblique the canal, the more favorable will it be; but even very fine and very

oblique wounds have proved rapidly fatal. In these latter cases possibly the injury of one of the great vessels hastened the fatal termination, yet in a patient, thirty years of age, it was shown that it is possible that a coronary artery which has been cut across may yet heal (Larrey, loc. cit.).

As regards the character of the wound, it is evident in general that death at a later period is more frequent in penetrating and incised wounds than in gunshot wounds; and in these latter it is a very striking circumstance that a deferred death is more frequent than an immediate one. Under all circumstances, simple penetrating wounds run the most favorable course. This coincides also with what has been observed in the numerous experiments by acupuncture, especially at the apex of the heart, both in animals and in men (Steiner[1]). In these cases, where the wound did not heal, the fatal end has occurred even as late as eight and a half months after the injury, and, in a case of rupture of the right ventricle, death occurred twelve days later.

What is the immediate cause of death in general? and what is its cause when death is deferred? Death, immediately after the injury, seems, as a rule, to be caused by loss of blood, and by the rapid development of anæmia in the central nervous system which it occasions. Among other circumstances the following fact would seem to strengthen that view, viz., that in wounds of the left ventricle death is apt to occur more quickly than in those of the right, probably because the anæmia of the brain, which is so fatal, is more speedily developed, and, moreover, the blood lost is arterial. The next most important cause of death is, doubtless, the interference with the heart's function produced by the accumulation of blood in the pericardial sac. If the objection should be made, that equally large pericardial accumulations are often well borne and even for a long time, to this we reply, that the objection is not valid, for there is a great difference in the rapidity with which the accumulation has taken

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[1] Billroth's Archiv. Bd. XII. 1871. See also on the same point: *Jung*, Schweiz. Zeitschr. Bd. XII., H. 2, 1841, who also gives the earlier experiments of *Bretonneau* and *Larrey;* later also *Cullender*, Berlin klin. Wochenschrift, No. 12, 1873; and *Demme* (a letter written by *Demme, Jr.*, to Prof. Billroth, in Steiner's paper).

place and with which the compression has been applied ; other organs also show a similar toleration of compression which is gradually increased. Yet these two explanations will not cover all cases, and where such cases occur, of immediate death, we can only explain them by the severity of the shock to the nervous system, perhaps caused by a special injury of the ganglia of the heart.

In those wounds in which death occurs at a subsequent period, the chief cause of death is the occurrence of more or less frequent and extensive secondary hemorrhages (these may occur at as late a period as eight weeks after the injury), for they still further increase the weakness of the patient, already existing; moreover, the development of any of the above-named complications, of which pericarditis, pleuritis, and pneumonitis are the most frequent, will be a new source of danger.[1]

In those cases in which healing has taken place, a cicatrix is formed of solid indurated connective tissue, which sometimes extends beyond the immediate limits of the wound ; the formation of a partial aneurism of the heart has also been observed, taking its rise from the induration which replaced the loss of substance (Mühlig[2]). After cicatrization of the wound the pathological changes which have taken place during recovery may remain for years, and sometimes permanent disturbances may last through life. It may result in cardiac lesions, or in very extensive adhesions of the heart to the pericardium, or to adjacent organs. Occasionally the patient complains of palpitation of the heart (as was observed in one case, where after death a foreign body was found encapsulated); in another case the patient suffered from a feeling of anxiety and distress in the chest ; finally,

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[1] Gazette Medic. d'Orient. 1860. Sept.

[2] The following communication is very interesting (*Steudener*, Berlin. klin. Wochenschr. 1874. No. 7) : A man, twenty-three years of age, with a bullet-wound through the heart, died fifteen weeks after the injury, by separation of the spinal cord. On the outer wall of the left ventricle was found an oblique cicatricial furrow beginning near the apex of the heart, 2 cm. wide in the middle; the muscular layer underneath, only a millimetre in thickness ; on filling the heart with water this portion bulges out considerably; the bullet must have struck at the moment of systole. Doubtless it would have resulted later in an aneurism of the heart.

the neighboring organs, suffering from the injury received at the same time, may give rise to permanent functional disturbances.

Prognosis.

When it has been established that the heart is injured, we can only give an uncertain prognosis. We cannot with certainty predict a recovery even with the slightest injury of that organ. The more robust the patient is, the less the nervous shock and the hemorrhage, the more favorable the direction and character of the wound, and the slighter the injury of neighboring parts, just so much more favorable will the outlook be. Profound syncope may act kindly, by putting a stop to the hemorrhage; so a moderate pericarditis may possibly serve to close the external wound and also that of the heart; so the retention of the foreign body in the wound may have a favorable influence as a hæmostatic. We may learn what an astonishing quantity of blood may be lost by reading the case of a soldier who received a rapier wound which passed through the left ventricle and septum into the right chamber. The patient died seventeen days later, after losing almost a pound of blood daily.[1] Foreign bodies, such as needles, the point of a knife, etc., remaining in the wound, do not, strange as it may seem, render the result more unfavorable; indeed, we have quite a number of authentic cases where recovery took place with the foreign body remaining *in situ.*

Treatment.

Even when we only suspect an injury of the heart, the greatest care and foresight will be necessary. Often enough, as experience has taught us, the importance of an injury of the thorax in the region of the heart has been overlooked, not only by the patient, but by the physician as well, but the sad termination soon showed that the heart had been wounded. Our first object will be to arrest the hemorrhage, close the wound, and keep up the strength of the patient. For the first purpose

[1] *Fantoni*, Giorn. dei litt. d'Italia. Tom. 21.

syncope may not be altogether undesirable under some circumstances, and accordingly we should be cautious about the too energetic employment of stimulants, and reserve them for the time when death seems imminent.

Phlebotomy, which certainly lessens the tension in the blood-vessels for a time, and thus gives an opportunity for the formation of a clot, can only be practised on a very robust individual. The removal of the foreign body, reasonable as it may appear from one point of view, is likewise a very dangerous proceeding, for there can be no doubt that its removal may set up a hemorrhage which will be instantly fatal. The most useful remedies will be absolute rest, the application of cold, and when the patient is very restless, narcotics, and of these the rapidly acting subcutaneous injection of morphine will be preferable, and, when the heart's action is greatly excited, digitalis. In the later course of the disease we must be guided by the general rules of surgery, and, when complications exist, by the rules which govern their treatment.

New Growths and Parasites of the Heart.

General.—The earlier works by *Andral*, *Bouillaud*, *Cruveilhier*, *Albers*, the more recent by *Rokitansky*, *Lebert*, *Förster*, *Virchow*, *Bamberger*, *Duchek*, *Friedreich*, *Dusch* u. s. w.

Special Treatises.—*E. Wagner*, Tuberkel des Endocardiums. Arch. f. Heilkunde. II. 1861.—*Heschel*, Theilweise Verkreidung Herzmuskulatur. Oesterr. Zeitschrift für pract. Heilkunde. No. 14. 1861.—*Kottmeier*, Fibröse Neubildung im Herzen. Virchow's Archiv. XXIII. 3. u. 4. Heft. 1862.—*Recklinghausen*, Myoma cordis. Monatsschrift f. Geburtskunde. 20. Bd. 1862.—*R. Law*, Cases of cancer of the heart. Dubl. Quart. Jour. of Med. Soc. May, 1863.—*Klob*, Metastatisches Cancroid d. Herzen. Wien. med. Wochenblatt d. G. d. Aerzte. XIX. 1863.—*Pietro de Venezia*, Storia di un tumore cancroso nel cuore. Giorn. veneto, Maggio. 1864.—*Williamson*, Echinococcus in Herz und Lungen. Allgem. med. Centralzeitung. No. 74. 1864.—*Virchow*, Congenitale cavernöse Myome des Herzens. Virch. Arch. XXX. 1864.—*Dr. A. Lüken*, Die patholog. Neubildungen des Myocardium. Zeitschrift f. rat. Med. XXIII. Heft 3. 1865.—*Paikert*, Medullarcarcinom des Herzens. Allg. milit. ärztl. Zeitung. No. 36. 1865.—*Haberling*, De tuberculosi myocardii. Diss. Breslau. 1865.—*E. Wagner*, Metastatisches Sarcom einer Lungenvene und des linken Vorhofs. Archiv der Heilkunde. Bd. VI.—*Virchow*, Die krankhaften Geschwülste, Melanosarcome

des Herzens. II. 289. 1863-67.—*Dr. Karl Bodenheimer*, Beitrag zur Pathologie der krebsartigen Neubildungen am Herzen. Diss. inaug. Bern. 1865.—*Feierabend*, Verknöcherung der vorderen Herzwand mit Lebercirrhose. Wien. med. Wochenschrift. No. 58. 1866.—*Waldeyer*, Tuberkulose des Myocardium. V. Arch. XXXV. 1866.—*Friedreich*, Beiträge zur Pathologie des Krebses. V. Arch. XXXVI. 1866.—*O. Wyss*, Herzkrebs. Wiener med. Presse. VII. 1866.—*Prudhomme*, Observation d'insuffisance aortique causée par une végétation cancreuse melanée emergeant des valvules sigmoides aortiques. Gaz des hôpit. No. 8. p. 30. 1867.—*Otto Oesterlen*, Ueber Echinococcus im Herzen. Mittheilung aus der Bruns'schen Klinik. Virchow's Archiv. XLII. 1868.—*Morgan*, Transactions of the Pathol. Soc. of London (Nodules of medullary sarcoma in the heart substance). 1868.—*Skoda*, Ueber die chron. Bindegewebsneubildungen im Herzen. Allg. Wiener med. Zeitschrift. Nos. 25 u. 26. 1869.—*Thomas P. Pick*, Fibroid degeneration of the heart. Transactions of the Pathol. Soc. XIX. p. 156. 1868.—*G. Burellai*, Observatione di una tuberculose del cuore. Annal. univ. di Med. Maggio, p. 346. 1869.—*Ch. Kelly*, Hydatid cyst in the heart. Transact. of Pathol. Soc. XX. p. 145. 1869.—*T. Whipham*, A heart showing extensive growth of fibrous tissue in the muscular walls. Transact. of Path. Soc. XXI. p. 115. 1870.—*W. Moxon*, Hydatid of the heart, obliterating by its pressure the coronary sinus. Transact. of Path. Soc. XXI. p. 99. 1870. —*W. W. Wagstaffe*, Fibrous tumor of the heart. Ibid. XXII. p. 121. 1871.—*J. F. Payne*, Cancerous growths on endocardium, etc. Ibid. XXII. p. 125. 1871. —*J. Coats*, Two cases of calcareous infiltration of the musular fibre of the heart. Glasgow Med. Jour. August, 1872.—*Peacock, Th. Bevill*, Hydatid cyst imbedded in the walls of the heart. Transact. of the Path. Soc. XXIV. p. 37. 1873.—*Kantzow u. Virchow*, Congenitales wahrscheinlich syphilit. Myom des Herzens. Virch. Arch. XXXV. 1873.

From a clinical point of view, this subject will afford us but little profit, and our knowledge hitherto gained belongs almost exclusively in the domain of pathological anatomy.

History.

We can only speak of a history of the subject within quite a recent period, for it is intimately associated with the development and progress of pathological anatomy, and especially of microscopy; only by these means can we explain the confused and varied accounts which have hitherto been thrown together.

Thus, for example, there can be no doubt that many tumors hitherto described as tuberculous should be classed, as Virchow has shown, among the gummy growths.

Pathological Anatomy.

We have spoken of the *connective-tissue growths* in the form of indurations under the head of chronic myocarditis, and also, under the same head, of gummy tumors.

Fibromas.

Growths of this nature have been very frequently observed. Luschka[1] described such a one of the size of a small hen's egg, situated in the substance of the left ventricle, in a boy six years of age. Albers[2] described one of the size of a pigeon's egg in the anterior wall of the left ventricle.

The case of Kottmeier (loc. cit.) is very interesting, where a fibroma, about two inches in length, springing from the septum of the auricles, hung down through the auriculo-ventricular opening into the left ventricle. An analogous case is found in the Gazette des Hopitaux, 1872, 101, where a fibrous polyp, springing from the septum, had so forced its way between the semi-lunar valves of the aorta, as to cause an insufficiency. Wagstaffe (loc. cit.) found in a little girl three months old, who had died suddenly after slight convulsions, a tumor the size of a hen's egg, lying between the muscular fibres of the septum ventriculorum, and the cavities very much contracted.

Formation of Concretions.

Aside from the bony scales or plates which sometimes cover the heart more or less extensively, these concretions are generally found in the form of calcification or ossification of connective-tissue growths which penetrate the substance of the heart, or else as the mortar-like residua of the healed abscess. Heschel (loc. cit.) observed in a case of Bright's disease a partial calcification of the muscular tissue of the heart. A case of idiopathic development of one of these growths is found in Lüken (loc. cit.),

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[1] Virch. Arch. Bd. VIII.

[2] Atlas der path. anat. III. T. 10. Fig. 1.

where a calcified tumor of irregular shape, and roughened surface, almost 3 cm. long and 2 wide, was found imbedded in the chordæ tendineæ of the anterior segment of the mitral valve, which reached far into the myocardium, and caused insufficiency of this valve.

Lipomas.

We have already spoken of the deposit of fat upon the heart and its penetration into the muscular tissue, as well as of the true fatty degeneration, under its own appropriate head. In the whole literature of the subject we find an account of only one lipoma imbedded in the muscular tissue; this is described by Albers.[1]

Cysts.

The accounts given of cysts in the literature are very confused, and probably, with the exception of the parasites which will be mentioned further on, they may be explained by considering them to be either the cavities of emptied abscesses, or else clots breaking down in the middle (Valsalva, Thebesius, Morgagni).

Myomas.

These are observed as tumors of greater or less size, usually of congenital origin (Virchow;[2] Recklinghausen, loc. cit.).

Carcinomas.

These are the most frequent of the new growths in the heart; thus in Köhler[3] in 9,118 autopsies, and in Tanchon[4] in 8,289 autopsies, cancer of the heart was observed six times by each; in Willigk[5] in 4,547 autopsies (of these 477 cases of carcinoma), cancer of the heart was observed nine times, and of the pericardium seven times. Most frequently carcinoma of the heart

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[1] Virch. Archiv. Bd. X.

[2] Virch. Archiv. Bd. XV.

[3] Ueber Krebs, Scheinkrebskrankheiten. Stuttgart. 1853.

[4] In Lüken (loc. cit.).

[5] Prager Vierteljahrschr. 1856.

is secondary, either developed by extension from neighboring organs (mediastinum, œsophagus, etc.), or occurring as a metastatic deposit, and here frequently following the course of the vein; thus we may see it extending from the lung along the pulmonary veins into the left auricle or along the venæ cavæ into the right auricle.

Primary cancer in the heart is commonly of the colloid and melanotic form; secondary cancer is usually also medullary in character. Occasionally the tumor is found already undergoing the process of destruction, and then becomes a focus for emboli.

Of forty-five cases collected by Bodenheimer, it occurred seven times in the left ventricle, three times in the right ventricle, and twice in the right auricle. In the remaining cases it was found in several portions of the heart at the same time. The new growth has its seat in the interstitial connective tissue, between the muscular fibres, and usually forms nodules of greater or lesser proportions, which frequently project into the pericardial sac as well as into the cavities. Here they may cause insufficiency of the neighboring valves or stenosis of the auriculo-ventricular opening.

Carcinoma may occur at any age. Billard found it in a newborn child, Segalas[1] in a child eleven years of age. Most frequently, however, it seems to occur after forty-five years of age.

It would seem to be more frequent in men than in women.

We find only one single observation of epithelial cancer. Paget[2] found it in the muscular substance of the apex of the right ventricle and in the septum after the extirpation of a carcinomatous bulbus oculi.

Tubercle.

Tubercle usually occurs as an accompaniment of general tuberculosis in the miliary form, with yellowish or gray nodules in the connective tissue between the muscular fibres. More rarely there are larger cheesy nodules always as a sequel

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[1] Both in Bodenheimer's collection, p. 42.

[2] Surgical Pathology. II. Vol. p. 449.

of chronic tuberculosis, and usually extending from the neighborhood into the heart (Waldeyer, Recklinghausen[1]). Townsend[2] describes a large tumor of this nature taking its origin from the left auricle, which compressed the trunks of the pulmonary veins.

Parasites.

Of these we find the cysticercus[3] and echinococcus. The latter is the more common, but, according to Davaine, of 160 cases of echinococcus occurring in organs other than the liver, it was found only ten times in the heart. It is found more frequently in the right than in the left ventricle; in the collection of Oesterlen, in twenty-one cases it occurred eleven times in the right heart, seven times in the left, and twice in the septum ventriculorum. The size varied between that of a pin's head and that of an orange. Usually the echinococcus is developed in the heart substance, but occasionally it hangs as a pendulous tumor into the cavity, attached to the inner wall. The free cysts or vesicles have been swept by the current of blood into the cavities of the heart. Coote[4] describes a case where an echinococcus cyst situated in the anterior wall of the left ventricle caused such an enlargement of the heart, that it extended from the third rib on the right to the eighth on the left, and pushed both lungs backwards.

In the cases of Oesterlen, rupture of the sac occurred six times.

Symptoms and Course of the Disease.

In many cases the neoplasm was found at the autopsy without giving any evidence of its existence during life, by signs or symptoms. In other cases there was, during life, a series of symptoms, which certainly could not be considered characteristic of the disease, and fully agreed with those of other diseases.

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[1] Virch. Archiv. Bd. XVI.

[2] Dublin Journ. of Med. Sciences, 1832. I. Vol.

[3] See Vol. III. of this Cyclopædia, pp. 586 and 604.

[4] Med. Times and Gazette. Feb. 1854.

Tumors, by their invasion of the cavities of the heart, lead to manifold disturbances in the circulation, to insufficiency and stenosis of the valves in their vicinity, to compression of the vessels, etc.; by their protrusion outwards they frequently set up pericarditis, or, when breaking down, or, if echinococcus cysts, by their rupture, they may by the loosened fragments give rise to embolism in various organs of the body. Cases of this nature are described where echinococcus cysts, situated in the right ventricle, by their rupture caused embolism of the numerous branches of the pulmonary artery, which was indicated by sudden gasping for breath, symptoms of asphyxia, and even immediate death.

Oesterlen gives the history of a girl, twenty-three years of age, who had gangrene of the right lower extremity, caused by the wedging of echinococcus cysts into the femoral and profunda arteries, carried thither by the current of blood from a tumor about two cm. long, situated on the posterior wall of the left auricle and bulging forwards into its cavity; the tumor was irregular in shape, and apparently made up of many spherical, bladder-like elevations, which were partially transparent, and of the size of a millet-seed.

Diagnosis.

In the present state of our scientific knowledge it will be impossible to establish the diagnosis, even in those cases where actual symptoms are observed, caused by the neoplasm or by the parasites; for they will differ in nothing from those of other heart diseases, which they exactly simulate, such as the compression of the venæ cavæ already mentioned, or an insufficiency and stenosis of the mitral valve, etc.

It will be equally difficult to ascertain which of the symptoms are caused by the primary disease, and which by complications. Under specially favorable circumstances, we might possibly be able to assert the probability of the existence of one of these diseases.

If, for instance, in the course of a general carcinomatous infection, or of an echinococcus of the liver whose existence had

been surely diagnosticated, there should suddenly appear symptoms of trouble with the heart, we might suspect a metastatic carcinomatous deposit in the heart, or that an echinococcus vesicle had been swept onward by the venous current into that organ. But here, again, we should not forget that the symptoms resulting from such an occurrence may take their origin from other complications of the primary disorder.

Heart Clots.

See the works on heart disease in general.—*Benj. W. Richardson*, Lectures on fibrinous deposition in the heart. Brit. Med. Journ. 1860.—*Gerhardt*, Ueber Blutgerinnung im linken Herzohre. Würzburg. med. Zeitung. 4. Bd. S. 150. 1864.—*The Same*, Thrombosis cordis dextri. Ibid. S. 221. 1864.—*Proust*, Polyp im rechten Vorhof. Gaz. méd. 52. 1864.—*Hayden*, Dubl. Quart. Journ. of Med. Sc. Nov. 1864.—*Gallard*, Blutgerinnungen im Herzen. Gaz. des hôpit. 71. 1865.—*Faure*, Recherches experimentales sur les caillots fibrineux et sur les produits d'inflammation du cœur. Arch. général. de Méd. Fevr. 1864. —*Vulpian*, l'Union medic. 1865. No. 18.—*The Same*, Bulletin et mém. de la Soc. médic. des Hôpitaux de Paris. 1865. Paris. 1866.—*P. Aronssohn*, Caillots dans l'intérieur du ventricule gauche, etc. Gaz. méd. de Strassbourg. No. 12. 1868.—*Nobiling*, Thrombose des linken Ventrikels. Bayr. Intell.-Bl. No. 24. 1869.—*Joseph Jones*, Heart clot, a clinical lecture. New Orleans Journ. of Med. July, 1869.—*J. S. Bristowe*, Softening clots in the heart in a case of renal disease. Transact. Path. Soc. XIX. p. 90. 1868.—*Bucquoy*, Concretion polypiforme ancienne du cœur. Gaz. hebdom. No. 10. Séance de la société méd. des hôp. 1869.—*George Gaskoin*, Polypus of the left side of the heart. Med. Times and Gaz. Sept. 4, 1869.—*John G. McKendrick*, A case of heart disease (cardiac thrombosis). Edinburgh Med. Journ. Nov. p. 396. 1869.—*Th. Neureutter*, Combustio, Thrombose und Embolie im Arteriensystem. Wiener med. Presse. Nos. 15 u. 16. 1871.—*Jules Dubois*, Kystes libres dans les cavités du cœur. Bull. de l'Acad. de méd. XXXV. p. 807 (changes in cardiac thromboses). 1871.—*Biermer*, Vortrag über polypöse Gerinnungen im Herzen. Corresp.-Blatt. Schweizer Aerzte. No. 9. 1872.—*R. Lawson*, Two cases of death from fibrinous concretions in the right side of the heart. Med. Times and Gaz. Feb. 8, 1873.—*Edwd. Crisp*, Heart clot and sudden death. Transact. of the Path. Soc. XXIV. p. 46. 1873.—*T. Whipham*, Old blood-clot adherent to the wall of the left ventricle and septum of the heart, producing thickening of the endocardium and degeneration of the muscular tissue. Lancet. Jan. 4, 1873. —*Mullier*, Concrétion sanguine du cœur. Arch. med. Belg. Avril, 1873.—*Raymond*, Concretion sanguine du cœur chez un malade atteint de diabète sucré.

Ibid. 1873.—*Vergely*, Observation de thrombose cardiaque chez un malade atteint de pleurésie chronique. Reflexions. Bordeaux médical. Nos. 22, 23, 24. 1873.—*J. Fayrer*, On fibrinous concretions in the right side of the heart as a cause of death after surgical operations. Med. Times and Gaz. Jan. 18, 1873. —*Richardson*, On fibrinous deposition in the heart. Ibid. June 14, 1873.

Interesting as the study of this subject is, as regards its origin and pathological anatomy, and at the bedside as regards its results, yet for the clinical teacher it affords little profit.

History.

It appears that Benivenius, at the beginning of the sixteenth century, was the first to give intimations of the existence of such a pathological condition; later, polyps of the heart, as they were first designated by Sebastian Pissinius (Milan, 1654), acquired considerable significance. A great number of symptoms were described as pertaining to this condition, which was generally considered to be an idiopathic disease, and numerous wonderful accounts were spread abroad, which clearly related to its various forms.

Kerkring (1670), Petit (1732), Pasta (1739), had only looked upon them as post-mortem conditions. Both Huxham and Chisholm wrote on the epidemic occurrence of polyps in the heart. Testa and Kreyssig associated them with inflammation, and the latter described a special "carditis polyposa." Morgagni and Senac were aware in their day that they might be formed not only after death or in the last moments of life, but also under certain peculiar conditions during life, views which were confirmed and elaborated by Laënnec, Bouillaud, Cruveilhier and Rokitansky. But only in later times has Laënnec's view been disproved, that the globulous vegetations upon the heart were due to an inflammatory process and contained pus (pus-cysts).

Etiology.

At almost every autopsy fibrinous concretions are found, and particularly in the right ventricle and auricle; they are formed either after death or when the patient is in articulo

mortis. On this account they are designated "post-mortem clots" ("Sterbepolypen"), and being fresh, as opposed to those older clots formed during life and called true polyps of the heart, they are termed false heart polyps. They are the result of the natural coagulation of the blood caused by the retardation and final cessation of the current, and since toward the end of life the blood is collected chiefly in the veins, they are most abundant in the right heart. If death has been protracted, and the contractions of the left heart have gradually become weaker, similar clots will be found in the left ventricle and auricle as well. These will not demand further consideration, but those formed during life are of clinical importance. The causes of their formation are various:

1. In this class belong all those clots which have been caused by a slackening in the current of blood, whether it be on account of some obstruction in its course, or because the heart contracts with diminished energy. They are termed thrombi of dilatation or marasmic thrombi. Moreover, they are found especially in those portions of the heart where the current is feeble, as in the appendix auriculæ, entangled in the trabeculæ carneæ, in partial aneurism of the heart, especially in the left auricle (here also far more frequently in the appendix), above a stenosis of the left auriculo-ventricular orifice, in the various diseases of the heart substance by which its contractile power is diminished (chronic myocarditis, fatty degeneration, pericarditis, extensive adhesions of the heart and pericardium, etc.). It is quite possible that the use of digitalis, too long continued, may, by slackening the current of blood, give rise to a more rapid coagulation.

2. Rough surfaces on the inner wall of the heart very readily occasion a deposit of fibrine, and from this, as a starting-point, an increased deposit may result. These irregularities arise from vegetations and inflammatory processes on the lining membrane of the heart.

3. With Rokitansky and Bamberger, as opposed to others, I hold that fibrinous coagula, set free from distant portions of the vascular system, may cling to the inner wall of the heart, remain there, and form a nucleus for more extensive fibrinous concretions.

4. Amongst the doubtful causes we may place the view that

the increase in fibrine in the blood, which attends certain diseases, leads to its more ready separation, with the additional fact that the fluid in which it is dissolved is diminished in quantity; and, finally,

5. That there may be such a thing as a morbidly increased tendency in the blood to coagulate.

Pathology.

Pathological Anatomy.

First we must consider the difference between fresh clots and those of older formation. The distinction is usually easily made, for the fresh clots are whitish-yellow, generally translucent, frequently jelly-like in consistency, having a moist shiny appearance, as if œdematous, and infiltrated with blood on their lower surface. Sometimes they extend into the vessels drawn out like strings or cords, sometimes they have many branches and are wound around the trabeculæ carneæ, and bear the impress of those parts of the heart with which they lie in contact. They can usually be easily separated from the subjacent parts without injury to either.

The true polypi of the heart are dull in appearance, dry, rotten, friable, of a whitish-yellow or whitish-gray color, often having many layers, in which case the layers may vary in color, since the older portions are stained by the coloring matter of the blood. They may reach a considerable size, and are sometimes flattened out, and are sometimes drawn out like cords.

Virchow found the left auricle, Rokitansky the left ventricle, and Housley[1], in a girl two years old, the right auricle, entirely filled with a firm clot.

Not unfrequently they project like a little cone, or apparently attached by a stem like a polypus, from the appendix auriculæ into the auricle, thence through the ventricular opening into the ventricle; or, in the same way, they project from behind the columnæ carneæ of the ventricle in the form of more or less

[1] Med. Times and Gazette. No. 408. 1858.

rounded tumors, from the size of a pea to that of a pigeon's egg, into the cavity, and form the so-called "végétations globuleuses." Since these frequently contain in their interior a puruloid fluid, they have received the name of pus-cysts. It is not really pus, but a mass made up of the detritus of the centre of the clot, arising from molecular destruction, mingled with white blood corpuscles. Sometimes in these old clots, lime salts are deposited, so that they may ultimately form firm concretions. Quite frequently single portions of the clot are found, which have been torn off. But the separation of the whole mass from its attachment is usually quite difficult. They are found particularly in the left ventricle at its apex, and in the auricles usually in the appendices.

Symptoms.

We have already mentioned the fact that the old physicians described a whole group of symptoms belonging to the formation of clots in the heart. If we investigate the subject thoroughly and free from bias, we must allow that polypi in the heart give rise to many and very important symptoms, but that these can in no way be distinguished from those which occur in a number of other serious affections of the heart.

Thus occasionally death may suddenly occur in a patient previously in good health. In other cases we observe more or less severe dyspnœa, cyanosis, or pallor of the whole surface, expectoration of bloody sputa, and coldness of the extremities, and death occurs after loss of consciousness, stupor, and convulsions. Generally the heart's action is diminished in frequency, and rarely is irregularly violent. If the patient survives some time, dropsical effusions occur. Even if these symptoms should appear in an acute and primary form, they would show nothing specially characteristic of the formation of a clot.

The difficulty is all the greater from the fact that the formation of thrombi in the heart is generally the sequel of other already existing diseases of this organ, and the symptoms of the two conditions are mingled.

Diagnosis.

Recently Richardson and Gerhardt have insisted on the possibility of a diagnosis of this condition; indeed, the former claimed to be able to recognize the location of the clot in the left or right ventricle, according to the different character of the dyspnœa in the two cases. But this position is untenable, from the fact that the symptoms are not characteristic of a clot. The same statement holds good with regard to what Gerhardt has contributed to the diagnosis, even though many of the symptoms which he relates may be occasioned by the formation of a clot. I do not feel disposed to lay special stress upon the symptom to which he has called attention, viz., the existence of a systolic murmur from the compression of the pulmonary artery by the left auricle distended by a thrombus. Aside from the fact that the existence of such compression is very doubtful, the sign might arise from various other causes as well.

From all this we learn that the diagnosis may often be suspected, indeed, may perhaps be given with a certain probability, but yet cannot be surely established. Even when we take into consideration what are the results of thrombus in the right ventricle—the infarctions of the lung following upon thrombus of the left ventricle; the suddenly occurring stenosis of the auriculo-ventricular orifice,[1] by becoming occluded by the fibrinous plug; embolism of the artery of the fissure of Sylvius (middle cerebral), or of some other artery of the body; or apoplexy—we shall see that all of these might occur in other diseases which cannot be diagnosticated with certainty. The physical signs on the part of the heart which may be recognized are of no value for diagnosis.

Prognosis.

It cannot be denied that a clot in the heart may often exist for a long time without giving rise to a single symptom, and the

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[1] Von der Byl. Lancet. 1855. A clot, detached from the left auricle, entirely occluded the mitral office, already the seat of stenosis, and thereby occasioned sudden death. McKendrick describes a similar case.

patient may die of some other disorder. Nevertheless the prognosis is in the highest degree unfavorable, inasmuch as the increased growth of the coagulum causes increasing disturbance in the circulation, and the danger of embolism in vital organs is ever present.

Treatment.

Even if it were possible to make a diagnosis of heart clot, we could suggest no treatment which would tend to cause an absorption of the clot, or which can render its passage into the circulation free from danger. Certainly we shall not accomplish this end by the use of the various salts of soda, lime, or ammonia (carbonate of ammonia was especially recommended by Richardson). Accordingly there remains nothing for us, except, under all those circumstances in which the formation of a clot is possible, to spare as much as possible the weakened heart, and to endeavor to secure the most complete contractions of the organ; but these are points which in practice are rarely considered. Hence, in a case of this kind, our treatment must be purely symptomatic.

Nervous Palpitation of the Heart.

See the works on heart diseases in general.—*Beau*, De l'angine de poitrine (in relation to smokers). Gazette des Hôpitaux. 1862.—*von Bezold*, Ueber die Innervation des Herzens. Leipzig. 1863.—*Valentin*, Versuch einer physiolog. Pathologie des Herzens. 2. Abthlg. Leipzig und Heidelberg. 1864.—*Remak*, Ueber centrale Neurosen des Herzens. Berl. klin. Wochenschrift. No. 25. 1865.—*Championnière*, Fumeurs, Journ. de méd. pratique. Juillet, 1865.—*Payne Cotton*, Notes and observations upon a case of unusually rapid action of the heart. Brit. Med. Journ. June 1, p. 629. 1867.—*Rob. Bowles*, Unusually rapid action of the heart. Ibid. July, p. 53. 1867.—*Edmunds James*, Unusually rapid action of the heart. Ibid. Aug. p. 97, 1867.—*Oppolzer*, Ueber Herzklopfen. Allg. Wiener med. Zeitung. Nos. 25–27. 1870.—*Fothergill*, Edinburgh Med. Journ. Dec. 1870.—*Da Costa*, On irritable heart, a clinical study of functional cardiac disorder. Americ. Journ. of Med. Sc. Jan. 1871.—*Green*, Disturbed action of the heart. Brit. Med. Journ. Nov. 25, 1871.—*Fr. B. Nunneley*, Observations on palpitation of the heart and its treatment. Lancet. Febr. 18, 25. 1871.

By the name palpitation of the heart we understand an in-

creased functional activity of the organ, without any pathological changes (at least any which we can recognize at the time). In the present condition of our scientific knowledge we might define it as an antagonism between different factors of innervation of the heart.

History.

In the works of the oldest physicians we find, as is quite natural from the prominence of this symptom, accounts and explanations of this condition. Together with some odd and fanciful views, we find some most ingenious attempts at an explanation of this symptom, as it is found both in the healthy and in the diseased. The most important work in this department has been done in very recent times, and especially, on the one hand, by the discovery of auscultation, by means of which a whole group of cases has been removed from the department of nervous diseases and put upon a more satisfactory basis; and, on the other hand, by the excellent experiments by means of vivisection (Goltz, Landois, von Bezold, Ludwig and Thiry, Cyon, Guttmann, and Eulenburg), which put us in a position to give at the present time, for many forms at least, a satisfactory explanation.

Etiology.

In order to make a comparatively clear statement of the causes of palpitation of the heart, we shall find it necessary, in accordance with the definition given above, to collect, first of all, what we know of the relations of the forces of innervation of the organ, as we learn them from the most recent investigations. The numerous discrepancies found in the various observations of different investigators are sufficient to show that we are not in a position at the present time to give an adequate explanation of all the facts which have been observed.

I shall confine myself to quoting the most important of them.

1. From the experiments on animals, where hearts were removed from the body and continued beating for a considerable time, it appears certain that the cause of the rhythmical contrac-

tions lies in the heart itself, in those ganglia which we find imbedded in its substance, both in the auricles and in the ventricles. Certain irritants may act upon this automatic apparatus of innervation so as to cause either an increased functional activity or paralysis (Landois[1]), and it is found that weak solutions of certain poisonous substances cause an excitement, and strong solutions a paralysis. We must, however, remember that what we learn by vivisection in animals does not always hold good in human beings, for the arrangement of the above-mentioned ganglia is very different in different classes of animals.

2. Other excitors of cardiac action are found in the cardiac branches of the ganglion stellatum, which pass down to the heart between the aorta and the pulmonary artery. They take their origin from the fibres which pass from the cervical portion of the sympathetic, as the long and the short root, to the above-mentioned ganglion (Roever and Bezold).

3. From the investigations of Bezold we learn that nerve fibres, originating in the medulla oblongata, run the length of the spinal cord, and that by irritating these, the functional activity of the heart is increased. They pass out from the cord with the spinal nerves, and become entwined with the sympathetic of the thorax and abdomen, whose branches in part extend from below upwards to the heart.

4. Finally, the sympathetic may exercise an influence upon the activity of the heart in still another way, viz., by causing a change in the tone of the vessels and an accompanying variation in the blood pressure. Irritation of the sympathetic causes a contraction of the vessels, and thereby increased blood pressure in the aortic system, and, as a result of the increased resistance, the labor of the heart is augmented, while a paralysis of the sympathetic, with the accompanying dilatation of the vessels, will cause a diminished resistance in the vessels, and a consequent diminution in the labor of the heart. Eulenburg and Landois[2] and Nothnagel[3] observed palpitation with symptoms of angina

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[1] Allg. med. Centralzeit. 1863. No. 89.

[2] Wiener Med. Wochenschr. 1868. No. 65.

[3] Deutsches Archiv für klin. Medic. III. 1867.

pectoris caused by a general spasmodic contraction of the arterial capillary system.

5. In opposition to these excitor nerves, we have the so-called restraining or inhibitory apparatus. This is made up of the pneumogastric and its ramifications. Thus if we irritate the vagus, the movements of the heart are slackened in frequency, and finally they come to a stand altogether in diastole, while after division of the vagi the heart begins to beat faster, for the power and influence of the automatic apparatus, so to speak, are set free. The fibres which cause this result, properly speaking, do not belong to the pneumogastric, but are fibres of the spinal accessory running along with it. The trick of the Indian juggler is now explained, for Donders has shown that he can slacken the heart's movement by the voluntary contraction of the muscles of the neck which are supplied by the accessory, since, by the irritation of those muscular branches of the nerve, the cardiac branches are simultaneously excited.

The same result can be attained by irritation of the fibres of the sympathetic in the abdominal cavity, for by such irritation (see investigations concerning palpitation by Goltz) the heart may be arrested in diastole, in the same manner as if we acted directly on the pneumogastric.

Since this ceases after section of both pneumogastrics or of the medulla oblongata, it must be that fibres of the sympathetic pass into the cord and along this to the medulla oblongata, from which the irritation is transferred to the pneumogastric (Goltz,[1] Bernstein[2]).

6. We must accord a wonderful influence to the *nervus depressor* shown by Cyon and Ludwig on guinea-pigs, and by the former also on horses, which nerve, arising from the pneumogastric or sup. laryngeal, by dilating the vessels, lessens the labors of the heart.

7. The blood has a powerful influence on the action of the heart and in various ways. Bezold[3] has shown by a very inter-

[1] Virch. Archiv. Bd. XXVI.

[2] Centralbl. für d. med. Wissen. 1863. No. 52. 1864. No. 16. Reichert's Archiv. 1864.

[3] Centralbl. für die med. Wissensch. 1863. No. 27.

esting experiment that the regularity of the heart's movements is dependent upon the constant and regular afflux of blood to the organ. If, after division of the pneumogastrics, the cervical cord, and the sympathetic in the neck, we compress the coronary arteries in the heart of a guinea-pig, the contractions are diminished in frequency, and, when we entirely close them, the heart's action will be completely arrested in a minute or a minute and a half. On withdrawing the pressure, the contractions begin again, and after a short time become regular.

The quantity of blood also has an influence on the movement of the heart. In regard to this point, however, observations are not all in harmony. It is certain that, after great loss of blood, palpitation occurs, and this agrees with the experiments of Bezold[1] on animals which he bled to death. He explains it by saying that, in consequence of anæmia, the brain is made abnormally irritable, and this irritability is transmitted to the excito-motor nervous system, and causes an acceleration of the heart's action.

Finally, the chemical combination of the blood is doubtless of great importance to the action of the heart, probably both by the disturbance it occasions to the sub-endocardial ganglia, and by its irritation, through the branches of the coronary arteries, of those which are situated in the muscular tissue.

As regards the gas which the blood contains, it is not yet settled whether the greater motor excitability lies in that containing more oxygen or more carbonic acid. We have already spoken of the effects of mingling various poisonous substances with the blood.

Now, if we look through the various causes of nervous palpitation of the heart, we find that mental excitements of the most various kinds, such as joy, fear, and anger, have a very sudden influence on the functional activity of the heart; nostalgia belongs in the same group.

What Livingstone writes concerning the free men of Central Africa who have been dragged away into slavery, is very interesting; they all complain of pain in the region of the heart, and

[1] Med. Wochenschr. 1867. No. 19. S. 291.

place their hands exactly on the spot where the organ pulsates, although they themselves believe that the heart lies much higher up under the sternum. With this as the prominent symptom, they speedily die. A boy of twelve years of age, who in a very short time languished in this way, declared that nothing ailed him but a pain in the heart. Doubtless, we have here a direct irritation, extending from the brain to the medulla oblongata, and thence to the excito-motor nervous system of the heart.

Diseases of the brain and of the spinal cord, or of its coverings, hyperæmia and inflammation of these organs, tumors within the calvarium and in the spinal canal, psychoses of the most various kinds, hypochondria and hysteria, may any of them occasion an excitement of the heart's action, partly by throwing out of play the restraining influence of the vagus, and partly by irritation of the excito-motor nervous system. Doubtless, sometimes the one and sometimes the other of these is the case. A temporary irritability of the central nervous system must be the cause of that kind of palpitation which we observe in individuals who are exhausted by protracted night-watching, or by great venereal or other excesses. In the same way tumors of various kinds in the course of the nerves in the neck and in the thorax, or inflammatory processes in the nerve sheaths, will have either an inhibitory or an exciting effect upon the movements of the heart. The same holds true of irritations which arise in the course of nerves apparently far removed from the heart.

Thus the most various diseases of the abdominal cavity, such as over-distention of the intestines with gas, worms, gall-stones, renal calculi, and diseases of the genital apparatus, transmit the irritation, through the fibres of the sympathetic which supply them, to the spinal cord, and, by the fibres running in this, up to the medulla oblongata, and thence out to the heart.

General plethora, especially in young persons, very readily occasions palpitation, probably through the increased irritation of the excito-motor nervous apparatus, perhaps through the greater pressure of blood in the coronary arteries, and perhaps purely in a mechanical way, by the increased resistance in the peripheral circulation. In the same way palpitation is undoubtedly produced by a temporary or partial hyperæmia in

the region of a given plexus of nerves. Thus palpitation may arise from a suppression of the menstrual flow, or of a bleeding from hæmorrhoids, through the consequent hyperæmia in the spinal canal.

We can fairly assume that it is a change in the quality of the blood which occasions the very troublesome palpitation observed in chlorosis and in anæmia after diseases of the most various kinds. Whether in these cases it depends upon paralysis of the inhibitory apparatus or irritation of the excito-motor apparatus, we cannot at present decide. Very frequently young persons in the first stages of tuberculosis, or those who afterwards become tuberculous, complain only of troublesome palpitation of the heart. Here it is possible that changes in the quality of the blood cause an irritation, and also that the immediate disease of branches of the pneumogastric in the lung destroys the power of the regulating apparatus of the heart.

Finally, there are a number of substances, the use of which will occasion more or less palpitation, different in different individuals. We should mention among them, especially, alcohol, coffee, tea, and strong tobacco. They certainly act in various ways upon the nerves of the heart. Perhaps we should speak in this connection of the palpitation which occurs in gout, for if we allow that the blood is laden with a poisonous material, we shall see that this of itself would be the cause of irritation. It is more probable, however, as changes in the peripheral ramifications of the nerves underlie the manifestations of this disease in other parts of the body, that the same holds true of the nerves of the heart, but wherein these changes consist is as yet unexplained. The palpitation which occurs in healthy men after severe physical exertion admits of various explanations, but as yet we can give nothing positive.

Pathological Anatomy.

On this point very little is known, for we are lacking in pathological discoveries in the bodies of such as have been under careful observation during life; the most important and valuable of these is a case described by Heine (Müller's Archiv für

Physiol. 1841), observed by Skoda, and on which Rokitansky made the autopsy, where the right phrenic nerve was found enclosed in a firm, blackish-blue nodule, which was partially calcified. The nervus cardiacus magnus was also embedded in a blackish nodule of the size of a hazel-nut, and, finally, the branches of the left pneumogastric, which ran down on the anterior face of the left bronchus to the plexus pulmonalis, were crowded and pushed aside by lymphatic glands, with the same blue-black pigmentation. (During life the patient had suffered from paroxysms in which the heart's action would be arrested for many seconds, accompanied by a most frightful feeling of distress and anxiety.)

Symptoms.

Palpitation usually comes on in attacks of variable duration, lasting several minutes, or hours, or even a whole day. Very frequently they occur without any apparent cause, even in the sleep of an individual otherwise healthy, or else they follow upon certain recognized conditions, such as overeating, constipation, or fright.

Together with the troublesome palpitation and hammering of the heart, the patients complain of more or less dyspnœa, distress, and even pain in the breast; not unfrequently the throbbing in the carotids or in the head is more troublesome than the palpitation itself. Many patients cannot lie down, or at least cannot rest on the left side, and only find relief in an upright position, which is natural with the existing dyspnœa. In another case we may find dizziness, a feeling of faintness, specks before the eyes, or flashes of light associated with the palpitation. Flushing of the face and elevation of the temperature certainly is not found in all cases, but frequently there is pallor and cold sweat. Sometimes the patients complain of palpitation without our being able to discover it, and this must be explained as being due to exaggerated sensibility.

The *percussion* sound is not changed. *Auscultation* discovers heart sounds of increased intensity, and this is true particularly of the first sound, which is also sometimes metallic in quality. The

second sound is wanting only in cases of tremendous acceleration of the heart's movements, where the heart has not had time fully to complete its diastole.

The *pulse* may vary greatly in its frequency; cases have been observed where the contractions ran up to 200 per minute, and over. J. Payne Cotton (Brit. Med. Journal, 1867, June) relates the case of a man, forty-two years of age, in whom the sphygmograph registered as high as 240 contractions per minute in his most severe attacks of palpitation. After getting rid of a considerable quantity of tape-worm, he recovered. Occasionally the change from an accelerated pulse to the normal is a gradual one, but often it is very sudden. The pulse may be very strong and regular, but often it is small, scarcely perceptible, and highly irregular. This last depends upon the fact, that the heart, in the rapid succession of systolic movements, has not sufficient time to complete its diastole, to provide itself with the adequate amount of blood, and accordingly can only throw a small stream into the aorta, whose impulse is not felt at any great distance in the arterial system.

Diagnosis.

This is usually easy, but it is sometimes difficult to distinguish palpitation from organic cardiac diseases. During an attack we are often unable to make an accurate diagnosis. But when the heart's action is again quiet, the absence of murmurs or of enlargement of the heart will establish it.

It is also important to distinguish it from cases of general disease, accompanied by accidental murmurs; of these we should specially mention chlorosis, in which, indeed, a temporary enlargement of the heart may also exist. Here the diagnosis will be made certain by the presence of the other symptoms of chlorosis.

As regards murmurs, we may lay down our rule, viz.: if there is a diastolic murmur we can no longer consider it simply palpitation, for such murmurs never occur without organic changes in the heart.

We have already said, in speaking of organic disorders, how

difficult it is to make a diagnosis of cardiac diseases which are not accompanied by a murmur; especially is this true of myocarditis and fatty degeneration.

Course. Prognosis.

Though these attacks are a source of great uneasiness, and though the patients suffer from the constant dread of their recurrence, yet it is rare that they have any serious results; this is the case only when other diseases are also present, as, for instance, atheroma of the arteries in old people. Here apoplexy may occur during the attack. When the attacks are very severe and frequently repeated, the patient's strength may be considerably reduced by them, but usually he recovers his vigor before another attack. Finally, the nature of the underlying cause and the possibility of its removal will enable us to decide as to the probability of a cure. If, for instance, the trouble has arisen from long-continued night-watching, or from the immoderate use of tea, tobacco, etc., when the cause is removed the heart will soon become quiet again, while if it be due to a tumor in the brain it is necessarily incurable.

Treatment.

It is not my purpose to enumerate here all the remedies which have been employed in palpitation, but only to speak of those which have proved more or less reliable. First of all, we must endeavor to cure or to remove the underlying cause, as far as possible; and to this end must employ the appropriate remedies, and lay down proper rules regarding daily life and habits. At the same time, we must employ a symptomatic treatment calculated to lighten the attacks or to quiet them as soon as possible. As one of the most desirable and often quickly effective, as well as completely harmless remedies, we should mention the application of cold, whether in the form of cloths wrung out in cold water or of ice-bags laid over the heart. The next most valuable is the use of digitalis and of the narcotics, and of these, as acting most speedily, of morphine, which, when

the attacks are very severe, we can best employ in the form of hypodermic injections. The preparations of hydrocyanic acid and hydrate of chloral are often very serviceable. We rarely find it necessary to resort to chloroform inhalation, and this should be done only as a last remedy, for patients readily acquire the habit of using it themselves. I am not disposed to accord such wonderful results to the use of the constant current as have been ascribed to it elsewhere.

CONGENITAL DISEASES

OF THE

HEART.

LEBERT.

THE CONGENITAL DISEASES OF THE HEART.

Complete collections of the numerous individual observations, at present far more than a hundred, are to be found in the larger works of *Peacock*, *H. Meyer*, *K. Stölker*, *Kussmaul*, and others. I content myself here with giving especially the works in which the collected observations are critically considered.

Meckel, De cordis conditionibus abnormibus. Dissert. Hallae. 1802—*Schuler*, Dissert. de morbo coeruleo. Oiniponte. 1810.—*Haase*, Dissert. de morbo coeruleo. Lips. 1813.—*Horner*, Dissert. de cyanosi. Monachii. 1823.—*Bertin*, Traité des maladies du cœur. Paris. 1824.—*Bouillaud*, Traité clinique des maladies du cœur. Paris. 1834, and 2. ed. 1841—*Louis*, Mémoires et récherches anatomo-pathologiques. Paris. 1826.—*Hope*, A treatise on the Diseases of the Heart, etc. London. 1832.—*Friedberg*, Die angeborenen Krankheiten des Herzens. Leipzig. 1844.—*Peacock*, Med. Chir. Transact. 1847; Monthly Journ. 1847; On malformation of the heart. London. 1858.—*Rokitansky*, Handbuch der pathol. Anatomie. Bd. II. 1854.—*Dorsch*, Die Herzmuskelentzündung als Ursache angeborener Herzcyanose. Erlangen. 1855.—*H. Meyer*, Ueber angeborene Enge und Verschluss der Lungenarterienbahn. Virchow's Archiv. 1857. Bd. XII.—*Bamberger*, Lehrbuch der Krankheiten der Kreislaufsorgane. Wien. 1858—*Friedreich*, Die Krankheiten des Herzens. Virchow's Pathologie. Bd. V. 1861.—*Duchek*, Die Krankheiten des Herzens u.s.w. Erlangen. 1862.—*C. Heine*, Angeborene Atresie des Ostium arteriosum dextrum. Beitrag zur Lehre von den angeborenen Herzanomalien. Tübingen. 1861.—*Halbertsma*, Med. Tijsdschr. v. Geneesk. VI. p. 45. 1862.—*Stölker*, Ueber angeborene Stenose der Arteria pulmonalis. Dissert. Bern. 1864.—*Kussmaul*, Ueber angeborene Enge und Verschluss der Lungenarterienbahn. Zeitschrift für rationelle Med. Leipzig. 1866.—*Lebert*, Ueber einen Fall von Ursprung der Aorta aus dem rechten Ventrikel mit Enge der A. pulmon. u. s w. Virchow's Archiv. 1863. S. 405.—*Mannkopf*, Ueber Stenose des Ostium arteriosum der rechten Herzkammer. Charité-Annalen. Bd. XI. Berlin. 1863.—*Lebert*, Ueber den Einfluss der Stenose des Conus arteriosus, des Ostium pulmonale, und der Pulmonalarterie auf Entstehung der Tuberkulose. Berliner klin. Wochenschrift. 1867.—*Schipmann*, Ueber angeborene Stenose und Atresie des Ostium atrio-ventriculare dextrum. Dissert. Jena. 1869. *Lebert*, A clinical lecture on congenital pulmonary stenosis. Medical Times and Gazette. London. 1870.

Brief Historical Remarks.

The observation of congenital malformations of the heart was formerly merely a matter of scientific curiosity. They were unreservedly placed among abortions, concerning which mystical and utterly unscientific views prevailed. Only since the end of the last century, and especially at present, have the anomalies of the formation of the heart been subjected to thorough and scientific research. Though Morgagni and Sandifort describe some cases with their usual precision, Meckel was the first to collect very carefully in his archives his own observations and those of others ; his deductions, however, were influenced by the erroneous doctrines which still ruled. It was an after effect of the unfortunate system of natural philosophy, that he compared abnormally formed hearts according to their construction and internal divisions, with those of insects, crustaceans, and amphibians. In those days, indeed, the human embryo and its individual parts were thought to travel upward through the rising scale of development of the animal kingdom, and arrests of development were thought to be due in part to the stopping of an organ's progress at the type of a lower form. Next, with similar impropriety, excessive and inadequate development came to be opposed to each other ; and, of course, primary and secondary changes, stenosis and atresia on the one hand, and the persistence of the openings for the fœtal circulation and of the ductus arteriosus on the other, were thrown together in "most admired disorder ;" indeed, the effect was often taken for the cause.

These two phases, of which one may be called that of curiosity, and the other that of purely anatomical research, were followed by the truly rational etiological one, and in the last ten years by a tendency which may properly be called the physiologico-etiological one. The first of these, which recognized the full importance of inflammation of the openings of the right side of the heart as a starting-point, opened a new road, but became perfect by the influence of the more eclectic and general view. Rokitansky was the first to put this causal relation on a firm

basis. Dorsch's excellent description of fœtal myocarditis, made under Dittrich's guidance, was a great step in advance, which brought a new and very important element into the discussion. Peacock has the great merit of having not only collected all earlier observations, but of having admirably annotated them. He was able to guard himself against the one-sidedness of the inflammation theory, and thus paved the way for the present eclecticism. H. Meyer, of Zurich, has, with great talent and remarkable anatomical knowledge, correctly divided into groups the various combinations of congenital cardiac malformation, and very acutely pointed out the causes of their presence or absence; but he carries the inflammation theory too far. C. Heine's and Halbertsma's objections to his views are in part well founded; but their theories are much less satisfactory. According to my convictions, it is Kussmaul, who by a deep understanding of anatomy and physiology, and by great comprehension of the signification of the whole question, has brought it to the limits of our present knowledge. Hence, I shall follow particularly his classification in the description which I am about to give. Even if I, too, could increase the number of observations by a very interesting one, I should still think to have done more to advance the study of stenosis of the pulmonary artery, by making its decided influence as a cause of the development of tubercle in the lungs the subject of thorough research. I wished also, by describing a case of congenital malformation of the heart—a pulmonary stenosis—in a child, which was examined only during life, to prove to my colleagues, by an example, that cases observed during life, even when the patient may survive many years, deserve to be made public. In fact, the clinical part of our knowledge of this subject is far behind the anatomical. Another anatomical gap, which is greatly to be regretted, is our incomplete acquaintance with intra-uterine cardiac defects, from the first appearance of life until birth.

Although, according to what has been said, the errors of formation and diseases of the ostia of the right side of the heart determine the pathology of congenital diseases of the organ, yet before we come to them we must speak of the anomalies which are not to be referred to these causes.

Acardia, usually accompanied with acephalia, is of only anatomical interest; the umbilical vein in the body of the mother supplies the circulation to the fœtus, which is unfitted for life. The heart may be abnormally small, either with a structure otherwise normal, or with only two or three cavities. The too great size of the heart usually shows, when pulmonary stenosis is not present, the transition to double heart, and then is commonly, with double vessels, a suggestion or even the consequence of a double monster. Changes of the apex of the heart are for the most part secondary.

Among the *abnormal positions* of the heart, that in which it lies *outside of the body* is particularly interesting. I have seen such a new-born child still alive, and have been able to study the movements of the heart very well on it. The unfitness of such children to live is self-evident, but cases of congenital fissure of the sternum, the heart being in other respects normally placed, may give rise to very interesting physiological conclusions. The heart is occasionally found *outside of the thorax*, in the abdomen or in the cervical region. The transposition of the heart to the right side of the thorax is less rare and more consistent with prolonged life. In such cases the liver is usually on the left, and the spleen on the right. In the cases which I have observed, this *transposition* had no bad effects. The heart, *consisting of a tube, of a ventricle and an auricle, or of a ventricle and two auricles*, is rather a matter of anatomical interest, and the heart of two or three cavities may often owe its peculiarity to anomalies of the ostia of its right side.

The real pathology of congenital malformations of the heart indeed depends in general upon these.

Congenital Narrowness and Closure of the Ostia of the Right Side of the Heart.

Here, also, the diseases of the tricuspid opening are much rarer and of much less consequence than those in the course of the pulmonary artery from the beginning of the conus to the subdivision, and even further.

In the first place, changes at the origin of the pulmonary

artery are of importance, according to the time of their development. If the inter-ventricular septum is wide open, we may infer that they occurred before the third month, but its closure allows us to assume that they were developed later. It is remarkable that Hunter[1] appears to have been aware of this fact. The current of the blood, of which little or none can escape by the pulmonary artery, necessarily presses through the open septum, making its closure impossible, and giving the opening the appearance of a round hole with smooth edges. Transposition of the great vessels works in a similar way, the current from left to right preventing the closure of the fœtal openings. In rare cases a congenital opening, usually a small one, persists in the septum of the ventricles, but for the most part it is not the only anomaly. Still more rarely do we meet, as von Dusch did, with the remains of inflammation here and in the neighborhood. A group of cases in which an inflammation has perforated the already closed septum and attacked the pulmonary opening, rests on a much more theoretical basis. Only one case, reported by Whitley,[2] can be certainly thus interpreted. An important remark of Kussmaul's, on which von Dusch and Mannkopf have laid great weight, is, that when the stenosis occurs at a very early period, the pulmonary artery remains exceedingly narrow behind the obstruction, but that when it occurs later the artery may be of normal capacity, and, if insufficiency exists as well as obstruction, it may even be dilated.

The classification according to position of the defects of development, which we are now considering, is of great importance, and especially the distinction which has been insisted upon by Rokitansky and Dittrich, between stenosis of the conus arteriosus and that of the pulmonary artery, to which, as not uncommon, are to be added mixed stenoses.

Stenosis of the conus appears either as a supernumerary ventricle cut off at its lower part, or as a more regular narrowing, or as a conical stenosis under the origin of the pulmonary artery. In this case, the chief point of narrowing, or the closure, is at

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[1] Med. Observations and Exp. Vol. 6, p. 305.

[2] Guy's Hospital Report. 1857, p. 252.

the ostium, and the pulmonary trunk is contracted or even transformed into a solid cord; the branches receiving their blood through the ductus arteriosus. If this latter is poorly developed or wanting, the branches, which can obtain their blood only through the enlarged bronchial arteries, are usually small. Mixtures of both groups of stenoses occur in many combinations.

The genetic difference proves, indeed, the great frequency of the inflammatory origin of stenosis or atresia in the course of the pulmonary artery, yet in a number of cases not only is this cause wanting, but it often is impossible to decide whether or not inflammation had occurred at an earlier period. Thus even the fœtal myocarditis, with its callous scars, stands opposed to non-inflammatory changes, caused by excessive growth, and here undoubtedly belongs Kussmaul's second case, in which he assumes an excessive muscular development as the cause of stenosis and atresia of even the venous ostia of the heart. Peacock's theory also is interesting in which he suggests hypertrophied muscular substance as the cause of a ring-like stricture at the apex of the conus in stenosis of the pulmonary artery.

Although in cases of stenosis of the pulmonary artery of the fœtus undeniable signs of inflammation of the endocardium of the valves can be demonstrated, such as growth, thickening, growing together, hardening, and calcification, nevertheless such signs are totally wanting in other cases, and, though rarely, the congenital non-inflammatory stenosis may lead to secondary endocarditis. If instead of three valves, two or but one are present, the inflammatory adhesions between the original ones can usually be shown. I have almost always found this to be the case, and also with the aortic valves of the adult.

Peacock traces the preference of fœtal endocarditis for the right side of the heart to the continuation of the pulmonary artery into the descending aorta and umbilical arteries. He believes that the frequency of inflammation at the origin of the pulmonary artery is to be explained by the readiness with which transitory interruptions of the current may occur in the umbilical arteries and placenta, just as in after life the manifold variations of blood pressure in the arterial system may give rise to disease

of the origin of the aorta. Here also should be mentioned the point that Friedreich insists upon, namely the greater pressure on the valves of the left side of the heart after birth, the reverse being the case during fœtal life.

Meyer maintains that fœtal entarteritis may also be a cause of stenosis, and even of obliteration and shrinking into a cord of the pulmonary artery; but satisfactory proofs are wanting in support of this view, which is theoretically possible. According to Gregory, cicatrices from pericarditis at the beginning of the pulmonary artery may cause stenosis; but this is certainly a very rare etiological occurrence.

Of much greater importance is Peacock's explanation; namely, the possibility of defective development of the branchial arch from which the ductus arteriosus is formed, as in that case the pulmonary artery of the fœtus would receive a much smaller quantity of blood. Then Kussmaul mentions the original defective and inadequate development of the pulmonary artery, which Ecker calls its primitive insufficiency. Defective development of the lungs may be followed by that of the artery, as also primary stenosis of the conus and primary atresia of the venous ostia of the right side are followed by secondary narrowing and closure of the pulmonary artery.

Thus becoming convinced of the number of causes, we renounce every exclusive theory and so necessarily come with Kussmaul to a classification of cases of this kind according to a mixed plan.

In the first class are the cases: *of stenosis and atresia of the pulmonary artery with closure of the septum.*

Narrowness and closure are in these cases primary.

The only difficulty is in deciding whether the stenosis is congenital or acquired after birth. There is less doubt in complete atresia than in stenosis; for in the former case, according to Kussmaul's analysis, the duration of life is usually very brief, one mostly of a few days or weeks, and never reaches an entire year; while with undoubtedly congenital stenosis an age of sixty-five years has been attained. There are eight observations of complete closure (Kussmaul). In these cases the right ventricle is contracted almost to nothing, yet a case of Rokitansky's is an

exception, as is one of Schuler's, in which the contraction was almost complete. The stunting (of the ventricle) is probably in relation with the early origin of the affection at a time when after the closure of the septum the ventricle was but slightly developed. In these cases the foramen ovale as well as the ductus arteriosus are always open. In atresia, either the artery is transformed into a cord, or the ostium is closed, with a narrowing of the artery above it, or the apex of the conus is closed below the atrophied valves.

Owing to a kind of compensation, the disease of the heart may remain latent for a long time, and in many cases it is only after a series of years that symptoms appear, which will be discussed later; at present the anatomical appearances are of much greater signification. The simultaneous persistence of the foramen ovale and of the ductus arteriosus is characteristic, especially when they are larger than usual. If only one of these openings persist, the latter is the more significant, as the foramen ovale may continue open to a slight extent without any disease; moreover, both these fœtal passages normally become closed only in the course of the first month of life, so that they may remain widely open in consequence of an inflammation in the right side of the heart, which did not begin till after birth. Nevertheless, it may be assumed that a marked narrowing of the orifice of the pulmonary artery has occurred before birth, and the same may be said of great stenosis of the pulmonary artery with a vein-like thinness of its walls. Kussmaul condenses all the elements excellently into the following propositions. This affection of the heart is the more surely congenital: 1st, when the birth was near the normal end of pregnancy; 2d, the sooner after birth cyanosis and other tokens of heart disease, collectively called physical symptoms of stenosis of the pulmonary artery, are perceived; 3d, when the foramen ovale and the ductus arteriosus Botalli are both open, or, indeed, only the latter; 4th, when the opening of the foramen ovale is proportionally large, the ductus being closed, and especially when its size depends on want of the fleshy substance of the septum; 5th, when the valves of the pulmonary artery show anomalies of structure that are evidently congenital; 6th, when the trunk of the pulmonary artery is decidedly con-

tracted and its walls are too thin; 7th, when the right ventricle appears contracted, or stunted.

Stenosis of the Right Conus Arteriosus, with an Opening in the Interventricular Septum.

The conus arteriosus has either the shape of an extra (third) ventricle constricted off from the others, or it is very much stunted and contracted, or its apex is narrowed by a ring of hypertrophied muscular fibres. For accounts of these particular forms, the various authors, and especially Kussmaul, are to be consulted. The constricting off of the conus into a supernumerary ventricle occurs with either a narrow or a free communicating opening. Also in the purely cicatricial strictures of the conus, endomyocarditis with consequent cicatricial degeneration of the walls of the heart may have followed the primary muscular stenosis, as is suggested by the preponderant frequency of the seat of the stenosis precisely at the point of passage of the conus into the sinus. Remarkable changes in the valves of the pulmonary artery are usually found in cases of an extra ventricle; the valves generally are fewer than normal, exceptionally more, and are still more rarely entirely wanting. In these cases, moreover, the pulmonary artery is generally decidedly contracted, and sometimes as thin as a vein; the sinus of the right ventricle is then commonly large and hypertrophied; the greatly expanded aorta arises usually from both ventricles, seldom from either the right or the left one alone; the persistence of the foramen ovale is much less common, and even the ductus arteriosus is usually closed. Life may endure for a considerable time—from twenty to forty years and more, often from ten to twenty. The uniform narrowing or closure of the right conus arteriosus is relatively rarer. In this case also often only two pulmonary valves are present. The pulmonary artery is as a rule much narrowed, occasionally with thin walls; the aorta springs from both ventricles; the right side of the heart is dilated, and the right ventricle hypertrophied; the foramen ovale remains open, the ductus arteriosus being sometimes open and sometimes closed. These patients also for the most part die from infancy to twenty-five years of age. The

annular narrowing of the apex of the conus is rare, and has been described only by Peacock and Oldham.

Simple Stenosis and Atresia of the Pulmonary Artery, with an Opening in the Ventricular Septum.

Kussmaul considers this a simple form only under the following conditions: 1st, When the differentiation of the pulmonary artery from the common truncus arteriosus is complete; 2d, when auricles and ventricles can be distinguished by their septa, though they are not fully separated; 3d, when the pulmonary artery arises from the right ventricle alone, and the aorta from both or only from the right; 4th, when the position of the aorta is normal in relation to the pulmonary artery, or is originally more to the right of the latter till it takes an antero-posterior instead of a lateral course; 5th, when there are no anomalies of importance at any of the other ostia of the heart.

Of all the stenoses and atresiæ in the course of the pulmonary artery, the simple ones are the most common. Kussmaul found ninety cases of this class, among which were twenty-six atresiæ. The pulmonary artery, in stenosis, is commonly narrowed as far as its bifurcation, indeed often beyond it, but chiefly at its origin; it has often very thin walls, and sometimes it is quite stunted. The valves also present anomalies in their number and development, and may be grown together so as to form a cone or a funnel; they are occasionally thickened, covered with growths or calcified, but their development is rarely arrested. When the pulmonary artery is completely closed, it may be transformed into a cord or a thread as far as its subdivision. The conus, too, may be more or less obliterated. Occasionally stenosis of the pulmonary artery is accompanied with narrowing of the apex or of the whole of the conus; but, as a rule, the conus is only moderately contracted by the hypertrophy of its walls, while the sinus of the right ventricle is hypertrophied and enlarged. The right auricle is usually dilated with thickened walls, the tricuspid valve is sometimes opaque or even thickened. The left ventricle is smaller and thinner than the right, and only exceptionally excentrically hypertrophied. The valves of the

left side of the heart are rarely opaque or thickened. The aorta is dilated and springs usually from both ventricles, sometimes chiefly from one or the other, but its relation to the pulmonary artery is generally normal. The interventricular septum may present an opening towards the base of the heart, either limited to the membranous portion or extending into the muscular. The foramen ovale has been found open in about three quarters of the cases, yet the statements of authors vary greatly on this point, and weighty authorities maintain that it is frequently closed. The ductus arteriosus is sometimes open, but more often closed. The closure, however, of *both* passages, the foramen and the duct, is uncommon. In thirteen per cent. of these cases, as well as in numbers of conus stenoses, the ductus arteriosus is wanting, and the coincidence of narrowness of the pulmonary artery with absence of the duct is the basis of Peacock's theory, according to which the narrowness of the artery is frequently the result of the defective development of the branchial arch, from which the ductus arteriosus is formed. Anomalies occasionally occur in the course of the duct.

We have already seen that many forms of these errors of formation are not inconsistent with prolonged life, and thus the collateral passages, through which a comparatively regular circulation may be carried on, become of great interest. The ductus arteriosus is naturally the highway by which blood from the aorta enters the pulmonary arteries; if it is wanting, or closed, or too small, the bronchial arteries are usually enlarged. Peacock has observed the enlargement of the œsophageal arteries, and Jacobsohn that of the artery supplying the pericardium.

The duration of life is much longer in stenosis of the pulmonary artery than in atresia. According to Kussmaul's analysis of sixty-four cases of stenosis, eight died in the first year of life, fourteen between the first and the fifth, nineteen from the fifth to the tenth, fourteen from the tenth to the twentieth, and nine from the twentieth to the thirtieth. Of twenty-five individuals with atresia fourteen died in the first year of life, and, indeed, ten in the first six months, five from the first to the fifth year, one from the fifth to the tenth, three from the tenth to the

twentieth, and two at twenty-one and thirty-seven years respectively.

When there is an opening between the ventricles, life may persist for years, in spite of narrowness or closure of the pulmonary artery; thus to twenty-one years in Stark's case, and to thirty-seven, with closure of the artery in Voss' case.

Combined Stenosis and Atresia of the Pulmonary Artery.

Kussmaul distinguishes the following combinations:

1. That with partial persistence of the common truncus arteriosus; in this case the pulmonary artery is not divided from the aorta; more frequently the common truncus arteriosus survives only partially, the pulmonary artery being generally very small.

2. Combination with a bilocular heart. The internal partitions of the heart are absent and the division of the truncus arteriosus incomplete; the pulmonary artery is narrow or closed.

3. Combination with a single ventricle, in which a septum is at most merely indicated, and with an auricular cavity more or less completely divided into two (*cor triloculare biatriatum*). In cases of this combination, with narrowness or stenosis of the pulmonary artery, death usually occurs soon after birth; nevertheless life may endure for years even with this very complicated arrangement.

4. Combination with a subdivided ventricle and a simple auricle (*cor triloculare, biventriculare*).

5. Combinations with special anomalies in the position of the two great arterial trunks of the heart. The most important of the anomalies thus occurring are: *A*, transposition of the great arteries; *B*, origin of the pulmonary artery from the left ventricle and of the aorta from both; *C*, origin of the aorta from the right ventricle and of the pulmonary artery from both; *D*, transposition of the two arteries in an antero posterior direction, both arising from the left ventricle; *E*, transposition of both arteries in an antero posterior direction, the aorta arising from both ventricles and the pulmonary artery from the right.

6. Combinations with primary anomalies of some of the other

ostia of the heart. *A*, combination with primary anomaly of the right auriculo-ventricular opening. *a*, combination with rudimentary development of the tricuspid valve, observed only in one instance, by G. Hannotte Vernon, in the case of a girl seven years old ; *b*, combination with stenosis and atresia of the right auriculo-ventricular opening. Unfortunately the condition of the pulmonary artery is not always accurately described in these cases. In them the stenosis or atresia of this three-cornered venous orifice is usually primary, and if the passages of communication between different parts of the heart are closed or nearly so, the right ventricle and the pulmonary artery must be contracted ; but if the fœtal openings are present, that is, if the stenosis of the auriculo-ventricular orifice occurred in early fœtal life, the pulmonary artery may attain its normal dimensions. If the stenosis of the tricuspid opening is slight, the hypertrophy of the right ventricle may act as a compensation ; *c*, combination with stenosis and atresia of the left auriculo-ventricular opening ; *d*, combination with stenosis of the left arterial ostium.

I take from Schipmann's dissertation some remarks concerning congenital stenosis of the right auriculo-ventricular ostium. When the muscular substance, at the time of the formation of the septa, is excessively developed at the borders of the right auricle and ventricle, the communication between these cavities may be prevented by a fleshy wall. This is one manner of origin of a congenital obliteration of the auriculo-ventricular opening, *a simple anomaly of formation.*

If the development of the septum is disturbed before the end of the twelfth week by endo- or myocarditis, which are commonly followed by cicatricial retraction, the tricuspid valve may shrivel into a cicatrix, and the opening becomes obstructed or closed by the growing together of the valves—*arrest of development from fœtal endocarditis.*

If the right side of the heart is attacked by disease after the organ is fully developed, and stenosis or closure of the right tricuspid orifice arises, the communications that have been closed may reopen or be reproduced by perforation—*endo- or myocarditis after the twelfth week.* The defect in these cases is small, and the left ventricle is hypertrophied. Again, an

abnormal communication between the right auricle and the left ventricle may follow the closure of the right venous ostium. According to Schipmann's analysis of twenty-three observations, fœtal endocarditis appears to occur most frequently after the twelfth week, and it is often very doubtful whether it began before or after birth. Indeed, the first beginning may be in fœtal life and the subsequent and complete development of the obstruction at a later period. Nevertheless, complete growing together of the valves, with closure of the orifice, indicates that the process was completed *in utero*.

The cause of the development of all these anomalies is very obscure. The high blood pressure in the right side of the heart during fœtal life is not alone a sufficient cause, for even when the pressure is still slight, deep-seated alterations may be already developing.

Symptomatology.

In spite of many peculiarities, truly pathognomonic symptoms are entirely wanting. This is, in part, the result of the varying degree of compensation, which is often sufficient for many years. Although the gravest malformations of the heart may kill before birth or in a few days, weeks, or months after it, I have seen cases in which a very tolerable life could be maintained by some precautions, although from the earliest childhood the signs of a malformation were evident, and accompanied with many inconveniences. Here, from the very beginning, the compensation was inadequate, yet so far sufficient that life was not directly threatened. I opened my policlinic in May, 1869, with such a case. I was enabled to demonstrate to the students all the symptoms I am about to describe of this remarkable disease, on a boy of four years, who from his birth had been moderately cyanotic, and had suffered much from dyspnœa, but otherwise was well nourished.

In one class of cases the compensation is for a long time so perfect that for many years the patients feel none of the subjective annoyances of heart disease till either the compensation gradually becomes insufficient, and the symptoms of heart

disease appear progressively, or till the equilibrium is disturbed by some acute, intercurrent disease, a wound, or any pathological cause. In the case which I described in Virchow's Archives in 1863, the patient, who was twenty years old, stated at the first examination that till six months before he had been perfectly well. At that time, on lifting a heavy weight, he was suddenly seized with a severe pain in the front and top of the right side of the chest, with oppression and dyspnœa. He had since then complained of palpitation of the heart, and of a beating in the second and third right intercostal spaces. We have, indeed, seen in the anatomical sketch how rapidly death must follow certain congenital defects of the heart; but the later anatomical condition of patients whose troubles appeared late, and who for years had suffered little or nothing, often gives unsatisfactory information compared with that of cases in which the patients soon become blue and short-breathed. The patient in Voss' case of closure of the pulmonary artery reached, it is true, the age of thirty-seven years; but here the collateral circulation was greatly developed. Moreover, it may be assumed, in cases with prolonged life, that the constantly increasing alterations are continually making the balance more difficult. Again, secondary troubles, and especially the progressive development of tubercles of the lungs, are liable to shorten life, not only by these serious complications themselves, but also directly, to increase by them the disturbances of the circulation. Thus, although the anatomical condition does not reveal much, and the course of the disease depends greatly on the individual predisposition and on accidental complications, it may in general be taken for granted that the fact that the stenosis is slight or moderate, the persistence of a sufficient opening through the fœtal passages, and a good and adequately developed collateral circulation, in most cases favor the long continuance of compensation, and are elements of a favorable course of the disease.

When attention was first directed to congenital heart disease, the blueness (cyanosis) especially attracted the notice of physicians, and for a time was considered pathognomonic. Yet blueness, from the very beginning of life, is observed only in exceptionally grave disturbances of the circulation without compensa-

tion. The cyanosis, that rapidly passes off from children that come into the world nearly asphyxiated, persists in these severe cases, and is accompanied with superficial and rapid breathing; it extends from the lips and cheeks over nose and ears, the whole face and the extremities, and is increased by strong movements or crying; indeed, these may occasion transitory attacks of suffocation, in which convulsive twitchings are not uncommon.

Such children grow continually weaker, the skin becomes cold, and so they die either asphyxiated or in a slow collapse. But if the disease is of a nature to permit the development of a collateral circulation, the danger of suffocation, the great dyspnœa and the cyanosis decrease, the child becomes tolerably well nourished, dyspnœa and palpitation, though occasionally severe, appear only on exertion, and the child thrives and lives a bearable life, till either the compensation becomes less and less adequate, or till he is greatly shaken by an acute intercurrent disease, in which case he may either die rapidly asphyxiated, or recover, but to be more short-breathed and cyanotic than before. These children are generally feeble and anæmic, with a tendency to headache, faintness, and dizziness, yet occasionally they are strong and show no signs of imperfect nutrition. The hemorrhagic tendency of these children or grown patients, which was formerly considered common, is relatively rare, if we except the later hemorrhages from progressive pulmonary tuberculosis. Palpitation of the heart is common in many cases, but is wanting in others, or occurs only irregularly, or on exertion. These patients are not more liable to catarrh than others; the warmth of the body usually suffers, the extremities are cold, and the patients very sensitive to external cold. If a feverish condition occurs, the thermometer rises, at least according to an observation of mine, in the usual way, and also in the absence of fever, the temperature shows no diminution that would be considered abnormal.

If the symptoms of heart disease do not appear till late, the patients grow up as usual, and both sexes pass normally through the period of development; the menstruation in females is regular and not excessive, but the body is usually small and weak. The mental development exhibits the usual variations. But

these patients, after passing the age of puberty, have the compensation usually disturbed again, so that dyspnœa and cyanosis increase, and either spontaneously or after exertion lead to paroxysms of suffocation, which, in children, are often accompanied with violent fits of screaming. In rare cases death occurs during such attacks. Without going here into the cause of cyanosis, it seems to me, as in other heart diseases, to depend chiefly on the mechanical disturbance of the circulation in the veins and in the capillaries, though the incomplete oxidation of the blood in the lungs is to be considered as more than a secondary circumstance, as in cases of equally serious malformation, or when an adequate collateral circulation is established, the cyanosis is trifling or absent, but becomes just so much more marked as the collateral circulation is insufficient. With regard to the frequency of cyanosis, I find the following statement in Stölker's excellent dissertation. In 57 cases in which it was present, it appeared at or soon after birth 32 times ; from the fourteenth day to the end of the first six months, 9 times ; in the first year, 3 times ; in the second, once ; in the fifth, 3 times ; and 4 times it did not appear at all.

I may mention, however, in this connection, that the number 57 represents hardly half the known cases of congenital disease of the heart, and that, according to my experience and reading, the complete absence of cyanosis, or its late appearance and slight development, is by no means rare. Even when it exists, it is only exceptionally very marked on the face or extending over parts of the limbs, or perceptible on such mucous membranes as are visible. The frequently mentioned clubbed ends of the terminal phalanges of the fingers occur as frequently with pyothorax and tuberculosis, but are not uncommon in cyanotic children, and are then characterized by the blueness under the nail. Cyanosis of the face, hands, and feet is often accompanied with great development of the superficial veins.

Whether it be in childhood, or at puberty, or later (even very much later) that the congenital defect of the heart leads, owing to a continual decrease of the compensation, to the combination of symptoms characteristic of organic heart disease, at that time the dyspnœa will become greater, the asthmatic attacks more

frequent, and cough, diffused bronchitis, albuminuria, swelling of the liver and spleen, together with dropsy in all its forms, more and more marked. A decided hemorrhagic tendency is occasionally observed. The patients become cachectic and sink exhausted, unless some acute disease cuts them off. I propose, at the end of this discussion of symptomatology, to dwell in particular on the decided tendency of these patients to become tuberculous.

The possibility of an accurate diagnosis in every phase of the disease is greatly increased by the physical examination. The shape of the chest offers nothing characteristic; it is generally slightly developed, and often has a bulging corresponding to the size of the heart. The impulse of the heart is often indistinctly seen, the veins of the chest are large, and the jugulars, which are often swollen, show occasionally distinct undulations, as in a case which I have observed. In one case Hodgson mentions pulsations in them. The impulse of the heart is sometimes strongly, sometimes slightly felt; and when there are distinct murmurs, which are usually systolic, they are felt as a thrill, with its greatest intensity at the point of its actual production. In one of my cases a distinct thrill could be felt behind the top of the sternum. The frequency of the contraction of the heart and the pulse is not perceptibly increased as long as the balance is maintained, and the degree of tension and fulness is not abnormal, but there is rather a tendency to smallness, while, when the compensation ceases, fineness, rapidity, and irregularity of the heart beat and pulse appear in all degrees. Of course in complications with pulmonary tuberculosis, the pulse is small and rapid.

Percussion shows a perceptibly increased cardiac dulness, especially in a transverse direction and to the right, on account of the hypertrophy of the right ventricle, in cases of long standing. In extreme instances the dulness may reach from the anterior left axillary line far beyond the right border of the sternum. Thus the spread of the dulness to the right depends upon the excentric hypertrophy of the right ventricle. Auscultation shows a pretty constant sharp systolic blowing over the region of the heart, as to the cause of which various explana-

tions can be offered. In the case which I observed clinically, its greatest intensity was in the second and third intercostal spaces to the left of the sternum and behind that bone. In the other case, which afforded an autopsy, and in which the heart lay more to the right behind the upper part of the sternum, the greatest intensity was in the second and third right intercostal spaces; and it is very probable that the cause was to be sought in the tumultuous rushing of the blood from the right into the left ventricle, through the large round opening which existed, as the subsequent anatomical examination did not permit the explanation of the murmur arising at the valves. If the maximum of the systolic bellows-murmur is at the base of the heart, at the sternum, it usually extends far over the limits of the heart, but is not continued into the arch of the aorta. The existence of a diastolic murmur, mentioned in many observations, tends to indicate insufficiency of the pulmonary valves, as well as stenosis. Not unfrequently the systolic blowing is so prolonged that it obscures a part of the diastole. Nevertheless, stenosis of the pulmonary opening can exist without a systolic murmur, indeed without any murmur at all, as is shown by some cases. In a case of Frerichs', a diastolic murmur, strongest on the right, and extending over the vessels, coexisted with the systolic sound. Here a thickening of the tricuspid was found besides stenosis of the pulmonary valves, with but a small opening; but to bring the physical manifestations of all these forms of heart disease to fixed and general rules, they must be established by a greater number of cases, which shall have been accurately observed, both clinically and anatomically.

But there remains even more to be desired for the physical diagnosis of congenital affections of the tricuspid. Not only are the usual symptoms, such as cyanosis, dyspnœa, chilliness, cramps, clubbed nails, etc., the same as those of stenosis of the pulmonary artery, but the diagnosis is made still more difficult by the fact that in most cases disease of the arterial ostium of the right side of the heart coexists with that of the venous one. The more prominent, however, is the group of symptoms of tricuspidal stenosis, the more clearly is hypertrophy of the left ventricle to be made out; hence the impulse of the apex is felt

deep in the sixth intercostal space in the mammillary line, or outside of it. The pulse is usually small and often irregular, together with an extended cardiac impulse, and a systolic thrill. The hypertrophy of the left ventricle is generally clearly shown by percussion, although in rare cases it is claimed that there is no abnormal murmur; there is a sharp prolonged systolic blowing, but it is strongest at the lower part of the left border of the sternum and under the bone, and does not extend into the great vessels; a diastolic or a double murmur may be found in cases of great insufficiency, joined with stenosis, and there is also a systolic pulsation in the jugulars, and probably, if attention were given to it, also in the inferior vena cava in the region of the liver.

We have already seen that congenital heart disease is frequently fatal soon after birth or in the first weeks or months of life, or else in early childhood, but that, in consequence of a favorable arrangement of the collateral circulation, life may persist for some decades; but it may be stated, as a rule, even for these favorable cases, that middle age will not be passed. In addition to all the other disastrous results that may follow organic heart disease, such as sudden death, death by asphyxia, or by progressive disturbance of the circulation, dropsy, cachexia, etc., there is also the very remarkable tendency to pulmonary tuberculosis which, especially in cases of congenital stenosis of the pulmonary artery, hastens the end. This complication was formerly only occasionally noticed, as by Farre and Travers, Gregory, Louis, and Crefeld; but since autopsies have been more thoroughly made, such cases have so multiplied that the relation formerly thought much rarer, has in the last twenty-five years been observed in one-third of all cases of stenosis of the pulmonary artery, and there is hardly a disease so often followed by tuberculosis. I give here the literature of twenty-four cases of pulmonary stenosis followed by tuberculosis of the lungs.

I. Stenosis of the Ostium of the Pulmonary Artery.

1. *Farre* und *Travers*, Meckel's Archiv. 1815. S. 235.
2. *Gregory*, Med. chir. Transact. Vol. XI. 1821. p. 296. Meckel's Arch. Bd. VII. 1822. S. 238.

3. *Creveld*, Hufeland's Journ. Dec. 1826. S. 74.
4. *Lexis*, Hufeland's Journ. 1835. Arch. générales de Méd. 4. Ser. T. XIV. p. 433.
5. *Louis*, Mémoires et recherches anatomo-pathologiques. Paris. 1826. p. 313.
6. *Deguise*, Bulletins de la société anatomique de Paris. T. XVII. p. 180. 1843.
7. *Bertody*, Philadelphia Medical Examiner. May, 1845. Arch. gén. de Méd. 4. Ser. T. XVI. 1848.
8. *Shearman*, Provincial Medic. and Surg. Journ. July. No. 31. 1845.—Canstatt'sche Jahresber. III. 1845. S. 287.—Arch. gén. de Méd. T. XXIII. 4. Ser. p. 508. (Arbeit von Chevers.)
9. *Spitta*, Med. Chir. Transact. T. XXIX.—Canstatt. Jahresb. 1846. III. Bd.
10. *Escalier*, Bull. de la Soc. anat. Année XX. p. 213. 1845.
11. *Le Gros Clark*, Med. Chir. Transact. T. XII. 1847.
12. *Peacock*, Report of the proceedings of the Pathological Society of London. Second Session. 1847–8. (18. October 1847.)
13. *Frerichs*, Wiener medicinische Wochenschrift. Nos. 52 und 53. 1853.
14. *Dorsch*, Die Herzmuskelentzündung als Ursache angeborener Herzcyanose. Dissertation. Erlangen. 1855.
15. *Le Page*, De la cyanose dépendant de la perforation de la cloison interventriculaire du cœur. Thèse. Paris. 1858. Canstatt's Jahresb. 1859. T. III. S. 226.
16. v. *Dusch*, Verhandlungen des naturforschenden, medicinischen Vereins zu Heidelberg. T. VI. S. 183.—Schmidt's Jahrb. T. CIII. 1859.—Canstatt, 1859. 179.
17. *Wilks*, Transact. of the Pathol. Society of London. Vol. X. p. 79. 1850.
18. *A. Stark*, Rigaer Beiträge. IV. S. 20. 1859.
19. *Schützenberger*, Gaz. med. de Strasbourg. No. 3. 1861.—Canstatt. 1862. III. 203.
20. *Frerichs*, *Mannkopf*, Ueber Stenose des Ostium arteriosum der rechten Herzkammer, besonderer Abdruck aus den Charité-Annalen. Bd. XI. Berlin. 1863.
21. *Lebert*, Virchow's Archiv. T. XXVIII. S. 405. 1863.

II. Inadequately reported Cases of Congenital Disease of the Pulmonary Artery.

22. *Bouillaud*, Nouveau Journal de Médecine. Vol. VI. p. 223.—*Louis*, Mémoire cité. Obs. III.
23. Unknown Author, Archiv für medicinische Erfahrung. Januar, Februar, 1847. Berlin.—Archives générales de Medecine. I. Ser. Vol. XXV. 1827. p. 110.

III. Congenital Disease of the Tricuspid Valve.

24. *Ebstein*, Verhandlungen der Schles. Gesellsch. für vaterländische Cultur. 16. Februar, 1866. Separatabdruck.

It appears from this analysis, which I published in 1867 in the Berliner klinischen Wochenschrift, that, if we include

Ebstein's case of congenital disease of the tricuspid valve, and throw aside the two insufficiently reported ones, thirteen of the twenty-two cases occurred in males and nine in females. The age at death was from five to ten years in three cases; from eleven to fifteen years in four; from sixteen to twenty years in nine; from twenty-one to twenty-five years in four; from twenty-six to thirty in one; and from thirty-six to forty in one; so that death usually occurs in later childhood or in youth, and life is only exceptionally prolonged beyond the twenty-fifth year. I would also mention, as important, the fact, which was noticed in these cases, namely, that the parents and their other children were healthy, and that in these observations there is no history of inherited tuberculosis, so that it would be a wilful and very improbable hypothesis to lay the chief blame for the tubercular process on inheritance.

If we now cast a look at the heart diseases themselves, we find only once that the contraction of the pulmonary artery was relatively slight. It is true that in Bouillaud's case there is question of dilatation of the pulmonary artery, but I have classed this case among the uncertain ones, because the ostium of the right heart and the conus were not described, and because, in true stenosis of the heart, dilatation of the origin of the pulmonary artery may occur. Moreover, as the foramen ovale was very widely open, we may plausibly assume an impediment to the emptying of the right side of the heart. The same is probably true of the 23d case by an unknown author, for the ventricular septum was almost wanting, the ostia were not described, and it is merely generally stated that the arteries arising from the heart were wide. In the rare cases that I am acquainted with of insufficiency of the pulmonary valves without stenosis of the ostium and of the conus, I cannot remember any statement of a tendency to tuberculosis of the lungs without a congenital narrowness of the pulmonary artery. In fact, as a rule, the stenosis of the pulmonary artery is either very marked, or the vessel which has but two valves is very small from the first. In the relatively more favorable cases the ostium hardly admitted the little finger, and the valves commonly were grown to the funnel-like entrance, leaving a triangular or round opening, varying in diameter from

a few millimetres to two or three lines, and frequently growths were found around this passage which was already so small. In one case the vessel was even obliterated as far as the ductus arteriosus, but this remained wide open, and carried the blood into the pulmonary artery. True stenosis of the heart, in Dittrich's sense, existed in the conus arteriosus in not less than five cases, and even in several of these the valves that lay higher up were grown together, and thus increased the stenosis. In Ebstein's case, indeed, the tricuspid valve was rudimentary, but a large membrane existed which was in part connected with papillary muscles and tendinous cords. This divided the right ventricle into two halves, one corresponding to the conus arteriosus, the other to the rest of the ventricle, and the two communicated only through a rather large elongated oval hole and several small openings, so that at all events there was a great impediment to the flow of the blood into the pulmonary artery. The imperfect closure of the valve of the foramen ovale permitted the passage into the left auricle of some of the blood that was forced back into the right one. The thickenings, and even osteoid plates on the tricuspid valve referred to, in many cases did not indicate any disturbance of the circulation, but simply complication with endocarditis.

The most common malformation accompanying stenosis of the pulmonary artery, and almost necessarily caused by it, and which indicates that the whole process occurred before the end of the third month of fœtal life, is the persistence of a large opening in the septum of the ventricles, occurring in not less than fourteen cases. The foramen ovale was widely open in nine cases, and moderately in six. Complete cyanosis was present in six cases, and a slight degree of it in two; in the others it was either altogether absent, or showed only the lighter characters which are observed in advanced disease of the heart. The collateral circulation, which is always insufficient, takes place through the enlarged bronchial arteries, and in part through other branches of the aortic system, among which the œsophageal arteries, the coronary arteries of the heart, the subclavians, and others are mentioned. In this case the aorta is usually enlarged at its origin. The state of affairs is similar to that following con-

genital stenosis of the aorta in the neighborhood of the opening of the ductus arteriosus, in which there remains a collateral circulation, that, especially in the long run, is very insufficient. Thus we can decidedly lay down the law that in stenosis of the pulmonary artery the pulmonary circulation is irregular and greatly disturbed, and hence that, in a number of observations, we find smallness and imperfect development of the lungs.

Here, then, is the very opposite condition from that of disease of the left side of the heart, and particularly of the venous ostium, for in the latter case, although the back pressure from the left auricle extends so far into the heart that an excess of blood is driven through the enlarged pulmonary artery into the lungs and stagnates in the pulmonary veins, so that even capillary ectasis is common, yet in spite of the great overfilling of the lungs with blood, progressive tuberculosis is one of the rarest complications of disease of the left side of the heart. It is an interesting fact in the study of inflammation, as well as of tuberculosis, that a direct disturbance of the supply of blood is much more irritating and conducive to inflammation than a great but constant and regular increase of it. In the case of pulmonary stenosis, however, I would attach more importance to the unequal distribution of the blood than to the general poorness of the supply. It is a difficult question to decide how much the excessive supply of arterial blood to the lungs eventually disturbs their nutrition. I would not estimate this factor too highly, as precisely on account of the obstruction at the pulmonary orifice, a certain amount of venous blood passes from the right heart through the open septum into the left ventricle, and mingles with the arterial blood. It is further to be noticed that this mixed blood, having been able to exchange its gases through only a part of the lungs, which has always been very small, now flows throughout the entire body, and yet consequent centres of inflammation are very rare in other parts than the lungs. There are, moreover, a number of cases of abnormal communications between the cavities of the heart without stenosis of the pulmonary artery, and yet with these tuberculosis is the exception. A final argument that pulmonary stenosis is the chief cause of this complication is that, though inheritance has been shown to be of

relatively slight influence, the numerical proportion of those affected by the complication is decidedly greater than that among the same number of healthy individuals, or of those affected by any other disease, excepting, perhaps, diabetes mellitus in its last stages. The latter is certainly an exceptional condition in the etiology of tuberculosis, and one in which we may assume that centres of inflammation arise less from a general weakening of the system than from the irritation of the capillaries and alveoli of the lungs by blood which is overcharged with sugar and no longer properly oxidized.

If we now consider this complication more with regard to its relations to the lungs, we find that both clinical and anatomical research shows most clearly that it depends on no accidental coincidence, as might be the case with few foci, or with the remains of old ones, but on a progressive active and destructive process, which becomes more and more prominent with the progress of the disease. Up to the tenth year it is comparatively rarely fatal; it reaches its height in the second decade, and is then frequently the cause of death, but is rare after the twenty-fifth year; so that it seems probable that this complication, which is the result of stenosis of the pulmonary artery, is the most frequent cause of death in cases of the very heart disease that caused it.

In considering the progress of the lung disease, which appears as a complication, we find that a relatively rapid chronic course is the exception, and though in two cases death occurred three and four months respectively after the appearance of lung symptoms, it is probable that the disease was previously latent for a time. The patient's history, however, usually shows that the disease has existed for years. Louis estimated the duration, in his cases, at ten years. Frerichs' first patient had his first hæmoptysis sixteen years before his death, and had always coughed after it, often expectorating blood with ordinary matter. We find in the records of other cases that the patients have coughed and grown thin, especially in winter, and improved again during summer. We shall soon see in the anatomical discussion how grave the alterations in the lungs may be, and how much, especially in the way of old and large cavities, may be

found. An interesting fact may here be mentioned, namely, that in our cases not unfrequently all the changes were more advanced on the left side, and that also the physical signs often first showed themselves on the left side, so that to the other causes of disturbed circulation is to be added the pressure of the heart, the right side of which is greatly hypertrophied.

Tuberculosis, as a secondary disease and as a complication, usually pursues a latent course, and becomes prominent only in some respects, but, on the contrary, we find that the hectic and marasmic symptoms of chronic disseminated pneumonia in cases of pulmonary stenosis are always progressive, and that a number of patients die with such fully developed marasmus that we may ask ourselves whether, in consumption, we do not often overlook the starting-point, namely, the pulmonary stenosis, especially when no cyanosis is present, and the patient comes into the hospital in the last stages. Hence is deduced the important rule, to examine carefully the heart, particularly its right side, during life and after death, in every case of chronic disseminated pneumonia accompanied with a hectic marasmic condition.

The frequency of bloody expectoration is remarkable among symptoms which otherwise are simply characteristic of disseminated pulmonary inflammation, such as cough, dyspnœa, mucopurulent expectoration, digestive disturbances, and gradually progressing marasmus with hectic fever. In no less than twelve cases, besides the occasional spitting of blood, true bleedings occurred even repeatedly, and in six other cases the expectoration had often either a bloody color or was pure blood. The diarrhœa, cough, hoarseness, and the local symptoms in the peritoneum and brain, that occur not unfrequently in these diseases, were repeatedly observed. The physical thoracic symptoms were the ordinary well-known ones, only with the peculiarity already mentioned, that, relatively frequently, not only the original catarrh of the apex, but also the subsequent dulness and crackling râles, bronchial breathing, bronchophony, cavernous breathing, and pectoriloquy, appeared first or were most marked in the left upper lobe.

The anatomical appearances, also, are quite characteristic of chronic disseminated pneumonia with increasing infiltration,

destruction of tissue and formation of cavities, or they are such as are found in the form characterized by the growth of granular cells (tubercle-granulations). Adhesions of the pleura were found most frequently strongest at the top of the left lung, and adhesions with the pericardium were common. In a relatively small number of cases, five times in all, many disseminated foci were found alone (tubercles without cavities), partly gray and small, partly yellow miliary tubercles or larger masses, and even here we find, in one case, the deposit chiefly in the left lung. The pulmonary tissue was usually of a dark red, especially in cases of cyanosis. In most cases the chronic pneumonia was found in all stages, from that of small yellow and gray tubercles, mostly in the lower lobes, to that of disseminated or confluent centres of destruction (abscesses) in the upper ones, and particularly frequent in twelve cases there were cavities as large as the egg of a pigeon or as that of a goose, or even as a fist, without counting the many smaller caverns from the size of a pea to that of a hazelnut. Small centres of softening and decay are frequently mentioned, around which the lung tissue may be found dense, the alveolar structure either persisting or being lost in growths of connective tissue.

Fluid exudation was frequently found in the pleural cavity, and its walls were in some cases covered with small miliary tubercles. The bronchi were usually inflamed and also enlarged, and in one case their mucous membrane contained many small yellow tubercles. A large laryngeal ulcer was found only once. In Louis' case, the surface of the right auricle presented small miliary tubercles, which could be removed, with a false membrane, under which the epicardium appeared thickened and of a cloudy, white color. In Deguise's case, a yellow tubercle as large as a cherry-stone was found in the right lobe of the cerebellum, and several large, softened tubercles in the spleen. There was, moreover, caries of the ribs and of the ilium, the cause of abscesses observed during life. In one case there were several abscesses, but no tubercles, in the brain, and in Peacock's, the intestinal glands contained small tubercles and there were occasional ulcers in the bowels. In Lepage's case there was a cicatrized and a fresh hemorrhagic cavity, and several yellow

tubercles in the brain. In Schützenberger's case there were many tuberculous granulations in the peritoneum and in the mesentery. In two of Frerichs' cases, miliary tubercles were deposited in the liver and kidneys, and follicular ulcerations were found in the jejunum and colon.

After all this, there can be no doubt that all the anatomical characteristics of tuberculosis existed in these cases of stenosis of the pulmonary artery, and it is specially to be noticed that in almost all the cases the alterations were extensive and repeatedly combined with manifold deposits in distant organs. Thus it may be considered a rule, that the tuberculosis dependent on stenosis of the pulmonary artery is general and progressive, and that it is contrary to its nature to be slight, or to remain stationary, or to end in cure—another reason for considering the tuberculosis as a sequel of the disease of the heart.

Diagnosis.

It is evidently impossible to diagnosticate those cases in which the disease is slight and in which a compensatory arrangement has occurred early, perhaps even during intra-uterine life, and has become more and more perfect. Inasmuch as the ostia of only the right side of the heart become diseased before birth, and inasmuch as even when the venous ostium is greatly changed, still the arterial one has usually suffered more or less, the diagnosis turns essentially on the question of congenital stenosis of the pulmonary artery. Only when the disease of the venous ostium greatly preponderates, may the hypertrophy affect the left ventricle, as will be indicated by dulness extending more to the left, the impulse of the apex being at once lower and more external. The systolic murmur is heard lower and more to the right, and, finally, the pulsation of the jugulars can determine the diagnosis of stenosis or insufficiency of the tricuspid valves.

Congenital hypertrophy of the right side of the heart and a systolic murmur over the base, which is not continued along the aorta, are of the greatest significance in the diagnosis of stenosis of the pulmonary artery; indeed, these objective symptoms

must be very marked even when subjective troubles are wanting, and compensation is almost complete. It is, no doubt, conceivable that in cases of early equalization and slight stenosis, even these symptoms may be wanting, but such cases must surely be very rare. Unfortunately most of the clinical observations are not so accurate nor from such competent sources that we can always be justified in accepting the absence of abnormal murmurs, because no mention is made of them in the history. When cyanosis exists from birth or from very shortly after it, this is a very valuable symptom, especially when it is constant and intense. The feeling of chilliness and the habitual difficulty of breathing acquire much significance in conjunction with the extensive cardiac dulness, the thrill and the murmur. It is remarkable that in the few cases of congenital stenosis of the aortic ostium, the symptoms very closely resemble those of pulmonary stenosis; this lesion, however, rarely appears quite alone. On the other hand, if the aorta is narrowed at the opening of the ductus arteriosus, the symptoms of heart disease, as I have shown twenty years ago in my paper in Virchow's Archives, are, for the most part, absent during childhood. It is not till later that symptoms appear, as a secondary disease of the left side of the heart is developed. Moreover, many superficial arteries, as the intercostals, some of those of the neck, etc., are developed into pulsating cords, which is not the case in stenosis of the pulmonary artery. A case which I have observed proves that the symptoms may resemble those of an aortic aneurism; they appeared late in life and quite suddenly on lifting a weight; their chief seat was high up under the sternum and rather to the right; a thrill and pulsation could be detected behind the incisura sterni. Still, as in this case, the high position of the heart, however abnormal it might be, corresponded precisely to the seat of the murmur, and as no other pulsation at some distance from it could be found, as that of the heart would be in a case of aneurism, the diagnosis of congenital heart disease was probable.

Apart from the objective symptoms already mentioned, the diagnosis in most cases can be reached by the early appearance of difficulty in breathing, for though disease of the left side of

the heart is not rare in children, it is so in those under six or eight years; but in this disease the difficulty of breathing is usually sufficiently marked even in the earliest childhood to demand an examination of the heart, which, at least in the great majority of cases, would quickly dispel every doubt.

Prognosis.

The prognosis must always be unfavorable, even where excessive cyanosis, danger of suffocation, and extensive objective symptoms do not announce that the disease will soon be fatal. Even though the severe disease of the ostium has been counterbalanced for years, yet the compensation gradually becomes more and more insufficient, while, on the other hand, the decided tendency which exists to pulmonary tuberculosis, shortens the duration of life in a marked degree. Hence, if the signs of increasing failure of compensation appear, or if tuberculosis of the lungs is clearly made out, there is no question that the prognosis, already very grave, becomes decidedly worse.

In regard to the duration of life, I take the following table from Stölker.

From the statistics of the duration of life in the ninety-nine cases in which the age at death is stated, we find that

4	died on the first day.				
4	in the first week.				
6	from the second week	to the end	of the	third month.	
10	" "	fifth month	"	"	first half year.
18	" "	first half year	"	"	tenth year.
14	" "	eleventh year	"	"	fifteenth year.
8	" "	sixteenth year	"	"	twentieth year.
7	" "	twenty-first year	"	"	twenty-fifth year.
2	" "	twenty-sixth year	"	"	thirtieth year.
3	" "	thirty-first year	"	"	thirty-fifth year.
3	" '	thirty-sixth year	"	"	fortieth year.

Of course, in cases in which the disease of the heart, and especially the consequent troubles, are but slight, great prudence in the order of life, care of the diet and hygiene, with proper

rest and attention, will make the prognosis less unfavorable. Experience shows that acute intercurrent diseases are here more indirectly than directly dangerous through the permanent disturbance of the equilibrium of the circulation which they may occasion. The apparent greater danger and consequently worse prognosis for males is remarkable. Stölker states that of seventeen individuals who passed the nineteenth year, twelve were male and five female; a difference that is greater than the relative proportion of pulmonary stenosis in the sexes, which is about seven to four.

Treatment.

Unfortunately there is little to be said, from a therapeutical point of view, on this state of affairs, which both anatomically and clinically is so interesting. Nothing can be done by way of prophylaxis. If a child is born with signs of congenital heart disease, as cyanosis, dyspnœa, etc., some relief may be obtained by quiet and careful tending, by good milk from the mother or a wet-nurse, by the greatest cleanliness and care for pure air in the chamber, and by care in carrying and moving the child. The medicines, such as digitalis, morphia, etc., which are useful later, must be used with the greatest prudence in this earliest period of life. Eight minims of infusion of digitalis, with a little cinnamon water, and syrup of orange-peel, may occasionally be given several times in the day, and in case of great dyspnœa a few drops of the water of bitter almonds may be given, and counter-irritation made by mustard pastes, poultices mixed with mustard and spirits, and mustard over the region of the stomach or lower extremities, when severe paroxysms of suffocation occur.

Even if compensation is early established, or is gradually developed, the children are nevertheless to be brought up with the greatest care. They must neither run, nor leap, nor walk fast, nor climb—indeed, it is to some extent impossible for them to do so; but in fine, or even tolerable weather, they should, with proper precautions, live much in the open air; they should not be made to tax their minds severely, and should receive a

nourishing, unirritating diet, of which milk, meat, and eggs, with easily digestible vegetables and fruit, form the chief part, while exciting drinks, as tea, coffee, beer, and wine, are to be avoided.

Constant attention should be given to keeping the bowels open, but without heavy dejections, which is best done by the diet or by mild laxatives, as the lighter preparations of senna, small quantities of Ofener bitter water, and later by laxative doses of aloes pills. It would be a great error to give much medicine to such children, or to give it frequently, as to adults, or to accustom them to the habitual use of digitalis, which is to be used only occasionally for the severest dyspnœa, when it may be given in the ordinary doses, according to the age of the patient, in infusion. The preparations of opium also are to be sparingly given, and used only in certain phases, and in attacks of great difficulty of breathing. When it is possible, their place should be taken by chloral hydrate, in doses of fifteen grains. Every intercurrent disease is to be treated with the greatest care, even through convalescence.

Nothing can be done to ward off the threatened tuberculosis of the lungs except by the hygienic precautions already mentioned; perhaps at the time of puberty a prolonged stay during the winter in a southern climate may be found desirable, and occasionally cod-liver oil, either with or without iron, may be given for a time as a prophylactic. The same may be done with commencing disease of the lungs, which is to be treated for the symptoms according to the known rules.

DISEASES OF THE VASCULAR SYSTEM.

(ARTERIES, VEINS, AND LYMPHATICS.)

QUINCKE.

DISEASES OF THE ARTERIES.

Bamberger, Lehrbuch. d. Krankh. d. Herzens. Wien. 1857.—*Bouillaud*, Traité clin. des mal. du cœur. Paris. 1835.—*Corvisart*, Essai sur les maladies et les lesions organiques du cœur et des gros vaisseaux. Paris, 1806; 3d ed. 1818, translated by Hebb. London. 1813.—*Crisp*, A treatise on the structure, diseases, and injuries of the blood-vessels. London. 1847. Also, Appendix to treatise on the blood-vessels. London. 1851.—*Cruveilhier*, Anatomie pathologique. Paris. 1829.—*Duchek*, Handb. d. Patholog. u. Ther. S. 222. Erlangen. 1862. Vide also his Untersuch. über d. Arterienpuls. Zeitschr. d. K. K. Ges. d. Aerzte in Wien. 1862.—*Förster*, Handbuch der pathol. Anatomie. 2d ed. Leipzig. 1865.—*Balthazar Foster*, The sphygmograph in the investigation of disease. Brit. Med. Jour. March, 1866; vide also his Clinical medicine. London. 1874. p. 277.—*Friedreich*, Krankh. d. Herzens. Virch. Handb. d. Spec. Pathol. V. 2. —*Guthrie*, On the diseases and injuries of the arteries. London. 1830.—*Hamernik*, Physiol. pathol. Untersuch. über die Erscheinungen an den Arterien u. Venen. Prag. 1847.—*Hodgson*, Diseases of the arteries and veins. London. 1815.—*Hope*, Diseases of the heart and great vessels. 3d ed. London. 1839.—*Jaccoud*, Pathol. int. Paris. 1873. I.—*Laënnec*, Auscultation médiate. Paris. 1837.—*L. Landois*, Die lehre vom Arterienpuls. Berlin. 1872.—*Lebert*, Krankheiten der Arterien. Virch. Handb. d. spec. Pathol. V. 2.—*Lorain*, Le Pouls. Paris. 1870.—*A. Luton*, Art. Aorte, Nouv. dict. d. méd. et de chir. prat. Paris. 1865.—*Marey*, Physiologie médicale de la circulation du sang. Paris. 1863.—*Niemeyer*, Handb. d. spec. Path. Berlin. 1875. Translated by Drs. Humphreys and Hackley. New York and London. 1871.—*Ranvier*, Manual d'histologie pathologique. Paris. 1873.—*Raynaud*, Maladies des artères, Nouv. Dict de méd. et de chir. prat. T. III., p. 193. 1865.—*Rokitansky*, Ueber einige der wichtigsten Krankheiten der Arterien. Wien. 1852. Vide also his Handb. d. patholog. Anatomie. Sydenham Soc. Transl. 1849–52.—*Stokes*, Disease of the heart and aorta. Dublin. 1854.—*O. Weber*, Krankh. d. Arterien in Billroth u. Pitha. Handb. d. Chirurgie. II. 2. S. 139.—*Wunderlich*, Spec. Pathologie. Stuttgart. 1853. II. S. 403. III. 2. S. 608.

Morbid Affections of the External Arterial Coat.

Inflammation of the External Arterial Coat, Arteritis Externa, Exarteritis, Periarteritis.

Bizot, Mem. de la Soc. méd. d'observat. T. I. 1837; quoted by Crisp, p. 25.—*Lebert*, Gefässkrankheiten. II. Aufl. S. 339.—*Spengler*, Virch. Archiv. 1852. IV. S. 166.—*Schützenberger*, Gazette Méd. de Strassbourg. 1856.—*Leudel*, Arch. gén. de méd. 5. serie. T. 18, p. 575, Nov. 1861.—*Kussmaul und Maier*, Deutsch. Arch. f. klin. Med. I. S. 484.—*M. Zimmermann*, Arch. d. Heilkunde. 1874. S. 167.—*W. Gull* and *H. Sutton*, On the pathology of the morbid state commonly called chronic Bright's disease, with contracted kidneys (arterio-capillary fibrosis). Med. Chir. Trans. Vol. 55, p. 273. 1872.—*Hayden's* Dis. of the heart and aorta. Dublin. 1875. P. 481.

Anatomy.

Capillary hyperæmia and cell infiltration are among the earliest phenomena observed in exarteritis, as well as in the inflammations of other structures composed of connective tissue, so that the adventitia speedily assumes a reddened and somewhat thickened appearance. Should this exarteritis have arisen from an external cause, we may find similar alterations in the surrounding cellular tissue ; and the vascular injection, swelling, and cell-infiltration may extend even to the media, and may thus ultimately give rise to disturbances of the nutrition of the intima (clouding, softening, and separation). The lumen of the artery is narrowed by the swelling of the media and adventitia, and the internal coat is thus thrown into puckerings. According to the cause and intensity of the inflammation, it may either result in hyperplasia of the connective tissue or in suppuration; should the latter occur, the pus is formed in the primary gelatinous exudation infiltrated in longish strips into the connective tissue, and but seldom runs into an actual abscess in the neighborhood of the artery. Should the middle coat take part in the suppuration, we may have a pustular protrusion of the intima, rupture into the cavity of the vessel, and mixture of pus with the blood. The rift in the wall of the artery may also become larger, and lead to the formation of an aneu-

rism (Rokitansky). Should the pus become inspissated or absorbed, or should the exudation into the adventitia exhibit from the first a greater tendency to organization, then we have the formation of dense cicatricial connective tissue, which unites the artery more firmly to the surrounding parts, and may also lead to compression and narrowing of its lumen, especially when the middle coat is implicated. Should any part of the wall of an artery be thus replaced by cicatricial tissue, subsequent distention of this part may lead to the formation of an aneurism.

Causes and Manner of Occurrence.

Simple exarteritis is seldom if ever spontaneous in its origin. It is almost always secondary, either the result of a wound or of the propagation of an inflammation from the surrounding connective tissue; in this way it may occur as a part of the pyæmic process (Bamberger).

Chronic inflammation of the intima is the most frequent cause of nutritional disturbances and inflammation in the media and adventitia.

While in the domain of surgery inflammations of the external coat of the arteries are of frequent occurrence and manifold character, in medicine the aorta is almost the only vessel that in this respect comes under our notice. Connected with the external coat of this vessel, and generally inseparable from the altered connective tissue surrounding it, we very frequently find dense fibrous bands and adhesions to the neighboring organs; much seldomer we discover abscesses the size of a lentil or hazelnut—sometimes on circumscribed patches of the periphery, less frequently extending for some distance up the aorta. Usually these alterations are found connected with chronic inflammation of the intima, as in aneurisms; at other times they are the result of pericarditis, of inflammation or neoplasmata in the mediastinum or in the cardiac muscle, of ulceration of the œsophagus or of the air-passages. These alterations are more frequent in the thoracic than in the abdominal aorta.

In three cases of suppurative inflammation at the origin of the aorta, there was coincident endocarditis.

Symptoms and Course.

Since exarteritis is almost exclusively a secondary affection, its symptoms are obscured by those of the primary disease; thickening and condensation of the arterial tube may possibly be detected in superficial vessels, and that all the more easily the less the surrounding connective tissue is implicated, and the more readily, therefore, the artery itself may be submitted to palpation. If the media be implicated, the swelling of the coats narrows the lumen of the artery, and the pulsation at and beyond the inflamed portion is less perceptible. When in the course of the disease the exudation is reabsorbed, the artery becomes dilated through paresis of its circular fibres, the result of the inflammatory infiltration.

It is scarcely possible to diagnose inflammation of the external coat of the aorta.

Coupling the anatomical results with general pathological experience, we may infer that the process is sometimes acute, when it leads to suppuration, more frequently chronic when it ends in condensation and the formation of cicatricial tissue.

Should an abscess open into the vascular lumen, this may originate pyæmia or metastatic processes.

This was the case in three cases observed by Spengler, Schützenberger, and Leudet. In these there was suppurative inflammation of the adventitia and media at the origin of the aorta, associated with endocarditis of the aortic valves. The patients died of pyæmic fever. (We must not, however, forget that in these cases the endocarditis may have been the original cause both of the aortitis and of the pyæmia.)

Treatment.

In accessible positions exarteritis may be—at its commencement—treated by leeches and cold applications; subsequently warm fomentations and mercurial inunctions may be employed.

It is impossible to treat a periaortitis.

In conclusion, I may refer to what Kussmaul and Maier have described as a

Periarteritis Nodosa,

on account of its peculiar anatomical and clinical phenomena. Of this these observers have described three cases. In the first case, which came to dissection, numerous arteries in the body—but only such as were larger than the hepatic artery—presented nodose swellings from the size of a poppy-seed to that of a pea, so that they resembled irregular strings of beads. The arteries of the mesentery, the stomach, the intestines, the head, the kidney, the spleen, and the voluntary muscles presented the most marked alterations; those of the liver and the subcutaneous cellular tissue were less changed. In the latter situation the altered arteries could be felt during life like pea-sized nodules, through the superjacent skin of the breast and belly.

Microscopically examined, the diseased patches exhibited hyperplasia of the nuclei in both of the external coats of the arteries, particularly in the adventitia, which was thickly infiltrated with round and spindle-shaped cells, many of them containing several nuclei. The muscular coat was similarly thickened, though in a less degree; the muscular fibres and their nuclei were enlarged. The intima was normal over the more recent nodules. In the larger and harder nodules the adventitia contained dense fibrous tissue; the muscular coat fatty and atrophic; the lumen of the vessel narrowed and the intima thrown into folds, thickened or atrophic; sometimes the vessel was thrombosed, more frequently its lumen was dilated (bellied). The fibres of the muscular coat had become granular or waxy.

There was also present diffuse gangrenous inflammation of the intestinal mucous membrane. Diffuse nephritis. Fatty degeneration of isolated nerve fibres. General anæmia.

The phenomena during life had been those of a tolerably acute illness; the patient, a strongly-built journeyman tailor, aged twenty-seven, was about the end of April seized with rigors, followed by increased temperature and sweating; and these were succeeded by diarrhœa and numbness of the right hand. In May, when admitted into hospital, he was already extremely feeble and anæmic. Pulse 120–130; temperature normal; great sweating; the urine contained blood, albumen, and tube casts. There was great pain in the muscles, and a rapid but irregularly progressing paralysis of all the muscles of the body; speedy disappearance of the muscular electro-contractility; the skin generally anæsthetic; hyperæsthetic here and there.

There was also frequent vomiting; colicky pains in the abdomen; diarrhœa alternating with constipation; delirium; and death occurred five weeks after the commencement of the disease.

Kussmaul and Maier regard the gangrenous enteritis, the muscular degeneration, and probably also the nephritis, as results of the arterial disease. No cause for this disease could be discovered; there was no history of syphilis. The second case was very similar, but not so severe; the patient was a young man in whom the most characteristic phenomena were irregularly progressive muscular paralysis accom-

panied by violent pain. This patient convalesced after many months' electric treatment. A piece of muscular tissue containing an artery was cut out, and found to exhibit similar alterations to those just described.

In a case described by Zimmermann a similar disease of the small arteries of the intestines produced, after a year's illness, a fatal gangrenous enteritis in an otherwise strong and healthy woman; cuticular hemorrhages, also present, probably depended upon the same cause. Further, Gull and Sutton have described an alteration of the arterioles which they have called

Arterio-Capillary Fibrosis,

in which the external coat of the arterioles is thickened and has a hyaline or fibroid, or hyaline-fibroid appearance, while the capillaries connected with them have a hyaline-granular aspect. Only rarely are the intima and the muscular coat thickened, the latter being often atrophied; the lumen is usually narrowed. These alterations are chiefly observed in arterioles of 0.013 mm. in diameter, whose lumen ought normally to be twice as great as the thickness of the walls on either side, and they are specially seen in the arteries of the pia mater, the kidneys, the stomach, lungs, heart, and retina. Gull and Sutton found this alteration most frequently in advanced age, and particularly when the kidneys were contracted; emphysema of the lungs was frequently present.

They regard this arterio-capillary fibrosis as a disease which occasions atrophy of the surrounding tissues, and usually commences in the kidneys, though occasionally also in other organs. The resulting contraction of the kidneys they look upon as only a concomitant phenomenon, the essential disease being the affection of the vessels, which cannot be referred to any antecedent change of the blood due to defective renal secretion; the clinical history varies according to the organs primarily and chiefly affected.

Further inquiries alone can determine how far these views are tenable; the measurements are undoubtedly in so far uncertain as that no attention could have been paid to the state of distention or contraction of the vessels. The contradictory views of Johnson and others will be referred to presently (vide p. 355).

Disease of the Middle Coat of the Arteries.

Of all the pathological alterations of the three coats of the arteries, those of the middle coat are most rarely of an inflammatory character. Should we find in the media young nuclei or cells (whether from proliferation of its own elements or from the migration of the white corpuscles), this is usually a secondary phenomenon depending upon inflammation of the external coat,

more rarely of the internal one. Under these circumstances we may have the development of new vessels in the media, or even a puriform softening of its structure. (Rokitansky; *vide* also p. 346.)

Köster[1] states that primary mesarteritis is not so rare, and, according to him, it occurs in the form of disseminated clear patches, caused by cell-accumulations round the small arterioles passing from the adventitia into the media; and this by and by gives rise to development of connective tissue and destruction of the muscular and elastic fibres. Distention of the resulting cicatrix frequently gives rise to an aneurism.

More frequently we find fatty or calcareous degeneration, occasionally atrophy, and less frequently hypertrophy of the muscular fibres. Fatty degeneration of the media makes it friable, easily torn, and somewhat opaque; fat globules are seen within the muscular fibres; and the concomitant muscular relaxation frequently occasions dilatations of the artery.

In calcareous degeneration of the muscular fibres of the media, the artery is found to have, in patches or streaks, a hard, stiff structure, and frequently a peculiar rigid appearance, depending upon an irregular distribution of the calcareous matter, most distinctly seen in the dried artery, and appearing like the marks in a chalk drawing. In such a case, the media is frequently thicker than usual.

Fatty and calcareous degeneration of the media occur in similar circumstances as chronic thickening of the intima, in advanced age and following increased functional exertion of the arteries, sometimes originating independently, at other times combined with endarteritis in various degrees. In many cases fatty degeneration seems to depend directly upon this endarteritis. It may be found in almost every artery of the body, while calcareous degeneration of the media is almost exclusively confined to the larger and medium-sized arteries of the extremities, particularly the inferior extremities.

Simple fatty degeneration of the media, according to Virchow, is also found in juvenile life where there is congenital narrowing

[1] Berliner klinische Wochenschrift. 1872. S. 322.

and thinness of the coats of the vessels, particularly the aorta and its branches, as so often occurs in fatal chlorosis.

During life we may reasonably suspect fatty degeneration when the heart and muscles of the body generally are beginning to fail in energy from commencing degeneration, and when those arteries which can be felt, such as the radial and temporal, exhibit a defective contractility, so that, in spite of the varying influences of such external agents as cold, the rigors of fever, etc., they present very trifling variations in their diameter, and therefore appear always soft and of uniform size to the feel.

Calcareous degeneration is much more readily recognized; the plates and rigid tubes resulting from it are easily felt on the superficial arteries, the pulse in such places being, on the contrary, very indistinct.

The recognition of these alterations is made considerably more difficult, when, as so frequently happens, the intima is coetaneously diseased, because on the one hand the softening of the media may be thus concealed, while on the other the intima itself may become the seat of calcareous deposit. In this situation, however, the deposit assumes more often the appearance of plates, but seldom is to any extent tubular, and is always accompanied by patches of simple sclerosis in other places.

The results of these alterations of the media to the circulation are entirely analogous to those of chronic endarteritis.

Atrophy of the media, either simple or more rarely combined with fatty degeneration, is very commonly found in conjunction with thickening of the intima. It sometimes affects uniformly the whole of the media, at others chiefly its inner layers. Only rarely in these cases have we a simple attenuation of the muscular element; usually the number of the alternate layers of muscular and elastic fibres are also diminished; frequently the muscular layers alone are specially atrophied, while the intervening elastic fibres are correspondingly developed.

Langhans,[1] who has accurately investigated these alterations, found that the number of the layers of the muscular and elastic fibres in the aorta were diminished to between 30 and 40 from

[1] Virchow's Archiv. 1866. Bd. 36.

their normal number of from 50 to 60 ; whilst the thickness of the media had diminished to between 0.4 and 0.6 mm. from its normal standard of 1.2 mm. Sometimes he found the muscular fibres changed into connective tissue infiltrated with cells.

Independent atrophy of the media, unconnected with endarteritis, is described as occasionally occurring in advanced age in connection with general atrophy of the body ; but more extended observations are required in regard to this subject as well as in respect to the defective development of the media in youth, usually found in connection with uniform narrowing of the vascular lumen throughout extensive tracts of the arterial area.

The vessels in such conditions are distinguished by their softness, the thinness of their coats, and the trifling variations in their diameters. The sphygmographic pulse curves resemble those produced where the media is relaxed, and exhibit tolerably distinct reflux waves ; on the other hand, those due to arterial elasticity are less perceptible.

What influence the atrophy of the vascular muscularity has upon the circulation and the nutrition of individual organs, or what may be the effect of extensive atrophy of this character upon the circulation as a whole, we cannot at present more accurately determine, because hitherto such conditions have only been considered incidentally and in connection with endarteritis, in which alterations of the arterial lumen and of the elasticity of the intima also play their part. Nevertheless, it is indisputable that atrophy of the media disposes to the formation of an aneurism when it occurs in a marked degree within a limited area ; when it extends over a wider stretch it leads to dilatation and changes in the elasticity of the vessel, and thus produces remora and other alterations of the blood current, and especially to an imperfect regulation of the blood supply, which, according to the experiments of Ludwig and his pupils, is constantly maintained in ordinary physiological conditions. The nutrition of the organs supplied is thus damaged ; they become less capable of resisting injurious influences, and become atrophied, degenerated, or soaked with dropsical effusion.

Should several large arterial districts become affected with

atrophy of their muscular coat, the blood pressure in the aorta becomes depressed, and the venous circulation altered; both of these causes react upon the cardiac energy.

Possibly the sphygmographic investigation of various arteries may yet aid us in the diagnosis of such cases.

Contrary to the opinions prevalent at the present time, Rokitansky regarded the relaxation, atrophy, and degeneration of the media as primary, and the alterations of the intima, the endarteritis, as secondary, the result of the remora of the circulation.

Hypertrophy of the media has been even less accurately investigated than its atrophy. According to Velpeau, it is sometimes observed in the neighborhood of true aneurisms. I have seen it in a few cases of aortic insufficiency, with considerable hypertrophy of the left ventricle, in arteries of a medium size, such as the brachial and radial.

Possibly, as happens in other more markedly muscular organs in powerful individuals, this may have been, in these cases, only the result of an increased and energetic antagonism to the action of the hypertrophied cardiac muscle, and probably more careful investigation will succeed in discovering it in other cases of cardiac hypertrophy.

In the smallest (microscopic) arteries Johnson [1] has observed hypertrophy of the muscular coat in cases of chronic nephritis with contraction of the kidneys and hypertrophy of the left ventricle, and he found this not only in the kidneys (in which the intima is also thickened), but also in the arterioles of the brain, pia mater, muscles, intestines, and subcutaneous connective tissue. He leaves it doubtful whether this hypertrophy is to be regarded as antagonistic to the cardiac hypertrophy, or whether it is due to the persistent irritation of these arterioles by abnormal contents (retention of the renal excretions), by which contraction and narrowing of the vessels and ultimately cardiac hypertrophy are produced. In many cases, according to Johnson, this hypertrophy varies in degree in different structures, so that the increased cardiac energy may give rise to hemorrhage

[1] Medico-Chirurg. Transact. LI. 1868. Med. Times and Gazette. July 2, 1870. No. 9. *A. L. Galabin*, On the Connection of Bright's Disease with Changes in the Vascular System. Thesis. London. 1874. Centralbl. 1874. S. 105.

from rupture of the less altered vessels (*e. g.*, to cerebral hemorrhage in one case).

A. L. Galabin found, like Johnson, cardiac hypertrophy associated with contracted kidneys (and epithelial nephritis), also hypertrophy of the muscular and external coats of the arterioles, but no alteration of their lumen. He regards these hypertrophies, as well as that of the heart, as concomitant results of increased capillary resistance due to an altered condition of the blood.

Johnson's views are antagonistic to those of Gull and Sutton, who found, in connection with contracted kidneys and similar conditions, a hyaline-fibroid thickening of the external coat, but the media only slightly altered, sometimes atrophied and at others hypertrophied.

Further observations of numerous cases are required to reconcile these contradictory observations, and the investigation is rendered all the more difficult that we have no positive knowledge of the actual size of the smallest arterioles in the various organs, nor of the normal relation between their lumen and the thickness of their walls, while the varying degrees of contraction of these arterioles, and the varying amount of cadaveric rigidity (quite irrespective of diversities in the methods of preparing the specimens), introduce serious complications into any attempts at comparative measurements.

Apart from the symptoms of the causal morbid process, we have as yet no diagnostic phenomena that can be regarded as indicative of hypertrophy of the muscular coat of the arteries.

The *prognosis* of those diseases of the media just described may be readily deduced from the anatomical description. The alterations are, for the most part, of such a character as to render an anatomical restitution impossible (atrophy, fatty and calcareous degeneration), but they are, moreover, chiefly of a secondary character, and their prognosis depends upon the endarteritis and the general condition of the body.

The *treatment*, also, must be directed against the original disease, against the cause of the arterio-sclerosis and the general atrophy, against the progress of the nephritis.

Whatever excites the circulation or interferes with the mechanism of its regulation, such as corporeal exertion, psychical excitement, or exposure to great degrees of cold or heat, must be avoided.

At present we possess no therapeutic means which may be profitably employed in diseases of the media. At the most, we may, in accordance with certain theoretic views, employ elec-

tricity locally in atrophy and degeneration of the muscular coat of the arteries, or we may use ergotine internally or subcutaneously.

Spasm and Paralysis of the Middle Coat of the Arteries.

The contractility of the muscular coat of the arteries is subject to continuous variations from physiological causes, which depend partly upon central innervation, and partly upon influences exerted directly upon the vessels themselves, by external agencies, or by their contents. The resulting continuous alterations of the arterial diameter and the blood-stream regulate the functions of the respective organs and organ groups, secretion, heat-production, and heat-dissipation, etc. The various links in this complicated mechanism have as yet been scarcely recognized; their disturbance and its results are only partially known. From their intimate relation with other disturbances of innervation, their clinical consideration in connection with individual vascular areas will be found in connection with other nervous diseases in the chapter on vaso-motor neuroses (Vol. XII.).

Here we confine ourselves to the consideration of spasm and paralysis as they occur in the circular fibres of the vessels generally; apart from nervous disturbances in the limited sense of the word, it is especially in febrile conditions that we find (the vaso-motor nerves being doubtless implicated) the greatest alterations in the contractile condition of the arteries, from the most violent spasm during the rigor of fever, to most complete paretic relaxation during and after critical defervescence.

Besides a number of narcotic agents, which irritate or paralyze the arterial muscles, and which may occasionally be therapeutically employed with success, there are two drugs, chronic poisoning by which very generally has an irritating action upon these muscles—lead and ergotine.

Finally, the local influences by which the arterial muscles may be excited to contraction, or relaxed, are specially cold and heat.

Should cold act for a considerable time continuously or repeatedly upon a vascular area, the primary contraction is

followed by a permanent relaxation of the vessels, accompanied by remora of the circulation (frost-bite, chilblains).

Symptoms.

The more firmly the muscular layer of the media is contracted, so much the narrower the arterial tube appears, its walls thicker and its lumen contracted; the latter, in small arteries, is even said to be occasionally entirely closed (?). When the arteries are superficial (as the radial and temporal), they appear to be narrower, they feel firmer, and the pulse is less distinct.

The sphygmographic pulse-curve is lower, its secondary waves, especially that of reflux, weakened, while those due to elasticity are small, though occasionally more numerous.

These alterations extend over the whole extent of the spasmodically contracted artery, often into its smallest branches; the capillaries are then imperfectly filled, the blood-stream scanty, the organ to which such an artery is distributed appears pale, collapsed, and cool, if it be, like the hand, capable of being investigated. The blood-stream is similarly altered, but in a less degree, when the smaller branches are unaffected, and the spasm and narrowing is confined to a limited extent of the main trunk.

The function of the organ supplied by the affected vessel is more or less disturbed according to the degree of spasm present; thus the muscles are feeble, the hand, the fingers, for example, are moved with difficulty and awkwardly, the skin is cool and dry; subjective sensations of cold, of formication, and of shooting pains, may be felt, combined with a diminution of the sensibility to touch.

When the spasmodically contracted artery is of moderate size, the flow through the collateral vessels or through those branches coming off anterior to the constricted portion, suffices to render the influence of the local disturbance scarcely perceptible on the general circulation. Should the spasm, however, affect a larger artery or several smaller ones, then the hindrance to the onward flow raises the blood pressure in the aorta, the heart is excited to more frequent and more forcible contractions, and it may thus partially overcome the obstruction—indeed, the

medium rate of the blood-stream in the aorta and its larger branches may become greater than it was before the occurrence of the arterial spasm.'

The arterial pulse in such cases is felt to be full and more or less hard, in proportion to the amount of the contraction in the artery examined.

Localized arterial spasm is exemplified in the radial artery, when the hand and forearm are exposed to the influence of cold; more extensive arterial spasm is observed in febrile rigor and in many cases of lead colic.

When the circular muscular fibres of an artery are relaxed or paralyzed, the vessel is broader than usual, its walls thinner, and its lumen dilated. A finger placed upon such a soft, large artery, superficially situated, perceives the pulse wave with extreme distinctness, and often enough this is very readily appreciated by the eye.

The sphygmographic tracing of such a pulse exhibits a well-marked systolic elevation, the line of ascension perpendicular, the wave of reflux very distinct, often manifold, because the relaxed arterial walls are affected by very trifling oscillations of the blood-wave; the waves due to elasticity are ill-marked and less numerous than usual, and may entirely disappear with increasing relaxation. The dicrotism is often perceptible by the finger.

Pulsation is frequently observed in those small arteries where it is not usually seen. The capillaries of the affected area are greatly distended, and in superficially situated organs this is evinced by turgescence, heat, and redness. The rapidity of the circulation throughout the entire organ is increased. The degree of relaxation of the arterial muscle varies very much, and this variation, as well as the share taken in it by the arterioles, causes the phenomena described to be more or less well marked. The arterial areas of the skin and of the extremities relax very readily under the influence of warmth, locally or generally applied; and a very general vascular relaxation takes place in the sweating stage of fevers. On account of the great capacity of the arterial system, and the facilitated outflow through the

[1] *Heidenhain*, Pflüger's Arch f. Phys. III. u. V.; *Slavjansky*, Arb. in der Physiol. Anstalt zu Leipzig. VIII. 1873.

capillaries, the blood pressure in such circumstances is usually diminished—unless, indeed, a compensating increase of the cardiac energy occurs. The average rate of the arterial circulation may thus fall so low, that in general vaso-motor paralysis, such as follows extensive burns of the skin or certain injuries to the central nervous system, depression of all the vital functions, gradual refrigeration and death, may follow.

Occasionally in cases of relaxation of the arterial muscles of the hands and forearms, a capillary pulsation is observed in the finger-nails, in the form of a post-systolic reddening and a diastolic paling of them; *pulsation may also be seen in the subcutaneous veins* of the back of the hand and forearm.[1] This venous pulsation, compared with that of the radial artery, is delayed even more distinctly than the capillary pulse of the nails; unlike the retrograde venous pulsation occurring in cardiac disease, it is propagated *centripetally* in the normal direction of the venous circulation, as can be readily proved by compression of a cuticular vein, when the peripheral portion continues to pulsate, while the centripetal portion is quiescent (provided the part compressed is so selected as to avoid on the one hand too great congestion of the peripheral part, and on the other the communication of the pulsation to the parts above, by anastomosing veins entering just above the part compressed). Sometimes all the veins on the back of the hand exhibit this phenomenon, at others, only some of them; occasionally this pulsation is propagated as high as the elbow. Evidently certain conditions are necessary for the production of this phenomenon; besides the arterial relaxation, we must also have venous relaxation, and a certain amount of congestion of the veins (depending upon the position of the extremity), delicacy of the skin, and finally a cardiac action sufficiently powerful to produce an efficient bloodwave. Hence the reason why this venous pulse *a tergo* is not more frequently observed, and when present it is easily overlooked, because of its evanescence, since a slight elevation or movement of the arm, a waft of cold air, etc., may cause it to vanish.

Nevertheless it is not so very uncommon; I have seen it in many patients affected with typhus, relapsing, and intermittent fevers, pyæmia, acute articular rheumatism, pneumonia, phthisis, and cholelithiasis; in all these cases it was coincident with the arterial relaxation, which accompanied certain stages of these fevers, particularly a rapid defervescence associated with sweating; in a limited sense nervous influences may also assist in producing it, as in cases of meningitis, spondylitis, encephalomalacia, and injuries to the cervical portion of the cord. Even in apyretic conditions I have frequently observed this form of venous pulse, as in cases of chlorosis, of carcinoma, in one case of gastric ulcer associated with profuse

[1] *H. Quincke*, Berl. klin. Wochenschr. 1868. Isolated notices of this form of venous pulsation are also to be found in Guy's Hosp. Rep. IV. XII (by *King*), in *Stokes'* work on Dis. of the Heart, and in the Arch. Gén. 1865 (by *Parrot*).

hemorrhage; in all these cases, as well as in an observation made on myself, the heat of summer had relaxed the peripheral vessels. Finally, this venous pulse was remarkably distinct in a series of cases of aortic insufficiency, in which relaxation of the vessels of the hand was produced by fever or other causes; the size and rapidity of the pulse wave in these cases evidently favored its distinct propagation. Only once in an extremely chlorotic girl, with an indistinct cardiac disease, this phenomenon was also observed in the veins on the back of the foot.

The explanation of this centripetal venous pulse is partially involved in the description of its mode of occurrence; by the relaxation and dilatation of the arteries even to their ultimate branches, the propagation of a powerful pulse wave through the capillaries into the veins is rendered possible; thus Claude Bernard, by dividing the sympathetic nerve and simultaneously irritating the lingual branch of the trigeminus, was able to observe pulsation in the veins of the submaxillary gland.

Perhaps also the direct communication between the arteries and veins, described by Sucquet,[1] may also have its influence in this respect, bringing about, as it does, a derivative circulation (in contradistinction to the circulatio nutritiva). By relaxation of the numerous muscular fibres, which, according to Sucquet, regulate this anastomosis, the propagation of the pulse wave into the veins is easily possible; Sucquet says, that on venesection he has frequently observed the blood escape in jets of a bright red color. But the existence of this anastomosis of Sucquet has not as yet been confirmed by any one, and has indeed been denied to be true on the faith of recent investigations made specially in regard to this matter.[2]

Should spasm or paralysis of an arterial area last for any time, days, weeks, or months, it gives rise to nutritional disturbances in the organs affected. The gangrene of the extremities, which occurs in certain forms of ergotism, is with the greatest probability regarded as the result of chronic arterial spasm.

When the arterial paralysis lasts for a long time, the primary dilatation of the vessels is not rarely followed by their contraction, accompanied by cooling of the organ affected, and diminution of its turgescence, blanching or cyanotic discoloration, followed by atrophy, especially of the muscles, and then of all the other tissues. Nevertheless it is doubtful whether all these disturbances result solely from the paralysis and subsequent contraction of the arteries, since they become developed in apparently similar affections of the arteries, in various degrees, and after divers

[1] Bull. de l'Acad. de Méd. Juin, 1861.

[2] *F. Berlinerblau*, Diss. Bern. 1875; and du Bois and Reichert's Archiv f. Anat. u. Physiologie. 1875.

intervals of time, or may, indeed, remain altogether absent, and further, because in such cases we have generally to do with extensive nervous disturbances, which may possibly enough be frequently of a trophic character.

Moreover, the anatomical results of paralysis upon the walls of the vessel itself have not as yet been accurately investigated.

Treatment.

The treatment of spasm and paralysis of the arteries must be specially directed against the causal influences, such as nervous affections, fever, the action of poisons, of cold, and of heat. But even a purely symptomatic therapeusis may be of much advantage in such cases; thus in every form of arterial spasm, whatever may be its origin, a most important part of the treatment consists in the application of external warmth (provided the arterial area affected is accessible to this remedy), in the form of poultices, tepid or warm baths, steam- or hot-air baths, swathing in cotton-wool, etc.

The use of the galvanic current applied through the nerve-trunks (such as the plexus brachialis, Nothnagel) seems to be useful in some cases. Friction, rubbing with irritating or volatile embrocations, shampooing, are all old, and often very efficacious remedies. In extensive arterial spasm the inhalation of from two to five drops of nitrite of amyl will be found to relax the whole of the arterial system in an extraordinarily short space of time; but the result is evanescent. Chloral, atropia, and other narcotics are occasionally employed, on account of their action on the vascular innervation.

When the arteries are paralyzed, we may attempt to regulate the vascular turgescence of the part affected by elevating the part of the body affected, by elastic compression, or we may endeavor—directly or in a reflex manner—to cause the dilated vessels to contract by the local application of cold, by inunctions, or by faradisation of the skin.

The internal remedies which may be tried in such an affection

are the preparations of lead and ergotine, and also occasionally certain narcotics (digitaline, physostigmine, etc.[1])

Diseases of the Internal Coat of the Arteries.

Inflammation of the Internal Coat of the Arteries.—Endarteritis.

a. *Acute Endarteritis.*

Corneliani, Opusculo sulle non-inflammabilita della membrana interna dei vasi arteriosi e venosi. Pavia. 1843.—*Durante*, Organis. d. thrombus. Wien med. Jahrb. 1872. IV.; Versuch ueber die Bez. d. Int. u. d. Blut. i. abgesch. Venensäcken, ebendaselbst. 1871. Bd. III.—*Ranvier et Cornil*, Histol. norm. et pathol. de la tunique interne des arteres et de l'endocarde. Arch. de phys. I. p. 551; Manuel d'histologie pathologique. Paris. 1873.—*Rokitansky*, Ueber d. wichtigsten Krankheiten der Arterien. Wien. 1852.—*Vanlair*, Rech. hist. sur l'endarterite gangrén. Arch. de physiol. IV. p. 223.—*Virchow*, Arch. I. S. 272. Ges. Abhdlg. S. 380.

The internal coat of the arteries seems incapable of undergoing any intense or rapid changes.

From the absence of nutritive vessels there is never any inflammatory redness, and the recorded observations of the older authors (Bouillaud, Dupuytren), referring to this, are either based upon their having mistaken cadaveric imbibition for inflammatory redness, or having erroneously referred the vascular injection of the adventitia to the intima through which it shone in thin-walled vessels.

Nevertheless, the arterial intima is occasionally affected by inflammation of the external and middle coats; for, by the swelling of these coats and the narrowing of the vessel thus produced, the intima is first of all thrown into folds, then softened by serous imbibition, and bulged into the vascular lumen by an exudation rich in cells; its epithelium is clouded, swollen, and softened, so that the arterial lining, even to the naked eye, seems

[1] Nitrite of amyl given internally in doses commencing with one-third of a minim thrice a day is often useful in such cases; its action when thus administered is more permanent than when it is inhaled.—*Trans.*

rough, velvety, or even puckered. If the external coats are inflamed to any considerable extent, the intima may even present gangrenous patches. (According to Durante this is always the case between two ligatures applied at no great interval from each other.) Wherever the nutrition of the intima is materially disturbed, the physiological obstacle to coagulation disappears, and deposits are found upon the arterial wall, which may lead to embolism.

The intima behaves itself in a similar manner when it is directly irritated (though the other coats are no doubt usually affected at the same time), as happens when the vascular lumen is blocked by an embolus, or when the artery is cut through, crushed, or torn, or when a ligature is applied. Experience in man and experiment on animals have shown that after embolism, as well as after ligature, all the three arterial coats are infiltrated with young cells, but that in the intima this proliferation is least marked.

Views differ, however, as to the behavior of the epithelium: according to some there is only cloudy swelling and softening, which either recedes or leads to the destruction of the cells; according to others (Thiersch, Waldeyer, Durante), the epithelial cells change to round ones, which take part in the organization of the thrombus.

The intima seems incapable of undergoing exudative inflammation, with effusion on its free surface (the so-called croupous endarteritis); floating fragments of the intima, necrosed and partially dissolved, seem to have been mistaken for this form of exudation; neither have we ever any adhesions between two opposite surfaces of the vascular wall, so long at least as the intima still exists. The formation of pus is also very rare, and though it does occur, the softening of a thrombus or the rupture into the vascular lumen of an exarteritic abscess is much more frequently mistaken for it.

The causes of acute inflammation of the intima are either an acute exarteritis or a wound (crushing or tearing), which leads to rupture of this coat, or an embolus. The intensity of the injury or of the external inflammation, and the condition of the embolus, are of material importance in regard to the future course of the inflammation of the intima.

The only deviation from the phenomena described is to be found in connection with the covering of the aortic semi-lunar valves, which, in its anatomical structure, and the nature and

frequency of its diseases, is more closely allied to the endocardium (vide diseases of the heart). Also in the ascending aorta, the intima (which is said to be richer in cells the nearer the heart it is[1]) sometimes exhibits soft excrescences resembling granulation tissue, which may give rise to adherent coagulation, or to emboli, etc.

C. O. Weber[2] relates a case ending fatally in three days, in which the whole of the lining membrane of the ascending aorta and of the arch was roughened with minute vascular granulations. Endaortitis, like endocarditis, is frequently of rheumatic origin.

H. Mayer and Buhl[3] found in a man, who had died pyæmic subsequent to the extirpation of a lympho-sarcoma, close above the origin of the aorta, and adherent to the icteric intima, a dark red vegetation of the size and shape of a mulberry; this "inflammatory growth" had a short stem, and was very soft and friable. Necrotic patches had been produced in the kidneys by embolism.

Meade[4] found in a girl, aged eighteen, the whole internal surface of the aorta injected and covered by a membranous coagulum; on isolated patches, the injection was more marked and the surface rough. Similar changes were found in the iliac and femoral arteries; the right brachial was obliterated.

Gordon[5] observed a circular deposit of soft, purulent-looking matter close above the aortic valves, which he described as aortitis purulenta.

Occasionally in chronic endarteritis there is a true suppuration in the deeper layers of the intima (Virchow, perhaps also Bizot); but this can only be distinguished from atheroma by the microscope.

Cornil and Ranvier[6] describe as acute endarteritis changes which have hitherto been looked upon as belonging to the chronic form, small longitudinal elevations and gelatinous patches, which may be one hundred times as thick as the normal intima,

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[1] Jaccoud (l. c.) I. p. 711.

[2] L. c. S. 164.

[3] Bair. ärztl. Intelligenzblatt. 1870. No. 40.

[4] Lancet. 1870. Dec. 10.

[5] Dublin Quart. Jour. 1868.

[6] L.c. p. 530.

and twice or thrice as thick as the media, and contain rows of small round cells. These are said to originate from the pre-existing cells of the intima, and are specially numerous in the *superficial* layers (this distinguishes it from chronic endarteritis). Sometimes we find superficial fungous ulcerations, to which fibrine and white corpuscles adhere. In the neighborhood of acute endarteritis, periarteritis (cellular imfiltration) is always found; the media is unaffected. This acute form exhibits every possible stage of transition to chronic endarteritis.

Symptoms.—Acute endarteritis is usually first recognized when it has produced coagula; should these adhere to the wall of the artery, they may produce narrowing or obliteration of the arterial lumen, and thus weaken or arrest the peripheral pulsation; occasionally they also produce a whizzing murmur. Should these coagula break loose and give rise to embolism, with all its diverse results, we may deduce from these phenomena the existence of an endarteritis, when other sources of embolism, especially endocarditis, can be excluded. But even when a thrombosis is certainly recognized, we are still left to determine, from the other phenomena and from the course of the disease, whether arteritis is actually present, and whether it is the cause or the result of the thrombosis.

In the aorta we never have complete occlusion, but only the formation of coagula which adhere to its inner wall, and which, from the force of the blood-stream, are very readily washed off and wedged into the peripheral vessels. In the few accurately observed cases the course of the disease closely resembled that of an acute endocarditis: pyæmic fever (once death in three days), embolism with necrosis, thrombosis of the larger peripheral arteries, and gangrene of the extremities.

The *treatment* of acute endarteritis of the peripheral arteries must be similar to that of exarteritis, but above all things we must endeavor to prevent the evil results of thrombosis (embolism, local disturbances of the circulation). In acute endaortitis the treatment must be symptomatic, anti-febrile and antiseptic.

b. *Chronic Endarteritis. Endarteritis Deformans seu Nodosa* (Virchow). *Arteriosclerosis* (Lobstein). *Atheromatous Process* (Förster). *Atherosis, Induration of the Arteries.*

Buhl, Zeitschrft. f. rat. Med. 1857. VIII. S. 97.—*Corneliani*, Opusculo sulle non-in flammabilità della membrana interna dei vasi arteriosi e venosi. Pavia. 1843. —*Conway-Evans*, Atheroma of the pulmonary artery. Transact. of the Path. Soc. XVII. 1867.—*Dittrich*, Beitr. z. pathol. Anat. d. Lungenkrankheiten. 1850. —*Donders* u. *Jansen*, Arch. f. phys. Heilk. VII. 1848.—*Guéneau de Mussy*, Étude clinique sur les indurations des artères. Arch. gén. Août. Sept. 1872.— *Hammernik*, Phys. pathol. Untersuchung. Prag. 1846.—*K. Hertzka*, D. ather. Process in seinen Beziehungen zum Gehirn. Stuttgart. 1875.—*O. Heubner*, Die luetische Erkrankung der Hirnarterien nebst allgemein. Erörterungen z. norm. u. pathol. Histologie sowie sur Hirncirculation. Leipzig. 1874.—*Kirkes*, On hypertrophy of the left ventricle of the heart. Med. Times and Gazette. Nos. 370, 371. 1857.— *W. Koster*, die Pathogenese der Endarteritis. Verhand. d. Niederl. Akad. d. Wiss. u Naturwiss. Abth. 2. A. Reeks. Th. 4. Centralbl. 74. S. 699.—*Langhans*, Beitr. z. norm. u. pathol. Histol. d. Arterien. Virch. Arch. 36. 1866.—*Moxon*, On the nature of the atheroma in the arteries. Guy's Hospital Rep. XVI. 1871.—*Polotebnow*, Sklerose des arteriellen Systems als Ursache consec. Herzerkrankungen. Berlin. klin. Wochenschrift. 1868.—*Ranvier et Cornil*, Manuel d'histol. pathol. Paris. 1873. Arch. de physiol. I. 551.— *Risse*, Obser. quæd. arteriar. statu normali atque pathologico. Diss. Inaug. Regiom. 1853.—*Rokitansky*, Ueber die Wichtigsten Krankheiten der Arterien. 1852.—*Tiedemann*, Ueber Verengerung und Verschliessung der Pulsadern. Heidelberg. 1843.—*Traube*, Berlin. klin Wochenshrft. 1871.— *Virchow*, Ges. Abhandl. 492–506.—*C. Wedl*, Beitr. zur Pathologie der Blutgefässe. Sitzungsber. der Wiener Akad. d. Wissenschft. 1863. 63. 66.

Anatomy.

Chronic endarteritis begins at one time in the form of localized and scattered elevations of the intima, of a bluish-gray or whitish-yellow color, which project into the arterial lumen, with but little retropulsion of the layer of the intima next the media (gelatinous and semi-cartilaginous thickenings); at other times these elevations are not localized, but extend over larger stretches of the vessel, and do not project so much (sclerosis). Or both of these forms may be combined; on the basis of the diffuse thickening, little localized prominences become developed. According to Langhans the latter only occur upon pre-existing diffuse conden-

sations, and never arise on the normal intima. The surface of these prominent patches, which resemble precipitated deposits, is smooth, but less lustrous than the surrounding tissue; their edges are sometimes abrupt and elevated, at others they are flat; the consistence may be that of the normal intima, often somewhat greater, resembling cartilage, seldom less, mucoid or gelatinous.

These flattened condensations are less commonly observed in the small arteries than in the aorta and larger vessels; they are more frequent and most strongly developed where the vessels divide or where branches are given off; the diffuse elevations of the intima are found in both large and small vessels, and, from the relation they bear to the lumen of the latter, they more readily attract the eye.

On examining a transverse section microscopically, it is readily seen that the alterations just described consist essentially in a hyperplasia of the normal elements of the intima, the connective tissue matrix, and its contained cells. The latter are partly star-shaped, somewhat larger than normal (Heubner), partly round or spindle-shaped, and show indications of fission. The matrix, as in the normal intima, consists of fibrous lamellæ, the fibres of which run parallel to the surface. In the semi-cartilaginous elevations, this matrix is dense and opaque; in the gelatinous patches (which are generally of recent date) it is soft and homogeneous, often even mucous in character, and containing mucine. The cells in tissue of the latter kind are no longer disposed in layers, but anastomose irregularly with one another.

Ranvier regards these as forms of acute endarteritis; Heubner, on the contrary, looks upon them as not quite recent, on account of the size of the contained cells; the gelatinous condition he supposes to be due to œdematous infiltration of the matrix.

According to Virchow and others the hyperplastic thickening is chiefly found in the deeper lamellæ of the intima, whilst the outermost fibrous lamellæ and the epithelium pass unchanged over the swelling. Langhans observed, in the aorta, at least, all the lamellæ of the intima uniformly altered, and the outermost layer sometimes most so.

Only in the smaller arteries, at the base of the brain, has Langhans observed hyperplastic thickening of the deepest layers of the intima, including the elastic lamellæ, so that the muscular coat itself was pushed back.

The diameter of the patches is from 2 to 15 mm. and more; their thickness from 2 to 3 mm.; larger masses are formed by confluence. Diffuse thickening of the intima is much more difficult to determine and recognize (and this can often be only done by microscopic examination of sections of the part), since the normal diameter varies so much. In the aorta Langhans obtained the following measurements:

Age.	*Thickness.*	
	Of the Intima. Mm.	Of the Muscular Coat. Mm.
4 days	0.015 to 0.02	0.5
1½ years	0.025	0.8
10 years	0.016 to 0.025	0.9
20 to 40 years	0.03 to 0.06	1.2 to 1.3
Over 50 years	0.05 to 0.12	1.5

L. reckons the normal thickness of the intima at 0.05, while Henle regards its maximum thickness as 0.03, and Heubner states that of a man aged twenty-four as 0.11 mm. According to Langhans the diffuse thickening of the intima never exceeds 0.5 mm., or tenfold the normal. In the smaller arteries the intima is thinner, and the pathological thickening relatively greater than in the larger vessels.

The new formations in the intima just described undergo in their further progress, with great regularity, the retrograde metamorphosis of fatty or calcareous degeneration. The commencement of the fatty degeneration is to be found in the cells, and in the intercellular substance; the cell contents either at once run together into large oil globules, or first of all they become granular, and the isolated oil-drops subsequently coalesce (Langhans); the intercellular substance is permeated by fine fatty granules. According to Virchow, the fatty degeneration begins in the superficial layers of the gelatinous thickenings, but in the deeper layers of the semi-cartilaginous plates; Langhans observed all the lamellæ to be almost uniformly affected, and the degeneration to commence sometimes in the middle. Macroscopically the fatty degeneration is manifested by an opaque, yellowish-white coloration, which sometimes commences in spots both in the isolated patches and in the diffuse thickenings, and at others is from the first more general. In its further course the

most degenerated parts become soft and friable. When this process occurs on the surface, the epithelium and the softened mass are both swept away by the force of the circulation, and a superficial abrasion results, the so-called fatty ulcer. When the deeper layers are chiefly degenerated, a pulpy accumulation of fatty granules and tissue detritus is formed, frequently containing crystals of margarine and cholesterine as well as myeline formations, over which the superficial lamella of the intima lies bladder-like (atheromatous pustule). This pulpy mass either becomes thickened and calcareous by absorption of its fluid part, or the degeneration gradually progresses till the superficial layer gives way and it ruptures like an abscess into the lumen of the artery; and thus an ulcer is formed with ragged and partially undermined edges, which becomes covered with a fibrinous deposit, and occasionally heals with the formation of cicatricial and pigmented tissue.

The healing of the ulcer is brought about partly by the organization of the thrombus which adheres to its surface, partly by granulation and cicatricial tissue originating in the media and adventitia.

The *calcareous degeneration* consists in a deposit of calcareous salts in the structure of the thickened intima; but it may also occur locally in any of the lamellæ of the intima, primarily, however, and most markedly in the deeper and middle layers, so that the resulting hard white plates are at least at first covered by the relatively normal superficial lamellæ. The sharp edges of these plates is usually the first part of them which breaks through the surface of the intima into the vascular lumen.

The thickness of these plates is about 0.5–2 mm., their diameter 5–15 mm. and more, and they often form regular rings round the vascular lumen. Most authors describe this process as a simple calcareous impregnation of the normal tissues (Förster, Langhans), by which the cells are destroyed (Rindfleisch). Virchow regards it as a kind of ossification.

The epithelium overlying the calcareous plates seems to disappear very early.

Fatty degeneration is very frequently combined with calcification, so that beneath, and sometimes within the calcareous

plates, we find athomeratous deposits, while the surrounding tissue is fattily degenerated.

In the same artery both forms of degeneration are not unfrequently found, in all their different stages, in different parts of its course.

Canalisation has been described by Rokitansky as a rare variety of thickening of the intima. In this the intima is perforated by pin-holes and canaliculi, into which the arterial blood passes, so as to form a kind of cavernous tissue.

The other coats of the artery take part in the changes of the intima just described in various degrees.

The media is almost always thinned, and more easily torn than usual; its muscular fibres have either simply disappeared or they are calcified or fattily degenerated; the latter is specially the case where the deepest layers of the intima are greatly altered (vide what has been already said as to atrophy of the media).

The adventitia, when it is altered in structure, is sometimes in a state of acute or chronic inflammation: hyperæmic, or fibrously thickened, or infiltrated with cells, and this is usually better marked the more intensive and extensive the changes of the intima are. In rare instances the formation of new vessels seems to pass through the media into the intima (Meckel).[1]

This seems to be the proper place for mentioning certain diseases of the arterial coats, which may occur independently, but are usually combined with chronic endarteritis, and lead to similar results and symptoms.

The first of these is simple fatty degeneration of the intima.

According to Virchow this commences on the internal surface of the normal intima, in small, sharply circumscribed spots, and leads to the formation of flat superficial ulcerations of an opaque, velvety appearance.

According to Langhans it is very easy to mistake for fatty degeneration any trifling thickening of the intima; and this observer has not been able to discover the fatty metamorphosis of the vascular epithelium described by Virchow.

Fatty degeneration and calcification of the muscular fibres of

[1] See also *Virchow*, Ges. Abhandlg. S. 498. *Lancereaux*, Gaz. Méd. 1864. The latter observer saw hemorrhage in and beneath the intima.

the media have been already described when speaking of the disease of this coat.

As appears from what has just been said, arterio-sclerosis depends upon anatomical alterations of the intima (the idea of a "deposit" from the blood has been given up by Rokitansky, its original propounder); nevertheless, there are various opinions as to how these alterations are brought about, and how far it is justifiable to regard them as of "inflammatory origin."

Whilst Virchow, Langhans, and Ranvier regard the new formation as proceeding from the normal, star-shaped cells of the intima, Heubner suspects that they may originate in the endothelium, and Traube has stated the hypothesis that the round cells of the sclerotic plates are white blood-corpuscles, which escape from the by-streaming blood between the cells of the endothelium into the interlamellar spaces of the intima, and there become changed into the star-shaped and spindle-formed cells, so that the whole of the thickening of the intima is a true inflammatory product in the Cohnheim sense of inflammation. Favorable circumstances for this immigration (from accumulation of lymph corpuscles on the inner wall of the arteries) are to be found in the remora of the blood, which occurs in the aorta during each diastole of the heart.

W. Koster has recently, on the strength of microscopic observations, in so far adopted this view, that while he regards cell hyperplasia and degeneration of the intima as primary, he yet adopts the idea of a secondary immigration of white blood-corpuscles from the vascular lumen, since he could not discover any connection between the groups of small round cells and the star-shaped cells of the intima; these immigrated cells may become partly developed into spindle-shaped cells; and when the immigration is very considerable the necrobiotic processes in the intima may be then hastened.

The question, whether in endarteritis chronica we have to do with a hypertrophy, a parenchymatous or a true inflammation in Cohnheim's sense of the word, has been thus as it were settled by compromise. Nevertheless, the final result of Koster's investigations, as well as that of the earlier authors mentioned, is that thickening of the intima by hyperplasia of its normal elements, consequently hypertrophy, or, as we may call it, parenchymatous inflammation, is the most important and the most characteristic part of the process.

The results of chronic endarteritis upon the external condition of the arteries affected and their functions are very various. In the smaller arteries the diffuse occurrence of the thickening leads to narrowing of the vascular lumen over wide tracts; its more limited development originates a local stenosis, or may even lead to complete closure of the vessel by fibrinous deposit; both result in producing diminution, or even complete stoppage, of the circulation in the corresponding vascular area.

In the larger arteries the indurated thickening is only rarely great enough to diminish the vascular lumen, and this is most apt to occur where branches are given off, as where the large arteries to the head and superior extremities pass out of the aorta; sclerotic and calcareous rings formed in these positions not unfrequently reduce the diameter of one of these arteries to that of the radial. More rarely, narrowing of the lumen of large arteries is produced by thrombi adherent to their walls, or by the secondary formation of cicatricial tissue in the media and the adventitia.

The diminution of the resistance and of the elasticity of the arterial walls is of much more importance than these alterations in the arterial lumen, and this diminution is produced by the thickening of the intima and by the atrophy and distention of the media, which is specially elastic, and, in the more severe cases, by the complete rigidity induced by calcareous degeneration. The normally expansile and elastic arterial tube is thereby more or less approximated to a rigid pipe through which the blood no longer flows uniformly, but, as it were, in jets; and it thus suffers a much more violent blow from each pulse wave. It is readily comprehensible that the texturally altered vascular walls are thus apt to be torn or at the least to experience a permanent dilatation, and in this way dilatations (simple or sacculated) of the arteries may be originated.

Such dilatations are sometimes confined to a limited extent or to only a part of the circumference of the artery, and thus are formed spindle-shaped, cylindrical, or sacculated dilatations; at other times entire arterial areas—as those of a limb—are uniformly dilated. Moreover, this latter form of dilatation may not be only passive, produced by the blood pressure, but may be brought about by the pathological alterations in the vessel itself, which may produce not merely a thickening of the intima, but an actual increase of its superficial extent.

Polotebnow[1] has published some experiments as to the diminution of the arterial elasticity. A piece of the femoral artery, 7½ cm. in length, was tied at each end; the one ligature was made fast, to the other a weight was suspended.

[1] Berl. klin. Wochenschr. 1868. S. 361.

	Sclerosed Artery. (Age of individual ?)		*Healthy Artery.* (Individual aged 22 years.)	
Suspended Weight in Grammes.	Length of Artery. Laden.	Unladen.	Length of Artery. Laden.	Unladen.
50	8.0 Cm.	7.6	10.5	7.5
100	8.5	7.8	11.2	7.5
200	9.3	7.9	12.1	8.0
500	9.6	7.95	15.0	8.1
1000	9.9	8.	16.5	8.1

With a similar load, therefore, the elongation of the sclerosed was less than that of the normal artery, whilst the permanent extension, persisting after the removal of the weight, was in both very much the same.

In other experiments, one end of a piece of artery was tied and the other fastened to a graduated burette, into which mercury was poured; sclerotic arteries with a thickened intima could not sustain a pressure of more than from 20 to 25 cm.; when the mercurial column reached 28 or 30 cm. the intima and media burst, and the mercury trickled through the adventitia.

When the artery contained calcareous plates, it burst at once at a pressure of 15 cm., and no dilatation preceded this rupture.

If the intima and media were atrophied, but the external coat indurated, the artery resisted a pressure of 120 cm.

According to Wedl, the changes in the elasticity of the arterial coats is also well shown by the results of sticking round needles through them. In the normal aorta such punctures leave only a lineal transverse fissure behind; in diseased patches round or elliptical openings remain, which gape more or less longitudinally or transversely.

The following considerations show how greatly the blood pressure must be increased within the area of any rigid artery: while a normally expansile artery, say the radial artery, permits a portion of the blood during the systole of the brachial to pass through it, and stores up the rest in an elastic reservoir, a perfectly rigid radial cannot fulfil the latter function; less blood, therefore, flows out of the brachial, and the pressure within it remains greater than normal during the diastole; at the same time there is a great increase of the blood pressure in the radial itself, which may be likened to a rigid manometer tube introduced into the brachial. A similar change, only to a less degree, occurs in simple diminution of the elasticity of the radial; similar considerations are referable to every other, large or small, arterial area in the body.

The alterations in the circulation just referred to, combined with the diminution of the blood stream from arterial stenosis, unite in producing changes in the nutrition of the organs within the range of any given arterial area.

But both these causes also make themselves felt in other directions: the increased resistance induces in the centripetal part of the circulation an increase of the blood pressure, which may be propagated back to the aorta itself, should a sufficient number of the arteries be thus degenerated; and thus, as well as by disease of the aorta itself, the resistance to be overcome by the heart is increased; hence we very frequently have dilatation and hypertrophy of the left ventricle, with all the results that follow from them, coexistent with long-standing endarteritis chronica.

Mode of Occurrence.—In contrast to most circumscribed acute inflammations of the arteries, chronic endarteritis is generally diffuse, and occurs over larger tracts and simultaneously at several parts of the arterial system.

It is most frequent in the aorta, especially at its arch and in the ascending aorta, as well as where the branches are given off; sometimes only isolated patches are thus altered, at others the whole inner surface is strewn with atheromatous patches or beset with calcareous plates (*aorte pavée*); the aorta is usually greatly dilated in the more severe cases. The iliac, femoral, and tibial arteries are also frequently diseased, as well as the cerebral arteries, the coronary arteries, the subclavians and brachials, etc.

Frequently (Bizot), but not constantly, this degeneration is found to be symmetrical in both sides of the body; sometimes it is more developed in one of the arteries mentioned than in the aorta, but only very rarely the latter is free when other arteries are affected. Almost the only arterioles thus affected are those of the brain, and cerebral hemorrhage and softening are in most cases dependent upon such arterial disease.

This degeneration rarely occurs in the pulmonary artery, and usually only in a trifling degree.

The various authors on this subject are pretty well agreed as to the scale of frequency in which the different arteries are affected; this enumeration is from Rokitansky: ascending aorta, arch of aorta, abdominal aorta, thoracic aorta, splenic, femoral, internal iliac, coronary arteries, cerebral arteries (trunks and branches) within the cranium, vertebral, uterine, brachial arteries and their branches, internal spermatic arteries, common carotids, hypogastric arteries; very rarely, the mesenteric, cœliac, gastric, coronaries, hepatic and epiploic arteries.

Fatty degeneration of the normal intima has about the same area of diffusion as that of the thickened intima.

Simple calcification of the media is specially seen in the arteries of the lower extremities, and next in those of the arm and head.

Etiology.

Endarteritis chronica is an extremely frequent disease, especially in advanced life; indeed, so common is it after fifty, that some have regarded it as a normal senile change; yet it is sometimes absent even in persons from eighty to one hundred or more years of age (Harvey, Bamberger, etc.). In youth it is very rare; its severer forms are uncommon before forty years of age.

The male sex is most frequently affected, but more or less favorable social conditions seem to make no difference. On the other hand, this disease seems to be less common in some places, such as Zurich (Lebert), and Lemberg (Duchek), than in others, such as Paris, Breslau, etc.

In contrast to the statement just made, Guéneau de Mussy considers, from one hundred and sixty clinical observations, that arterio-sclerosis is just as frequent and just as severe before the forty-fifth year as after it. He also thinks he has made out that this disease develops itself more rapidly and with greater intensity in men, and more slowly in women.

Chronic alcoholism is regarded by many authors as a cause of arterio-sclerosis, and it appears specially to favor its development in early life (Guéneau de Mussy). The influence of lead-poisoning is more doubtful, but this is often associated with alcoholism; —gout,[1] rheumatism, syphilis, and chronic disease of the kidneys have all been claimed as causal agencies. Virchow has observed simple fatty degeneration of the intima, chiefly in connection with anæmia.

The influence of gout and rheumatism in the production of this form of disease is brought prominently forward by Lobstein, Andral, and others. Guéneau de

[1] *Landerer* (Buchner's Repertor. Bd. 45) once found fourteen per cent. of uric acid in a concretion from the aorta. *C. E. E. Hoffmann* never found anything like this in the course of numerous investigations.

Mussy thinks he can prove this in at least the half of all cases, and that he has several times seen a severe degree of arterio-sclerosis, instead of a cardiac disease, originate in an attack of acute rheumatism. Wilks[1] and Seitz[2] have both testified as to the frequent simultaneous occurrence of renal disease and chronic endarteritis. Dickinson found disease of the arteries in 52 per cent. of cases of contracted kidneys. Syphilis is, especially by English authors, regarded as a frequent cause of chronic endarteritis of the aorta (vide Aitken).[3] This form is distinguished from that produced by mechanical causes, in that it is more extensive, and not limited to the ascending and transverse portions of the arch of the aorta; that the intima is not so distinctly corrugated, but has a worm-eaten appearance, and exhibits more distinct elevations from deposits beneath it (? Q.).[4]

As to the influence of lead-poisoning, the reader is referred to—amongst others —Duroziez, Gaz. des Hôpit. 1867. Nos. 146—50.

Given the conditions just mentioned—most probably due to the composition of the blood and the general nutrition of the tissues—the constitutional disposition to an endarteritis, yet to this we have very frequently superadded a mechanical agency, a functional strain of the arteries (Rokitansky). In particular, tearing and stretching must be regarded as the direct cause of the more frequent and more severe affection of individual parts. In the aorta, at least, the parts most frequently affected, the ascending and transverse portions of the arch, and the parts where the branches are given off, are those most exposed to tearing and stretching; and a similar remark may also be made in regard to the arteries of the heart, spleen, and uterus. The pulmonary artery is only affected with this form of disease when it has become greatly distended by an obstruction to the circulation, and its coats thus injured; and the truthfulness of the etiology just propounded is confirmed by the facts that processes similar to endarteritis occur in veins affected with aneurism by anastomosis, and in the pulmonary veins when a high degree of mitral stenosis is present.

Sometimes this affection seems to originate in the propagation of a chronic inflammatory process from the endocardium to the inner surface of the aorta.

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[1] Guy's Hosp. Rep. 1853. [2] Arch. f. Physiol. Heilk. IV. S. 53.

[3] Science and Practice of Medicine. *Davidson*, Army Med. Rep. 1863. *Myers*, Transact. Path. Soc. XX. 1870.

[4] *Myers*' Diseases of the Heart among Soldiers. London. 1870. p. 78.

Traube regards remora of the blood-stream (such as occurs in the aorta during each diastole) as favorable to the development of endarteritis ; because this favors the view adopted by him as to the adherence and emigration of the white corpuscles (*vide antea*).

But "functional strain" of the arteries is not only of importance in regard to the positions of preferential occurrence, but in relation to the development of this disease generally. Thus Kirkes and others look upon the frequently concomitant hypertrophy of the left ventricle, and the consequent more forcible systolic dilatation of the arteries, as a frequent cause of this disease, an opinion which is undoubtedly true in many cases, while in others the causal sequence of events is precisely the reverse.

It is possible, also, that habitual excitement of the heart's action by bodily exertion, physical excitement, and spirit drinking, may be followed by similar results.

Endarteritis sometimes originates in the propagation upwards of an endocarditis.

Symptoms.

In most cases of endarteritis the alterations are not so great as to lead, during life, to any marked disturbance in the health of the individual. When the process occurs in advanced life in moderate intensity and to a limited extent, no other symptoms are observed but those of commencing age, senile involution generally. Widespread and far advanced arterial degeneration may even be not unfrequently found in old persons, who to the very last were apparently perfectly healthy.

Nevertheless, the anatomical description already given is sufficient proof that this disease is of the greatest significance in relation to the continuance of the organism and the integrity of its individual organs ; indeed, it seems highly probable that at least part of the phenomena of senile marasmus is the direct consequence of arterial degeneration.

Contrasted with these, as it were, normal cases of the gradual and imperceptible development of this condition in advanced age, there are other cases in which more or less evident symp-

toms are present, symptoms which may indeed be very various, according to the locality and stage of degeneration. These phenomena are usually specially noticeable in younger individuals, perhaps because the course of the disease in them, though still chronic, is yet relatively more rapid, perhaps because the nutrition of young and still active organs suffers more quickly.

In the heart, dilatation and hypertrophy of the left ventricle occurs frequently in those cases where the larger arteries are narrowed, or where considerable peripheral arterial areas have become sclerotic and inelastic. The greater resistance to the circulation thus produced has all the effect of a stenosis of the aorta; the more powerful the individual, and the better nourished his heart is, so much the more complete will be the development of the compensating hypertrophy of the left ventricle, while in opposite circumstances, in weakly individuals, dilatation prevails. If large tracts of the arteries are rigid, at the commencement of each systole the pressure within the ventricle rapidly increases, and its walls are dilated, as it were, by desultory impulses; and, doubtless, these continually repeated impulsions contribute to hasten the subsequent degeneration of the cardiac muscle.

Usually the hypertrophy is moderate and generally proportional to the intensity and extent of the arterial disease; but in individual cases, as in cardiac hypertrophy generally, this accurate proportion is not always to be found. The state of the nutrition generally, and many other circumstances, as yet unknown, are of importance in regard to this.

A. L. Galabin[1] found seventeen cases of moderate cardiac hypertrophy among twenty-three cases of pure arterial sclerosis (without nephritis).

The characteristic phenomena of hypertrophy of the left ventricle—displacement to the left and downwards, with a corresponding increase of the cardiac dulness, strong heaving impulse, and increased loudness of the first sound—[? *Tr.*]—are not unfrequently obscured or effaced by the coexistence of ossification of the costal cartilages and pulmonary emphysema.

These complications obscure the diagnosis still more when

[1] On the Connection of Bright's Disease with Changes in the Vascular System. Thesis. London. 1873.

the heart is atrophied, with pigment deposit in its muscular fibres (brown atrophy). Local nutritional disturbances of the cardiac muscle from endarteritis of the coronary arteries and general cachexia seem to favor the occurrence of this condition.

As the disease progresses we frequently find, both in the atrophied and in the hypertrophied heart, fatty degeneration of the muscular fibres and also indurations.

In the more advanced stages of this muscular affection, the first sound is (certainly not always) feeble or impure, and palpitations, dyspnœa, and disturbances of the general circulation set in.

The heart becomes sympathetically affected after another fashion when the inflammation is propagated from the ascending aorta over the aortic valves into the endocardium. Simple thickening and fatty degeneration lead to no very remarkable functional disturbances, but more deeply invading sclerosis and calcification lead to stenosis or to insufficiency of the aortic valves, more rarely of the mitral valve, with all the results that flow from such lesions.

But there are other reasons why valvular cardiac disease should not rarely coexist with arterio-sclerosis: because the valves are exposed to increased pressure as well as the cardiac muscle, and because the causes of endocarditis and of endarteritis are in a great measure the same.

Should the ascending portion of the aorta be dilated, this can sometimes be detected by dulness on percussion at the right edge of the sternum. The sounds heard at the origin of the aorta (in the aortic area) exhibit many alterations. The first sound is usually blunt and faint or altogether absent, from diminution of the vibratility of the aortic walls. Not unfrequently it is accompanied or obscured by a systolic murmur, which is either due to thickness and rigidity of the aortic valves or to the dilatation of the aorta, and is most distinctly audible over it.

This murmur is louder the more rapid the transition from the normal calibre of the vessel to the dilated part. In this case it is the alteration in the diameter of the vessel that gives rise to eddies in the current, whilst in thickening and rigidity of the valves these project across the orifice and render it irregular. The older idea,

that the roughness of the lining membrane of such diseased arteries was the cause of the murmur heard, was first of all opposed by Hamernik, and is now given up by almost all observers. The rapidity of the circulation in the peripheral stratum of blood is not great enough to produce an audible murmur. Such a murmur may be distinguished from that of aortic stenosis by its minor intensity and sharpness [?]. When the stenosis is considerable, the hypertrophy of the left ventricle is much greater, and the pulse small, hard, and slow.

The aortic second is also frequently dull and clangless—when the valves are thickened and non-vibratile; followed by a murmur—when insufficiency is developed. At other times the aortic second is accentuated, the valves are closed with a slam, and the sound has an almost metallic clang, and is audible over a wider area than usual. Bamberger thinks this is specially the case when the aorta is dilated, its walls rigid and calcified, and the valves enlarged but little thickened.

The alterations produced in the peripheral arteries by arteriosclerosis are frequently perceptible on palpation, and may sometimes even be seen. The artery is longer and wider than normal; this increased length of the vessel produces a tortuosity of its course, more evident during each cardiac systole and to some extent disappearing during each diastole; this mobile tortuosity renders the temporal artery specially visible; it is also readily seen in the radial, the carotid, and other arteries. The vessel seems hard to the feel, even during the diastole, as its resistance is so much greater than that of the surrounding tissues; the thickening of the coats is perceptible on compression, and like a dense cord the artery may be moved about beneath the skin. Sometimes the calcareous plates may be felt, or the artery seems irregular in its diameter, like a string of beads (rosary); in the most extreme degree it is changed to a rigid calcareous tube.

The pulse exhibits many and various alterations from the normal. It is frequently abnormally delayed in the peripheral arteries from slowing of the pulse wave.

This is very distinctly observed when we examine corresponding arteries simultaneously; the reason for it is to be found sometimes in the diminished vibratility of the coats of the affected arteries, at others in stenosis of the main branch leading to them (as stenosis of the subclavian, when the pulse is delayed in the

radial); when the latter cause exists, the pulses are not only asynchronous, but of different strengths.

The pulse is also felt to be feeble when the artery has thickened walls (whatever be the state of its lumen); and when these are calcified, the pulse may become quite imperceptible, though the circulation through the artery may still continue.

Should the dilatation, however, be in excess of the thickening of the walls, the pulse in the artery affected is abnormally large and full.

From the nature of the concomitant cardiac affection (hypertrophy, myocarditis, etc.), the pulse may be strong and jerking, or it may be small and irregular.

Besides these more readily detected abnormalities, the pulse in arterio-sclerosis also exhibits changes in the form of its wave, which according to their degree may either be detectable by careful palpation, or may be only recognizable by means of the sphygmograph; these changes are partly due to alterations in the arterial coats themselves and partly to secondary affection of the heart.

From the diminished expansibility and elasticity of its sclerotic walls, the arterial tube is neither so rapidly nor so fully distended as formerly, and it also returns more slowly to its former volume; the pulse wave therefore has a lower line of perpendicular ascension, a flatter (often rounded) apex, and (after a short perpendicular ascension line further on) a more gradual declension than usual. The reflux wave, but still more the waves of elasticity in the line of descent, are much diminished, or entirely wanting. The (so-called anacrotic) vibrations in the line of ascent are, on the other hand, abnormally well developed. On the whole the pulse curve much resembles that produced during great distention of the artery with contraction of its muscular coat.

Of all the various qualities ascribed to the pulse since the olden times, the pulsus tardus is that which corresponds most with the pulse of arterio-sclerosis.[1]

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[1] *Marey* points out the resemblance between the curve of the pulse of the senile radial and that of pressure in the left ventricle (within the heart of a healthy horse), in their

Coexistent hypertrophy of the left ventricle modifies the pulse, so that the wave is ampler and the line of ascent more perpendicular and the anacrotic vibrations all the more readily produced.

The chief of these alterations in the form of the pulse wave are also readily perceived by the finger, as a pulsus tardus, or pulsus tardus et durus.

According to the degree of the arterial disease, these alterations in the pulse curve are more or less developed, and exhibit every possible stage of transition to the normal in those cases in which this change is only commencing. In general, as age advances, the pulse approximates to this form, even where no other symptoms of chronic endarteritis are present; and though it may not be true in every case, yet in most it is right enough to conclude that the loss of expansibility and elasticity of the arterial walls, shown by this form of pulse, is dependent on chronic endarteritis. Moreover, this type of pulse also exhibits many variations due to physiological and pathological (such as febrile) changes in the vascular tonicity and repletion—exactly as the normal pulse does, but in a less marked degree.

It is evident that the characteristic phenomena of the arterio-sclerotic pulse may vary in the different arteries of the body, according to the amount of alteration in the artery in question, and also that the condition of other portions of the vascular system, particularly the centripetal and peripheral portions, may have an important influence on the form of the curve. Dilatation of the centripetal part of the arterial area may efface the details of individual curves;—stenosis has a similar result, at the same time the effect of medium repletion is trifling; rigidity or stenosis of the peripheral arteries hinders the outflow, raises the pressure in the part examined, and thus produces displacement of the katacrotic vibrations towards the summit of the wave, etc. The number of the possible combinations is increased, in that, on the side of the heart, besides hypertrophy and dilatation, other alterations of the muscular action (produced by degeneration or disturbances of innervation) may influence the form of the curve in a most important manner. A more accurate study of these conditions in artificial arterial schemata, and more extended sphygmographic investigations of different arteries in the same individual, will probably by and by enable us more accurately to define arterio-sclerosis, in relation to its degree and position in the arterial system; at present sphygmographic investigation is only preferable to simple palpation in reference to the diagnosis of trifling alterations of the elasticity of arterial walls.

The signs of sclerosis of the ascending aorta have been

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systolic portions. It is very doubtful, however, whether this resemblance is not accidental and actually (as *Marey* supposes) depends upon the fact that the cardiac curve is exactly reproduced in the periphery by the rigid arterial tube.

already narrated. If the transverse portion of the arch is also affected, this can often be detected by pressing the finger behind the sternal notch—if the arch be dilated or pushed upwards by elongation of its ascending portion.

Apart from aneurism, diseases of the descending thoracic aorta cannot be diagnosed; on the other hand, dilatation and alterations of the walls of the abdominal aorta, and of the iliac arteries, can in many cases be detected through the abdominal walls.

In the arteries of the extremities, besides the results of palpation, we have also those of disturbed circulation, particularly in the fingers and toes. There are subjective feeling of cold, often an actual diminution of temperature and of the normal turgor, feeling of formication and of numbness. Trifling injuries, such as a moderate exposure to cold, may in these circumstances lead to inflammation and subsequent death of the part. In other cases, apparently without any cause, gangrene and mummifying of a part may occur, and, as the result of that, an inflammatory line of demarcation may form.

Most frequently single toes or even a part of the foot may be thus affected, either because the blood has ceased to pass through the rigid narrow vessels, or because a local thrombus or an embolus has cut off the blood supply.

These senile or spontaneous gangrenes are frequently symmetrical, because the arterial disease is often bilateral. Enfeeblement of the circulation from cardiac debility, intercurrent febrile attacks, or digestive disturbances, may act as exciting causes.

In other cases we find a disposition to œdema in the lower extremities, most likely because the rigid arteries can no longer maintain a uniform circulation in the capillaries, and the nutrition and elasticity of the tissues suffer.

Varicose dilatation of the veins is frequently associated with this condition.

It is much more difficult to diagnose the diseases of the arteries of internal organs. It is only when functional disturbances occur, associated with other symptoms of an arteriosclerosis, that the diagnosis has any foundation to rest on.

Thus, as we know, the greater number of cerebral hemorrhages depend upon disease of the cerebral arteries, and also most cases of cerebral softening, especially in advanced age, are due to stenosis or obstruction of the arteries supplying the part affected. Since the cerebral arteries are those most frequently early affected, an attack of apoplexy or of paralysis is frequently the earliest evidence of an existing arterio-sclerosis. At other times the whole brain suffers from the gradually-increasing disturbance of the circulation and atrophies; the vacuum thus formed in the cranial cavity is filled by serous effusion into the ventricles and between the meninges; and in such cases the earliest symptoms are a gradual failure of intelligence and memory, while headache and dizziness may also occur.

Further, many cases of epilepsy, of dementia paralytica, and of multiple cerebral sclerosis, depend upon endarteritis of the cerebral arteries (Hertzka).

The other organs are much less sensitive than the central parts of the nervous system to abnormal changes in their blood supply, the most sensitive being the heart, which is frequently affected with disturbances of its innervation when the coronary arteries are atherosed. This is sometimes evinced by attacks of palpitation, at others by irregularity and inequality of the individual contractions, and at still others by a general feebleness of the heart's action.

In many cases abnormal sensations accompany the preceding phenomena, or may form the prominent symptoms in the case, such as a feeling of oppression and pain in the region of the heart and all the complex symptoms which make up angina pectoris.

The anatomical alterations in the intracardiac nervous apparatus, which lie at the bottom of these abnormalities, are not known; on the other hand, the degeneration and atrophy of the cardiac muscle already described are sometimes seen to result from disease of the coronary arteries.

Occasionally the (perforating) gastric ulcer seems to be the result of disease of the gastric arteries, inasmuch as the alteration of the circulation associated with it, which possibly enough is sometimes a local thrombosis of the arterioles of the gastric

mucous membrane, deprives this membrane of its power of resistance, and so paves the way for auto-digestion of the part.

In the other organs sclerosis of the nutrient arteries gives rise to alterations, which are to some extent of even greater importance; but the symptoms of them are but faintly expressed. Throughout them all we have the characters of depressed functional energy and atrophy of the organ. These disturbances are produced partly by narrowing of the arteries and diminution of the blood supply, and partly by the already frequently referred to alterations in the blood-wave, depending upon alterations in the elasticity of the arterial walls, and which seem to influence the diffusion between the blood and the organic juices.

Serous transudation, fatty degeneration, or simple atrophy, are the results of these disturbances in the circulation, which may also lead to a remora of the blood in the abducent veins, and to consequent dilatation.

Thus we frequently find the liver flabby and atrophic (the so-called red atrophy, Rokitansky, or brown atrophy, Wilkes —when accompanied by venous hyperæmia), the spleen being small, with a corrugated surface and thickened capsule. The kidneys are frequently atrophic, and may exhibit parenchymatous alterations; only the latter changes produce albuminuria.

Moreover, it must be expressly stated that the intensity of the organic diseases narrated is by no means always proportionate to the amount of lesion in the nutrient arteries.

Many of those phenomena, whose totality constitutes the typical senile marasmus, may be referred to a more or less extensive sclerosis of the arteries, or at least they are developed simultaneously with it, and may therefore be regarded as indications of its presence; amongst them we may reckon wrinkling and dryness of the skin, wasting of the muscles and of the fat, the arcus senilis of the cornea, ossification of the costal cartilages with the resulting rigidity of the thorax, senile kyphosis, senile emphysema of the lungs, and, lastly, various changes in the joints, especially the arthritis sicca, which so often occurs in the hip-joints of old people.

The *course* of arterio-sclerosis is chronic, but varies in rapidity in different cases. Whilst in many the disease advances so

slowly, that life may, notwithstanding, be prolonged for twenty or thirty years, in others the phenomena of general marasmus or local disease set in within a few years. The amount of danger threatened in such cases to the integrity of organic function, or indeed to the organism itself, has already been referred to when speaking of the symptoms. At present, we have only to consider a few of the sequential phenomena which may arise from arteriosclerosis, particularly aneurisms, in the limited sense of the word, which so often lead to death by rupture, and thromboses, the formation of which is favored by the alterations in the lining membrane of the vessels and the remora of the blood stream, and which may give rise to embolism in the peripheral arteries.

When this disease is developed in advanced life, it usually progresses more slowly, and is less dangerous than when it occurs in younger individuals. The greater activity of the circulation in the latter seems to hasten the process, and of course involves a greater likelihood of danger from the formation of aneurism and of rupture.

Death in this or some similar manner is the direct result of the arterial disease, or it may occur independently from some intercurrent disease, such as pneumonia, bronchitis, or the like.

There is no possible cure of this disease.

Any *treatment* must be directed to preventing the development of the disease, and to delaying its progress. Everything hurtful and likely to favor the development of the disease must be avoided, and that all the more, so soon as symptoms of its commencement have been already shown. The customary use of alcoholic fluids is specially to be avoided, as well as every violent excitement of the vascular system, since this favors the increase of the disease, and may cause rupture of the affected vessel.

Otherwise, a symptomatic treatment is the only one indicated.

Hypertrophy of the Arterial Coats.

Hypertrophy of the arterial coats occurs as a uniform increase in size of all the three coats, usually combined with dilatation of

the vessel, and it occurs specially in hypertrophying organs and morbid new formations. Thus in enlargement of the thyroid gland we find the thyroid arteries more dilated and with thicker walls than usual; often they are also longer, and hence tortuous; carcinomatous growths are often provided with large nutrient arteries, arteries which, under normal conditions, are of trifling dimensions and nameless. The development of the arteries in the pregnant uterus comes also under this category.

Thickening of the walls and corresponding dilatation are also found in arteries which are developed in the formation of a collateral circulation in compensation of the stenosis or obstruction of another vessel; this is well marked in surgical ligature of the trunk of the femoral or brachial artery, as well as in the compensatory dilatation of the mammary and intercostal arteries in stenosis of the aorta at the ductus Botalli (ductus arteriosus).

A similar condition occurs in the trunk of an artery above a contracted portion, as well as in an entire arterial area above or anterior to some obstructed capillary area, for instance in the pulmonary artery.

Finally, under this head we must reckon that form of arterial disease known as a (surgical) cirsoid aneurism.

All these alterations in the arteries correspond to what we call in the heart "hypertrophy with dilatation." It has not yet been determined whether there is any such thing in the arteries as "simple hypertrophy" (without alteration of the lumen), or "concentric hypertrophy" (with narrowing of the arterial lumen); and, indeed, as is well known, it is not easy to determine even in the heart, in any special case, whether the one or the other form of hypertrophy is present. In the arteries this is specially difficult, on account of their trifling calibre, chiefly, however, because there exist no accurate measurements as to the physiological variations in the thickness of the arterial coats, in the arterial lumen itself, or in the relations of the one to the other. When once these have been definitely ascertained for every part of the arterial system, then we shall be in a position to decide approximately as to the existence of hypertrophy or dilatation in any artery; but even then the state of the arterial contraction at the moment of death, and at the commencement of cadaveric

rigidity, must have an influence which it will be difficult to calculate; possibly weighing the artery may help to a decision in such cases.

Henle (Anat. III. S. 72) gives the measurements ascertained in various arteries of five bodies; these differ very much from one another, but may serve as a provisional basis.[1]

The hypertrophy of the individual arterial coats has been already treated of under the respective heads; pure hypertrophy of the middle coat seems to be the

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[1] As Henle's "Anatomy" is accessible only to a few English and American physicians, the measurements here referred to are given below.—*Tr.*

"In the following table I have added some data of my own to those which have thus far been furnished on the thickness of arterial walls. The arrangement is according to the calibre of the vessels. The Roman figures indicate hundredths of a millimetre; those in brackets refer to the thickness of the circular fibre coat. The inner coat should not be taken into consideration, because its diameter has only a narrow range of variation, and even in the largest vessels, in the normal condition, scarcely amounts to 0.03 mm. The only exception to this rule is in the coronary arteries. Their inner coat, like the external, is composed of connective tissue and layers of longitudinal elastic fibres, which gradually diminish in size the nearer they are to the inner surface of the vessel; it has a thickness of from 0.1 to 0.3 mm., while that of the circular fibre coat averages 0.2 mm.

"In this table we notice at first the lack of agreement among observers in their statements about the same vessel. This is partly explained by the difference in the vessels in individual cases, and partly by the difference in the methods of preparing them. The measurements were all made from softened sections of dried vessels; and here the thoroughness of the softening, on the one hand, or of the hardening, on the other, are of importance. Of the external coat it is to be said, that it is sharply defined where it joins the circular fibre coat or rather the elastic; but that its outer boundary is uncertain, because its tissue gradually blends with that of the surrounding connective tissue. Reliable results, therefore, can only be expected from a comparison of the elastic fibre coats; but even in this case it is hard to avoid error. Thus, for example, to introduce a single instance only, the thickness of the circular fibre coat can increase to almost double size, and varies with the contraction of the artery, which after death only slowly disappears, and then returns with the rigor mortis. It is hardly necessary to remind the reader of the precautions that should be taken in such measurements; namely, to select for comparison only healthy individuals, and to take the averages only from a large number of observations. It seems to me a law will be deduced out of the comparison of these measurements of different arteries in one and the same body. Of the figures tabulated by Donders and Jansen, most, according to their account, were derived from the same body; mine, given under division I., were all from a young man, and all under division II. were from a young woman, both of whom had suffered a violent death. The figures relating to the superior mesenteric agree with those tabulated by earlier observers; the thickness of the inferior mesenteric, however,

only one which occurs, while thickenings of the adventitia, but especially of the intima, which were formerly described as hypertrophies, are now by most anatomists

is at variance with the law referred to above."—(From Henle's "Anatomy," Vol. III., Part 1st, pp. 71 and 72.)

	Donders and Jansen.*	Koelliker.†	Gimbert.‡	My own Observations.	
				I.	II.
Sinus of the aorta	67 [54]				
Ascending arch	84 [80]				
Thoracic aorta			[77]		
Abdominal aorta	75 [65]	95 [60]	[45]		75 [60]
Innominate	100 [60]		[61]		
Common iliac	72 [40]	60 [33]	100 [55]		57 [42]
Subclavian	58 [38]	60 [51]	[33]		
Superior mesenteric	50 [10]	47 [18]		50 [25]	[20]
Cœliac axis	53 [12]	43 [15]	[16]		
Common carotid, origin	70 [45]	49 [37]	[44]		56 [36]
" middle		51 [39]			
External iliac		49 [26]			
Femoral under the crural arch	62 [34]	58 [26]	[33]		52 [27, in places 47]
" middle	48 [21]	56 [22]	[22]		
Axillary	65 [35]				
Internal iliac	54 [27]		76 [26]		[38]
Profunda		45 [22]			
Popliteal	43 [22]	72 [35]			[40, in places 60]
Brachial, middle		43 [16]	[33]		[22]
" at elbow		47 [22]			
Internal carotid	44 [31]	31 [13]			44 [32]
External carotid	55 [33]	29 [11]	67 [22]		[45]
Splenic			[20]		
Renal	38 [13]			47 [15]	[35]
Hepatic				56 [36]	
Post. tibial		41 [18]	[11]		
Ulnar	65 [35]		[33]		
Vertebral	34 [20]				
Basilar	25 [14]				
Internal maxillary			32 [16]		
Left coronary				35 [20]	
Radial, above	64 [34]	30 [13]	[33]	39 [22]	
" below		22 [9]		35 [23]	
External maxillary			[18]		
Inferior mesenteric				34 [18]	42 [22]
Lingual			[16]		
Superior thyroid					45 [30]
Anterior tibial		35 [18]	[17]		
Middle sacral					[8]
Temporal			[11]		
Ovarian				11 [6]	
Dorsalis pedis			[5]		
External plantar			[8]		
Palmar arch			[35 b.44]		
Radialis indicis		24 [11]			
Digital of middle finger		15 [6]			
Digital of 0.45		7 [3]			

* Nederlandsch Lancet. 2. Ser. II. 476. † Microskop. Anatomie. II. 512.

‡ Journ. de l'Anatomie. 1865. p. 526 et seq.

regarded as the result of inflammatory processes, and have accordingly been treated of under that heading.

No symptoms referable to hypertrophy of the arterial walls are known. In those arteries which can be submitted to a sphygmographic investigation, we ought to have a diminution in the line of ascent and a disappearance of the secondary wave.

Atrophy of the Arterial Coats.

Atrophy of the arterial coats with a corresponding reduction in the calibre of the vessel is found in the arteries of atrophying organs and in the stumps of amputated limbs. It is also observed generally extended over the arterial system in cases of general marasmus and atrophy, further in depression of the circulation from stenosis of the aorta or atrophy of the heart, and, finally, in cases of congenital hypoplasia of the arterial system, which shall be presently alluded to.

How far it is possible for atrophy of the arterial coats to occur, independently of changes in the arterial calibre, is but little known; careful measurements are as requisite to determine this as they are in regard to arterial hypertrophy.

Vide also the chapter on hypoplasia of the arteries.

Degenerations. Neoplasmata of the Arterial Walls.

The degenerations of the arterial coats have been already partially noticed in connection with the diseases of the individual coats themselves.

Fatty degeneration occurs most frequently, partly in connection with endarteritis, partly as one of its results. It is most frequently found in elderly individuals as a fatty infiltration of the endothelium, of the cells of the connective tissue, and of the muscles, and also as a granular fatty deposit in the interstitial tissue of these structures. In this way the epithelial cells may be destroyed and the intima laid bare. Fatty degeneration is most common in the aorta.

The adventitia only undergoes fatty degeneration in the smallest arteries. Fatty degeneration of the intima, as well as

of the connective-tissue cells, and of the endothelium, is further of no uncommon occurrence in young persons, particularly when anæmic.

Ponfick[1] has observed fatty degeneration of the endothelium alone, in erysipelas, and in other infective diseases, especially in relapsing fever, and this may even be recognized by an examination of the blood in these diseases during life.

Calcification is a frequent senile alteration of the muscular fibres of the media (*vide antea*); in the intima it occurs as the result of chronic inflammation.

Küttner[2] has described an extensive metastatic calcification of the intima. The case was one of rarefying ostitis, so-called scrofulous caries of all the dorsal and lumbar vertebræ, of a man aged nineteen; the liver and spleen were amyloid; there was purulent nephritis and meningitis. All the arteries were calcified, and more so the further they were from the heart, and the smaller the artery in relation to the trunk from which it sprang. The heart, thoracic aorta, innominata, subclavians, common and internal carotids, and the cerebral and meningeal arteries alone were unaffected. The veins were also exempt. In the liver and spleen all the small arteries were incrusted. The arterial tubes were annularly rigid, but everywhere normally translucent; the adventitia and media were exempt. Only in and upon the intima there were transverse calcareous plates, which in the larger arteries were to be felt as transverse bands, and in the smaller ones were seen microscopically to be finely granular and strongly refractive; hydrochloric acid cleared up the preparation with the development of gas, so that the intima was distinctly seen, only somewhat thickened. There was no pathological deposit in any other organ or tissue. Between the plates the epithelium was uninjured.

The calcification of the perceptible arteries had taken place in the course of the last few weeks of the patient's life, under the very eyes of the physician, so that at last the pulse could only be felt in the femoral artery.

During life the amyloid degeneration of the liver and spleen was all that could be diagnosed with certainty.

K. attributes the calcareous metastasis to a deficiency of carbonic acid in the blood.

Amyloid degeneration, which is more frequently found in the small arteries, such as those of the renal glomeruli (Malpighian bodies), and of many other organs, than in any other part of the

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[1] Deutsche Klinik. 1867. Virch. Arch. Bd. 60. 1873. Centralbl. f. d. med. Wiss. 1874. S. 384.

[2] Virch. Arch. 1872. Bd. 55. S. 521.

frame, and seems chiefly to affect the muscular coat (Virchow, Kyber), is not unfrequently observed in the large arteries, particularly in the aorta and pulmonary artery (never on the far side of the axillary and common carotid, and never in the femoral). In these cases groups of muscular fibres, particularly from the middle of the intima, as well as the nutrient arterioles of the adventitia, are found degenerated.

This degeneration occurs in chronic cachexiæ, suppurations, syphilis, and malarial poisoning, with concomitant affections of other organs.

New formation of connective tissue occurs in the adventitia as the result of chronic inflammation, chiefly propagated from the intima ; and in the intima itself, as cicatricial tissue, at the seat of former ulcerations, and in the formation of these it seems probable that the elements of the thrombus take a share.

Carcinoma occurs both primarily and secondarily in the arterial coats. A carcinomatous growth only rarely implicates an artery in its neighborhood, and usually only the adventitia. In very rare cases all the coats of an artery may become perforated and infiltrated by carcinoma, and then the vessel either becomes closed by the formation of a clot, or hemorrhage occurs.

Broca[1] describes a case of cancer at the commencement of the aorta; it grew from the arterial coats themselves, and led to perforation of the pericardium.

In a case observed by Wernher[2] of medullary cancer of the tibia, there were found, in the chief and in many of the smaller branches of the pulmonary artery, beaded strings of cancerous matter, which were connected with the inner coat of the artery by means of fine (vascular ?) threads, and narrowed or obstructed the lumen of the affected vessel. Most probably they originated in wandering embolic cancerous particles which had become developed at the part implicated.

Brodowski[3] has recently described a case of primary sarcoma of the thoracic aorta, which as yet remains quite unique. This commenced in the adventitia and passed into the media, and had given rise—without any evidence of continuity—to confluent secondary neoplasmatic masses in the intima, which, when cut into, were found to be gelatinous and about 1 cm. thick, and which narrowed the aortic

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[1] Bull. de la Soc. Anat. 1850.

[2] Zeitschrft. f. rat. Med. V. S. 109. 1854.

[3] Pamietnik towarz lek. Warz. IV. 388. Jahresber. v. Virchow-Hirsch. 1873. II. S. 243.

lumen. The lower part of the thoracic aorta was changed by this neoplasm into a flattened oblong tumor about 11 cm. in length.

Microscopically the tumor was found to contain very large spindle-cells and a small amount of fibrous matrix. Secondary masses, probably produced by emboli from the aorta, were found in large numbers and of various sizes in all the abdominal organs, in the peritoneum, and in the ilium. The patient, a man aged fifty-two, was seized only six weeks before his death with gastric disturbances, accompanied by a feeling of burning in the scrobiculus cordis and pain in the region of the left kidney, which by and by extended over the whole abdomen. Gradual diminution of strength, and quickening of the pulse, but no fever. Death from exhaustion. Three years before his death he had received a blow from the pole of a carriage in the left side.

Parasites in man have as yet been only observed within the vascular lumen, such as young trichinæ, filaria sanguinis (in cases of chylous urine), distoma hæmatobium. Of the larger parasites, echinococcus cysts are sometimes found in the larger arteries, even in the trunk and branches of the pulmonary artery and in the aorta, into which they have escaped from the liver or cardiac muscle. These, like other emboli in such vessels, produce considerable obstruction of the circulation, and even death. Such a case has been described by McCall Anderson ;[1] in it cysts were found in the aorta, iliac, and left internal carotid arteries.

In the arterial coats parasites have as yet only been found in animals ; thus Lewis found, in an Indian dog, pea-sized tumors along the aorta, and somewhat smaller nodules situated within its coats, projecting internally, and some of them ulcerated ; within these tumors filariæ lay coiled up.

In the abdominal arteries of horses (and especially of asses—*Tr.*), particularly in the anterior mesenteric artery and its branches, the sexually immature young of the palisade worm, sclerostomum armatum (strongylus armatus minor), is found in great numbers and very frequently (in about ninety per cent. of all horses—[but not so commonly in Britain.—*Tr.*]). They seem to give rise to aneurisms by injuring the arterial coats, and are found within the aneurismal cavity. They are generally found in considerable numbers within the aneurism, inclosed in a blood-clot. The coats of the aneurism are strengthened by

[1] Glasgow Med. Jour. 1872.

hypertrophy of the media. The clot within the aneurismal cavity not unfrequently gives rise to embolism of the intestine, and thus occasions symptoms of colic.[1]

Syphilitic Diseases of the Arteries.

Vide also Vol. III. of this Cyclopædia. p. 217.

Clifford Albutt, St. George's Hosp. Rep. Nos. III. and IV. Transactions of the Pathological Soc. XXIII. p. 16. 1872.—*Heubner*, Arch. d. Heilk. XI. 1870; and Die luetische Erkrankung der Hirnarterien. Monographie. Leipzig. 1874. (Bibliography here given in full.)—*Lancereaux*, Traité hist. et pratique de la syphilis. Paris. 1866. and New Sydenham Soc. Trans. London. 1868.—*O. Steenberg*, Den syphilit. Hjirnelideloefs. Kjobenhavn. 1860. Canst. Jahresber. 1861.—*Virchow*, Arch. XV. S. 243. Geschwülste S. 444.—*E. Wagner*, Archiv d. Heilk. IV. S. 167. u. V. VII. 1866. S. 524. *O. Weber*, Syphilit. Neubildung. in d. Wand. d. A. pulmonal. Med. Centralzeitung. 1862. No. 52. Schmidt's Jahrb. 123. S. 180.—*S. Wilks*, Guy's Hosp. Rep. III. Ser. Vol. 9. 1863.—*Wilks and Moxon*, Pathological Anatomy. London. 1875. p. 147.

That syphilis is one of the causes of chronic endarteritis and thus of aneurism, has been already narrated; English authors in particular have described this causal sequence, and have ascribed to it certain variations from the ordinary form (*vide antea*). But these phenomena do not seem characteristic enough to constitute a distinct difference between the two.

In one case related by Virchow, extensive endarteritis seemed to be connected with syphilis.

Amyloid degeneration of the arteries may be the result of syphilis.

C. O. Weber, E. Wagner, and Virchow have published notices of syphilitic neoplasms of the arterial coats.

The first-named author found in a girl, with syphilitic disease of the cranial bones and the liver, an oval nodule in the right branch of the pulmonary artery, 2–3 cm. long, and 5 mm. thick, projecting into the lumen of the vessel, and so considerably narrowing it that it was reduced to a mere slit. The nodule was covered by the uninjured intima; it was soft and gelatinous, and consisted of granulation tissue springing from the media. The latter was for some distance around thickened. Both lungs, especially the right one, were beset with extravasations of blood, but free from tubercle. Death resulted from hæmoptysis.

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[1] *Bollinger*, Die Kolik der Pferde und das Wurmaneurysma. München. 1870.

Virchow and Wagner also found smooth (both hard and soft) nodules in the pulmonary artery, which seems thus to be a favorite position for these neoplasms.

Besides these morbid affections, others occur in the intima in the course of constitutional syphilis, especially in the cranial arteries, which from their most important result—cerebral softening—have been long well known. By most authors this affection has been looked upon as an ordinary chronic endarteritis, whilst others, such as Wilks, Lancereaux, Clifford Albutt, and Heubner, regard it as anatomically different and peculiar to syphilis.

The latter author bases the following description of this disease upon cases which have been observed by himself and a comparison of those recorded by others.

The arteries at the base of the brain have none of those striated lamellæ containing cells which are found in the intima of the aorta and carotids, but the endothelium is closely applied to the membrana fenestrata (which belongs to the intima). At the commencement of the syphilitic affection of the arteries, between these two lamellæ we find spindle-shaped nuclei and cells, which spring from the cells of the endothelium, and are ranged in longitudinal rows corresponding to the striæ of the membrana fenestrata. These cells, which have but little intercellular matter between them, acquire processes by which they are connected together; giant cells are also occasionally formed.

When this neoplasm has attained a certain development, we find associated with it groups of small round cells—white blood-corpuscles which have emigrated from the vessels of the adventitia. The endothelial growth is always limited, confined to one side of the arterial circumference, so that the arterial lumen is excentrically displaced. On the other hand, it frequently extends longitudinally along the vessel primarily affected or into its diverging branches, so that long stretches of the vessel may be narrowed or even obstructed (by thrombosis, which is a frequent result). This neoplasm but rarely oversteps the region between the endothelium and the membrana fenestrata.

When this thickening has progressed for a few months, new capillaries become developed in its peripheral zone, which anas-

tomose with the vessels of the adventitia; on its internal surface, that next the arterial lumen, a secondary membrana fenestrata is formed, whilst the cell mass of the tumor separates into two layers—an inner one in which the cells are disposed circularly, and an outer one resembling embryonal connective tissue, in which the irregular cells which anastomose with each other are widely separated by a homogeneous intercellular substance. This neoplasm, therefore, repeats within itself the normal structure of the arterial coats, so that Heubner is justified in terming it an "arterioma."

This condition may be permanent, or in the course of years it may lead to the formation of a cicatricial mass of connective tissue.

H. rigorously differentiates syphilitic disease of the intima from the ordinary form of chronic endarteritis, because the former takes only months to develop, the latter years; the former is an almost purely local disease, while the latter extends over wide districts, and usually occurs also in other arteries; further, the syphilitic neoplasm is richer in cells and poorer in intercellular matter, contains a greater number of emigrated round cells, and leads to a peculiar form of organization, or to the formation of cicatricial tissue, and in no stage of its growth exhibits any tendency to fatty degeneration so constantly found associated with endarteritis. Referring to recent observations as to the participation of the endothelium in the organization of a thrombus, H. defines syphilitic disease of the arteries as originating in irritation of the endothelial cells, subsequently associated with inflammation (cell immigration). He finds no resemblance between this and other forms of syphilitic disease.

This affection has hitherto been solely observed in the basal cerebral arteries, and most frequently, indeed, in the anterior vessels coming from the carotids; still occasional observations have made it probable that other arteries also may become similarly diseased, such as those of the intestinal submucous tissue, of the umbilical cord, the liver, and the placenta.[1]

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[1] Vide *Heubner*, l. c. S. 168.

This form of arterial disease belongs to the tertiary period of syphilis, and only occurs after the lapse of years.

Only in isolated cases does it appear to have been developed within a year after the primary infection, and then in the neighborhood of and in connection with neoplasms of the subarachnoid space.

It occurs in both sexes, and (unlike arterial sclerosis) at any age.

H. supposes the existence of a neuropathic disposition as directly predisponent to this form of arterial disease.

During life we can diagnosticate syphilitic disease of the arteries only from the cerebral symptoms, because our knowledge of it is as yet confined to it as affecting the cerebral arteries, and these symptoms have reference only to the narrowing or obstruction of certain arteries, and in their totality present a certain resemblance to those due to chronic endarteritis of the cerebral arteries, only that in syphilis the disease is more often circumscribed, and therefore leads to distinctly local symptoms. To understand these symptoms, it is of importance to remember the differences shown by Heubner to exist between the ramifications of the arteries at the base and over the cortical part of the brain; for while in the latter part the smaller arteries anastomose freely before their branches pass into the brain substance, at the base many small nutrient vessels pass at once from the larger trunks into the brain tissue. Diseases of the latter arterial area, which syphilis preferentially attacks, is therefore speedily followed by injury to the nutrition of the great basal ganglia, and therefore by hemiplegia, which, according to the extent and degree of the stenosis, may be complete or incomplete, temporary or persistent. According, also, to the energy of the cardiac contraction, etc., the circulation varies within the affected area, and the paralysis is more or less marked and associated with hemiplegic contractures, clonic spasms, or pain; should the obstruction be complete, necrotic softening and persistent irreparable paralysis is the result.

In the cortical portion the effect of the narrowing of any artery is at least partially compensated by the network of anastomoses, so that serious disturbances of the consciousness and of

the mind are usually only temporary, while, on the contrary, the stenosis is quite sufficient to produce slighter functional disturbances, such as giddiness, pain in the head, changes in the disposition, and diminution of the mental energy. These are usually the earliest prodromata. With these the paralytic phenomena are sometimes associated in a somewhat sudden manner, in the form of an apoplectic attack with loss of consciousness; at other times they are more gradually developed (with occasional temporary improvements); later on, the mental functions become more and more affected, though even in this disease, as well as in senile softening, the incompleteness and variation in the functional disturbances are remarkable. General epileptic attacks but rarely coexist.

The duration of the manifest cerebral symptoms may be days, weeks, or months, sometimes—under appropriate treatment—more than a year. A cure, though possible, is but rarely seen.

For the treatment, the fundamental principles of anti-syphilitic therapeutics are all-sufficient; Heubner recommends mercurial inunction. Along with that, stimulants must be given, and the head elevated with the body in the horizontal position, so as to diminish the blood pressure in the cerebral vessels.

(General) Dilatation of the Arteries.

Beneke, Ueber d. lumina d. arteriellen Gefässe. Sitzungsber. d. Ges. z. Beförd. d. ges. Naturwiss. zu Marburg. 1868.—*W. Ruckert*, Ueb. d. Lumina d. art. Gef. Diss. Marburg. 1868.—*Joh. Kimpen*, Diss. Marburg. 1874.

We possess but few measurements of the normal width of the more important vessels of the body; and the measurements to hand[1] have only reference to physiological differences, and not to pathological variations; and even though the anatomist, from daily experience, may have an approximate idea as to the medium calibre of individual arteries, yet deviations from the mean can only be recognized as physiological or pathological by

[1] *Krause* (Handb. der menschl. Anatomie. Hannover. 1848. S. 638) determined the arterial diameter in recent, well-injected bodies, and gives the mean of several measurements. He frequently found a difference of $\frac{2}{7}$ of the diameter between the circum-

the results of numerous measurements; and, indeed, the calibre of every individual artery during life undergoes many changes from the variations in the blood pressure and the state of contraction of its circular muscular fibres.

In respect of measurements made on the body, many important circumstances require to be considered, such as the state of contraction or distention at the commencement of the rigor mortis, the intensity and rapidity of occurrence of this rigor mortis, as well as the degree of elasticity of the artery itself—as in a very elastic vessel the size of its lumen before and after the cessation of the blood pressure will, of course, be perfectly different from what it is in a rigid tube. Measurements in the dead body can, therefore, as in regard to all hollow muscles, give us only a very imperfect idea of the state of the artery during life; measurements of the diameter ought in such cases to be always combined with measurements of the thickness of the walls. Only after we have obtained a large series of such measurements for all the various arteries of the body at various ages, in both sexes, and in bodies of various sizes, made with special reference to pathological conditions, shall we be in a position to determine what alterations are to be regarded as pathological.

It is most probable that we shall thus obtain results having a most interesting bearing upon the diseases of individual organs, because the calibre of the vessels influences the blood supply in a most important manner, and therefore the nutrition and power of resistance of individual organs.

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ferential measurements of the arteries cut open and the calculation of their diameters. The following are, according to him, the diameters of the

Ascending aorta	28.0 mm.	Commencement of external carotid	5.6 mm.	Superior mesenteric	10.1 mm.
Arch of the aorta	22.6 "	Internal carotid	6.2 "	Inferior do	3.8 "
Upper part of abominal aorta	20.3 "	Right subclavian	11.3 "	Renal	5.6–6.7 "
Lower part of do	16.8 "	Left do	10.1 "	Common iliac	7.3 "
Innominata	13.5 "	Upper part brachial	6.8 "	Femoral	9.0 "
Right common carotid	9.0 "	Lower part of do	5.6 "	Popliteal before its division	6.2 "
Left do	8.6 "	Commencement of radial	3.9 "	Posterior tibial at the internal malleolus	3.4 "
		Cæliac artery	9.0 "		

When alterations in the vascular calibre are extended over an entire arterial area, this reacts upon the whole of the blood mass and upon the nutrition of the body generally.

Influenced by such considerations, Beneke and his pupils have made a large number of measurements of the aorta and its chief branches.

The results of such measurements made upon 96 and 125 bodies, respectively, have been published by Ruckert and Kimpen. The arteries were slit open, laid flat, their internal circumference determined in millimetres, and, taken in connection with the length of the body from which they were obtained, they were uniformly reduced to the measurements belonging to an individual measuring 100 centimetres in length. In this way it was found that the internal circumference of the

	Maximum.	*Minimum.*
Aorta, 1 ctm. above the edge of the valves, measured	58.8 mm.	28.7 mm.
Thoracic aorta, 10–12 ctm. below the giving-off of the subclavian	40.7 "	19.8 "
Abdominal aorta, 1–2 ctm. above its bifurcation	33.3 "	10.6 "
Pulmonary artery, 1 ctm. above the edge of its valves	58.0 "	28.8 "
Left subclavian artery, 1 ctm. above its origin	18.6 "	10.0 "
Left carotid, 1 ctm. above its origin	14.5 "	8.4 "

The authors named have not given any average measurements. The very great differences between the circumferential measurements are very remarkable, amounting, as they do, to a full half of the maximum. Slighter differences were found in twelve soldiers, who had died of wounds or disease during the last war; in particular, their maximum measurements were not so great. Sex had no apparent influence, but, on the other hand, the dilatation of the vessels seemed to increase with age, and the greatest width was attained beyond the thirty-fifth year.

In 53 per cent. of all the cases the pulmonary artery was wider than the aorta.

The vessels were much dilated in most cases of carcinoma, remarkably narrowed in pulmonary phthisis and allied scrophulo-tubercular conditions.

It has been known for a long time, not from exact measurement but from approximate valuation in a large number of cases, that the arterial diameter increased with age, and that it also increased with improved nutrition and increase in the size of the body; but it is not known whether the latter form of increase depends upon simple distention from increased blood pressure, or upon an actual increase in size of the arterial coats.

That hypertrophy of the left ventricle, in so far as it leads to

a permanently increased blood pressure in the arteries, may also produce a permanent dilatation of these vessels, seems possible, but it has never been proved.

De Renzi[1] has published a case bearing upon this subject, in which a man aged twenty-three, who from youth up had labored under aortic insufficience, had, besides cardiac hypertrophy, a general dilatation of all the peripheral arteries.

Uniform dilatation of a larger or smaller number of arteries down to their smallest branches is very frequently observed in arterio-sclerosis, sometimes alone, at other times associated with stenoses (*vide antea*).

Paresis and paralysis of the circular muscular fibres of the arteries, as well as atrophy of the muscular and elastic elements of their coats, which so frequently accompany arterio-sclerosis, also lead to dilatation in the affected arterial area, and that all the more readily the less the blood pressure has deviated from the normal.

Dilatation of individual arteries is, moreover, found generally combined with hypertrophy of their coats (vide this subject), in the arteries of neoplasms and hypertrophying organs, as well as in those vessels which go to form a collateral circulation, and, finally, in its most exquisite form in the arteries constituting a so-called (surgical) cirsoid aneurism. Dilatations are also often observed in those vessels which lie immediately in front of a stenosed artery, or whose arterioles and capillaries are destroyed or in any way obstructed, as, for example, in cases of partial induration of the lungs.

The (surgical) cirsoid[2] aneurism (an. racemosum, varix arterialis) belongs practically to the domain of surgery, since it occurs most frequently in the course of such superficial arteries as the temporal, occipital, supraorbital, and radial; this form of dilatation only really affects the carotids, or the iliac arteries. In this condition the arteries are elongated as well as dilated, down to their ultimate arterioles; they pursue, therefore, a tortuous course, their walls are thinned, whether as the result of chronic inflammation, or of impulse or pressure, which often exist

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[1] Virchow-Hirsch, Jahresb. 1873, I S. 215.

[2] In contra-distinction to a simply dilated artery with bulgings, which in the aorta specially Rokitansky has described as a (medical) cirsoid aneurism. Handbuch. 1844. Bd. II. S. 555.—*Tr.*

as occasional causes; the arterial walls are paralyzed and are only passively dilated. From the pressure of the dilated arteries the neighboring parts, skin and bone, are atrophied; and by the same means abnormal communications may be formed with veins or with other dilated arteries.

In rare cases the veins as well as the arteries of the vascular area affected are dilated; it is questionable whether this dilatation is always only the result of an abnormal communication with the arteries.

Symptoms.

The uniform dilatation of an artery is recognized during life by its being felt as a thicker cord with a more distinct and forcible pulsation than usual. Thus, in cirsoid aneurisms, small, nameless arterioles may attain the size of a crow quill, pulsate visibly, and give rise to a whizzing murmur.

In dilatation of the ascending aorta, the normal lung sound to the right of the manubrium sterni may be duller (of higher pitch and shorter duration) than on the left side; a slight pulsation may even be felt in the same situation when the dilatation is somewhat greater. In such a case the aortic second sound is often very loud and accentuated. If the ascending aorta is elongated as well as dilated, or if the transverse portion of the arch is coexistently dilated, then the latter may be felt in the supra-sternal notch (while normally its highest point does not extend above the plane of the middle of the first costal cartilage), and the subclavian arteries are pushed upwards into the supra-clavicular fossa, and are thus more readily felt.

The uniform dilatation of the descending aorta can only be recognized in its abdominal portion, where it is capable of being palpated; the abdominal pulsation already referred to as so often distinctly to be felt and even seen in women, besides other favoring circumstances, frequently originates in a (permanent or temporary) dilatation of the abdominal aorta (of a paralytic character).

We have but little pathological knowledge of the results and symptoms of extensive dilatations of the arteries of the body. It is not improbable that any considerable degree of this may lead to the accumulation of a relatively excessive amount of the

blood in these dilated arteries, and hence to a diminished repletion of all the capillaries and to general nutritional disturbances; but in such cases there is almost always present arterio-sclerosis, alteration of the elasticity of the arterial coats, from which a much more important interference with the circulation may originate, which may give rise to general nutritional disturbances.

Krauspe [1] has described a case of extensive arterial dilatation accompanied with the formation of many aneurismal bulgings. The patient was a shoemaker, aged sixty-four, afflicted with emphysema and bronchial catarrh. The most dilated (and tortuous) vessels were the carotids and several of their branches (the thyroids, linguals, and maxillaries); both of the external jugular veins, as well as the right internal one were dilated. The abdominal aorta was tortuous. There were spindle-shaped aneurisms on the axillary and femoral arteries. The left ventricle was hypertrophied. Probably there was arterio-sclerosis.

Dilatation of the pulmonary artery from primary disease of its walls is of rare occurrence; but, on the other hand, dilatation of the trunk of this artery, as well as of all its branches, is of very frequent occurrence in the course of every kind of obstruction to the lesser or pulmonary circulation, such as occurs in mitral insufficience, in collapse and emphysema of the lung, etc., as well as in insufficience of the pulmonary valves. Usually, hypertrophy of the right ventricle and accentuation of the pulmonary second are combined with this dilatation. When the dilatation is considerable, relative to the diameter of its conus arteriosus, a systolic murmur may be produced.[2]

The edge of the lung may also be forced aside by the dilated pulmonary artery, and then we may have a double impulse, a simple systolic pulsation, or a systolic whirr perceptible in the left second intercostal space.

Still, for the proper estimation of the diagnostic value of this sign, it is important to remember that it may occur without any dilatation of the pulmonary artery and without any hypertrophy of the left ventricle, when, from inflammatory contraction or

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[1] Berlin. Klin. Wochenschr. 1873. No. 11.

[2] Vide *Quincke*, Berlin. klin. Wochenschrift. 1870. No. 21.

shallow respiration, the edge of the left lung does not cover the heart to its normal extent, as not unfrequently happens in phthisical patients, in women, in young people of feeble muscularity, in anæmic individuals, in pregnant and puerperal women, convalescents, and those affected with acute rheumatism.

In such individuals the pulmonary second frequently seems abnormally loud when contrasted with the aortic second, without this being any indication of hypertrophy of the right ventricle. The determination of the position of the lung certifies the diagnosis in such cases.

On the other hand, the signs of an actual dilatation of the pulmonary artery may be marked when the heart and blood-vessels are covered to a greater extent than usual and also pushed back from the thoracic walls by an emphysematously dilated lung.

Treatment.

The treatment of a uniform dilatation of the arteries, when it is extended over a large area, and is the result of arterio-sclerosis, can only be directed against that. In relaxed conditions of the arterial muscle, cold, electricity, and the other remedies formerly mentioned, may be employed.

[The use of iodine externally and of iodide of potassium internally has been apparently useful in many cases of dilatation, especially of the aorta, subclavians, and carotids, possibly in that of other arteries also. Vide *Balfour*, Clinical Lectures on Diseases of the Heart and Aorta. London. 1876. Pages 223 and 407, etc.—*Tr.*]

In severe forms of cirsoid aneurism, surgical interference may become necessary. In most cases of dilatation no treatment is possible ; in collateral dilatation any interference is likely to be dangerous.

Of much more frequent occurrence and of more importance than the arterial dilatations just referred to, are those localized and partial dilatations of the arteries to which the term aneurism —in the limited sense of the word—is applied.

ON ANEURISM.

Cruveilhier, *Rokitansky*, *Förster*, etc. Handbooks of pathological anatomy.—*Wunderlich* (II. 403. III. 2. 680), *Niemeyer*, *Duchek*, and *Jaccoud*, Handbooks of special pathology.—*Corvisart*, *Laënnec*, *Hope*, *Stokes*, *Bouillaud*, *Bamberger*, *Friedreich*, *Hayden*, *Balfour*, Treatises on Diseases of the heart.—*Crisp*, *Lebert*, Diseases of the blood-vessels.

A. Richet, Art. aneurysme. Nouv. Dict. de méd. et de chir. prat. Paris. 1865. Vol. II.—*A. Luton*, Art. Aorta. Ibid.—*P. Niemeyer*, Schmidt's Jahrbücher. Bd. 110. S. 237. Bd. 125. S. 234.—*F. Helmstedter*, Du mode de formation des aneurysmes spontanés. Diss. Strasburg. 1873.—*A. Legroux*, Sur les dilatations simples circonscrites des artères dites aneurysmes vrais. Arch. gén. Nov. 1874. p. 585.—*W. S. Church*, On the formation of aneurisms, especially intra-cranial aneurisms, in early life. Barthol. Hosp. Rep. VI. p. 99. 1870.—*R. W. Smith*, Cereb. aneur. Dub. Quart. Journ. Nov. 1870.—*D. Ogle*, St. George's Hospital. Rep. 1867. II. p. 285. Aneurism of the coronary arteries.—*Ponfick*, Embolische Aneurismen. Berlin. klin. Wochenschr. 1873. No. 40. S. 481. Virchow's Archiv. Bd. 58.—*J. Lidell*, On internal aneurism and its relation to sudden death. Am. Jour. of Med. Sc. 1867. Jan. p. 46 (Etiological). *Myers*, Remarks upon the prevalence of aortic aneurism in the army. Transact. of the Path. Soc. XX. 1870. p. 117. Lancet, 1869. Feb.—*Do.* On the etiology and prevalence of diseases of the heart among soldiers. London. 1870.—*Axel Key*, Om aorta aneurysmes aterwerkampa hjirtat. Nord Med. Arkiv. Bd. I. 1870. Ber. II. S. 102.

Bellingham, Dub. Med. Press. 1848. Bd. 19. Vide *Stokes*, p. 546.—*Lyons*, On the motions and sounds of aneurism. Dub. Quart. Jour. of Med. Sci. 1850. Vol. IX. Vide *Stokes*, p. 548.—*Mayne*, Dub. Quart. Jour. Nov. 1853.—*Schrank*, Zur diagnostik der an. d. aorta desc. Thoracica. Allg. Wiener med. Zeitg. 1872. Nos. 34–36. Ber. II. S. 109.—*Fischer-Dietschy*, Experim. Beitr. z. diagnose d. aneurysmen. Deutsch. Arch. f. klin. Med. 1869. VI. S. 530.—*E. Leudet*, Gaz. méd. 1864. No.25. Diseases of the œsoph. caused by aneurism. Schmidt's Jahrb. 125. S. 127.—*J. Ogle*, Cases of an. of the thor. aorta affecting the bronchial tubes. Transact. of the Pathol. Soc. XVII. 1867. p. 99.—*Page*, Lancet. 1867. I. p. 43.—*Atlee*, Eructation as a symptom of thoracic aneurism. Americ. Jour. of Med. Sci. 1869. July.—*Kohts*, Aneur. d. Trunc. anonym. Ber-

liner klin. Wochenschr. 1873. Nos. 1 and 2.—*Coats*, Aneurism of the thoracic aorta and hæmorrh. into the spinal canal. Glasgow Med. Jour. Feb. 1872.—*M. Seidel*, Deutsche Klinik. 1868. Nos. 1 and 2. (5 cases of loss of voice.)

Thurnam, Med. chir. Trans. XXIII. 330. Aneur. arterio-venosum.—*V. Czerny*, Virch. Arch. Bd. 62. 1874. S. 464.—*Peacock*, Transact. of the Path. Soc. of London. 1868.—*Chaboud*, Lyon. médical. No. 26. 1873.—*Jacoby*, Berliner klin. Wochenschr. 1870. No. 13.—*Hayden*, Dub. Quart. Jour. 1826. p. 434.

Anatomy.

Those dilatations which are termed aneurisms affect at one time the whole of the artery for a certain distance, and at others only a portion of its circumference, and they may be cylindrical, spindle-shaped, or sacculated. Sometimes they are sharply defined (possibly by a margin projecting internally), as is the case with many sacculated aneurisms ; at other times they pass gradually into the normal lumen of the vessel, as the spindle-shaped and cylindrical aneurisms usually do, and in this way they form transitions to simple uniform dilatations.

Saccular aneurisms exhibit a great variety of forms, from the simply globular to such as are composed of several intercommunicating cavities ; sometimes the sac includes some extent of the vascular lumen, at others it is attached like an ampulla to the artery by a narrow neck. Not unfrequently the saccular form is combined with the cylindrical in the shape of numerous sinuous bulgings, so that irregular tumors result (*An. à bosselures* of Cruveilhier, cirsoid aneurisms of Rokitansky).

The size of an aneurism may vary from that of a pin's to that of a man's head.

The walls of an aneurism are almost constantly formed by the diseased arterial coats. The alterations in the walls are usually the results of a chronic inflammation of the intima : thickening, hyperplasia of the connective tissue, atrophy, fatty degeneration, ulceration, calcification ; usually several or all of these changes are present together, combined with complete or incomplete absorption of the media.

Besides the division of aneurisms just given, relative to their form and relation

to the artery (cylindrical, fusiform, saccular—peripheral or total, semi-peripheral or partial), an etiological division has also been made into spontaneous and traumatic, and finally an anatomical division has also been made in reference to the condition of the walls of the aneurism. With due respect to the actual facts, it seems best to retain only a fraction of the ingeniously contrived and very various nomenclature[1] referring to this matter, and to speak only of *true* and *false aneurisms* (an. vera and spuria). True aneurisms are such as are limited by one or more of the arterial coats, whilst false aneurisms are bounded by the neighboring tissues alone; consequently in them we have to do with a hæmatoma, an effusion of blood, which is either diffused into the neighboring tissues or is surrounded by a connective-tissue membrane of recent formation. In most cases these so-called aneurisms originate in wounds of the arteries.

Aneurisms are called mixed when the sac consists of one or two only of the three arterial coats; in an aneurysma mixtum externum, the adventitia alone is preserved; in an an. m. internum (the actual occurrence of which is denied by many authors) the intima remains, and the media and adventitia are ruptured.

Should the blood, after rupture of the intima and media, force its way between the layers of the latter or between the media and adventitia, and a blood tumor be thus formed between the arterial coats, this is termed a dissecting aneurism (*vide postea*).

Moreover, these divisions have a greater anatomical than clinical interest, and even at the dissection of the body they are often not easily made out, when the individual arterial coats have been pathologically changed, and are therefore not easily separated from one another.

It is quite usual for all the three coats to be distinctly recognizable at one part of a large aneurism, while at another only one or perhaps two may be distinguishable. The external coat being the most distensile, usually survives longest; while, according to the mode of origin, sometimes the intima, and at others the media, is the first to give way. When an aneurism attains a certain size and extent, all the three coats disappear, so that connective tissue, muscle, and bone, etc., may form the sac, and thus we have a so-called false aneurism (an. spurium consecutivum, in opposition to the an. sp. traumaticum). By long continuance even these organs suffer from the pressure of the aneurismal tumor, partly in the form of simple atrophy (the older writers supposed that the osseous tissue was dissolved by a corrosive fluid secreted by the aneurism), partly by the development of

[1] Vide *Duchek*, p. 235.

connective tissue, which forms a membranous limitation to the aneurism.

An aneurism is termed arterio-venous when it opens into a vein, and we have an aneurismal varix when the communication is immediate, and a varicose aneurism when there is a saccular tumor interposed between the arterial and venous openings.

The cavity of an aneurism rarely contains only fluid blood; usually there is also more or less of a fibrinous coagulum, which is deposited in layers over the lining membrane; of these the most interior is soft and reddish, more externally they become dry, hard, gray, or of a yellowish color, and finally they may develop into fibrous tissue, and combine intimately with the lining membrane of the artery. In this way the aneurismal sac may be filled up and obliterated.

In other cases there are only partial organizations of the blood clot, and between these we have cavities through which the blood stream passes. Or part of the coagulum may calcify or break up into a puriform detritus, which may be encapsuled by the harder masses, and only rarely gets into the circulation. Should the sac be rapidly filled by the copious formation of coagula, these may project into the arterial lumen and may narrow or occlude it; a similar result may be produced by the mere pressure of a sacculated aneurism filled with clots on the arterial trunk from which it springs. Finally, pieces of the clot breaking loose may lead to embolism.

In general, laminated clots are of more gradual formation, firmer, and therefore more capable of resistance and of organization than large lumps of coagula (Broca calls the former active, and the latter passive, coagula).

From the greater rapidity of the circulation in the spindle-shaped and cylindrical aneurisms, these in general contain fewer clots and of less size than the sacculated.

The arterial branches springing from the sac, or from its neighborhood, frequently take part in the dilatation; at other times their place of exit is distorted and narrowed, wholly or partially occluded by fibrinous clot, or an emerging branch may be compressed by the aneurismal sac.

The statements of the various authors upon this subject differ

extremely in regard to the state of the heart. Thus, while Wunderlich looks upon cardiac hypertrophy as an almost certain accompaniment of an aneurism of the aorta, and Bamberger finds in such cases hypertrophy and dilatation of the left ventricle at least more often than not, Stokes declares that aneurism has scarcely any influence in producing enlargement of the heart.

In eighteen cases of aneurism of the ascending and transverse portion of the arch, Axel Key found in a few of them slight dilatation of the left ventricle, but never hypertrophy. It seems therefore that an aneurism (even an aortic aneurism) never of itself produces hypertrophy; and we can readily understand this, because the existence of an aneurismal sac does not present any important obstacle to the circulation.

Assuredly in many cases of coexistent cardiac hypertrophy, other affections, extensive arterio-sclerosis, valvular lesions, etc., will be found an all-sufficient cause.

Moreover, even in cases of large aneurisms, atrophy of the heart has been actually observed.

In many cases the aortic valves are simultaneously diseased, or the heart is depressed or dislocated by a voluminous sac. Pericardial effusion, or degeneration of the cardiac muscle may also occur as a complication.

An existing aneurism has within itself, in the persistent pressure of the blood on its internal surface, and the attenuated and often otherwise diseased condition of its coats, all the conditions necessary for progressive enlargement. The cure of an aneurism is of rare occurrence, and is brought about by the filling of the sac with coagula, their organization and the subsequent obliteration of the sac by contraction or inflammation; or the artery on which the aneurism is situated may be obstructed, and the tumor may thus be cut off from the circulation.

Czerny found in a four months' old traumatic aneurism of the femoral artery, which was extirpated by him, that the sac was formed of a layer of connective tissue, 2–7 mm. thick, which was dense, laminated, and rich in cells; externally it was firmly adherent to the arterial adventitia; its internal layer resembled granulation tissue, and this passed gradually into a fibrinous coagulum.

In spindle-shaped and flat sacculated aneurisms, the coagula adhering to their walls frequently so strengthen them that the

tumor does not increase in size, and the blood-stream passes through the persisting canal almost in the normal manner.

Should death not result from some intercurrent disease, the progressive growth of the aneurism at last induces rupture, and effusion of blood into the surrounding connective tissue, or into any cavity with which it may be connected by position and possibly by adhesions, such as one of the chambers of the heart, a vein, one of the serous cavities, into the air-passages, or the digestive tract, or finally externally by rupture of the skin.

I endeavored to produce aneurisms experimentally in dogs, by wounding the internal surface of an artery. For this purpose I made use of a small chisel with a rounded cutting edge of 1½ mm. in diameter; this was introduced into the carotid for a few centimetres (either towards the periphery or towards the heart). This chisel passed through a silver tube, above which the vessel could be tied. After its introduction, the cutting edge was protruded as far as was permitted by a screw in the handle; the internal surface of the artery was scratched, the instrument removed, and the vessel tied. The aneurisms thus manufactured were but small, and were speedily filled with blood coagula. For example, one sixteen days old was the size of a pea; its walls formed by the adventitia, the entrance closed by a plug of coagulum 2 mm. in diameter. The media and intima were cut through; the laminæ of the former, close to the opening, were separated from each other by blood. Whenever, in my experiments, only the inner layer of the media was injured, no aneurism was produced, but merely a flat clot adherent to the arterial wall; in these cases also blood had passed between the layers of the media in the neighborhood of the cut, but more in the longitudinal than in the transverse direction.

According to Crisp,[1] Amussat, Jones, and others had previously torn the internal and fibrous coats of the arteries, without producing any aneurisms.

Etiology.

In the larger number of cases, the cause of the formation of an aneurism is to be found in alterations in the arterial coats, particularly of the media, as that upon which principally depends the strength and firmness of the arterial tube.

Aneurisms are therefore of frequent occurrence in chronic endarteritis, which so commonly leads to atrophy and fatty degeneration of the media. Loss of substance, thinning or ulceration of the intima, favor the occurrence of aneurism, because

[1] l. c. p. 124.

the blood then acts directly upon the media, forcibly separating its elements. Acute ex- and endarteritis may be followed, though more rarely, by the same results as chronic endarteritis.

Thus aneurisms have been seen to result from circumscribed inflammation of the external and middle coats (periarteritis nodosa, *vide antea*).

In many cases simple (senile) atrophy of the arterial coats, particularly of the media, seems to lead to the formation of aneurisms.

Even paralytic relaxation of the arterial coats (from paralysis of the vaso-motor nerves) has been credited with the like result (Corvisart, Rokitansky).

In many cases aneurism seems to be produced by a primary disease of the media.

Thus Rokitansky[1] relates two cases, where, from suppuration of the media, an ulceration (the size of a sixpence) of this coat and of the intima was produced, and a sacculated aneurism resulted. H. Helmstädter describes several cases of aneurism with loss of substance in the media, so that with a normal intima the interior of the aorta exhibited irregular depressions (several centimetres in diameter); at the edges of these depressions the elastic fibres broke off suddenly with irregular ends. In another case these depressions were smaller, did not occupy the whole thickness of the media, and were partly filled with connective tissue of recent growth. No degeneration could be seen in the elastic fibres at the edges of these crevices. Helmstädter looks upon a molecular alteration of the elastic fibres of the media as the commencement of this rupture and aneurism formation.

Köster, in a recent communication,[2] endeavors to show that the most frequent cause of aneurism is a primary mesarteritis, which occurs in the muscular coat in the form of numerous clear patches. This is said to occur in the course of the nutrient vessels coming from the adventitia, and gives rise to cell accumulation and hyperplasia of the connective tissue; consecutively the muscular and elastic fibres disappear. The adventitia and intima are finally conjoined to one highly vascular membrane.

As yet it is not possible to decide whether primary or secondary alterations of the structure of the media most frequently give rise to the formation of aneurism. Hitherto the former supposition has been most frequently accepted, and chronic endar-

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[1] L. c. S. 16.

[2] Berliner klin. Wochenschr. 1875. S. 322.

teritis has been regarded as the most important cause, probably because alterations of the intima have received more attention than those of the media, and because endarteritic alterations in the neighborhood of aneurisms have been usually regarded as primary, although they may be also of secondary origin. In opposition to the prevalent idea of the endarteritic origin of aneurisms, it has been truthfully urged that, while aneurism most frequently occurs between the thirtieth and fortieth years, endarteritis is a disease of advanced life; the latter, therefore, cannot be the most important cause of the former. But this objection cannot be regarded as altogether successful, when we reflect that, besides the predisposition, many accidental causes concur in producing an aneurism.

These accidental causes are sometimes a fall, a blow, a concussion, or a violent exertion, by which the affected artery is directly or indirectly injured, inflamed, forcibly stretched, or partially ruptured. In vessels already diseased such violence acts of course more readily. Even the rupture of a few fibres or laminæ may suffice for the commencement of further dilatation.

Bamberger reports the case of a miller aged thirty-two, who, while unlading heavy sacks of grain, was suddenly seized with a violent stinging pain in the sternum, with palpitation, and loss of strength; ten weeks later a tumor had developed in his thorax. Albutt[1] also relates many cases where a bruise of the chest or a fall with a heavy burden produced an aneurism.

In a sailor, aged twenty-eight, engaged in hoisting a mast, a sudden sensation of something giving way in the abdomen was felt, followed by pain and faintness, and in two days by a feeling of pulsation in the epigastrium; an aneurism of the abdominal aorta was subsequently developed. (Greenhow.) Uterhart[2] relates a similar case.

Frequently repeated stretching and bending of the arterial wall is equally favorable to the production of aneurism, as is proved by its common occurrence in the neighborhood of joints.

The distending force in the formation of aneurism is—except in those rare cases where it originates in the contraction of an external cicatrix—the blood pressure on the internal surface

[1] St. George's Hosp. Rep. London. 1872.

[2] Berlin klin. Wochenschr. 1867. S. 501.

of the vessel, which leads to the distention and stretching of its diseased parts. Increase of this blood pressure, therefore, such as results from cardiac hypertrophy or frequently repeated bodily exertion, is specially favorable to the occurrence of aneurism. Thus Lebert[1] observed an aortic aneurism developed by hypertrophy of the left ventricle resulting from aortic insufficience. In a similar state of matters, Friedreich[2] observed the development of many peripheral arterial aneurisms, and Legroux,[3] aneurisms of both axillaries, and dilatation of the arch of the aorta. A local increase of the blood pressure, such as occurs above an arterial stenosis, is referred to by Duchek as an occasional cause of aneurism.

Finally, Ponfick has pointed out that injury to the inner surface of an artery may frequently lead to the formation of an aneurism—when sharp-pointed emboli are wedged in by the blood-stream.

Ponfick describes seven cases, in which, in medium-sized arteries (a. fossæ Sylvii, a. lienalis, a. mesent. sup.), twelve sacculated aneurisms were situated close above embolisms; in each case there was a hard, sharp-pointed embolus (coming from a calcareous cardiac valve), which had stuck close behind the bifurcation of a vessel. Ponfick supposes that in these cases there was a perforation of the arterial wall by the sharp-pointed embolus (favored by the increased blood pressure), consequently that there was a traumatic aneurism from an internal cause (an. spurium). He is inclined to assume a similar cause for many aneurisms occurring in the smaller arteries; particularly when these, as at the base of the brain and in the mesentery, are situate in loose and yielding tissue. This idea seems all the more probable that it is precisely at the base of the brain that it is often impossible to connect aneurism with chronic endarteritis.

Church[4] had already deduced, from their frequent coincidence, the probable causal connection between embolism and aneurism; but he looks upon embolic disturbance of the nutrition of the arterial walls, and not perforation of them by the embolus, as the proximate cause of the aneurism.

R. W. Smith[5] also, and Ogle[6] have likewise pointed out the connection between embolism and aneurism (in the coronary arteries). On the strength of one observation of his own and two by Corvisart,[7] Lebert describes a "cystogenous" aneurism

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[1] L. c. S. 402.

[2] Herzkrankheiten. S. 253.

[3] Arch. gén. Nov. 1874. p. 585.

[4] Barthol. Hosp. Rep. VI. p. 99. 1870.

[5] Dublin Quart. Jour. Nov. 1870.

[6] St. George's Hosp. Rep. 1867, etc.

[7] L. c. p. 312. Obs. 45.

as a rare variety. In this variety a cyst (dermoid or atheromatous?) forms in the arterial wall, and afterwards opens into the vascular lumen.

From what has just been said, it follows that in many cases there is disease of the arterial coats, consequently a "spontaneous" origination, existing coetaneously with the occurrence of an injury; that, therefore, it is impossible to adhere strictly to the etiological division of aneurisms into spontaneous and traumatic;—in general, various etiological causes coincide in the origination of an aneurism, so that it is not always possible to determine the special importance of any one cause in any given case.

Position and Ratio of Occurrence as to Occupation, Sex, etc.

Aneurisms are almost confined to the aortic arterial system, and only rarely occur in the pulmonary artery.

In 551 cases of aneurism Crisp found

The thoracic aorta affected in	175	cases	41.7 %.	The common iliac in	2	cases	0.3 %.
" abdominal aorta and its branches in	59	"		" external iliac	9	"	1.9 %.
				" gluteal artery	2	"	0.3 %.
" pulmonary artery	2	"	0.3 %.	" temporal artery	1	"	0.1 %.
" femoral artery	66	"	11.9 %.	" ophthalmic "	1	"	0.1 %.
" popliteal artery	137	"	24.9 %.	" subclavian "	23	"	4.1 %.
" posterior tibial artery	2	"	0.3 %.	" axillary "	18	"	3.2 %.
" innominata "	20	"	3.6 %.	" subscapular "	1	"	0.1 %.
" carotid "	25	"	4.5 %.	" brachial "	1	"	0.1 %.
" vertebral "	7	"	1.8 %.				

In 109 cases Myers found

Aneurism of the ascending aorta	.....	37 times.
" arch of the "	.....	38 "
" descending "	.....	19 "
" abdominal "	.....	15 "

Aneurisms are of most frequent occurrence in middle and advanced life; before the twentieth year aneurisms of the larger arteries are very rare.

Crisp analyzed 551 cases of every kind of aneurism; Lebert, 324; and Lidell 243 cases of internal aneurisms. They found:

	Crisp.		*Lebert.*	*Lidell* (New York).
Under 10 years	1 case	15 %.	16 under 20 years.	2 cases.
Between 10 and 20 years	5 cases			8 "
" 20 " 30 "	71 "		42	31 "
" 30 " 40 "	198 "	39 %.	80	81 "
" 40 " 50 "	125 "	25 %.	75	69 "
" 50 " 60 "	65 "	13 %.	70	24 "
" 60 " 70 "	25 "	5 %.	35	20 "
" 70 " 80 "	8 "	2 %.	7 after the 70th year.	6 "
" 80 " 90 "	2 "			2 "
" 90 " 100 "	1 "			
Age not given	46			
In all	551 cases.		324	243

The greatest number of aneurisms is, therefore, by all these observers found to occur in the fourth decennium of life, and the next greatest number in the fifth decennium. The differences between these three series of statistics probably depend upon differences in the materials from which they were collected (situation of the aneurism, nationality, etc.).

Aneurisms are more frequent in men than in women; in particular, aneurisms of external arteries are rarer in females.

Coupled with other observations, this also indicates the influence of occupation. Aneurisms are more common among the poorer classes, and especially among those whose occupations are laborious, such as smiths, porters, etc.

The influence of occupation is well shown in the statement of Lawson,[1] that in 1866 the deaths from aortic aneurism in the English army were eleven times more frequent than among the civil population. A statistical comparison of the occurrence of all forms of aneurism gave:

0.36 per thousand among 176,320 men of the Footguards and Line in the United Kingdom during 1862–65.

0.10 per thousand among 217,170 men in the Navy during the same time.

0.09 per thousand among 507,405 men (?) of the civil population of London in the year 1861.

0.9 per thousand among 29,930 men of the white troops at Cape Colony during 1860-66.

0.7 per thousand among 42,482 men of the white troops in New Zealand during 1860-66.

[1] Army Medical Reports. 1866.

Strenuous corporeal exertion, inappropriate clothing and accoutrements, which compress the arteries of the neck and arms, are said to be the causes which lead to the more frequent occurrence of aneurism in the army compared with the navy and civil population.

Heredity (Lancisi), the abuse of alcohol and excesses generally (Lancisi), as well as the influence of exposure to cold, and of acute rheumatism (Lebert), have been also insisted upon as occasional causes of aneurism.

Aneurism is a comparatively rare disease, and its frequency varies in different countries. In Germany it is less common than in France, and in Italy it is very rare.[1] It seems to occur with extreme frequency in England (Britain generally). Whether a fondness for gymnastic exercises, a more copious use of alcoholic fluids, and a greater frequency of endarteritis, have anything to do with this, may indeed be suspected, but it has not been proved.

Since it sometimes happens that certain individuals have more than one aneurism simultaneously, it has been assumed that a (certainly very obscure) constitutional disposition to aneurism may exist, an "aneurismal diathesis." In many cases this disposition seems simply to depend upon a wide-spread chronic endarteritis (as in Cruveilhier, Livr. 28); but it is indeed inexplicable why this affection should have precisely in this (or any given) case have led to the formation of aneurism (whether single or multiple).

Symptoms.

On the external arteries aneurisms form perceptible and sometimes visible tumors of various shapes, which increase in size with each cardiac systole, expanding in every direction at each pulsation, when they contain fluid blood, and frequently communicating to the hand a sensation of whirring, which may also be heard.

The symptoms of aneurism of the internal arteries are often very obscure, sometimes entirely absent, and they only become distinct when the aneurism has attained a certain size, has approached the external wall of the cavity in which it lies, and contains (at least in part) fluid blood, or when it disturbs the functions of neighboring organs.

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[1] Vide *Crisp*, p. 129, etc.

Aneurism of the Aorta, particularly of the Thoracic Aorta.

When an aneurism of the thoracic aorta is in contact with the thoracic walls, a pulsation may be felt at this spot; if the thoracic wall is flexible, and the extent of contact somewhat considerable, then the pulsations become visible; it seems, as Stokes has well observed, "as if two hearts were beating within the chest." By and by this point of contact projects, at first flat and diffusely, but gradually the tumor becomes more and more prominent, varying from the size of a nut to that of a child's head, and is hemispherical, oblong, or irrregular.

The more distended an aneurism becomes, the more its coats become atrophied and ruptured, and the neighboring organs form the limit of the blood sac, so much the thinner its external coverings are felt to be; the ribs are separated from each other and absorbed, the muscles are atrophied, the skin becomes tense, permeated by enlarged veins, and finally inflamed and necrotic. The resistance of the tumor is considerable, corresponding to the blood tension in the aorta; and it can only be diminished in size by considerable and often very painful pressure. It is elastic, and fluctuation may be perceived in it (during the diastole.).

The pulsation is all the more distinct the more prominent the tumor is, and the thinner its walls are; it follows the apex beat at an appreciable interval, which is greater the further the aneurism is removed from the heart. The expansion of the tumor in every direction during its pulsation is of diagnostic importance; it is to be felt not only at its upper part, but also at its sides, differing in this respect from abscesses or other tumors resting on an artery or on the heart, which simply rise and fall.

In occasional cases, indeed, an abscess, an emphysema just about to burst, may exhibit expansile and yet only communicated pulsations.[1] In such a case the diagnosis must rest upon the history of the development of the disease, the trifling

[1] *E. Müller*, Berlin. klin. Wochenschr. 1875. *L. Traube.* Ibid. *Ph. Plogge*, Memorabilien. 1872. Vide also *Reiher*, Berlin. klin. Wochenschr. 1868. No. 41. *Richardson*, Jahresber. von. Virchow- Hirsch. 1868. II. S. 74.

tension of the tumor, and the great extent of the thoracic dulness.[1] Empyema most frequently perforates the chest at the lower part of the left side, aneurisms more frequently on the right side above the fourth rib.

A systolic dilatation of the sac is often visible in large and thin-walled aneurisms.

Not unfrequently a second feeble impulse is felt to succeed the primary pulsation; there is as it were a perception of a double shock analogous to the dicrotic wave of the pulse; and in truth it is nothing else than the sensation produced by the reflux wave caused by the recoil of the blood column from the aortic valves closed at the moment of the commencement of the cardiac diastole. This double shock is most distinct in the neighborhood of the valves, consequently in an aneurism of the ascending aorta; it may, however, be also felt in aneurisms of the descending aorta.

According to some, the double shock felt over an aneurism may be caused by the reflux of the pulse wave within its own cavity, a view which may be correct enough in some cases, certainly not in all.

A sphygmographic investigation of superficial aneurisms (from the large surface they present) usually furnishes us with well-marked curves, whose form, in general, corresponds with that of the artery affected—some of the details being effaced when the soft parts covering the aneurism are thick, when clots fill its cavity, or when the communication with the artery is very narrow. Moreover, vibrations originating in the wall of the sac may modify the form of the curve.

Sometimes the hand laid upon an aneurismal sac feels a sensation of whirring, and the vibrations thus produced may also be heard. (In regard to the mode in which this whirring is produced *vide postea.*)

The *percussion sound* over the aneurismal sac is quite dull, so far as the sac is in contact with the chest-wall; around this, so far as the sac is overlaid by the lung, it is merely higher in pitch and of shorter duration than normal. It is often impossible to separate the aneurismal dulness from that of the neighboring organs, such as the liver, heart, spinal column, etc.

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[1] For other diagnostic phenomena, vide *Balfour*, Dis. of Heart. p. 341; and *McDowall*, Dublin Quarterly Journal for March, 1864.

On *auscultation* we usually hear over an aneurism of the thoracic aorta, just what we hear over that vessel itself—a systolic and a diastolic sound. The latter is always present when a double shock is felt, but often also when the pulsation is only systolic; it is sometimes absent in aneurisms of the lower part of the thoracic aorta. The systolic sound is produced by the forcible expansion of the walls of the sac as well as of the aorta near it; the diastolic is propagated from the aortic valves. Any abnormalities in the aortic sounds are usually perceptible over the aneurism, particularly any accentuation in their character, or any murmurs which may replace them in an insufficiency of the valves or stenosis of the orifice.

But even when the aortic valves are perfectly sound, murmurs (chiefly systolic) may be heard over an aneurism. They replace the normal sound, and are produced by the systolic expansion of the heterogeneous walls of the sac (?), or, as happens in most vascular murmurs, by a sudden alteration in the diameter of the blood current, which, from the varying form of aneurisms, consists at times in a simple dilatation, and at others in a narrowing of the arterial lumen—when the aneurism is placed on one side of the vessel—, or by compression of the aneurism by some adjacent organ. At other times coagula in the aneurismal sac give rise to irregularities in the channel of the blood current, or the sac itself compresses the aorta or some other artery (the subclavian, pulmonary, etc.), and so produces a murmur in it.

That a diastolic murmur may be produced in an aneurism by the elasticity of the sac producing reflux of the blood into the artery through a narrow opening, seems a possible occurrence, but in any case it is very rare; no sufficient explanation has been given for the origination of a diastolic murmur within the sac itself; it seems always to be propagated from the aortic valves; it is certainly sometimes heard louder over the sac than over the valves themselves, but this seems to be due to resonance within the cavity of the sac itself, and better conduction to the ear of the observer.[1]

[1] An interesting case of diastolic murmur in a case of aortic aneurism will be found in *Balfour*, On Diseases of the Heart, etc., p. 400, with subsequent dissection.

Moreover, murmurs must not be regarded as common signs of an aneurism of the thoracic aorta; in most cases they are absent.

Scheele[1] relates three cases of aneurism with diastolic murmurs: the diastolic emptying of the sac into the artery was favored by insufficience of the aortic valves, and the murmur was thus intensified. When the aneurism is forcibly and suddenly expanded and then gradually contracts, the reflux eddies produced during the equalization of the blood pressure in the sac are said to give rise to a diastolic murmur.

According to some authors, all the sounds and murmurs heard over an aneurism originate within itself from the friction of the blood on its walls (Bellingham), or at the opening of communication with the artery (Gendrin, Lyons). Williams supposes that the diastolic sound is caused by the reflux of the blood from a large and elastic sac, through a wide opening into the vessel, etc.

It is certain, that of all the various explanations given by different authors, no one—from the manifold character of the phenomena present—can be right in every case. Whether we have a sound or a murmur, very often depends, as is well known, upon very trifling anatomical differences. In no case can we suppose the active cause to be a contraction of the aneurismal sac itself, since the muscular elements of its walls have generally disappeared, or if they still exist they are no longer capable of a sudden sound-producing contraction.

In respect to the origination of murmurs at dilated parts of blood channels, we must remember that Nolet[2] (as well as Weber and others) have been able to produce them experimentally, but the murmur became weaker or disappeared when the dilatation exceeded a certain relative size (eighteen times the normal diameter).

Eddies in the fluid seem first of all to be produced by the inequalities of the channel, and these then throw the vascular walls into irregular vibrations. It has already been stated that these are not only to be heard, but can even be felt as a sensation of whirring by the hand laid over the sac.

All the phenomena just related as belonging to aneurisms, are either entirely or partially absent wherever the sac is situated deep within the thorax, or is covered by other organs, particularly by the lungs.

Even then the pulsation may still be felt as a diffuse succussion over that part of the surface lying nearest the sac, most readily by palpating with some force during expiration; or the aneurismal pulsations may be conducted to the surface by super-

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[1] De Strepitu diastolico, quem in aneurysmatibus animadvertimus. Dissert. Regiomonti. 1867.

[2] Archiv d. Heilkunde. Bd. XII. 1870.

jacent dense organs (such as infiltrated or airless portions of lung, mediastinal lymphatic glands, etc.).

The results of percussion are of great importance in the detection of such deep-seated aneurisms; such as an abnormally high pitch, a tympanitic character of the sound, or any alterations of the normal organic limits.

A deep-seated aneurism may be recognized by auscultation, when both of the aortic sounds (or, at any distance from the heart, only the systolic sound) can be heard in any abnormal position of the thorax, every other cause of such a phenomenon being capable of exclusion. The recognition of murmurs, which, like the sounds, are frequently propagated from a distance, is of still more importance.

From what has just been said, it is evident how uncertain may be the direct symptoms of an aneurism of the thoracic aorta. In many cases we are indeed compelled to rely for our diagnosis upon the indirect symptoms, upon the sequential phenomena, presently to be referred to.

In the heart there is, from the extensive arterio-sclerosis which so frequently accompanies aneurism, sometimes dilatation and hypertrophy of the left ventricle; at other times, on the contrary, there is atrophy or degeneration of the cardiac muscle, and consequently great variations in the extent of dulness and the force of the impulse. Sometimes we have the signs of stenosis or insufficience of the aortic valves; more rarely we find the mitral coetaneously affected. If the aneurism has attained any considerable size, then the position of the heart, and hence the dulness and the apex beat, may both be altered. In aneurisms of the ascending aorta, the heart may be pushed to the left or downwards; in those of the descending aorta the heart may be pushed to the right or forced against the anterior chest-wall; in the latter case the area of absolute dulness is enlarged, the cardiac impulse is broader and more forcible, and at times even a double shock may be felt from propagation of the aneurismal impulse. Cardiac hypertrophy may be simulated by a displacement of this character.

The peripheral arteries are sometimes normal, at others they feel sclerotic, and from this cause, as well as from the state of the

heart, the pulse may vary very much in its quality; it may be normal, hard, small, jerking, slow, or irregular, etc.

Sometimes the pulse exhibits a marked difference above and below the aneurism in the affected artery itself, as well as in the branches coming off above or beneath the sac. If the size of the aneurism is considerable relative to that of the vessel from which it springs, its cavity acts as a reservoir for part of the blood coming from the heart during its systole; and in the succeeding diastole the elasticity of the sac drives this portion on towards the periphery; so that the aneurismal sac acts as an air-chamber in making the blood-flow in the arteries situated peripherically beyond it more uniform and less subject to pulsatory variations of pressure. The sphygmographic pulse curve, taken beneath such an aneurism, is therefore of a more uniform medium elevation, the systolic elevation and the diastolic depression being less marked than normally. The details in the form of the individual waves, the secondary elevations, are obscured or obliterated; the line of ascension is not perpendicular but gradual; the apex of the curve is therefore rounded and postponed relatively to the preceding diastole.

It is easily understood that this alteration in the pulse is all the better marked the greater the size of the aneurism in relation to the artery, and that on the other hand repletion of the sac with coagula and rigidity of its walls (calcification, tendency to ossification) act so as to preserve the normal appearance of the pulse wave. The alterations of the pulse referred to are therefore more readily produced by aneurisms of the peripheral arteries of comparatively small dimensions than in aneurisms of the aorta and its larger branches.

We can much more easily determine whether the pulse is altered or not, when we examine simultaneously the affected artery itself or a branch from it, both above and below the aneurism. When, for instance, in an aneurism of the descending aorta, we compare the pulse in the upper half of the body with that in the lower. And our object will be most certainly attained by comparing symmetrically situated homonymous arteries. In aneurisms of the aortic arch and its branches, trifling variations in the pulse curves of both radials, carotids, or temporal arteries, are

readily recognized, and are of importance in determining the seat of the aneurism anterior or posterior to the place of exit of one of the carotids or subclavian arteries.

Moreover, we must not forget that narrowing of an artery may alter the form of the pulse curve after much the same fashion as a simple aneurismal dilatation, and that an aneurism itself may produce the narrowing (or occlusion) of any emerging branch by distortion of its place of exit, twisting, compression, or the formation of coagula (that finally asymmetry in the width of an artery may be of physiological origin, and may depend upon the tonicity of the arterial muscle).

If the alteration in the pulse, particularly that of homonymous arteries (pulsus differens of authors) be considerable, it may be felt by the finger.

Besides the differences in the size of the pulse above and below an aneurism, a difference in time has been also observed, so that the pulse in arteries coming off beneath the sac is (more than normally) delayed when compared with the pulse in the arteries coming off above it. This observation is also more easily made in similar arteries which usually pulsate synchronously than when we, for instance, compare the carotid pulse with that in the femoral, which are normally asynchronous. According to Marey the delay is only apparent, since the line of ascension of the pulse curve beneath the aneurism commences at the proper time (*vide antea*), its summit alone being delayed (the degree of arterial distention requisite for the distinct perception of the wave being that only which is delayed). Fischer-Dietschy has adopted this view, and by measuring the interval between the cardiac impulse and the pulse at the periphery in healthy persons and in one case of aneurism of the ascending aorta, he found it to be the same in both. Nevertheless, an actual delay of the pulse, especially in spindle-shaped aneurisms of some length, is not improbable, inasmuch as the velocity of the propagation of the blood wave materially depends upon the elasticity and tension of the arterial wall.

Marey has been able artificially to imitate the alterations in the pulse waves just described, by attaching a caoutchouc ball as an aneurism to his caoutchouc imitation of the circulatory apparatus (schema), while on the other hand a rigid

glass ball had no effect in modifying the pulse curve—a proof of the truth of the theory formed by him as to the action of an aneurism as an elastic reservoir.

Marey has also pointed out that by manual compression of a superficial aneurism, the blood tension in those arteries lying beyond it may be temporarily increased, and this falls at once on the remission of the pressure (this of course is most easily detected by the sphygmograph); whilst in the case of tumors only passively pulsating, pressure narrows the artery and diminishes both the pulse and the arterial tension in the parts beyond.

As in arterio-sclerosis, so also in aneurism, the comparative sphygmographic investigation of different arteries may undoubtedly contribute much to the precision of our diagnosis of the situation and extent of these tumors, yet in estimating the results we must never forget the many other possible anatomical sources of similar alterations; and particularly we must never lay too much stress upon delay of the pulse as a diagnostic sign of aneurism, nor indeed in most cases need we expect to find it. Marey observed very distinct dicrotism of the pulse in one case of aneurism of the ascending aorta (*vide postea*). Wade also observed a most exquisite double beat of the pulse (alongside of the normal dicrotism as sphygmographically brought out) in a colossal aneurism of the abdominal aorta, and has referred the secondary wave, which coincided with the collapse of the tumor, to its elastic contraction.

The great veins of the thorax are very often narrowed or completely closed by the pressure of an aneurism; this happens less seldom to the v. cava inferior than to the v. cava superior and its branches, particularly the innominata, the subclavian, and the jugular veins. Dilatation of the veins and—in its severer forms—cyanosis, alterations of temperature and œdema in the area of origin of the vein compressed, as in one arm or in one half of the body, are the resultant phenomena, and may be of diagnostic importance.

Aneurisms of any considerable size influence injuriously the circulation through the great veins without in any way compressing them, merely by diminishing the negative pressure within the thorax, and thereby diminishing the flow of blood towards the heart.

Many other disturbances of the venous circulation are also observed in cases of aneurism; but they are not due to it, but to coincident disease of the heart and blood-vessels.

A capillary pulse in the face has been seen by Lebert and by the author. It seems specially to occur in aneurisms of some considerable size; a sudden alteration of the blood pressure, and with this the capillary pulse, seems to arise from diastolic

regurgitation into the aneurism, just as similar phenomena occur in regurgitation into the left ventricle from insufficience of the aortic valves.

A pulsation in the retina (without any disease of the eye), occurring under similar conditions as the capillary pulse, has been observed by Becker in cases of aneurism.[1] In one case in which an aneurism of the aortic arch between the innominata and the left carotid was suspected, the arterial pulse in the retina was much more distinct in the left eye than in the right one.

The *subjective phenomena* referable to the vascular system, as well as those specially due to the aneurism, may be wholly wanting, even when the objective symptoms are present; more frequently they occur earlier than the latter. The patient then feels pain in the region of the aneurism, sometimes indistinct, dull and heavy, at others more severe, tense, stinging, boring, pulsating; and this pain occasionally occurs in paroxysms, especially on any increase in the arterial blood pressure by movement, mental emotion, etc., by pressure from without, and at times, when the aneurism increases rapidly, stretches the neighboring organs and gives rise to inflammation in the parts around it. Besides the feeling of pulsation in the region of the aneurism, the patient frequently has palpitation of the heart and a feeling of tightness in the chest, which may eventuate in all the well-marked phenomena of angina pectoris. Certain positions of the body—such as lying on the left side—originate or increase these sufferings, while in other positions they disappear.

Besides the disturbances in the organs of circulation, those most commonly observed in aneurism are referable to the organs of *respiration*, and of these dyspnœa is most frequent; this is partly due to the diminution of the thoracic capacity by the tumor; occasionally a part of the lung may be completely compressed (evinced by dulness on percussion and bronchial breathing), or a large or medium-sized bronchus, or the trachea itself may be narrowed by the pressure of the aneurism.

On account of its contiguity to the arch of the aorta, the left bronchus (and its branches) is that most frequently affected. On the corresponding half of the thorax, or over that part of the lung to which the affected bronchus is distributed, the respiratory murmur is less distinct, sometimes accompanied by a whistling

[1] Graefe's Archiv f. Ophthalmolog. Bd. 18. 1872. Cases 16 and 17.

rhonchus due to stenosis of the bronchus; when the compression is considerable, the respiratory movement on the side affected may be visibly diminished, and from the diminution in the contained air the percussion note of the lung is shorter and duller (of higher pitch).

In a lung so compressed, a catarrh is very readily developed, —and this all the more readily where there is coincident congestive hyperæmia due to compression of the pulmonary veins, which may itself give rise to hæmoptysis.

In rare cases gangrene of the lung may occur (probably from occlusion of a bronchial artery).

Pleurisy with adhesions or with serous effusion, may also result from aneurism.

In many cases we have a paroxysmal dyspnœa quite disproportionate to the comparatively trifling objective alterations in the lungs, and this seems to be truly neurotic in character, and dependent upon a lesion of the pulmonary branches of the vagus nerve by the aneurism. Should the recurrent laryngeal nerve be compressed, we have unilateral paralysis of the laryngeal muscles, hoarseness (vox anserina), and dyspnœa upon exertion. In such cases a laryngoscopic examination is of importance for the diagnosis of the aneurism; the left recurrent, which winds round the arch of the aorta, is that which is most frequently compressed by the aneurism.

At other times, on the contrary, we have spasm of the glottis, induced in a reflex manner by irritation of the pulmonary branches of the vagus, or œdema of the glottis, from pressure on the veins of the neck. In rare cases compression of the pulmonary artery by the aneurism may also be a cause of dyspnœa.

In the digestive tract, disturbances of *deglutition* are of frequent occurrence; sometimes these depend upon simple compression of the œsophagus, at others upon spasm or other disturbance of its innervation from irritation of the vagus nerve; and from the latter cause we also frequently have eructation, actual vomiting, and other disturbances of the digestion. Sometimes the dysphagia varies with the position of the body, and is greater in the supine posture.

According to the position of the aneurism, disturbances of

function may arise in other nervous areas, as in pressure upon the intercostal nerves or on the brachial plexus, especially on the left side, when we may have neuralgiæ, formication, numbness, and paretic phenomena in the arm, breast, and neck.

When the vertebræ are eroded, movement and pressure on the spine are painful; in severe cases there may be symptoms of a spinal meningitis, or paraplegia may occur from compression of the spinal cord.

The inconstant alterations of the pupils observed in some cases (sometimes on one side, at others on both, sometimes appearing as mydriasis, at others as myosis) may arise from direct or reflex irritation of the cervical sympathetic (of the nerves issuing from the cord in the cilio-spinal region).[1]

In cases of large aneurisms, which have lasted for some time, the general well-being and nutrition of the body suffer; there is diminution of the strength, wasting of the body, and anæmia or even œdema may set in. And though part of this cachexia may be due to the separation from the circulation of the blood accumulated in the aneurism, still the largest share is no doubt due to the usual accompaniments of aneurism, extensive endarteritis, disturbances of the heart's action and of the respiration.

The persistent increase of an aneurism leads finally to rupture and death, either from the hemorrhage alone, or, as may happen from the site of the rupture, from coincident disturbance of some vital function.

Generally the rupture is so large that copious hemorrhage and rapid death result (in a few minutes [seconds, *Tr.*]); but occasionally only small openings are formed, through which the blood flows but slowly, and which are occasionally closed by coagula, so that repeated small bleedings occur.

Gairdner[2] relates a case where repeated hemorrhages occurred into the air-passages during the course of nearly five years, and another where two years before death there had been a small rupture into the duodenum.

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[1] Vide *Ogle*, Medico-chir. Transact. Vol. XLI.; *Gairdner*, Edinburgh Med. Jour. Jan. a. Aug. 1855; and for the physiology of the subject, an interesting paper, by Dr. *Argyll Robertson*, Ed. Med. Jour., Feb. 1869.

[2] Med. Chir. Transact. Vol. 42, p. 189.

Sometimes the patient feels a violent, deep-seated, painful sensation of something having burst; pallor, a feeling of anxiety and giddiness occur, the pulse becomes small and frequent, syncope and death follow. When the rupture takes place into one of the pleural cavities, great dyspnœa is also present, and, if the patient can be examined, we have all the signs of the effusion of fluid.

When the rupture takes place into the pericardium, death is usually still more rapid from interference with the heart's action.

In one case in which the rupture was no bigger than a needle-hole, the patient lived for fifteen hours.[1] Bennet describes a case in which the rupture of one of the sinuses of Valsalva took place into the adherent pericardium, and formed an aneurismal sac, which lasted for some time.[2]

Rupture into the air-passages is followed by extreme dyspnœa, hæmoptysis, and aspiration of the blood into the smaller bronchiæ; death results partly from suffocation and partly from hemorrhage.

Rupture into the large veins (v. cava superior, or inferior, or the innominate veins) produces great distention of these and of their primary branches with blood, and even pulsation within them; dropsy and cyanosis may result.

Similar phenomena may follow rupture into the right side of the heart, or the pulmonary artery; only in such a case distention of the right heart, palpitation, dyspnœa, or hæmoptysis may occur. Sometimes a murmur is audible over the situation of the perforation, which may vary in its character and duration.[3] In many cases a loud, continuous systolic murmur is audible.[4]

When the rupture takes place into the œsophagus, blood gurgles up into the mouth or is vomited.[5]

Should the aneurism lie close to the thoracic wall, it may rupture externally; the coats of the tumor become gradually

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[1] *Beaugrand*, Rec. de mém. de méd. milit. 1867.

[2] Dublin Quarterly Journ. 1867. Nov. p. 444.

[3] Vide *Peacock's* Collection of 43 Cases, Transact. of the Path Soc. of London. 1868.

[4] *Thurnam*, l. c.; *B. Salomon*, Diss. Berlin. 1863.

[5] *Bourgeois*, Mouvement medical. 1867. p. 148.

thinner, the pulsations more distinct, the skin tense, very painful, inflamed, at length livid and necrotic; beneath the gangrenous scab blood trickles out; for a few days, perhaps, the opening may close, but at last profuse hemorrhage occurs either at this or at some neighboring spot. This catastrophe is often brought about by some accidental cause, some bodily exertion, a cough, or straining at stool; or a fall, a blow on the tumor, the exploration of the œsophagus, or violent vomiting may occasion it. Peripheral arteries may be occluded by coagula escaping from an aneurismal sac, and gangrene of an extremity or hemiplegia may be the result; palpation of the tumor has sometimes been the cause of this.

To this general description we now append an account of the dilatation of the several parts of the aorta, and in doing so only the characteristic symptoms shall be specially referred to.

Aneurisms of the Ascending Portion of the Aorta.

Simple dilatation of the aorta is of most frequent occurrence, namely, a uniform, cylindrical, or spindle-shaped dilatation implicating all the three coats. But sacculated aneurisms are also most frequent in this position, and are most commonly developed on the right (convex) side of the ascending aorta. The preferential site of such an aneurism is at the origin of the aorta, close above the valves, which are often coincidently diseased, rigid, thickened, or insufficient. Sometimes one of the sinuses of Valsalva alone is affected.

If the aneurism be still small, or the uniform dilatation of the trunk of the aorta moderate, we have a slight shortening of the percussion note to the right of the sternum from the second rib upwards; from concomitant elongation of the aorta, or from participation of its arch in the dilatation, the latter is felt pulsating in the jugular fossa. In proportion as the dilatation increases, the anterior border of the right lung gets forced aside, the percussion dulness becomes more distinct, and a (double—*vide antea*) pulsation becomes perceptible in the first and second intercostal spaces.

The aortic sounds are remarkably distinct, the first often

dull, the second clanging and metallic (accentuated); from coincident valvular disease these sounds are very frequently replaced by murmurs; but, independent of any valvular disease or of any stenosis of the aorta, it is precisely in this situation that we so frequently hear a loud systolic blowing in simple dilatation of the aorta; the absence of the characteristic, hard, small, and usually slow (tardus) radial pulse prevents such a case from being mistaken for one of aortic stenosis.

A diastolic murmur is almost invariably due to insufficience of the aortic valves, which are so frequently concomitantly altered. In rare cases, as I have clearly ascertained, a relative insufficience may be brought about by dilatation of the aorta alone, the valves remaining normal.

Marey observed a loud diastolic murmur, without valvular insufficiency, in one case of (dissecting) aneurism of the aorta, in which the communicating opening was situated in one of the sinuses of Valsalva.[1] The regurgitation of the blood into the aneurism produced a diastolic murmur and a rapid descent of the wave in the radial pulse curve, which, owing to this, and to the forcible reflux wave which succeeded, exhibited a peculiar form. Stokes[2] also mentions a similar case.

As the tumor grows larger, the thoracic walls are more and more vaulted outwards; from the pressure on the costal cartilages, the right edge of the sternum itself may be absorbed, and the pulsation becomes more distinct; finally, it is in this position that external rupture of the aneurism, as above described, is most frequently observed, after the tumor has reached the size of a man's head, often with a nodulated surface. Such aneurisms rarely ascend higher than the clavicle, or descend below the fourth costal cartilage.

In aneurisms of the ascending aorta the heart is sometimes displaced downwards and to the left; the left ventricle is frequently hypertrophied as the result of the valvular disease; the cardiac impulse is then more forcible and extends over a wider area than usual; yet between it and the aneurism there is always a part wherein pulsation is either entirely absent, or at all events faint.

Apart from murmurs of valvular origin, those murmurs

[1] L. c. p. 458.

[2] L. c. p. 550.

originating in the aneurism may be propagated over the cardiac area and heard faintly there. Cardiac palpitation, often increasing up to attacks of actual angina pectoris, are not unfrequent.

A deeply situated aneurism may give rise to pericarditis.

The cardiac muscle, and with it the cardiac energy, often suffer from concomitant disease of the coronary arteries.

The peripheral arterial pulse may be abnormally delayed relative to the cardiac impulse (but this is not constant); moreover, it always exhibits the peculiarities characteristic of any concomitant cardiac lesion (aortic insufficiency, hypertrophy, etc.). If the innominate artery be implicated in the aneurism, the pulse in the right carotid and subclavian may be small and delayed relative to that in the left carotid and subclavian arteries.

From compression of the vena cava superior, or of the right innominate vein, we have dilatation of the veins and œdema of the upper half of the body, or of the right arm, and (in a slighter degree from venous anastomoses) of the right side of the body.

Dyspnœa is common, and is partly evidently due to physical compression of the upper lobe of the right lung, partly to concomitant catarrh and disturbances of the cardiac energy.

Numbness, formication, and a feeling of weakness in the right arm, are produced by pressure on the right brachial plexus.

In those rarer cases in which the aneurism springs from the concave side of the ascending aorta, it only attains a comparatively trifling size, and gives rise to indefinite symptoms, those of compression of the pulmonary artery being the most important.[1]

Rupture of an aneurism of the ascending aorta occurs most frequently into the pericardium, the right pleural sac, the lungs, or externally.

Aneurisms of one or more of the sinuses of Valsalva, occasionally associated with aneurisms of the aortic valves themselves, have been described by A. H. Weber,[2] Jennings,[3] Rokitansky.[4]

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[1] Vide the case of *Rindfleisch* and *Obernier*. Deutsch. Arch. f. klin. Med. V. 539. 1869. *Colberg*, ibidem, S. 565. *Roberts*, Brit. Med. Journ. 1868. May. *Peacock*, Lancet, Feb. (Rupture into the Pulmonary Artery.) *Ebstein*, Wien. Med. Presse. 1869. 3.

[2] Transactions of the Pathol. Soc. 1867. Vol. XVII. p. 88.

[3] Dublin Quart. Journ. 1867. Aug. p. 229.

[4] Wien. Med. Jahrb. 1867. Bd. 13. S. 174.

Aneurisms of the Transverse Portion of the Arch of the Aorta.

Aneurisms of the aortic arch are spindle-shaped or saccular, and frequently extend into one or more of the emerging arteries. They are most frequently situated on the convex surface of the arch, and extend towards the right or upwards to the neck. If only of moderate size, an aneurism in this situation is frequently latent; its earliest sign is the perception of pulsation by the fingers pushed into the jugular fossa. When larger, compression is exerted on the anterior edges of both lungs, and hence there is dulness at the upper part of the manubrium sterni and at its edges; finally, there is absorption, from pressure, of the manubrium, of the collar bones, and of the uppermost ribs; a pulsating tumor then appears, rising into the neck, and, in one case of Bamberger's, reaching even the chin. The vessels given off from this part, the innominate and the left carotid and subclavian, frequently undergo alterations in their lumen, which may be dilated, or narrowed by distortion or displacement of their place of exit, or by coagula; therefore aneurism of the arch of the aorta is the most frequent cause of alterations in the size and chronological succession of the pulse in the arteries of the head and arm on both sides.[1]

In regard to unilateral arterial pulsation of the retina, vide p. 425.

Compression of the innominate veins or their branches leads to œdema, compression of the brachial plexus to disturbances of motility or sensibility in the area of distribution of one or more of its branches. From the comparative narrowness of the upper thoracic aperture compression of the trachea and difficulty of deglutition (the latter partly from injury to the vagus) are of no unfrequent occurrence. Paralysis of the left recurrent laryngeal nerve, which winds round the arch from before backwards, is not seldom present, and leads to paresis or complete paralysis of the laryngeal muscles on the left side, and thus produces hoarseness, aphonia, and impediment to the inspiration.[2]

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[1] Vide Lancet. 1866. I. 3. *Fergusson.*

[2] *Traube*, Deutsche Klinik. 1860. No. 41, etc.

Should the aneurism implicate the concavity of the arch, the left bronchus may be compressed; the inspiratory movements of this side are then less extensive than usual, the respiratory murmur weaker than normal and often accompanied by stenotic whistling.

Rupture occurs into the air-passages, into a pleural cavity, into the œsophagus, or into the mediastinum, more rarely into the pulmonary artery or into one of the large veins.

Aneurism of the Descending Thoracic Aorta.

In this part of the aorta, cylindrical, spindle-shaped, and saccular aneurisms occur; the latter most frequently bulge posteriorly or to one side of the vessel.

From their deep-seated position, these aneurisms are more frequently latent than others, and remain so for a longer period. Often their presence is first indicated by pain in the region of the left shoulder-blade extending towards the arm. Should the obtuse border of the left lung be forced aside, dulness and a murmur may be detected to the left of the spinal column; as the sac enlarges, pulsation may be detected, and finally a pulsating tumor makes its appearance; this comes to the surface between the shoulder-blade and the spinal column, or somewhat lower down at the level of the angle of the scapula, after it has eroded the ribs from within. Delay of the femoral pulse relative to that of the radial artery has been observed in occasional instances.[1]

Should the aneurism grow into the thoracic cavity, the heart may be pushed to the right, or it may be compressed against the anterior wall of the chest; in the latter case the cardiac impulse is felt to be more forcible and its area increased; sometimes a shock propagated through the heart from the aneurism is perceptible.

An actual increase of the cardiac force and palpitation sometimes accompany this compression of the heart.

Pressure on the hemiazygos, the azygos, and the intercostal

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[1] This, however, is wanting in a well-marked case now under my care.

veins, may lead to collateral dilatation of the veins on the anterior wall of the chest.

Intercostal neuralgia is not unfrequently the result of pressure upon the intercostal nerves ; pressure on the vertebræ produces erosion of them and pain (which is not, however, constant). This pain is increased by movement and by pressure upon the spinous processes, and is sometimes attended by nocturnal exacerbations. When the erosion is considerable, curvature of the spine may result, or even paraplegia from compression of the spinal cord.

Interference with deglutition is of frequent occurrence in aneurisms of the descending thoracic aorta.

Rupture most frequently takes place into one of the pleural cavities, or into the lungs, and next to these into the œsophagus.

Aneurisms of the Innominate, Carotid, and Subclavian Arteries.

Aneurisms of the innominate are rare, but sometimes occur in connection with aneurism of the arch of the aorta, and resemble the latter in position and symptoms. The tumor is situated at the right edge of the sternum, beneath the cartilage of the first rib and the clavicle, and extends upwards into the right supraclavicular fossa, behind the sterno-cleido-mastoid muscle.

In this situation we hear the sounds and murmurs, and perceive the pulsation, the latter usually as a double shock. The arteries of the head and arm commonly exhibit deviations from the normal in respect of the size and chronological succession of the pulse ; the murmurs are also propagated into the first portions of the carotid and subclavian arteries.

The functional symptoms are pain in the region of the aneurism, and, radiating from it, disturbances of innervation from compression of the right brachial plexus or the right recurrent laryngeal, dysphagia, and dyspnœa from compression of the trachea.

Pressure upon one of the innominate veins produces venous distention on one side of the neck and in one arm. Since the left innominate vein runs across the front of the innominate artery, it may be occasionally compressed earlier than the right one.

How uncertain the diagnosis of an aneurism of the innominate artery is from a concomitant or even a simple aneurism of the arch of the aorta, is well shown by numerous cases recorded in the English literature of this subject, in which this matter is discussed in relation to the question of operative procedure.

Aneurisms of the carotid, subclavians, and vertebrals are still rarer than those of the innominate, and are partly indeed to be regarded as external aneurisms. They occur as tumors in the right or left supra-clavicular fossa, and their connection with one artery or another is to be deduced partly from their anatomical position and partly from the alteration of the pulse (delay, smallness) in the peripheral portion of the vessel.

Regard must also be paid to any unilateral pulsation of the retinal artery which may be present (*vide antea*, p. 425). According to Duchek, the pulsation in an aneurism of the carotid or subclavian arteries is double, according to Gendrin single.

Aneurism of the vertebral artery is to be distinguished from a carotid aneurism in that pressure above the transverse process of the sixth cervical vertebra stops the pulsation only in a carotid aneurism, while pressure beneath this point stops the pulsation in aneurisms of both arteries.

Moderate dilatation of considerable stretches of the subclavian and carotid arteries are of tolerably frequent occurrence in arterio-sclerosis as well as in aneurism of the aorta.

Aneurism of the Pulmonary Artery.

Rokitansky, Krankheiten der Arterien. S. 30. Beob. XII.—*Dlanhy*, Prag. Vierteljahrschr. 1848.—*Dowse*, Lancet. 1874. II. p. 660.—*Gilewski*, Wien. med. Wochenschrft. 1868. Nos. 33-38.—*Goldbeck*, Diss. Giessen. 1868 (a collection of twenty cases).

Aneurisms as well as chronic endarteritis are extremely rare in the pulmonary artery (out of 915 cases of aneurisms generally, Crisp relates only four).

They may be spindle-shaped or sacculated, of moderate size, and usually situated on the arterial trunk.

Small aneurisms of the intra-pulmonary branches of the artery occasionally occur in tubercular cavities (originating in the

diminished resistance of the surrounding tissues and in external traction), but they are solely interesting anatomically.[1]

According to the observations hitherto recorded, in which, however, the diagnosis has been but seldom made during life, aneurism of the pulmonary artery may exhibit the following signs: dulness, with more or less considerable prominence in the second intercostal space on the left side and beneath the second left costal cartilage; above this loud sounds are heard, and a systolic—usually also a diastolic—impulse is perceptible.

Instead of the sounds, or, along with them, murmurs, especially a systolic one, may be heard and their thrill felt; these are propagated in the direction of the pulmonary artery, to the left and upwards.

Such an aneurism is very readily mistaken for an aortic aneurism. But the consecutive phenomena (dilatation and hypertrophy), which in the one case are confined to the left ventricle, and in the other to the right one, are important aids to the diagnosis; as may be also the proofs of consecutive changes in the lungs (hemorrhage, embolisms, etc.).

Rupture usually takes place into the pericardial sac.

Aneurisms of the Ductus Botalli

Are merely of anatomical interest; they are not above the size of a hazel-nut, and usually communicate only with the aorta, not with the pulmonary artery; they occur in new-born children, and undergo a spontaneous cure by the formation of a clot.[2]

Aneurism of the Abdominal Aorta.

Lebert, Aneur. d. Bauchaorta und ihrer Zweige. Berlin. 1865 (an analysis of one hundred and three cases).—*Moore*, Medical Press and Circular. March, 1869.—*Sallé*, Thèse de Paris. 1869.—*Wade*, British Medical Journal. 1868 (Dicrotic pulse).—*Murray*, Medical Times and Gazette. 1867. II. p. 647.—*Do.*, On the rapid cure of aneurism by pressure. London. 1871.—*Chancel*, Gaz. des Hopi-

[1] Vide *Fearn*, Transact. of the Path Soc. 1868. p. 45. *Moxon*, ibidem, p. 55.

[2] *Thore*, Archiv gén. IV. Sér. Tom. 23. p. 30.

taux. 1866. No. 37.—*Chatin*, Gaz. méd. de Lyon. 1866. No. 4,—*Daly*, London Hosp. Rep. 1866. III.

Aneurisms of the abdominal aorta are of frequent occurrence in the neighborhood of the tripus Halleri (cœliac axis), more commonly below than above it; they are spindle-shaped or saccular (from three to six inches in mean diameter); often of very considerable size (containing as much as ten pounds), so that they can be felt.

According to Stokes they are mostly "false" aneurisms.

They chiefly project from the anterior surface or sides of the artery, and compress or push aside the neighboring intestines, vessels, and nerves, or produce erosion or curvature of the spinal column. They more frequently develop downwards and to the left than upwards and to the right, because of the liver and diaphragm.

Adhesions of the sac to other organs also occur. Rupture takes place in about two-thirds of all cases, most frequently into the retro-peritoneal cellular tissue, between the layers of the mesentery, into the peritoneal or one of the pleural cavities, or into the intestinal tract.

Crisp observed the abdominal aorta affected 105 times out of 915 cases of aneurism.

Symptoms.

The objective facts in aneurisms of the thoracic and abdominal aorta exhibit many analogies, but from the more yielding character of the walls an abdominal aneurism becomes perceptible or even visible while still of but moderate size; it is most usually found to the left just above the navel, at first covered by the stomach and intestines; when the abdomen is but little distended, the aneurism comes in contact with its anterior wall and communicates to it a systolic concussion. On palpation of the tumor a forcible pulsation is perceived, somewhat posterior to the apex beat, and sometimes accompanied by thrill. Along with this a sound or a murmur is heard. A double shock, such as is felt in aneurism of the thoracic aorta, though possible

(Duchek), is only very rarely observed; a double sound must be generally propagated directly from the contiguous heart.

The murmur is sometimes audible in the aorta beneath the aneurism, or in the lumbar region. Diastolic murmurs are never heard in aneurisms of the abdominal aorta. The heart is very frequently free from alteration.

When the aneurism is situated high up, it cannot be palpated; and we can only perceive the pulsation communicated through the contiguous organs (liver, spine, etc.), and hear the murmur similarly propagated.

In the femoral and other arteries of the lower extremities the pulse may be delayed more than normal relative to the apex beat.

Functional disturbances may be entirely wanting in aneurism of the abdominal aorta, or they may be very severe. The first and most common of these is pain in the belly, particularly in the epigastric and hypochondriac regions, in the lower part of the dorsal and the upper part of the lumbar regions. This pain is deep-seated, dull, sometimes attended by colicky exacerbations, radiating backwards or to the sides. Vomiting, distention of the abdomen, constipation, and cessation of the pain upon assumption of the prone position, are often associated with pain of this character as well as with cardialgia. Diarrhœa, icterus, persistent dyspepsia with vomiting, obstinate constipation, and difficulty of swallowing may also occur. These disorders are partly produced by the simple pressure of the tumor upon the œsophagus, stomach, intestines, liver, or pancreas, but they probably also partly depend upon disturbance of the cœliac plexus, and possibly also the vagi, by the aneurism. These phenomena, in their totality, frequently resemble those presented by an ulcer or carcinoma of the stomach.

When the aneurism is situated lower down, particularly if it presses upon the vertebræ, the pain radiates more over the lumbar and sacral regions, or even towards the testicles or the upper part of the thighs, or (from pressure on the nerves or on the femoral arteries) paralysis of both extremities may occur.

The general condition is sometimes unaltered, at others a progressive cachexia sets in, which finally proves fatal without presenting any remarkable phenomena. Rupture of the aneu-

rism is, however, more frequent, so that death may occur as in any other internal hemorrhage, or we may have hæmatemesis, or bloody stools. The sudden occurrence of peritonitis, or of a painful tumor in the belly, gives a special indication as to the direction of the perforation towards the stomach, the intestines, the peritoneum, or the retro-peritoneal cellular tissue.

An aneurism of this kind may also rupture into the left pleural cavity.

Aneurisms of other Arteries in the Abdomen.

These aneurisms are comparatively rare, and are usually solitary; among thirty-nine cases collected by Lebert, the superior mesenteric artery and the splenic artery were affected ten times, the hepatic artery and its branches eight times, the cœliac and the inferior mesenteric arteries three times, the renal artery twice, and in three cases the aneurisms were multiple.

Such aneurisms are seldom larger than a hen's egg, generally globular; those of the mesenteric arteries are, according to Ponfick, not unfrequently of embolic origin.

Whitmarsh[1] describes an aneurism of the right ovarian artery. It proved fatal by rupture into the peritoneal cavity.

These aneurisms, from their small size, frequently give rise to no symptoms at all. If they are larger, the phenomena resemble very much those of aneurism of the abdominal aorta; this is particularly the case when the cœliac or superior mesenteric arteries are diseased, whilst in aneurisms of any of the other abdominal arteries the pain and swelling usually have a somewhat different position. These aneurisms frequently rupture, and then give rise to the phenomena of internal hemorrhage already described, which from the small size of the aneurism is not always fatal.

In aneurism of the hepatic artery, besides pain in the right hypochondrium, icterus is often present from compression of the gall duct or of one of its branches. In one case observed by the

[1] Brit. Med. Journ. 1867. II. p. 177.

author, there was an aneurism of the right principal branch of the hepatic artery the size of a hazel-nut, which had grown into the gall duct, that ran close to it. Repeated hemorrhages occurred into the gall duct, accompanied by all the symptoms of hepatic colic (pain, rigor with fever, collapse) and escape of blood by stool ; blood was also vomited a few times, and the patient finally died from exhaustion.

Aneurism of the Superior Mesenteric Artery: *Heppner*, Oesterr. med. Jahrb. 1868. I. p. 3.—*Chauffard*, Union méd. 1864. No. 54.

Aneurism of the Hepatic Artery: *H. Quincke*, Berl. klinische Wochenschr. 1871.—*Babington*, Dublin Jour. 1856.—*H. Wallmann*, Virch. Arch. XIV. S. 387.

Aneurism of the Cœliac Axis: *Aronsohn*, Gaz. méd. de Strassbourg. 1866. No. 12.—*Bulley*, Med. Times and Gaz. 1863. Dec.—*Concato*, Annali univ. Agosto, 1862.—Schmidt's Jahrb. 120 S. 303.

Aneurism of the Cerebral Arteries.

Lebert, Aneurysmen der Hirnarterien. Berl. klin. Wochenschr. 1866 (80 cases).—*Gongenheim*, Thèse de Paris. 1867.—*Durand*, Des aneurysmes de cerveau, etc. Paris. 1868.—*J. Coats*, Glasgow Medical Journal, August, 1873. (87 cases).—*Hayem*, Gaz. méd. de Paris. 1866. No. 29.—*Paulicki*, Deutsche Klinik. No. 48. 1867.—*R. Bartholow*, Amer. Journ. of Med. Sc. 1872. Oct. p. 373.—*S. Gee*, Bartholomew's Hosp. Rep. VII. p. 147. 1871. (2 cases).—*Russell*, Brit. Med. Journ. July 23, 1870 (1 case).—*R. W. Smith*, Dub. Quart. Journ. Nov. p. 443. 1870 (3 cases).—*W. J. Church*, Barthol. Hosp. Rep. VI. p. 99. 1870.—*Ch. Bastian*, Transact. of the Pathol. Soc. XX. p. 106. 1870. (Ibidem, several other cases by various authors.)

Aneurisms of the Internal Carotid.

Bezold, Deutsche Klinik. No. 24 et seq. 1867.—*Adams*, Lancet. 1869. December 7.

Aneurisms of the cerebral arteries are by no means rare.[1]

They occur most frequently at the base of the brain, in the neighborhood of the circle of Willis, and more commonly in connection with the carotid than with the vertebral arteries, especially on the artery of the fossa Sylvii (40 per cent. of all

[1] *Lebert* could collect eighty cases in 1866.

cases); and more frequently on the left side than on the right. Of all the other arteries the basilar is most frequently affected with aneurism.

Their medium size ranges from that of a bean to that of a hazel-nut; the opening communicating with the artery is wide; their site frequently where the artery divides. The arterial coats in the aneurism are attenuated and often defective, otherwise usually healthy, especially they are free from endarteritis; rupture took place in about 50 per cent. Circumscribed inflammation or softening was a frequent result of their pressure on the contiguous part of the brain.

The frequency of multiple aneurism of the cerebral arteries (about 25 per cent.) is remarkable, also the frequent concomitance of endocarditis and embolic lesions of the intestines.

According to Lebert, aneurism of the cerebral arteries is tolerably uniformly spread over all ages; those of the anterior cerebral arteries are most common in youth, the others in later years.

In a few cases the preoccurrence of a tumor was proved. English authors ascribe their origin chiefly to syphilis.

These facts in regard to the occurrence and etiology of aneurism of the cerebral arteries, mostly taken from the work of Lebert (1866), make it extremely probable that at least the greater part of them are of embolic origin (predominance in the area of the carotid, on the left side, youthful age, and coincidence of endocarditis), and this view has recently been expressed by Ogle, Ponfick, and others, and in some cases distinctly proved.

Symptoms.—Cerebral aneurisms are prone to develop themselves latently, and either to remain latent till their rupture or to give rise only to indeterminate and very gradually increasing phenomena, which resemble those of a tumor at the base of the brain. Pain in the head, sometimes limited to one part of the cranium (corresponding to the site of the aneurism), a sensation of pulsation, giddiness, or even epileptic attacks may occur. Pressure may even produce paralytic phenomena in the range of certain cerebral nerves, particularly the third pair; in aneurism of the internal carotid we have compression of the trigeminus, and neuralgiæ and anæsthesiæ in the area of distribution of its first

branch; when the aneurism is situated posteriorly, we have interference with speech and deglutition.

Hemiplegia is rare and usually only imperfect; pressure of a basilar aneurism on the pons may originate paralysis on both sides of the body, with predominance on one.

After such phenomena have persisted for some time in varying intensity, or without any prodromata whatever, an apoplectic attack occurs, to which the patient succumbs often within twenty-four hours, under symptoms of general cerebral compression, produced by the considerable effusion of blood into the arachnoid and subarachnoid spaces.

More rarely the hemorrhage is trifling, and its absorption is followed by improvement of the symptoms.

The *diagnosis*, as may be seen from what has been already said, is in many cases impossible, and at all events can only be defined as some disease at the base of the brain. Variations in the intensity of the symptoms occur also in other cerebral tumors. The fact that many of these aneurisms are due to embolism permits us to regard existing valvular lesions or a previous attack of rheumatism, etc., as of diagnostic importance.

In a few rare cases of aneurism of the internal carotid and of the middle meningeal artery, a systolic murmur is heard over the cranium.

Thus Hutchinson[1] reports the case of a woman, aged forty, who had an aneurism of the internal carotid the size of an egg, which was spontaneously cured by the coagulation of the blood within it. It lay external to the carotid, with which it communicated by a narrow opening; it had flattened the Gasserian ganglion, and destroyed the third nerve. The patient had long suffered from headache; then pulsation in the temporal region set in, accompanied by disturbance of vision. A murmur was audible over the head; the pulsation gradually ceased; the general condition improved. The patient lived for eight years with this aneurism, and finally died of a dissecting aneurism of the abdominal aorta. From the superficial position of the meningeal artery, aneurism of it (which is very rare) has somewhat different symptoms. In one case described by Kremnitz[2] the aneurism appeared half a year after a blow on the right side of the head on the edge of the bed. A year afterwards, above the right ear there appeared a soft, fluctuating tumor (7 and 9 ctm. in diameter), which could be reponed; pulsation and thrill could both be felt over it. Compression of the right common carotid caused the tumor to

[1] Med. Times. 1875. I. p. 563.

[2] Deutsche Zeitschr. für Chirurgie. IV. S. 473. 1874.

disappear, compression of the left carotid produced increase both of the size of the tumor and of the loudness of the murmur (!, in fungus of the dura mater compression of either carotid is said to have a similar effect). One year subsequently a cure was effected by ligature of the right common carotid and the application of a silver plate over the osseous defect.

The *prognosis* in aneurism of the cerebral arteries is tolerably unfavorable.

When the diagnosis can be made, the treatment must be conducted in accordance with the principles applicable to the treatment of aneurism generally. In particular we might try the use of ergotine, acetate of lead, or iodide of potassium (*vide postea*); perhaps also compression or ligature of one of the carotids (provisional compression must be first employed to ascertain the influence of the collateral communication through the circle of Willis, which in itself makes the result somewhat problematical).

In what has just been said we have left unconsidered those aneurisms of the minute cerebral arteries which frequently occur in advanced life (Brummerstaedt, Pestalozzi, and others), to which recent French authors (Charcot and Bouchard[1]) have ascribed an important influence in the production of cerebral hemorrhages. Similar small aneurisms of the retinal arteries are frequently concomitant, and may be recognized by means of the ophthalmoscope.

Multiple Aneurisms.

It has already been stated that several aneurisms are not unfrequently found in the same individual, and that the supposed existence of a so-called aneurismal diathesis for the explanation of such cases is, to say the least, superfluous, seeing that in them there is always to be found extensive endarteritis.

Cruveilhier has described such a case (Livr. 28) with several aneurisms of the aorta and of the superior mesenteric artery; Rokitansky (Beob. 3, Tafel. VI) a case of numerous aneurisms from the size of a millet-seed to that of a hazel-nut, affecting all the medium-sized and smaller arteries, particularly the mesenteric arteries, and caused by rupture of the intima. C. O. Weber, p. 178 and Crisp, p. 188, quote many cases of multiple aneurisms. I myself have found in a girl aged twenty, who

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[1] Archiv. d. Physiol. I. 1869.

[2] *Liouville*, Gaz. des hôp. 1870. No. 36.

died of nephritis and endocarditis, multiple aneurisms of the coronary arteries (about twenty), from the size of a linseed to that of a pea; but no others throughout the whole arterial area. During life there was no indication of the existence of these aneurisms.

In the case related by Krauspe (*vide antea*, p. 403) there were, besides spindle-aped aneurisms of the brachial and femoral arteries, diffuse dilatations of several arteries, particularly the carotids.

Macleod [1] relates the case of a man aged thirty-seven who had six aneurisms on his two inferior extremities.

Loomis, New York, Med. Record, 1868, July.

Porter, Dublin Quart. Jour. 1867, p, 441.

Pelletan, clin. chirurgic. 1810 (sixty-three aneurisms in one individual).

In horses multiple aneurisms of the mesenteric arteries are extremely common [2] and frequently give rise to attacks of colic (*vide antea*, p. 393). [This is not the case in Great Britain.—*Tr.*]

Diagnosis.

The diagnosis of internal aneurisms is difficult, so difficult tnat in most cases they are not discovered till after death. Their diagnosis will always remain impossible when, from their small size or concealed position, they are removed from objective examination, and also give rise to no functional disturbance. How diverse the latter may be according to the site of the aneurism, and how indefinite, may be readily deduced from what has been already said ; so that even where arterio-sclerosis and other etiological causes are present, we can only base upon them a very indefinite suspicion as to the existence of an aneurism. And, besides, in advanced life we have arterial changes, disturbances of the respiration, and of the cardiac action, very frequently occurring from various causes. It is only when the patient has a distinctly localized feeling of pressure or pain in a definite part of the course of the aorta, when pulsation is felt, and when a tumor is perceptible, that the idea of aneurism becomes probable.

Yet, even in such a case, we must beware of mistaking for aneurisms tumors of quite a different character, such as intra-

[1] Glasgow. Med. Jour. 1873. May.

[2] According to *Bruckmüller* (Oesterr. Vierteljahrsch. f. Veterinärkunde. Bd. 38. 1871), in more than 75 per cent.

thoracic neoplasms, especially of the mediastinum, collections of pus proceeding from the ribs, the spinal column, the pleura, etc.; and we must beware of mistaking for abdominal aneurisms the various tumors originating in the liver, kidneys, lymphatic glands, pancreas, stomach, and intestines, especially carcinomata of those organs, encysted peritoneal exudations, fæcal tumors, displaced kidneys, etc.

It is of the utmost diagnostic importance to ascertain that the tumor pulsates; and we must carefully ascertain its expansile pulsation in all directions, and avoid being misled by the simple rising and falling of a solid tumor to which arterial pulsation is propagated. In the abdomen we can occasionally put a stop to this up and down pulsating movement of a solid tumor by pushing it to one side.

Dislocation of the heart or propagation of its impulse by a contiguous organ or tumor may also give rise to mistakes. In doubtful cases the absence of continuity between the cardiac impulse and the pulsating tumor, and also delay of the pulsation relative to the cardiac impulse, are in favor of the diagnosis of aneurism.

Pulsation at the upper part of the abdomen, *pulsatio epigastrica*, may arise from various causes, and not unfrequently misleads the tyro as to the existence of abdominal aneurism. Sometimes this pulsation occurs from simple propagation of the cardiac pulsation (through the left lobe of the liver), from simple increase of the force of the heart's beat, or from hypertrophy of the right ventricle; at other times it originates in the abdominal aorta; it is then delayed relative to the impulse of the cardiac apex, is often jerking, and is propagated to the anterior wall of the abdomen through the left lobe of the liver, the stomach, or intestines.

An empty condition of the latter organs, emaciation and anæmia (!—[more probably spanæmia—*Tr.*]), relaxation of the abdominal walls, and strong projection of the lumbar vertebræ, are combined with excited cardiac action—favorable conditions for the production of this phenomenon; hence this is specially observed in old women, and is described as nervous pulsation of the abdominal aorta; it is possible that in such cases there may be also dilatation of this vessel, and the variations in the phenomena in such cases would be thus in part explained. Usually we can palpate the abdominal aorta in its normal position, a little to the left of the median line, down to its bifurcation (at the level of the navel), and thus determine its actual size, and so avoid mistaking this pulsation for that of an abdominal aneurism, which is either of larger dimensions than the normal artery, or forms a circumscribed tumor upon it.

There may even be a difficulty in explaining the nature of any murmur which may be present. We must remember, however, that an aneurismal murmur is almost exclusively systolic in character. The heart must, first of all, be carefully examined, so as to avoid the possibility of any mistake arising from the presence of any valvular murmurs. A dilatation of the ascending aorta, too trifling in character to be reckoned an aneurism, may nevertheless be sufficient to give rise to a systolic blowing murmur, as may also trifling vegetations or rigidity of the aortic valves, without there being any actual stenosis present; in such cases, therefore, we must specially seek for other signs and symptoms in order to determine the existence of an aneurism with certainty. And in making the necessary examination, we must also not forget that narrowing of the arterial calibre, from the compression of a tumor, or from the results of chronic endarteritis, at the origins of any of the large arteries, may also give rise to systolic murmurs. Finally, when the artery auscultated is only covered with soft parts, as, for instance, is the case with the subclavian artery and the abdominal aorta in part of their respective courses, compression by the stethoscope may give rise to an artificial stenosis and a murmur. In such cases it is important when the murmur is always heard exactly at the same spot, and also with the upper part of the body bent forward.

We must always remember that even in large aneurisms most of the signs already described are inconstant, and that their partial or total absence cannot therefore be regarded as excluding the presence of an aneurism; in making the diagnosis, therefore, we must duly consider all the local and other phenomena as well as the history of the development of the disease. Frequently we can attain a somewhat probable diagnosis of aneurism only by excluding the possibility of any other disease.

The differential diagnosis from simple atherosis of the arteries is all the more difficult that it very frequently coexists with aneurism, and because it is upon this disease that those disturbances of the general nutrition depend which occasionally accompany aneurism; we ought, therefore, only to assume the existence of aneurism when characteristic local symptoms are present, or when appropriate functional disturbances of thoracic

or abdominal organs have become relatively suddenly developed in connection with long existing atherosis.

The subjective ailment not unfrequently resembles simple angina pectoris; but in the latter disease the pauses are more frequently quite free from pain, while in aneurism the dull feeling of oppression is continuous, though its intensity may vary; the pain of the latter also does not always shoot directly from the region of the heart as in angina pectoris.

Mediastinal neoplasms are distinguished from aneurisms of this region by the communicated character of their pulsation, and by the usually somewhat triangular form of their dulness; they ordinarily give rise more rapidly to cachexia, involve other lymphatic glands (those of the neck, axilla, etc.), and early, and before they have attained any great size, give rise to compression of the œsophagus, the bronchi, the vagus, etc.

Should the mediastinal tumor be of secondary origin, the organ primarily diseased indicates its nature.

Course and Result.

The course of an aneurism is with few exceptions a chronic one, and its commencement cannot usually be determined. At first indefinite phenomena occur, which are but little regarded: shortness of breath, slight feeling of oppression in the chest; to these succeed attacks of asthma, and of palpitation; the general nutrition suffers, and the tumor may then become perceptible. In aneurisms of the abdominal aorta, pains in the epigastrium or in the back usually constitute the equally indefinite primary phenomena.

In but a few cases only can the patient definitely date the origin of his disease from a certain time, from an injury, or an over-exertion, etc.

Finally, there are cases which run their course wholly latent, and which either succumb to some totally different disease, or in which the primary occurrence of definite symptoms is at once rapidly followed by death.

In all ordinary cases the disturbances already mentioned gradually increase, the phenomena due to pressure upon the con-

tiguous organs are developed, and thus we have serious interference with the respiration, the circulation, or the digestion, so that some complication on the part of these systems leads to a fatal result; or the patient may die from marasmus, or from some accidental intercurrent disease.

Not unfrequently, during the course of an aneurism, there occur periods of remission of all these disturbances; in particular, the phenomena due to pressure often undergo remarkable variations, which may be due to the formation of coagula and consequent changes in the plasticity of the tumor.

In regard to the duration of the disease, we know but little, on account of the uncertainty as to its commencement. In thirty cases of aneurism of the thoracic aorta, collected by Lebert, the disease lasted from half a year to four years, with a mean of one and a quarter years; but aneurisms have been known to last for a much longer time, and this is at least highly probable in many of those which are latent. Aneurisms of the abdominal aorta are credited with a much longer duration, as much as eight years; but this may be only apparent and may depend upon earlier diagnosis.

Of thirty-six cases of "internal aneurism" analyzed by P. Niemeyer, he ascribes to three a duration of from 10 to 20 years, and to eight a duration of from 3 to 6 years.

In young patients with a powerful cardiac action, the disease seems to make more rapid progress than in old people.

In one case observed by myself, the patient, aged fifty-seven, with an aneurism of the descending thoracic aorta the size of two fists, could, one year subsequent to the occurrence of symptoms (probably much longer after the commencement of the aneurism), still pursue his calling as a guide in the Bernese Alps, and make journeys of from 8 to 10 German miles (32 to 40 British) in length, such as the passage of the Grimsel.

A weaver, aged thirty-two, lived and labored for twelve years[1] with an aneurism of the aorta projecting through his sternum.

In by far the larger number of cases the disease proves fatal, either, as already related, by marasmus and local complications, or suddenly by paralysis of the heart, or more frequently by rupture of the sac.

[1] *Jamieson.* Med. Press and Circular. Feb. 1873.

Rupture is very common in aneurism of the cerebral arteries, and death then follows under the phenomena of cerebral apoplexy.

A cure is very rare, and all the rarer the larger the aneurism, the larger the artery from which it springs, and the larger the opening of communication between the aneurism and the artery is.

Cures of peripheral aneurisms are most frequently observed. Cured aneurisms of the aorta are only found accidentally at post-mortem examinations, but even during life diminution in size and cessation of growth, consequently a comparative cure, have been observed in aortic aneurisms.

According to Stokes, the cure of an aneurism may be simulated by the pulsation becoming less or disappearing, the tumor, nevertheless, extending in other directions.

Prognosis.

From what has already been said, it is evident that the prognosis in aneurism is mostly unfavorable, all the more unfavorable the less open it is to surgical treatment, and the more voluminous and thin-walled it is, because then the compression of contiguous organs is more important, and rupture itself more imminent. Extensive arterio-sclerosis makes the prognosis even worse, on account of the marasmus connected with it. Further, the prognosis materially depends upon whether the aneurism threatens to impair the functions of any vital organ.

Treatment.

COMPRESSION: *Murray*, On the rapid cure of aneurism by pressure. London. 1871. —*Moxon* and *Durham*, Case of abdom. aneur. cured by compression of the aorta. Med. chir. transact. Vol. 55; and Lancet, April, 1872.—*E. H. Greenhow*, Med. chirur. transact. Vol. 56, p. 385; and Lancet, June, 1873.—*Bryant*, do., Lancet, April, 1872. Med. chirur. transact. Vol. 55. April, 1872.—*T. Holmes*, Surgical treatment of aneurism. Lancet, June to Nov. 1872.

ERGOTINE: *von Langenbeck*, Berl. klin. Wochenschr. No. 12. 1869.—*Rouge*, Bull. de la Soc. méd. de la Suisse. Rom. 1869.—*Dutoit*, Arch. für klin. Chir. XII. H. 3. —*Wolff*, Heilung eines Aneur. aortae abd. Berlin. klin. Wochenschr. 1873. No.

27.—*Catiano*, Ueber die subcut. Anwendung des Ergotins. Berlin. Diss. 1873. —*P. Vogt.* Beh. v. Varicen mit Ergotininjection. Berl. klin. Wochenschr. 1872. No. 10.

Coagulating Injections: *Pétrequin*, Bull. de la Soc. de Chir. Vol. III. p. 524. 1849.—*Lenoir*, Gaz. hebd. I. No. 2. 1853. Oct.—*Gontaux et Giraldès*, Expér. sur les inj. Ibid. April, 1854.

Foreign Bodies: *Moore* (twenty-six yards of iron wire), Lancet. 1864. I. April; and Med. chir. transact. Vol. XLVII. p. 129. London. 1864.—*Levis* (Philadelphia). Introduction of horse hair. Philadelphia Med. Times. 25th Oct. 1873.—Ibid. 1874. No. 126.—*T. Bryant*, Guy's Hospital Gazette. 6th Dec. 1873.—*Baccelli*, Treatment of aneurisms. Gaz. med. ital. Province Venete. 12th April and 24th May, 1873.

Galvano-Puncture: *Pétrequin*, Comptes rendus. Bd. 21. p. 992. 1845.—*Bruns*, Galvano-chirurgie. Tübingen. 1870.—*J. Abeille*, L'électricité appliq. à la therapeutique chirurg. 1870.—*Duncan* and *Fraser*, Edin. Med. Jour. 1867. Aug. p. 102 (experiments upon the action of galvanism on albumen, etc.).

L. Ciniselli, Dell'azione chimica dell' Electrico, etc. Cremona. 1862.—*Do.* (report of twenty-three cases), Annali univers. Nov. 1870. L'Imparziale. April, 1871. Gaz. méd. de Paris. 1872. No. 27.—*Do.* (report of five cases), Il Galvagni. 1873.—*Do.* (one case), Gaz. Med. Lomb. 1868. Nos. 39 and 44.—*Duncan*, Edinb. Med. Jour. 1872. Dec. 1866. April. p. 920.—*Lincoln*, New York Med. Rec. May 15, 1871.—*Bastian*, Brit. Med. Jour. Nov. 22 and 29, 1873.—*M. Beck*, Lancet, Oct. 18, 1873.—*M. C. Anderson*, Lancet, Feb. 22, 1873.—*Macchiavelli*, Gaz. med. ital. Lomb. No. 38. 1870.—*De Cristoforis*, Rendicont del R. Inst. Lomb. Ser. II. Vol. III. 1870.—Gaz. med. it. Lomb. 1870.—*Mazzachelli*, Gaz. med. it. Lomb. 1870.—*F. dell'Acqua*, Ibidem.—*Zdekaner*, Petersburg med. Zeitschr. I. Heft. 1869 (five cases).

Cure by Starvation and the Recumbent Posture, Together or Separately: *Morgagni*, De sed. et caus. morb. Epist. XVII. No. 30.—*J. Tufnell*, Successful treatment of internal aneurism. London. 1875.—*Do.*, The treatment of aneurism by position. Lancet. 1873. Dec.—*Nicolaysen*, Nord. med. Arkiv. 1869. Jahresber. II. S. 85.

Acetate of Lead: *Dusol et Legroux*, Arch. gén. de méd. 1839. III. Sér. V. p. 443. —*Höegh*, Norsk. Magazin. f. Læg. Bd. 22. H. 6. 1868.

[Iodide of Potassium: *Chuckerbutty*, Brit. Med. Jour. 1862. July.—*Roberts*, Brit. Med. Jour. 1863. January.—*Bouillaud*, Gaz. d. Hôpitaux. 1859.—*Balfour*, Ed. Med. Jour. 1868, July; and 1869, and 1871.—*Do.*, Transactions of St. And. Med. Graduates, 1861. p. 68; and for a full account of this treatment, based upon over thirty cases, with numerous cases and dissections, vide *Balfour*, Clinical lectures on diseases of the heart and aorta. London. 1876. p. 367.]

The treatment of aneurism must have for its object the obliteration of the sac; only thus can a perfect cure be obtained, as

we learn from spontaneous cures and from the results of surgical interference in the aneurisms of peripheral arteries.

When a perfect cure of this kind is aimed at, it is necessary that a proper supply of blood to the parts lying beyond the aneurism be duly secured, either by patency of the arterial lumen or by the collateral circulation.

While therefore most of the methods of treatment proposed have for their object the formation of coagula within the aneurismal sac, the coagula formed do not all seem to be of equal value in respect of their power of organization.

Coagulation may be induced in three modes: by slowing the blood stream; by the production of an unequal and uneven surface on the internal wall; and, finally, by causing inflammation of the wall of the sac. By many modes of procedure, several of these causes are put in action simultaneously.

An advance in the mode of treating aneurism is chiefly to be looked for from accurate experimental investigations as to the conditions under which we shall not only be able to produce coagula at will within the sac, but to produce organizable coagula, and as to the conditions under which they actually do become organized.

In the large arteries of cavities of the trunk the customary methods of ligature—above the aneurism, below it, or both above and below it—must of necessity be almost exclusively restricted to the second method, and even then ligature is only applicable in exceptional cases, as in aneurism of the innominata, where the carotid and subclavian arteries may be tied, and in aneurisms of the iliac arteries or of the abdominal aorta, where the femoral may be ligatured.

In a woman with a sacculated aneurism of the arch of the aorta, which during life was thought to be an aneurism of the innominata and pulsated at the junction of the right clavicle with the sternum, Christopher Heath [1] tied the subclavian artery and the common carotid. The tumor diminished in size, the patient was discharged cured, and four years subsequently died from rupture of the aneurism externally.

In one case operated on by Fearn, the patient died of pleurisy three months after the operation; the aneurism was filled with old coagula.[2] These relatively successful results ought to lead to further similar trials.

[1] The Lancet. July, 1870. [2] *Chr. Heath*, Transact. of the Pathol. Soc. 1868.

A similar operation was performed by Holmes,[1] in the case of an aneurism of the aorta and of the innominata, with temporary benefit. The patient died seven weeks subsequently.

Cockle and Heath [2] ligatured the left carotid in an aneurism of the arch of the aorta, and observed as its result remission of the subjective symptoms and diminution of the pulsation. Cockle [3] quotes similar cases, which had, however, been mistaken for aneurism of the innominata.

Even the abdominal aorta has been a few times ligatured (from the left iliac region)—but unsuccessfully— (Cooper; also in a case of aneurism of the left common iliac by W. Stokes.[4])

Instead of ligature, compression has been frequently employed in peripheral aneurisms. Whilst it has not been found possible to keep up direct compression of the aneurism itself by bandages etc. for any length of time, indirect compression—on the artery leading to the sac—has been more successful. This has either been carried out by means of variously modified tourniquets or by the finger, applied constantly for several days, or daily for a few hours only, during a longer period. The pulsation becomes gradually feebler, and finally disappears, as the sac becomes filled by coagula. Complete suppression of the blood stream is not necessary for the production of this result, but only slowing of it by lessening its quantity; hence the compression does not require to be complete.

Recently this procedure has been employed in the treatment of aneurisms of the abdominal aorta, Lister's compressorium having been applied above the aneurism during chloroform narcosis. [So early as 1864, Dr. Murray, of Newcastle, successfully operated in this way in a case of abdominal aneurism. An ordinary tourniquet was employed; the first séance lasted for two hours, the second—three days subsequently—for about five hours; the result was completely successful; the patient, three months subsequently, was following his trade as an engine fitter, and died six years afterwards from rupture of another aneurism. The dissection proved that complete obliteration of the aorta had occurred. Vide op. cit.—*Tr.*] Moxon and Durham compressed in one case for ten hours, Greenhow for several hours repeatedly; in both cases diminution of the pulsation occurred, in a few weeks complete cessation, and finally a cure. The radial pulse during the compression exhibited increased frequency; it was tense or small and dicrotous; in one case pain and coldness of the lower extremities, vomiting of blood, and temporary albuminuria were observed, and in it, indeed, judging from the absence of pulsation in the femoral arteries and their branches, obliteration of the aorta had been produced. In two other cases (by Bryant and Bloxam) the compression proved fatal by inducing peritonitis.

This method, though thus not without danger, has been followed by good

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[1] St. George's Hosp. Rep. VI. 1873.

[2] Brit. Med. Jour. May, 1872.

[3] Lancet. 1869. April. *Sands*, New York Med. Rec. 1869. Dec. *Maunder*, Lancet. 1867. II. p. 324.

[4] Dub. Quart. Jour. 1861. August.

results. Should the aneurism lie too high, distal compression may be tried (as was done by Bryant), and in this case also coagula formed within the sac.

Kneading of an aneurism, whereby it is sought to loosen a clot and get it so wedged as to block up the arterial opening, is but rarely employed even in aneurisms of the peripheral arteries, and ought to be entirely rejected in the case of aneurisms of the larger arteries on account of the great danger of embolism.[1]

Instead of employing mechanical compression, the effort has been made to diminish the sac by producing contraction of its muscular elements; at least this idea seems to have been the primary inducement to the employment of the subcutaneous injection of ergotine. From one-half to four and a half grains of watery extract of ergot dissolved in a mixture of one part of glycerine to five of alcohol, or in glycerine and water, equal parts, have been injected over or in the neighborhood of the tumor, at intervals varying from half a day to several days. The tumor is said to have become gradually small and hard, and the pulsation to disappear.

Langenbeck cured in this way an aneurism of the subclavian in six weeks, and one of the radial in twenty-four hours. Good results are also reported by Dutoit, Catiano, etc. Wolff cured an aneurism of the abdominal aorta by injections in the mesogastric region, whilst Rouge (as well as the author) was unsuccessful in aortic aneurisms.

At the place of injection indurations or suppurative inflammations frequently occur; whilst the latter may favor rupture of the aneurism, the former may lead to a desirable compression and strengthening of the walls of the aneurism. And in this way Schwalbe[2] has explained the good results observed, which he regards as produced not by the specific action of the ergotine, but by its property of exciting inflammation, partly due to the vehicle employed. The author agrees in this opinion, since any permanent contraction of the arterial muscle by ergotine has not been proved, and is not probable; moreover, because in the walls of the aneurismal sac the muscular element is very defective. And this is specially the case in aneurisms of the larger arteries, particularly the aorta. If, in consequence of the recorded results, any one should desire to try in any case the injection of ergotine, great caution is recommended in regard to the amount and quality of the fluid injected, on account of the danger which may arise from the possible occurrence of an abscess. [Boiling the fluid to be injected, or dissolving a small quantity of chloral in it, so as to destroy existing bacteria, is said to be an effectual method of preventing the occurrence of inflammation or abscess.—*Tr.*]

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[1] Vide *Esmarch.* Virch. Arch. 1857. XI. S. 470.

[2] Virch. Archiv. 56. S. 360. 1872.

It has further been attempted to produce coagulation within the aneurismal sac by chemical means. Alcohol, acetic acid, and sulphuric acid have been recommended for this purpose. Salts of iron, particularly the solution of the chloride, have been employed, a few drops being introduced by means of Pravaz's syringe into the cavity of the aneurism. The deposit of albumen thus produced is supposed to combine with the rest of the blood in the sac coagulated by the injection, and to form a solid mass. Since, however, the ferro-albuminous precipitate is of a tolerably loose consistence, there is a probable danger that part of it may be swept off by the blood-stream and driven into some of the peripheral arteries; and this may occur even when the superficial arteries lying above and below the sac have been compressed for some time. This will happen all the more easily, the larger the opening communicating between the sac and the arterial lumen, and the greater the amount of the fluid injected. The results of this procedure, which has specially been tried in France, have not been encouraging; besides embolism, inflammation and gangrene of the sac have occurred. This method should at the most be only attempted in superficial sacculated aneurisms with a narrow neck, and even in these there is great danger of suppuration of the sac and of the superjacent tissues.

Attempts have also been made to produce coagulation within the aneurismal sac by the introduction of foreign bodies, and at Velpeau's recommendation needles have been introduced into the sac (acupuncture); but even in small peripheral aneurisms the result has been very doubtful and the complications many and dangerous.

Stromeyer [1] recommended the injection of molten wax or spermaceti into the sac, isolated by pressure and emptied of blood; such an attempt could, however, only be carried out in peripheral arteries.

Moore, of the Middlesex Hospital in London, seems to have made the first practical experiment in the introduction of a large mass of foreign substance into an aneurism; he introduced, through a canula, twenty-six yards of fine iron wire into a projecting aneurism of the ascending aorta; the patient died with signs of exacerbation of the local symptoms. Recently, Baccelli, of Rome, in a similar case, passed a trocar (one seventeenth of an inch in diameter) obliquely through the skin to a depth of one and a half inches into an aneurism, and through it introduced a

[1] Handbuch d. Chirurgie. S. 399.

piece of watch-spring fourteen inches long and one twenty-fifth of an inch broad into the sac, within which it coiled itself up. The wound was closed by collodion. Temporary diminution of the pulsation occurred, but this was soon followed by inflammation of the skin and gangrene; the patient died on the thirtieth day. During this period repeated attempts were made to remove the watch-spring, and pieces of it were actually taken out. At the dissection the rest of the spring was found in six pieces, associated with coagula of various ages and fluid blood. The opening between the sac and the aorta measured four-fifths of an inch in diameter.

Other surgeons have employed horse hair instead of iron wire. Levis, of Philadelphia, treated in this way an enormous aneurism of the subclavian which projected considerably both above and below the clavicle. Through the canula of a fine trocar he introduced more than twenty-four feet of hair into the aneurism; in fourteen days the tumor was hard, and pulsation had ceased both in the tumor and in the axillary and radial arteries; the pain had disappeared; nevertheless bloody expectoration occurred, and the patient died suffocated. The hair lay in the posterior part of the sac, which was filled with old and recent coagula.

T. Bryant treated after a similar fashion a popliteal aneurism, in which, on account of valvular cardiac lesion, no other treatment seemed possible. The pulsation diminished, but the patient died.

However dangerous the introduction of a foreign body into an aneurism may appear at the first glance, yet further experiments seem quite justifiable from the insufficiency of all our other remedies. Iron wire seems preferable from its freedom from any possible organic infection, yet, as Baccelli's case proves, it may wound the sac from within. Possibly carbolized catgut may be employed with advantage.

Soon after the introduction of acupuncture, it was attempted (first of all by Pétrequin, 1831) to pass a galvanic current through the needles—*galvano-puncture.* In this way their mechanical action as foreign bodies was combined with a chemical action, produced by the electrolytic decomposition of water and of the salts in the blood.

Whilst at the negative pole hydrogen (2 vols.) and alkalies were separated, at the positive pole oxygen (1 vol.) and acids were separated, and the needle was oxidized unless made of one of the noble metals. When the needle-shaped poles of a battery are dipped into albumen or serous exudation, there is formed on both of them a white spongy mass consisting of bubbles of gas and molecular albumen, and of these that formed at the negative pole is considerably more voluminous, looser in texture, and more easily separated from the needle than that formed at the positive pole. If, instead of a solution of albumen, blood be employed, the mass separated at the positive pole is of a brownish-black color. It is certain that the acids and alkalies electrolytically separated, from their influence upon albumen and on hæmoglobin, have a share in the formation of these masses, but it must remain undetermined whether the

direct influence of the galvanic current may not have contributed to the formation of the "coagulum;" at all events, this must be regarded as perfectly distinct from a "fibrinous coagulum," or from a coagulum produced by the action of chemical reagents. Nevertheless, such a coagulum may initiate a further "fibrinous coagulation."

Experiments on living animals have proved that even in living and circulating blood local coagulation may be produced by galvano-puncture.

When employed therapeutically, this coagulation certainly occurs to a varying extent and in divers degrees of rapidity; in each indivdual case, indeed, the conditions vary considerably.

According to Ciniselli, the clot produced by electricity within the sac of an aneurism differs from that ordinarily found there in its density, the absence of lamellæ in its structure, its irregular edges, and its yellowish, pale-red color. Besides the direct action on the blood in the sac, the inflammatory excitement in the wall of the sac must also have an influence upon the subsequent formation of the clot. Interrupted currents have been employed a few times, but they have been for various reasons found unsuitable, and have accordingly been given up.

Ciniselli recommends for galvano-puncture a current of low intensity (to avoid cauterization), but of a sufficient degree of tension, for the production of which a column of many elements, possessing but a small superficies, and but little electromotor power, is best suited. He employs a voltaic column consisting of thirty zinc and copper plates (each from two to four inches square), or also from ten to thirty small Daniell elements. These will develop in water acidulated with sulphuric acid, from twelve to eighteen one-hundredths of a cubic inch of detonating gas (2 parts of hydrogen to 1 of oxygen[1]) in five minutes. He prefers needles one twenty-fifth of an inch in thickness, sharp and well polished, and made of steel, so that they may become oxidated, and corrosion of the tissues by the electrodes may thus be avoided. The needles were introduced to a depth of from one to two inches, and at a distance of at least four-tenths of an inch from one another; in intrathoracic aneurisms from two to four needles were employed, in extrathoracic ones not more than six. Instead of employing a continuous current, he prefers to allow both poles to act upon each needle alternately, and indeed at first he unites the positive pole with the first needle, and employs as the negative pole a damp electrode applied in the neighborhood; after five or six minutes he unites No. 2 needle with the positive pole, No. 1 needle with the negative, then No. 3 needle with the positive pole, and so on. The whole of the séance should last only from twenty-five to forty minutes. By changing the poles the corrosion of the tissues is said to be diminished. *C.* changes the pole so soon as a black ring has formed round the positive needle, and changes again whenever (at the now negative pole) a white zone has become developed within the black circle. The needles must be at once removed when the operation is finished, and this is most easily done by needle forceps, as their oxidized surface produces considerable friction at the punctures. The body

[1] Vide *Watts'* Dictionary of Chemistry. London. 1866. Page 285.

must be maintained in perfect rest, and the tumors must be fomented with a lead lotion or covered with a bladder of ice; by this and by compression any trifling bleeding from the punctures is at once suppressed. The galvano-punctures should not be renewed till after the lapse of several weeks.

A few writers on the subject (as J. Duncan) lay considerable stress upon the needles being isolated, except at their points, by some covering material (such as enamel, glass, gutta-percha, or vulcanite). But the needles are thus made thicker and less smooth, and it is also questionable whether we may not thus prevent a slight cauterization of the punctures, and a slight coincident inflammation which may serve to prevent hemorrhage. Gilt needles and platinum needles have also been employed; these, because incapable of oxidation, are more easily removed, but are more objectionable than the steel needles, because the punctures made by them are more extensively corroded.

Whether the changing of the poles recommended by Ciniselli is of advantage, may be doubted (according to Duncan the coagulum thus obtained is firmer but of slower formation). The author (at the recommendation of Prof. Hitzig) has used needles (long Carlsbad needles) connected with silver wire and attached to the positive pole, whilst a broad moist electrode was employed as a negative pole and applied over the skin at a distance of from three to six inches; no evil results from cauterization were observed. The positive pole deserves to be preferred on account of the greater density of the resulting coagulum.

It is readily understood that the closing and opening of the current must be gradually made to avoid shocks, and that the needles must be so introduced that they shall not come in contact with each other.

If we follow the preceding rules, evil results are scarcely ever observed. If the sac has very thin walls, considerable hemorrhage may follow; when the cauterization has been too great, or when the tension of the skin has been extreme, suppuration of the punctures may ensue.

Of twenty-three cases of electro-puncture for aneurism of the thoracic aorta related by Ciniselli, five were cured; only one case from defective procedure proved fatal by gangrene of the punctures. Moreover, improvement of the objective phenomena of the aneurism occurred often after the lapse of days or weeks (by the secondary formation of a coagulum). Under favorable circumstances, one single electro-puncture may be sufficient; according to Ciniselli (and others), this is to be expected when the aneurism is of medium size, is still intrathoracic and only communicates with the artery by a small opening. The neighborhood of emerging branches is unfavorable. Temporary results from electro-puncture are frequently observed (Duncan, Zdekaner, etc.). Of three cases of strongly projecting aneurisms of

the thoracic aorta treated by the author by repeated electro-punctures, only one presented an objective retrogression of all the symptoms; no evil results were ever observed.

In most cases it is either wholly or partly impossible successfully to carry out these attempts at the cure of an aneurism, and we are therefore forced to employ an indirect method of treatment, either by itself or in aid of the direct local treatment.

Under all circumstances, the *general treatment* of the patient is of the greatest importance, and this, as a whole, must be similar to that of a patient laboring under cardiac disease. Bodily exertion, all excitement, whether produced by psychical or alcoholic agency, etc., in short, whatever increases the cardiac force and the arterial blood pressure, must be avoided; and this must be all the more strictly insisted upon, the greater the development of the aneurism; exception must, however, be made in favor of some few individuals, such as the case of aneurism of the abdominal aorta related by Stokes, where active movement and the use of alcohol relieved the violent neuralgic pains.

Exertion of muscles in the neighborhood of an aneurism are chiefly to be avoided.

The *diet* must be moderately nutritious and suited to the strength of the patient; when general marasmus is commencing, the chief object of treatment must be to delay it by the administration of nutritious food.

An opposite opinion was acted upon by many of the earlier physicians, who prescribed repeated bloodletting and a starvation diet, from the apparently very rational idea that they could thereby diminish the mass of blood and the amount of blood pressure; the slowing of the blood stream and the artificial hydræmia thus produced were believed to favor the coagulation of the contents of the aneurism. In this method of cure, which was recommended by Valsalva and Albertini, and also employed by Morgagni, Chomel, and others, small bloodlettings were made every one, two, or three days, for from six to ten times, or larger bleedings, even to fainting, were employed at longer intervals.

Along with this, rest in bed was insisted upon, and food and drink were given in gradually decreasing quantity, till the patient was reduced to the utmost extremity of weakness. In a few cases a cure is said to have resulted. More recent observations have shown that by this procedure the aneurism is not obliterated, that at the most in a few cases the subjective ailments, dyspnœa, palpitation of the

heart, etc., are diminished, but that marasmus and dropsy only the more rapidly set in. This method of cure has therefore been entirely given up.

Moreover, the experimental physiology of the present day has shown how little influence bloodletting has upon the diminution of the blood pressure, and how rapidly vascular tonicity exerts in such a case its controlling power.

The cure by a restricted diet (six ounces of milk, two ounces of roast meat, six ounces of white bread and butter) and complete bodily rest, recommended by Bellingham, has a less spoliating effect on the frame.

Tufnell relates (in his last edition) nine cases of aortic aneurism (three intrathoracic and six abdominal), in all of which, after an average treatment of a little more than eleven weeks, a marked improvement had taken place, and in some an apparently perfect cure; nevertheless, he has not found many imitators.

Vanzetti [1] cured an aneurism of the carotid artery by a restricted diet and nine months' lying in bed.

The treatment by saline or drastic purgatives, recommended by Hope, is similar in its tendency to the hunger-cure, with which it is sometimes combined; in some cases is said to have been useful.

The objection to all attempts at cure by a restricted diet is, as has already been pointed out by Astley Cooper and Dupuytren, that the irritability of the heart is thereby much increased, and the nutrition of the arterial walls impaired, so that so much injury may be produced in this way as quite to counterbalance any good effected by the diminution of the blood pressure; moreover, by a restricted diet the blood becomes more watery and less capable of coagulating.

Astringent remedies, such as tannin from four and a half to fifteen grains, alum from thirty to sixty grains, acetate of lead from three to thirty grains (!) in the day, have been employed for a long time continuously, partly with the intention of increasing the coagulability of the blood, and partly with the view of inducing contraction of the walls of the sac. The latter action has been expected from the state of the arteries found in many cases of lead poisoning, particularly with acetate of lead.

Dusol and Hoegh have reported cases (four in all) of distinctly projecting and indubitable aneurisms of the arch of the aorta which have been cured. The acetate of lead was given in gradually-increasing doses of from one and a half to nine grains per day. The treatment was continued for many weeks, and by Hoegh it was five times repeated in the course of five years. In all of the cases temporary symptoms of poisoning occurred, so that the remedy had to be stopped for a time.

In a far advanced case related by Legroux, the dyspnœa, the cyanosis, and the distention of the veins in the neighborhood of the aneurism were at least diminished.

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[1] Gaz. des Hôp. 1867, p. 508. No. 128.

Vide also Daly, London Hosp. Rep. III. 1866.

Bamberger and Lebert speak less favorably of the remedy.

Ergotine has also been employed internally, with the view of exciting contraction of the sac.

Iodide of potassium has been recommended by some as a purely empirical remedy (Bouillaud[1]) and has recently been specially praised by Balfour.[2]

B. gives ninety grains (sometimes more) daily for months, and has reported several cures, some of them proved anatomically; thus, amongst others, one of an aneurism two inches in diameter,[3] whilst he has frequently observed arrest of the progress and relief of all the symptoms (possibly the effect of the potash? Q.).

[No other salt of potash has, however, the same effect. Iodide of sodium seemed to have a similar action in a few cases in which it was tried, but as it possessed no special advantage, in particular as it was found to be not a bit less apt to produce hydrœa, it was not persevered with. Since my work on "Diseases of the Heart" was published, only a few months ago, additional experience enables me to confirm the statements therein made, that the effect of iodide of potassium is to produce diminution of the cardiac force and of the blood pressure, and secondarily diminution of the size of the sac and thickening of its walls, in which all the coats present may take part, but which probably mainly affects the adventitia. Any coagulum present seems to be accidental, or at least not dependent on the action of the iodide. For the production of all the phenomena recumbence is not necessary, though it is always a most useful adjuvant, and they are the invariable result of the use of the iodide of potassium in sufficient doses for a sufficient length of time.—*Tr.*[4]]

Moreover, we must never forget that the spontaneous cure of aneurism without any medication has been observed, and in particular that a temporary arrest of progress and remission of all the symptoms very frequently occurs.

From the trifling results as regards the actual cure of aneurism obtained by these various remedies, the relief and palliation of symptoms is in most cases the most important object of treatment.

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[1] Gaz. des Hôp. 16. 1859.

[2] Ed. Med. Jour. 1868. 69. 71.

[3] Vide Berlin klin. Wochenschr. 1871. No. 18. and Ed. Med. Journal, April, 1871, p. 936.

[4] Vide *Keith*, Ed. Med. Jour. 1873, June, p. 1077; and *Mathews*, Am. Jour. Med Scien. Jan. 1875; or Ed. M. J. April, 1875; also Ed. Med. Jour. June, 1876, 954.

In cases where the cardiac action is excited, giving rise to subjective palpitation, etc., perfect rest and a carefully-regulated diet must be prescribed, also mineral acids, digitalis (not often), or an ice-bladder over the cardiac region.

Dyspnœa may at times be relieved by the same remedies, at others by the treatment of a consecutive catarrh; in most cases, however, it is produced by the mechanical pressure of the aneurism, or is of nervous origin. Against this form of dyspnœa, as against the pain, the pulsation in the tumor, etc., we can employ with benefit only narcotics, such as morphia, conium, hydrocyanic acid, exceptionally also alcohol, particularly in abdominal aneurism; occasionally also determination to the skin of the chest, or of the extremities, or to the intestinal mucous membrane, may be resorted to.

Any dropsy present must be treated by regulating the cardiac force, and by diuretics and drastic purgatives.

Should a tumor project externally, this must be protected from friction and pressure by a suitable arrangement of the clothing, or by some covering. Pain, a feeling of tension, or pulsation in the tumor, are to be treated by cold compresses or a bladder of ice; the cold fomentation may be made with a solution of acetate of lead, as its astringent properties may be useful. We must carefully watch that the skin over the aneurism does not inflame or necrose under the influence of the cold and damp; for this reason, therefore, they ought not to be applied for too long a time continuously.

The application of cold can scarcely be reckoned as a direct method of curing an aneurism, since, even should its action extend sufficiently deeply, yet coagulation of the blood is rather delayed than hastened by lowering its temperature. The utmost good effect that could be expected from its use would be in aneurisms of superficial arteries, when the muscular fibres of the artery or possibly of the aneurismal sac itself might be excited to active contraction, and the blood pressure within the aneurism thus reduced.

The application of a few leeches to the tumor or in its neighborhood is said to be useful when the pain is violent (Stokes).

Venesection may be justifiable in rare cases, for the purpose of palliating special symptoms (such as violent dyspnœa, great cardiac excitement, etc.).

The thinner the soft parts covering the aneurism become, the more imminent is its rupture, and the more careful must we be in its treatment; besides the local application of cold, we may employ internally digitalis, the mineral acids, astringents (acetate of lead, chloride of iron, tannin or alum), also narcotics to relieve the violent pain. Perfect rest must of course be maintained.

Should rupture actually occur, we can sometimes arrest the hemorrhage, at least for a time, by the application of light compresses of pinajaremba, or of astringents, over the bleeding part. Should the hemorrhage still go on, or should it occur internally, stimulants alone can be of use.

Aneurism of the abdominal aorta must be treated upon similar principles; but in such cases we must also, as may be readily understood, pay particular attention to the regularity of the bowels and the selection of the diet (in regard to its digestibility, consistence, and temperature).

Narcotics are very frequently necessary in these cases, on account of the violent attacks of pain; moreover, each case must be individualized, as sometimes bladders of ice, at others poultices on the abdomen, uniform pressure, bodily movement, or certain positions of the body, alleviate the pain; tepid baths or a plaster over the region of the stomach may occasionally be useful (in regard to the treatment by compression, *vide antea*).

Aneurisms of the cerebral arteries (when they can be diagnosed) must be treated in accordance with the general principles just laid down. We may especially try the internal use of ergotine, iodide of potassium, or acetate of lead, possibly also the compression or ligature of one of the carotid arteries (a preliminary compression having first been made to ascertain the potency of the collateral circulation through the circle of Willis, which might certainly render the result very problematical). Rupture of such an aneurism must be treated like any other cerebral hemorrhage.

Finally, if we take a comprehensive survey of all the methods recommended for the treatment of aneurism, it seems that of all

the local methods of treatment, next to ligature and compression, galvano-puncture is most deserving of confidence. But in most cases we are restricted to indirect and palliative treatment, and in this category the principal factors are, appropriate nourishment, avoidance of all excitement of the vascular system, the employment of astringents and narcotics, and also the local application of cold.

Narrowing of the Arteries.

Beneke, Ueber die Lumina der arteriellen Gefässe. Sitzungsber. d. Ges. z. Bef. d. ges. Naturwiss. zu Marburg. 1868.— *W. Ruckert*, Ueber die Lumina d. art. Gef. Diss. Marb. 1870.—*Joh. Kimpen*, Ein Beitrag zur Lehre von der Weite der arteriellen Gefässe und deren Beziehung zu einzelnen Krankheitsformen. Diss. Marburg. 1874.—*Morgagni*, Lib. II. Ep. 18. Nos. 2, 4. Ep. 30. A. 12. (refers to narrowing of the arteries as a cause of disease).—*J. F. Meckel*, Observ. d'anat et physiol. cons. une dilatation extraord. du cœur. qui venait de ce que le conduit de l'aorte était trop étroit. Hist. de l'Acad. des Sciences de Berlin. 1750.—*Andral*, Clin. méd. 4 édit. 1836. I. p. 49.—*Fr. Tiedemann*, Von der Verengerung und Verschliessung der Pulsadern in Krankheiten. Heidelberg u. Leipzig. 1843. S. 42, etc.—*Rokitansky*, Patholog. Anatomie. 1856. II. S. 337; or Syd. Soc. Transl. Vol. II. p. 303.—*R. Virchow*, Ueber die Chlorose und die damit zusammenhängenden Anomalien im Gefässapparate. Berlin. 1872. — *Geigel*, Ruptur d. Aorta. Wurzb. med. Zeitschrf. II. 2. S. 107. 1861.— *Wilkinson King*, Cases of contraction of the Aorta. London Med. Gaz. Vol. XXVII.—*F. Riegel*, Ueber regelwidrige Enge des Aortensystems. Berl. klin. Wochenschr. 1872. Nos. 39, 40.— *Jacobs* (Brussels), Presse médicale. XXII. 24. 1870.—*Stoll-Krotowski*, Stenosis Aortæ congenita. Diss. Inaug. Berl. 1873.—*Kulenkampf*, Ueber regelwidr. Enge des Aortensystems. Berl. klin. Wochenschrft. 1873. No. 4.

The contractions of the arteries, like their dilatations, may be conveniently divided into *local* and *diffuse*. The latter occur as contractions of individual arteries or of large arterial areas, or even of the whole arterial system.

In regard to the determination of the fact whether an artery has an abnormally contracted calibre or not, I must refer to the details given in the section treating of the dilatation of the arteries. The relative thickness of the walls must, in these cases also, be duly considered in all measurements which may

be made. In most cases, contraction of the calibre is associated with a corresponding thinness of the arterial coats.

1. Abnormal contraction of the calibre of limited arterial areas is found in all mutilated or deformed limbs, and hence in amputation stumps, in atrophied extremities, in other atrophied organs, whether these are atrophic from original development, or have become so by disease; the contraction certainly is not in all cases proportionate to the atrophy; in many cases the contraction of the vessels may be the cause of the atrophy. Also, beneath the place of ligature or of obliteration of an artery from any other cause, its calibre is unusually diminished up to the nearest collateral branch. These arterial contractions (in all cases where their diagnosis is of consequence) are recognizable by the smallness of their calibre and of their perceptible pulse; also in certain cases by the anæmia, coldness, relaxation, and atrophy of the parts to which these contracted vessels are distributed.

2. All the branches of the arterial system appear contracted in anæmic individuals, in general emaciation the result of acute and chronic diseases, and in cases of permanent diminution of the blood-pressure, such as is associated with aortic stenosis, mitral stenosis, and atrophy of the heart; but it has not yet been accurately determined whether this is a simple contractile adaptation of the vessels to the diminished quantity of blood, or whether, as is probable, atrophy of the arterial coats is also associated with it.

All these cases during life exhibit a uniform diminution in the size of all the arteries which can be felt, and a feebler pulse than normal.

A very remarkable condition, which was also known to the older authors (Morgagni, Meckel, and others), but which has recently received more full consideration from Virchow, is that which he has described as

CONGENITAL UNIFORM STENOSIS OF THE AORTA AND ITS BRANCHES.

In these cases, which most commonly come under observation

in youth or early manhood, the aorta is of infantile dimensions, being no wider than the femoral artery or the little finger (in one of Virchow's cases, a girl aged twenty-four, its circumference at its origin was 2.2, at the commencement of the descending part 1.8, at the beginning of the abdominal portion 1.4, at its division 1.2 ctm.). The arterial wall is in such cases very thin, remarkably extensile, and elastic; this thinning affects all the three coats, but particularly the two internal ones. The emerging arteries, particularly the intercostals, exhibit frequent abnormalities in their origin; on the intima, especially of the abdominal aorta, there are ripple-like and latticed projections; the media and also the intima very frequently exhibit the commencement of fatty degeneration of their cellular elements, and the latter also, in spite of the youthful age, often shows commencing sclerosis. The branches springing from the aorta are all correspondingly reduced in calibre and in the thickness of their walls; the peripheral ramifications are similarly affected (measurements in proof of this are, however, wanting).

Etiology.

Congenital narrowing of the aorta occurs more frequently and in a more extreme degree in women than in men, and especially in those who during life have exhibited the phenomena of chlorosis. Very frequently (Rokitansky), yet by no means constantly (Virchow), there is a simultaneous defective development of the genital organs, an infantile condition of the uterus and the ovaries; a few cases, on the other hand, exhibit an abnormal increase in size of the ovaries and in the abundance of their follicles.

The heart varies very much; at times it is abnormally small, corresponding to the dimensions of the aorta, like the heart of a child; at other times it is enlarged, dilated, and hypertrophied. In the former class of cases we have to do with defective development, a dwarfed growth of the whole of the central vascular apparatus; in the second class of cases it seems as if the aorta alone remained undeveloped, while the heart was normal, and its left ventricle only became dilated and compensatorily hypertro-

phied in endeavoring to overcome the resistance opposed to the blood-stream by the contracted condition of the aorta; the degree and reciprocal relations of the dilatation and hypertrophy may be very various, just as happens when similar conditions arise from other causes. In these cases we may also find, further back in the course of the circulation (without any coincident valvular lesion), other tokens of its obstruction, such as brown induration of the lungs, dilatation and hypertrophy of the right side of the heart, etc.

Symptoms.

In cases of congenital contraction of the aorta the entire development of the body only sometimes suffers, so that the individual affected remains small in stature and infantile in development; frequently, on the other hand, it happens that in an otherwise apparently normally developed individual, there is a remarkable paleness and bloodlessness of the skin and visible parts of the mucous membranes; and with these purely external phenomena there are frequently associated other characteristic symptoms of chlorosis, tendency to syncope, attacks of palpitation, shortness of breath, cardialgia, disturbance of the menstruation, etc.

If we analyze the phenomena individually, we find the following:

The radial artery is usually contracted and the pulse small, but often nothing remarkable is to be observed, and this in itself depends partly upon the varying condition of the heart, while both of these conditions depend upon the quantity of the blood. Should this be small, corresponding with the calibre and capacity of the aortic system, then a normal heart or even one arrested in its growth may be sufficient to maintain the circulation; should, on the contrary, the quantity of blood from better nutrition be greater, and approximate to the relative norm of the body, then, as the result of the increased obstruction, we have a dilatation, and should the nutrition be favorable, also a compensating hypertrophy of the left ventricle occurring, and this all the

sooner the greater the call made upon the heart by corporeal exertion.

Most of these patients have complained during life of a tendency to cardiac palpitation from trifling causes; this tendency never quite disappears, even when compensatory hypertrophy is developed, and it recurs the more markedly the less enduring the compensation thus induced is, and the greater the fatigue or the degeneration of the hypertrophied muscle. We recognize the deviations in the size of the heart by means of palpation and percussion. On auscultation, a systolic murmur is sometimes heard over the aorta, depending upon the vibrations of the thin arterial walls (Bamberger).

Along with the palpitation, and also apart from it, the patient complains of shortness of breath, which depends partly upon the anæmia, and partly on the pulmonary hyperæmia caused by the defective emptying of the left ventricle, which by and by leads to chronic catarrh.

The tendency to syncope, which Laënnec supposed in many cases to depend upon abnormal smallness of the heart, is really partly explicable by this cause, and partly by the obstruction of the flow of blood to the brain.

The attacks of coldness of the extremities with abnormal sensations are also to be referred to the defective blood-current that goes to these parts.

In spite of the great elasticity of the arterial walls, their tenuity is such that lacerations and partial distentions are more readily brought about than usual, especially when the heart is hypertrophied.

Bruberger describes a case of spontaneous rupture of a contracted and thin-walled aorta ascendens; Geigel another, in which there was rupture of an aneurism of the thoracic aorta, all the rest of which was much contracted. This peculiar lacerability seems in many cases to extend into the smaller arteries, and thus to give rise to the morbid phenomena we term hæmophilia; at least Virchow found contraction of the aorta in almost all the cases of bleeders dissected by him.

The very profuse menstruation of many chlorotics, as well as the frequency of gastric ulcer in them, may be connected with

this disposition to hemorrhage. On the other hand, there are many cases of contracted aorta in whom the menses are either deficient or entirely absent, which may depend upon the defective development of the genital apparatus already referred to.

The phenomena just narrated occur in connection with congenital contraction of the aorta in various degrees of frequency and intensity. They may either be entirely absent, or, from their trifling character, they may attract no attention, so that the condition is for the first time accidentally discovered at the dissection of the body; or the patient may exhibit all the symptoms of a more or less well-marked chlorosis, which may disappear on appropriate treatment, but may indeed all the more readily recur the greater the abnormality of the aorta.

A third series of cases first come under the physician's care in the guise of a cardiac affection; such are those cases in which there has been a primary hypertrophy of the left ventricle, in which disturbance of the compensation has resulted from degeneration of the cardiac muscle, and in its turn has given rise to congestion of the lungs, of the right side of the heart, and finally of the systemic veins.

Very frequently indeed it happens, as Virchow has stated, that an actual valvular lesion is developed in individuals who have a contracted aorta, since they seem to have a peculiar disposition to endocarditis—a disposition which he refers partly to the congenital debility of the vascular apparatus, partly to the increased tension of the cardiac walls and of the valves induced by the obstruction. [It seems possible, also, that the reverse of this may occasionally be true, and that congenital hypoplasia of the aortic system may be due to congenital mitral stenosis—vide Balfour, op. cit. p. 149.—*Tr.*]. In such a condition, as well as in ordinary states, the puerperal condition is frequently the cause of the development of endocarditis.

These are all the anatomical and functional disturbances which as yet are known to arise from congenital contraction of the aorta; undoubtedly the number of these will be greatly increased by further observations, especially when a larger number of cases have been compared with one another, and considered in reference to the fact that this condition may very effectually

predispose to certain morbid states. In this investigation due attention must be paid to those less exquisite cases of arterial contraction which constitute the transition to the normal state. Already Virchow has pointed out the frequent occurrence of pulmonary phthisis in chlorotic individuals, and the statistical inquiries of Beneke also exhibit the great probability of a connection between arterial contraction and pulmonary phthisis, as well as with other allied scrophulo-tubercular conditions.

Possibly, in connection with general arterial contraction, the predominant contraction of any individual artery may give rise to a peculiar proclivity to disease in the organ supplied by it.

Diagnosis.

In accordance with what has been already stated, it is in many cases impossible to diagnose congenital contraction of the aorta, and in even the best-marked cases this diagnosis is only hypothetical. Although this condition frequently presents the well-marked phenomena of chlorosis, yet we must not, on the other hand, assume that all cases of chlorosis depend upon a contracted condition of the aorta. It seems certain that under the collective name of chlorosis there are associated various anæmic [spanæmic] conditions, differing from one another in negative phenomena, and depending upon diverse disturbances, the due recognition of which will by and by lead to a subdivision of chlorosis into several varieties; and as one of these subdivisions, we may regard that depending upon contraction of the aorta. This form is apparently distinguished from others, depending upon disturbances of the blood formation, by its greater intensity and by its readily explicable obstinate resistance to treatment.

The occurrence of cardiac hypertrophy in early life, and without any obvious cause, is sufficient to justify the suspicion of contraction of the aorta, especially when chlorosis either exists simultaneously or has been previously present, and if a disposition to cardiac palpitation has preceded the development of hypertrophy.

Contraction and thinness of the walls of the peripheral arteries will strengthen this supposition. Possibly also the form or

the chronological succession of the pulse wave may in the future afford us additional means of diagnosis.

The defective development of the sexual organs is also of importance in regard to the diagnosis.

Prognosis. Course.

The prognosis in congenital contraction of the aorta materially depends upon its degree, and also upon the external conditions of the patient. The more he is exposed to injurious external influences, the more readily—from the diminution of his corporeal resistance—will organic diseases, such as pulmonary phthisis, etc., become developed. The greater the amount of exertion he is compelled to make, so much the earlier may cardiac hypertrophy be developed—first of all, indeed, as compensatory, but presently inducing of itself the secondary dangers of arterial rupture, of aneurism, and of degeneration of the cardiac muscle. Ceteris paribus, therefore, this condition will be best borne by females leading a sedentary life. Most of these patients, however, die early, usually between seventeen and thirty, and generally from implication of the cardiac valves.

Treatment.

When contraction of the aorta is suspected, the treatment must chiefly consist in a regulation of the mode of life : moderation in corporeal movement, avoidance of all violent exertion, and great care in any accidental sickness. Food and remedies should be the same as in chlorosis generally; but we must be prepared to put a stop to too nutritious a diet or too stimulating remedies so soon as the occurrence of cardiac palpitation and other symptoms leads to the suspicion that the blood is so much increased in quantity as to threaten the heart with too great a relative amount of work.

LOCAL CONTRACTION OF THE ARTERIES.—OBLITERATION OF THE ARTERIES.

Fr. Tiedemann, Von der Verengerung und Verschliessung der Pulsadern in Krankheiten. Heidelberg u. Leipzig. 1843.—*Eppinger*, Zusammenstellung von 60 Fällen angeborner Aortenstenose. Prager Vierteljahrschrift. Bd. 112. S. 31–67. —Stenosis of the Aorta at the Depression Corresponding to the Ductus Botalli: *Fr. Erman*, Berlin. klin. Wochenschr. 1873. No. 19.—*M. Purser*, Dublin Journ. of Med. Sc. Dec. 1873.— *W. Redenbacher*, Bair. ärztl. Intelligenzbl. No. 7. 1873.—*Scheele*, Berlin. klin. Wochenschr. 1870. No. 3.—*Degen*, Deutsch. Arch. f. klin. Med. III. S. 614.—*Schrötter*, Wochenbl. d. Zeitschr. d. Ges. d. Aerzte in Wien. 1866. No. 43.—*Lallemand*, Gaz. Hebd. 1866. No. 5. Obliteration of the Aorta abd.—*Kussmaul*, 2 Fälle von spontaner allmählicher Verschliessung grosser Halsarterienstamme. Deutsche Klinik. 50, 51. 1873.—*Immermann*, Strictur beider Hauptäste d. A. pulmonalis in Folge chron. interstit. Pneumonie. D. Archiv. f. klin. Med. V. 1868.—*K. Bettelheim*, Stenose eines Astes d. Pulmonalarterie. Wien. Med. Presse. 1869. No. 42.—*Tommasi*, Case of stenosis of the pulmonary artery (at its bifurcation). Rivista clinica. 1868.—*Ollivier*, Observation pour servir à l'histoire de la clandication intermittente chez l'homme. Gaz. méd. 1872.—*Charcot*, Id. Comptes rendues. 1858.—*Craigie*, Path. anat. Edinb. 1848. p. 110; and Ed. Med. and Surgical Journ. Vol. LVI. p. 427. Instances of obliteration of the aorta beyond the arch. A collection of the ten most authentic cases recorded, up to date (1841), of obliteration of the aorta at what he terms "the definite point," just beyond the ductus arteriosus, etc.

Local contractions or obliterations of the arteries occur:

1. As a congenital condition.

2. As the result of disease of the arterial tunics (arteriosclerosis, acute inflammation with the formation of an abscess, tumefaction of the walls of the artery.)

3. As the result of external pressure (from tumors or cicatrices).

4. From coagulation of the blood (thrombosis and embolism).

As the last-mentioned cause shall be treated of along with similar alterations in the veins in a section specially devoted to it, I shall for the present confine myself to a consideration of those contractions which depend upon the three first-mentioned causes, which are always of gradual occurrence. They may lead to complete obliteration of the arterial lumen.

Coincident with the development of these coarctations, a collateral circulation is equally gradually established, since the

blood pressure above the contracted portion acts with greater force than usual upon the walls of the artery itself and on those of its branches, till both are so dilated that the outflow becomes as great as formerly, and so the uniformity of the blood pressure is restored. When the position of the contracted portion of the artery is withdrawn from observation, the abnormal collateral circulation may afford important diagnostic evidence. Small and perfectly unimportant arteries are found to possess a considerable diameter and to exhibit distinct pulsation; they are often tortuous, and a purring thrill may be both heard and felt in them. Often they show signs of endarteritic disease, no doubt as the result of the abnormally high blood pressure exerted on their walls.

At the contracted portion there is frequently audible a systolic blowing murmur; at other times this blowing murmur is continuous and only increased at each systole; of course all this disappears upon the occurrence of complete obliteration.

Beneath the contracted portion, the pulse is either not to be felt at all, or, at all events, it is much weaker than above it, because the equalization of the systolic increase of the blood pressure in the central artery only takes place gradually through the contracted portion and the collateral vessels; hence the circulation in the peripheral parts, instead of consisting of a series of rhythmic waves, is more uniform in its character.

Hence the characteristic form of the sphygmographic pulse curve is obliterated; the wave is lower, and both rises and falls gradually without any secondary elevations. It is recognized as more than usually delayed, partly because it is actually delayed by its collateral circuit, partly because the rounding of the summit of the pulse wave simulates the appearance of delay.[1]

There are but few arteries of a medium size (3–5 mm. in diameter), the obliteration of which cannot be compensated by the development of a collateral circulation.

The arteries of the spleen and of the kidneys are, however, exceptional in this respect; contraction of the splenic or of one of the renal arteries leads therefore to persistent anæmia and atrophy of the organ implicated, its tissue being gradually

[1] Vide Aneurism, p. 424.

changed into connective tissue. In other parts of the body, such as the extremities, contraction of the main artery may lead to anæmia, coldness, and atrophy, when the development of a collateral circulation is prevented by the diseased condition of the other arteries.

CONTRACTION AND OBLITERATION OF THE AORTA AT THE JUNCTION OF THE DUCTUS BOTALLI (DUCTUS ARTERIOSUS).

This contraction is most usually observed just where the ductus arteriosus joins the descending aorta, that is, a few centimetres below the origin of the left subclavian; more rarely it is met with a little above or below this point; this contraction may extend over a space of 0.5–1 ctm. in length, and may vary greatly in degree up to complete occlusion; in the latter case the portion of the aorta lying above and that lying below are, at the part occluded, only connected by a mere cord of cicatricial tissue. The stricture is usually annular, as if formed by a duplicature of the internal coats of the artery, over which the external coat is continued; in other cases the vascular lumen is contracted on each side like a double cone towards the stricture. Apart from a dilatation of the aorta above the stricture, which is commonly found (being frequently associated with chronic inflammation of the intima), other abnormalities (general contraction, dilatation beneath the constriction) are but rarely seen;—the ductus arteriosus is usually closed.

This condition has hitherto been observed in about sixty cases of all ages [up to ninety years. Craigie—*Tr.*], and more frequently among men than among women. In all probability it originates at a very early period of extra-uterine life in connection with the shrivelling and obliteration of the ductus arteriosus.

Under normal conditions, when the ductus arteriosus is closed, the blood supply of the lower half of the body, which has hitherto been conveyed by it to the descending aorta, must pass by the arch of the aorta, and consequently through the commencement of the descending aorta lying between the left subclavian and the ductus arteriosus, that is, through the connecting link between the fourth and fifth arteries of the primary vas-

cular arches (aorta and ductus arteriosus), and which, up till now, has been narrower than the rest of the aorta. Instead, however, of dilating to the calibre of the rest of the aorta, this so-called isthmus aortæ sometimes remains contracted.

By the shrivelling associated with the obliteration of the ductus arteriosus, an influence is exerted on the isthmus which results in its complete or incomplete occlusion (Rokitansky). It is true, indeed, that this explanation leaves the true cause of the phenomenon as incomprehensible as the physiological closure of the ductus arteriosus itself; and it is specially insufficient for the explanation of those cases in which the ductus arteriosus remains pervious. Still less satisfactory are the explanations of the stenosis by a thrombosis of the ductus arteriosus with extension into the aorta, or by compression of the ductus arteriosus by the left recurrent laryngeal nerve. According to Bochdalek, the obliteration of the artery is more probably brought about by proliferation of the fibre-cells of the media which gradually occlude its lumen. Possibly a local inflammation may have something to do with it; moreover, the stenosis is probably not always brought about in the same manner. [Whatever the cause of this obliteration, and it may vary, Rindfleisch regards it as always occurring during uterine life while the heart is still single. For, as he says, "a contraction of the aorta in opposition to the high pressure of the blood would be contrary to all our experience of the mechanics of aneurism."—Path. Hist. Syd. Soc. ed. Vol. I. p. 297.—*Tr.*]

Along with this abnormality, other congenital malformations are not unfrequently found, such as supernumerary or incomplete semi-lunar valves, deficiency of the cardiac septum, stenosis of the conus arteriosus from myocarditis, cleft palate, etc.

Stenosis of the aorta leads, in the first place, to an increase of the amount and pressure of the blood in the arch of the aorta and its branches, which is usually neutralized by means of a collateral circulation leading to the lower half of the body. This occurs through the following channels:

1. Through the subclavian, internal mammary, superior and inferior epigastric arteries into the lumbar and femoral arteries.

2. By anastomoses between the first intercostal, the internal mammary, the dorsalis scapulæ, the subscapularis, and the external thoracic arteries, on the one hand, and the intercostal arteries, on the other.

The arteries named are found to be correspondingly dilated and with thicker walls than usual.

Should the collateral circulation be insufficient for the out-

flow of the blood, then the increased pressure produces dilatation of the arch of the aorta and its branches, and secondary dilatation and compensating hypertrophy of the left ventricle.

Symptoms.

Stenosis of the aorta in the region of the ductus arteriosus may run its course wholly without symptoms, and may be only accidentally discovered at the dissection of the body; this happens when it is moderate and its effects upon the circulation are compensated by the development of collateral routes and by cardiac hypertrophy. But even in such cases the diagnosis may be made by an objective examination, especially by the discovery of the dilatation of the above-named collateral arteries; of these the dorsales scapulæ, the epigastric and the mammary arteries may be both seen and felt as thick, often tortuous, pulsating cords. A purring murmur often originates in these vessels, and may be both felt with the finger and heard on auscultation. Over the thorax this murmur is heard loudest over the intercostal arteries at the first and second dorsal vertebræ, also over the mammary arteries at the edges of the sternum, and it may be distinguished from a cardiac murmur by its postponement relative to the cardiac impulse.

In all the arteries coming off below the stenosis (provided they are not implicated in the collateral circulation), the pulse is feeble, slow, and delayed,[1] or it may be entirely absent, since the pulse wave through the contracted portion is enfeebled and is propagated circuitously by collateral routes (by these alone when the occlusion is complete), and the blood stream is thus rendered more uniform (less pulsatory).

This change of the pulse in regard to time and strength, which is specially remarkable in the femoral arteries, is decidedly the most important sign of this stenosis, since collateral dilatation in some cases cannot be discovered even when carefully sought for.

Sometimes, at the back, over the site of the stenosis (the left

[1] Vide *Scheele*, Berl. klin. Wochenschr. 1870.

side of the fourth dorsal vertebra), and downwards from that point, a systolic murmur may be heard.

The dilated arch of the aorta may often be felt in the tracheal fossa, and the existence of hypertrophy of the left ventricle, in the absence of other causes, must be regarded as of diagnostic importance.

Should the stenosis be considerable, and the dilatation of the collateral arteries insufficient, or, should the hypertrophied heart be paralyzed, then the same symptoms occur as in chronic cardiac diseases: palpitation, dyspnœa, bronchial catarrh, dilatation of the right side of the heart, and all the phenomena of remora in the systemic veins. This disturbance of the compensation often depends upon the degeneration of the hypertrophied cardiac muscle; at other times the aortic or mitral valves become diseased from the abnormally high pressure to which they are exposed.

In a few cases, rupture of the aorta occurred, in others sudden death from cardiac paralysis.

Prognosis.

Those affected with stenosis of the aorta may reach a very advanced age, and may die from some intercurrent disease. When, however, this condition has been recognized, when, therefore, for the most part, the subjective signs of circulatory disturbance of any kind must have been present, the patients have generally died in a few months or years from dropsy, or suddenly from rupture of the aorta, or paralysis of the heart.

Treatment.

When this condition has been early recognized, the cardiac complications which threaten life may be prevented or postponed by leading a quiet life, free from violent exertion. In other respects the treatment must be similar to that for cardiac disease generally.

NARROWING OF THE AORTA,

In its ascending portion from external pressure, is of rare occurrence, since tumors in the mediastinum, as well as aneurisms, are more apt to embrace the circumference of the vessel than to compress it.

Narrowing of its calibre by an adherent thrombus is in this situation usually unimportant, and presents no other symptoms than those of endarteritis generally.

Cohn has observed sudden obstruction of the ascending aorta by a clot; in one case, perforation of a carcinoma cordis into the left ventricle; in another case, rupture of a myocarditic abscess gave rise to the formation of adherent thrombi, which, when suddenly broken off, obstructed the aorta; the patients suddenly became pale, fell collapsed, and died instantaneously; the left heart was found to be greatly dilated.

Narrowing of the descending aorta, both of its thoracic and its abdominal portions, is occasionally of congenital occurrence, and leads to precisely similar symptoms as those of contraction at the ductus Botalli; in forming the collateral circulation, all the intercostal arteries above the constriction convey the blood from the spinal column towards the mammary arteries; all those beneath it transmit the blood in the contrary direction.

The descending aorta is but rarely narrowed by any tumor, because of the amount of blood pressure present within it throughout its course.

This narrowing may, however, be brought about by aneurism of the aorta itself, or by a carcinomatous mass (Velpeau). In one case related by Axenfeld[1] the aorta of a kyphotic patient was narrowed by being bent.

Obstruction by an embolus or autochthonous coagulum occasions a sudden or more gradual diminution of the amount of blood in the lower half of the body, and thus produces paraplegia or paresis, a feeling of numbness and pain, as well as an absence of the pulse and a lowering of the temperature in the same.

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[1] Bull. de la Soc. anatom. 1850.

Sometimes these phenomena are better marked in one extremity than in the other, when the coagulum, situated at the place of bifurcation, obstructs the entrance of one iliac artery more than the other. When a collateral circulation is set up, all the phenomena of obstruction may gradually disappear; at other times complete obstruction is followed by swelling, hemorrhage and gangrene in the lower half of the body.

The phenomena attending the narrowing or obstruction of one common iliac are very similar to those just described, only of course limited to one side. When the collateral circulation is in such cases only imperfectly developed, we sometimes have developed the peculiar phenomenon of intermittent lameness, as has been observed by Charcot (case of obliteration by aneurism) and Ollivier (case of obliteration from an unknown cause, probably lues). In these patients there occurred occasionally, sometimes spontaneously, but always after long walking, a sensation of numbness and formication in the whole leg, also a cramped feeling of stiffness combined with weakness. The functions of the sensible and motor nerves seemed to suffer from the anæmia. One of the cases resulted in permanent paresis, the other in atrophy of the leg.

Narrowing of the innominate, the subclavian, and carotid arteries at their origin, is by no means rare in arterio-sclerosis, and is manifested by smallness and delay or by failure of the pulse in the arm, or in the corresponding side of the head, more rarely by other functional disturbances, such as diminished temperature and slight numbness of the affected extremity, because even in the case of complete obstruction a collateral circulation is readily set up (by the vertebral, mammary, and inferior thyroid arteries). The subclavian arteries may also be narrowed or obstructed by a coagulum, an aneurism of the arch, or a tumor of the mediastinum or of the supra-clavicular lymphatic glands.

Similar causes may produce narrowing or obstruction of the carotids, which in many cases causes no uneasiness whatever, but in others induces disturbance of the cerebral circulation, giddiness, convulsions, and paralysis. According to two cases observed by Kussmaul,[1] this seems to depend upon defective development of the arteriæ communicantes of the circle of Willis. The author observed one case of obstruction of the left carotid with absence of pulsation from all its branches in a case of right hemiplegia, probably due to softening, but in it there was no post-mortem examination.

In a case of obliteration, occurring at the origin of the superior mesenteric artery (in a man aged sixty), Tiedemann saw the collateral circulation carried out by the hepatic and the pancreatico-duodenal arteries. In a case related by Chiene[2] of an aneurism of the abdominal aorta, the cœliac axis and both the mesenteric arteries

[1] Deutsche Klinik. 1872. Nos. 50, 51.

[2] Jour. of Anat. and Physiol. 1868. Nov.

were obliterated; the collateral circulation was carried on by:—1. The phrenic and intercostal arteries on the left side. 2. The colic, renal, and supra-renal arteries on the left side. 3. The internal hemorrhoidal and the internal iliac arteries.

NARROWING AND OCCLUSION OF THE PULMONARY ARTERY IN ITS TRUNK OR MAIN BRANCHES.

(We here leave entirely unconsidered the very frequent, and often manifold, occlusion of the smaller branches of the pulmonary artery by thrombi.)

Narrowing of the trunk or of one of the main branches of the pulmonary artery is of rare occurrence, as already narrated; endarteritis is a very unfrequent event in the pulmonary artery; narrowing of it, therefore, by an abnormal growth of the intima is quite exceptional.

(Case of Tommasi: stenosis of the pulmonary artery at its bifurcation from cartilaginous thickening and calcification of the intima.)

Aneurisms of the ascending aorta or of its arch, and also enlarged mediastinal or bronchial lymphatic glands may compress and narrow the pulmonary artery; and this may also be brought about by cicatricial shrivelling of a patch of lung or of the bronchial glands (Immermann).

During life, narrowing of the trunk of the pulmonary artery gives rise to phenomena very similar to those produced by stenosis of its orifice in the region of the valves: to anæmia of the entire area of the pulmonary capillaries, and, as the result of that, to persistent dyspnœa with occasional exacerbations; to congestion, dilatation, and hypertrophy of the right heart, and as the consequence of this to cardiac palpitations and ultimately to general congestion of the systemic veins. The enlargement of the right heart is revealed by increased dulness and increased force of the cardiac impulse. The commencement of the pulmonary artery quite up to the beginning of the narrowed part may share in this dilatation the result of increased pressure (it may even as a consequence of this become secondarily affected with endarteritis); the second sound over the pulmonary artery is thus rendered abnormally loud and accentuated, while, when the

orifice itself is stenosed, the whole of the pulmonary artery is diminished in calibre, and the second sound is abnormally weak. In these cases a systolic murmur produced by the stenosis is also audible, with its position of maximum intensity not therefore over the pulmonary valves, but above them (in Immermann's case, indeed, it was loudest in the second intercostal space on the *right* side); this may be propagated upwards to the neck, or it may be heard in the interscapular space close to the spinal column—when one of the main branches is more contracted than the other, it is probably most distinct on the side to which that is distributed.

When the commencement of the pulmonary artery is dilated, its pulsation may sometimes be both seen and felt in the second intercostal space on the left side.

It is important for the diagnosis to determine, besides the foregoing phenomena, the existence of glandular swellings, of an aneurism, or of pulmonary contraction (Immerman's case was one of stone-hewer's phthisis).

The prognosis is unfavorable, and treatment of little use; it must be similar to that of stenosis of the pulmonary orifice.

Rupture and Perforation of the Arteries. Dissecting Aneurism.

RUPTURE: *Morgagni*, Epist. LIII. 35 and 36.—*H. Schnabel*, Günsb. Zeitschr. X. 1859. S. 421. Schmidt's Jahrb. 110. S. 243. Traumatic rupture of the healthy aorta.—*Ollivier*, Dict. en XXX. Vol. XXVI. 305. Rupture of the pulmonary artery.—*Wallmann*, Ueber Ruptur d. innern und mittlern Arterienhaut. Oesterreich. Zeitschr. für pract. Heilk. IV. 6, 7. 1858. Schmidt's Jahrb. 99. S. 230.

DISSECTING ANEURISM: *Laënnec*, Traité de l'auscultation. 4. édit. 1837. III. p. 420.—*Peacock*, Edin. Med. and Surg. Jour. 1843, April and October. Edin. and London Monthly Jour. 1847, Nov. Experiments.—*Peacock*, Report on cases of dissecting aneurisms (eighty cases). Pathological Transactions. Vol. XIV. 1862-63.—*Peacock*, Ibid. XVII. 1867. p. 50.—*Rokitansky*, Krankheiten d. Arterien. S. 41.—*Do.*, Oesterreich. med. Jahrb. Bd. 16. 1837.—*Do.*, Wochenblatt d. k. k. Ges. d. Aerzte zu Wien. 1866. No. 28.—*Crisp*, L. c. pp. 178 and 310.—CASES: *O. Barth*, Arch. d. Heilk. XII. S. 253. 1871.—*Chauvel*, Gaz. méd. de Paris. 1866. No. 16.—*G. Fischer*, Diss. Würzburg. 1872 (two cases).—*H. Fagge*, Med. Chir. Transact. Vol. 52. 1869.—*F. E. Geissler*, Ueber die als Aneurysma dissecans bekannte Ruptur der Aorta. Würzb. Inaugural. Diss. Bremen. 1862 (eighty-

five cases).—*Heschl*, An. Diss. der Brust- und Bauchaorta mit Compression des Aortenlumens. Wien. med. Woch. No. 90. 1867.—*Mandron*, Thèse de Paris. 1866.—*O. Wyss*, Arch. der Heilkunde. 1869. X. S. 490.

Rupture of the arterial continuity may be occasioned by :

1. *Wounds.* In peripheral arteries these are most frequently caused by stabbing or cutting instruments; but in the aorta this is of rarer occurrence.

A preparation in the pathologico-anatomical collection at Berne exhibits a wound of the abdominal aorta by a dagger-knife, which had gone through one of the vertebræ.

Injuries to the arch and to the aorta descendens by a set of artificial incisors and by a piece of wood, which had stuck in the œsophagus, have been recorded by James Duncan and Kreyser.[1]

Wounds of the intima of the smaller arteries (at the base of the brain and in the mesentery), seem to be occasionally produced by embolic calcareous plates coming from the cardiac valves or the intima of the aorta, and lead to the formation of aneurisms (Ponfick).

The coats of the arteries are capable of being ruptured by direct violence (contusion or laceration), just as they also are by violent concussion of the whole body, by a fall from a great height, etc. The intima seems to be thus singularly easily ruptured, as we are taught by the results of ligature of the arteries.

The experience of Wallmann has proved that a violent blow from a blunt weapon ruptures during life as well as after death, when the arteries are tense and full—first of all the intima, next the media, and with greatest difficulty the adventitia.

In this way the great arteries of the extremities and even the aorta itself may be ruptured. Thus Morgagni saw the aorta descendens torn across by a blow from a stick on the back.

2. Rupture is favored by a certain degree of *tension* of the arterial coats ; therefore the filled arteries of the living body are more readily torn than the empty ones of the cadaver.

Tension of the arterial coats from the blood-pressure alone may lead to rupture, especially where these coats are degener-

[1] Vide *Hugues*, Lyon médical. 1870. No. 17. Vide also Northern Jour. of Med. 1844. p. 15.

ated in any way, and the blood-pressure is increased by hypertrophy of the left ventricle and by increased force of the cardiac action.

3. *Disease of the arterial coats* is of the greatest importance in regard to the production of rupture. Suppuration or carcinoma only rarely implicates an artery *ab externo.*

Ogle saw ulceration of the abdominal aorta as the result of caries of two lumbar vertebræ.[1] Bucquoy and Lancereaux[2] observed perforation of the aorta descendens produced by epithelial carcinoma of the œsophagus. The aorta was ulcerated, but not carcinomatous; vomiting of blood occurred.

Rokitansky and others have seen ulceration of the femoral artery the result of the suppuration of a bubo; Crowfoot, ulceration of the pulmonary artery from an abscess of the lung.[3]

On the other hand, the arterial coats are not unfrequently destroyed by the action of the gastric juice; this happens most frequently to the gastric artery itself, but the splenic artery and others may be opened into by a gastric ulcer.

Stich[4] has recorded the opening of the (atheromatous) abdominal aorta by an ulcer in the lower transverse portion of the duodenum. The old woman died eighteen days after the commencement of the hæmatemesis.

Abnormal thinness of the walls, either congenital or the gradual result of dilatation above a contracted part (as, for instance, at the isthmus aortæ), predisposes to rupture, whether that be finally produced by violence or occur "spontaneously" from the blood pressure.

Fatty degeneration of both the internal coats frequently, but chronic endarteritis more frequently still, gives occasion to rupture of the arteries, insomuch as they lead to ulceration of the intima, to brittleness of the intima and media, as well as to thinning of the arterial coats by atrophy and dilatation.

With arteries so predisposed, violence or increase of the blood pressure very readily produces rupture.

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[1] St. George's Hosp. Rep. 1867. II. p. 375.

[2] Bull. de la Soc. anatomique. 1855 and 1861.

[3] Medico-chir. Transactions. XXVI. p. 154.

[4] D. Arch. f. klin. Med. 1874. XIII. S. 191.

Should the rupture be complete and embrace all the three coats, then the blood escapes either into an adjoining cavity, the pericardium, pleura, etc., or into the surrounding cellular tissue, within which it both forms and fills a hollow, larger or smaller, according to the amount of resistance present.

In many cases the rupture at first is only incomplete, generally only implicating the intima; by and by, under the influence of the blood pressure, the external coats stretch and at last give way; should this occur very gradually, then an aneurismal sac is formed by condensation of the surrounding connective tissue; should it happen after hours only or days, then the results are the same as in simple rupture.

Sometimes the blood forces its way through the rupture not only outwards, but also between the arterial coats, so that the adventitia is separated from the media by a blood sac, forming a dissecting aneurism. At other times the blood forces its way between the layers of the media, so that the external wall of the sac is formed by the adventitia plus one of the layers of the media.

According to Peacock, the latter is the rule, since he could only produce dissecting aneurisms artificially in the dead body by injecting water, when the media had been imperfectly cut through; when it had been completely divided, the only result was diffuse infiltration of the neighboring tissue; a true sac is, however, always produced when, as so often happens in arterio-sclerosis, the adventitia is condensed. Vide *supra*, the experiments on animals in regard to the artificial production of aneurism, p. 410.

The blood sac lying between the arterial coats is often of considerable size, embraces a large part of the arterial circumference, and extends for some distance along it, as far, for instance, in the aorta as to its bifurcation. Finally, the rupture may become complete by the giving way of the adventitia, or a second communication with the arterial lumen may be formed by the rupture of the inner layer of the sac. In certain rare but well established cases a cure has resulted by the formation of coagula on the inner surface of the adventitia, and the thickening of the latter coat.

In one case of dissecting aneurism of the aorta, described by

Helmstedter,[1] in which the media was for a considerable extent divided into two layers, the inner surface of the sac, both on its central and peripheral sides, was covered by a layer of connective tissue of recent formation, by which the origins of the intercostal arteries were partly narrowed, and partly occluded.

The rupture is usually transverse (especially in the ascending aorta), sometimes oblique or parallel with the axis of the vessel. Its length at the most is two-thirds of the arterial circumference; usually it is less (Peacock).

Spontaneous rupture of the arteries (inclusive of dissecting aneurisms), like chronic endarteritis, occurs most commonly in advanced life, and most frequently in the aorta, particularly in its ascending portion; rupture takes place most frequently into the pericardium. Rupture of the pulmonary artery has been occasionally seen, but dissecting aneurism never.

Symptoms.

In rupture of the aorta, as in rupture of the heart, death is preceded by a feeling of anxiety, pallor, and loss of consciousness, when the rupture is large and the blood escapes freely into any cavity (pericardium, pleura, or peritoneum); when the opening is small and the bleeding takes place into the mediastinal or retro-peritoneal connective tissue, the phenomena of internal hemorrhage do not occur with such lightning-like rapidity; the effused blood may be recognized by percussion, and by its pressure may injuriously affect the cardiac force and the respiration; may occasion violent pain, and by compression of arteries or nerves may give rise to local disturbances of the circulation, motility, or sensibility in one of the extremities. Disturbance of the cerebral and renal functions has also been observed as the result of obstruction to the blood-flow thus produced (Peacock). The patients have themselves sometimes had a sensation as of something giving way internally.

Such a slow progress is most apt to be observed in cases of dissecting aneurism, and in them death may follow in a few days,

[1] Diss. Strassburg. 1873.

complete rupture, or, in spite of the threatening symptoms, a cure may occur in certain rare cases, or finally the symptoms may ultimately come to resemble those of a simple aneurism. A dissecting aneurism of one of the main branches of the aorta may be sometimes recognized by palpation, from the consequent change in the form of the artery.

The treatment is the same as that of rupture of an aneurism.

A case narrated by Verneuil is very remarkable,[1] in which, after being run over by a railway wagon, the patient had both the internal coats of the left internal carotid torn across (apparently by violent twisting of the head). Thrombosis of the carotid as far as the middle cerebral artery, softening of the temporal lobe, and paralysis of the right side resulted; death occurred in five days.

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[1] Bull. de l'Acad. de Méd. 1872. No. 2, p. 46.

DISEASES OF THE VEINS.

Literature of the subject generally and literature of phlebitis.

Vide the handbooks of pathological anatomy by *Cruveilhier*, *Förster*, *Rokitansky*, and others; and the handbooks of special pathology and of diseases of the heart, by *Bamberger*, *Bouillaud*, *Corvisart*, *Duchek*, *Friedreich*, *Hope*, *Jaccoud*.

Bollinger, Endophlebitis verrucosa of the portal vein of the horse. Virch. Arch. Bd. 55. S. 279. 1872.—*Bouillaud*, Arch. gén. 1824. Dec. 1833. June.—*Crisp*, Treatise on diseases of the bloodvessels. 1847.—*Duchek*, Krankheiten der Venen. Wiener. Allg. med. Zeitg. 1863.—*Do.*, Prager Vierteljahrschrift. Bd. 41. S. 109. —*John Hunter*, Transact. of the Soc. for the Improvement of Med. and Chir. Knowledge. 1793. Vol. I. p. 18.—*Lee*, Inflammation of the pulmonary veins. Med. Chir. Transact. XIX. 44.—*Meinel*, Archiv f. physiol. Heilkunde. 1848.—*Paget*, On gouty and some other forms of phlebitis. Bartholomew's Hosp. Rep. 1866. II. p. 82.—*Do.*, Clinical lectures and essays. 1875. p. 292.—*Tuckwell*, Bartholomew's Hosp. Rep. 1874. Vol. X. On clotting of blood in gout and chlorosis.— *Pelvet*, Phlébite rhumatismale. Gaz. des Hôp. 1866. No. 28.—*Puchelt*, Das Venensystem. II. Aufl. Heidelberg. 1843. *Stannius*. Ueber Verschliessung grosser Venenstamme. Berlin. 1839.—*Velpeau*, De la phlébite. Paris. 1829.—*Virchow*, Arch. Bd. I. Spec. Pathol. Bd. I.—*C. O. Weber*, Billroth, Pitha's. Handb. d. Chir. II. 2. p. 97.—*Zahn*, Ueber Thrombose. Virch. Arch. 1875.

Phlebitis.

1. *Acute Phlebitis.*

Acute phlebitis generally begins in the adventitia with redness and swelling of it, as well as of the surrounding connective tissue; sometimes there is only serous infiltration, at others a denser swelling with cell proliferation. The media and intima may also be implicated in these changes, and may become cloudy and thickened. By and by there occurs either complete absorption of the exudation, or it may become organized with a new growth of connective tissue in the external coats and the adjacent

tissue (chronic, sclerosing phlebitis), or the exudation may soften with a more or less copious formation of pus, the normal elements of the venous coats becoming granular and breaking up into a fatty detritus. In the former case the intima is more rarely, in the latter more frequently, altered, either thickened and plicated, or projected like a pustule by the solid or fluid exudation in the media; or it may partly fall into molecular detritus, and partly disappear by ulcerative absorption due to the pressure of accumulated pus.

Very early in the disease a blood coagulum may form on the diseased part of the venous wall—so soon, namely, as the endothelium of the intima has become altered in the slightest degree from its normal (Zahn); coagulation occurs therefore more readily in the destructive than in the sclerosing form of inflammation. According to the nature and intensity of this inflammation, according to the size of the vein affected and the force of its blood current, this coagulum either remains adherent to the wall or obliterates the lumen of the vessel and undergoes further changes, which lead either to its organization or to softening, and which vary in character with the nature of the phlebitis. When the intima is destroyed, it depends upon the extent and firmness of the coagulum whether the contents of the inflamed part are at once let loose, or whether they are gradually mingled with the blood current and are swept away by it.

Though the phlebitis just described is primary in its occurrence, and the thrombosis a secondary and by no means necessary result, yet, on the other hand, we frequently meet with precisely a reversed condition: a primary thrombosis and a secondary phlebitis, which is then primarily associated with important changes of the intima, and will be particularly described under the heading of "Thrombosis."

From the thinness of the venous coats, we cannot readily subdivide phlebitis, as we do arteritis, into inflammation of the external, middle, or internal coats. The predominant importance of the individual coats in the various forms of venous inflammation may be gathered from what has been already said.

The most common cause of phlebitis is inflammation in the neighborhood of the veins: suppuration of the cellular tissue (as

the result of pelvic, intermuscular, or subcutaneous cellulitis) following bruises, fractures of the bones, or operations; or direct injury to the coats of the veins by bruises, lacerations, ligature, or wounds from cutting-instruments, as in venesection.

Further, primary thrombosis of a vein, as already said, frequently leads to inflammation of its coats. Previous dilatation, varicosities, or chronic inflammation of the coats of a vein dispose to acute phlebitis.

(Primary) phlebitis frequently occurs without any apparent local cause; *e. g.*, in pyæmia, or in gout. In such cases it is frequently associated with other gouty inflammations, especially in the lower extremities, according to Paget. The same author further describes cases of widely diffused phlebitis, which he regards as having been produced by putrid infection from sewer gases.

Pelvet has an idea that there is an acute specific rheumatic phlebitis, analogous to the endocarditis which accompanies acute rheumatic affections of the joints.

The *symptoms* of acute phlebitis are in themselves of a very indefinite character; they resemble on the one hand the symptoms of inflammation of the parts in the neighborhood of the vein, and on the other those of thrombosis, presently to be referred to.

According to the extent of the inflammation of the surrounding connective tissue, there is either only a uniform diffuse infiltration to be felt, or the periphlebitic thickening of the tissues runs along the inflamed vein, and is felt like a cord, provided the vein is superficial; at the same time the vein is painful on pressure or movement. Thrombosis of a vein can only be recognized by palpation in trifling degrees of inflammation of its coats, at other times it is revealed by consecutive disturbances of the circulation (venous congestion, œdema, lowering of the temperature).

Fever may be present or not. The former is the case when the inflammation of the surrounding tissues is somewhat extensive, or when the inflammation has led to destruction of the coats of the vein and to the entrance of the resulting detritus into the circulation; while, on the other hand, a limited phlebitis, or one tending to organization, whether the accompanying

thrombosis has been primary or secondary, runs its course without any or with but little fever.

The entrance of pus or of the detritus arising from tissue destruction into the circulation is usually followed by metastatic inflammations, particularly in the lungs.

Treatment.—In every case in which suppuration occurs in the neighborhood of a vein, the prevention of phlebitis is a primary and most important object, and this necessitates the most careful and painstaking treatment of the parts primarily diseased, the opportune opening of abscesses, removal of foreign bodies, etc. Should the inflammation approximate a vein, or should it be necessary to wound a vein in any operation (even only in venesection), then we must still more carefully endeavor to prevent the entrance from without of any infective material, because the entrance of such into a vein directly, or by means of the breaking down of a thrombus, leads to the danger of general infection. On the other hand, a benign phlebitis, which leads to thickening of the coats and the formation of an organizable thrombus, is a process often necessary for the healing of a wound, and has a tendency to prevent the occurrence of infection.

The detraction of blood by leeches applied along the course of the inflamed vein, as well as the application of cold, are in most cases unnecessary, and are only indicated by the occurrence of a considerable amount of pain and swelling. On the other hand, poultices tend to relieve both pain and tension and to favor absorption. The envelopment of the part affected in cotton wool, with or without repeated frictions with mercurial ointment, is recommended by some.

Under all circumstances, the part affected must be so placed as to secure the fullest possible rest, to prevent tension or pressure on the inflamed vein, and to promote the return of the blood from the peripheral parts by means of the collateral circulation and the influence of gravity.

2. *Chronic Phlebitis.*

Chronic phlebitis occurs either as the result of an acute in-

flammation, or in cases of permanent dilatation (from congestion, the influence of collateral flow, varicosity, etc.), or in cases of primary thrombosis. It leads to thickening of the coats, especially of the external layers, whilst the intima remains intact. In the parts affected there is proliferation of the nuclei and hyperplasia of the connective tissue; in the media there is also hypertrophy of the muscular coat; the vasa vasorum are also strongly developed as far as the media, and often so dilated that in the coats of the vein there are venous sinuses of the dimensions of cuticular veins. The intima is, however, unaltered, and its epithelium uninjured.

In opposition to this description of varicose veins (from the investigations of Soboroff and others), Cornil states, as the result of his own independent examination of varicose veins, that the new formation of the connective tissue chiefly takes place between the media and intima; in other respects his observations agree with those just stated.

Secondly, we may have a chronic endophlebitis, analogous to endarteritis, in those veins which have been exposed to great pressure, as in the pulmonary veins in disease of the mitral valves, in the large systemic venous trunks in cases of obstruction to the emptying of the right ventricle, or in peripheral veins in aneurism by anastomosis.

The anatomical alterations in endophlebitis are analogous to those of endarteritis, and like these result in thickening, calcification, rarely in fatty degeneration and atheromatous destruction of the intima, and are secondarily accompanied by chronic inflammation of the external coats, or by calcification of these. In it the intima is beset at times with isolated plates, and at others it presents an extensive diffuse thickening, so that the inner coat of the vein presents an appearance like that of an artery.

Clinical symptoms are absent in most cases of chronic phlebitis. After a preceding attack of acute phlebitis, the persistence of the disease in a chronic form may be suspected, from the continuance of congestion and dilatation; but we can only ascertain this certainly in superficial veins, in which, besides a

uniform or varicose dilatation, we can very often feel a thickening or calcification of the walls.

Most commonly attention is first directed to the existence of this affection, by the fact that chronically inflamed veins are specially predisposed to thrombosis and to acute phlebitis.

The treatment must be directed to the prevention of such complications, as well as of the further progress of the chronic inflammation, by warding off every source of injury or of external irritation. The cause of the venous congestion must therefore, as much as possible, be removed, and the part affected must be so placed as to favor the return of the venous blood by means of gravity.

The endophlebitis verrucosa, observed by Bollinger in the portal vein of horses, possesses an anatomical interest. In these cases, the internal surface of the portal system (apparently as the result of sclerosis of the perivascular connective tissue) exhibits, over several square centimetres, a velvety appearance produced by minute warty elevations of the intima, which contain, besides almost homogeneous connective tissue, connective-tissue cells, and round cells, but no vessels.

Hypertrophy, Atrophy, Degeneration, Neoplasms of the Venous Coats.

Pure *hypertrophy*, with a uniform thickening of the coats, with dilatation, elongation, and tortuosity of the venous tube, occurs in veins which have been long engaged in carrying on a collateral circulation. Further, hypertrophy of the venous coats, as just described, occurs in connection with chronic inflammation, and as one of its results, especially when accompanied by persistent increase of the pressure of the venous blood.

Similar conditions may lead to *atrophy* of the walls, when combined with considerable stretching and dilatation of the venous tube. Persistent external pressure, persistent diminution or cutting off of the blood stream from a vein (as, for example, in atrophying organs), leads also to atrophy of its walls. The venous coats are less subject to degeneration in general than the

arterial coats; thus *fatty degeneration* is somewhat rare, but *calcification* is more common. By it, plates or rings are formed, which are commonly imbedded in considerable numbers in the venous walls, especially in dilated veins, in the inferior extremities. Calcification of blood clots (phleboliths) also frequently occurs in these situations.

In extensive *amyloid degeneration* the large systemic venous trunks, and also the portal vein, are often affected, sometimes throughout the entire thickness of their coats, at others only the media or the vasa nutrientia.

Of these degenerations we can only recognize—and only in the superficial veins—hypertrophy, atrophy, and calcification, which alter the resistance of the venous tube, in relation to the finger palpating it, as well as its extensibility in regard to central compression. Atrophy favors rupture from violence or sudden increase of the blood pressure.

Carcinoma affects the veins almost always only secondarily, spreading from the neighborhood, but attacking them more frequently than the arteries, on account of the thinness of their walls. Sometimes the carcinoma breaks through into the lumen of the vein, and grows there, producing thrombosis; sometimes coagulation of the blood occurs previous to the breaking in of the carcinomatous growth. In this way both large and small venous trunks may be affected by carcinoma. Sometimes the carcinoma grows forwards cylindrically within the venous lumen, as in the portal vein. Should particles of the cancerous mass be swept away by the blood stream, they may, as emboli, give rise to secondary cancers (in the lungs or liver).

The results and symptoms during life are the same as those in simple thrombosis of the veins.

Perl and Virchow[1] have described a primary sarcoma of the vena cava inferior in a woman aged thirty-four. It was a myosarcoma with giant cells, and formed several tumors, the size of the fist, in the space between the kidneys and the lower edge of the liver. The vena cava was surrounded by the growth, and, as well as the renal veins, was almost completely filled by it; the aorta was compressed.

[1] Virch. Arch. Bd. 52. 1871.

Syphilitic Disease of the Veins.

Oedmansson, Cases of syphilitic disease. Nord. med. Ark. I. 4. (Virch. Jahresber. 1869. II. S. 561.)—*Schüppel*, Ueber Periphlebitis syphil. bei Neugebornen. Arch. d. Heilk. XI. S. 74. 1870.—*Heubner*, S. 169.—*Birch-Hirschfeld*, Beitr. z. pathol. Anat. d. hereditären Syphilis Neugeborner. Arch. d. Heilk. 1875. XVI. S. 166. —*Winckel*, Ber. u. Studien aus dem kgl. sächs. Entbindungsinstitut. 1875. S. 307.

A circumscribed disease of the intima, similar to that described by Heubner in the arteries, especially those of the brain (*vide supra*), has been observed by Anderson and Birch-Hirschfeld in the umbilical vein within the cord, and has been by them connected with congenital syphilis. But, indeed, in some of these cases no other more certain proof of the existence of syphilis could be ascertained. Oedmansson regards this affection of the intima of the umbilical vein, even in actual syphilitics, as still belonging to atherosis. This disease is in so far of considerable importance that the stenosis of the umbilical vein, thus produced, is, according to Oedmansson and Winckel, a frequent cause of the intra-uterine death of the fœtus in syphilitic women.

In new-born children, Schüppel has also observed a peculiar alteration of the portal vein at its entrance into the liver, produced by syphilis.

In these cases the vein was changed into a thick dense cord with a much contracted lumen; its coats consisted of a central grayish yellow opaque zone, and a peripheral one, gray and more transparent, which gradually passed without any definite boundary into the surrounding tissue; a microscopic examination exhibited a fibrous tissue, finely granular masses, and many partially atrophied lymphoid cells. This *gummy peripylephlebitis* affected either the trunk of the portal vein or one of its main branches, and extended along the capsule of Glisson into the liver, so that this organ appeared to be permeated in every direction by firm, fibrous, and, for the most part, perfectly solid cords. In the three cases observed, the umbilical vein was unaffected; in one, the ductus venosus Arantii was also diseased. In this solitary case the ductus hepaticus was also compressed by the gummy inflammation, so that (five days after the birth, and three days and a half before death) jaundice from obstruction was produced. In the second case, icterus was also present from three to five days before death; the third child only lived eight hours. The spleen in

all the three cases was considerably enlarged. In one case there was intestinal hemorrhage, in the other peritoneal effusion.

Dilatation of the Veins.

Briquet, Arch. gén. VII. pp. 200 and 396.—*V. Cornil*, Sur l'anatomie pathol. des veines variqueuses. Arch. de physiol. 1872. p. 602.—*John Marshall*, On the extirpation of varicose veins. Lancet. IV. 1875.—*Ravoth*, Ueber Druckbehandlung d. Varicocele und d. Phlebectasie. Berl. klin. Wochenschr. 1874. S. 222. 1875. S. 317.—*Soboroff*, Ueber den Bau normaler und ektatischer Venen. Virch. Arch. Bd. 54. H. 3. 1872.—*P. Vogt*, Beh. d. Varicen mit Ergotininjectionen. Berl. klin. Wochenschr. 1872. No. 10.

We have much less accurate information as to the normal diameter of the veins than as to that of the arteries, because in them the physiological distention is subject to much greater variation, while the tension of the contents has a much greater influence. Indeed, it seems as if the great venous trunks, from their distensibility, were intended to serve as reservoirs for the blood under certain physiological conditions, such as limitation of the peripheral circulation (by cold, etc.). The diameter of the veins, therefore, varies very considerably within physiological limits, according to the amount of blood present in the individual.

The *dilatation* is either uniformly *cylindrical* (with or without coexisting elongation, and hence tortuosity), or it is circumscribed and *saccular* (varix, corresponding to the usual forms of aneurism in an artery), which like an aneurism may either affect the whole of the circumference of the vein, or only a part of it, and at one time is attached to the vein by a narrow neck, at another by a wider one; it may develop secondary bulgings, and may vary in size from a pin's head to a walnut. These varices are most frequently found on the central side of the venous valves; when venous congestion sets in, the venous tube is by means of the valve temporarily changed to a cul-de-sac.

Varices much more commonly than aneurisms occur together in considerable numbers. This latter form of dilatation is not commonly found alone, but associated with the cylindrical.

The coats of the dilated veins are sometimes normal, more frequently thinned, or thickened by chronic inflammation (*vide*

supra). Very often both thinning and thickening are simultaneously present in different parts of the vessel. The surrounding connective tissue is not unfrequently in a state of chronic inflammation, sclerosed, and the vessel thus adherent to surrounding tissue is less mobile than usual, or, if it be considerably dilated, the pressure of the neighboring tissues, such as the skin, or the subcutaneous cellular tissue, may cause it to atrophy in circumscribed patches.

The vein is often simultaneously elongated, and therefore tortuous.

From these tortuosities, as well as from the projecting varices, the vein assumes a necklace- (rosary-) like appearance; when the individual projections come in contact with one another, or with those of a neighboring vein, their coats may atrophy at limited portions, and so give rise to abnormal communications. The valves in the dilated veins are also distended and subsequently very often insufficient, atrophic, or fenestrated.

Blood coagula, which more or less completely occlude the lumen of the vessel, are of frequent occurrence in dilated veins, especially in the varices and in the peripheral trunks.

Etiology.

Dilatation of the veins is chiefly brought about by the persistent accumulation of blood in the veins, whether this is caused by obstruction to the onward flow (congestion), or whether it happens, because by the occlusion of certain veins a larger quantity of blood than usual is forced to pass through other collateral vessels. Dilatation of the veins is consequently present in all cases of disease of the heart or lungs, accompanied by congestion of the right side of the heart, or by diminished aspiration of the venous blood towards the thorax.

In such circumstances those veins are chiefly dilated which pass their blood onwards in direct opposition to the force of gravity, such as the veins of the lower half of the body.

Long-continued retention of the body in one uniform position may also produce dilatation of the veins of certain parts; continuous standing, for instance, occasions dilatation of the veins of

the lower extremities, especially of the legs. Obstruction to the outflow from certain venous trunks (by the pressure of a tumor, or of a contracted muscle) causes dilatation of their radicles; thus we have dilatation of the hemorrhoidal veins as the result of pressure on the portal vein.

Further, the veins are dilated when flushed with arterial blood, as in arterio-venous aneurism.

A relaxed and atonic condition of the surrounding tissues, which gives the venous coats but little support, favors the occurrence of every form of dilatation.

It is for this reason partly, and partly simply because of the increased flux of blood through the part, that we so often find venous dilatation in connection with repeated local hyperæmiæ, as well as in the neighborhood of new formations.

It has been also generally supposed that a diminution of the tone of the venous coats themselves, a paretic condition of the muscular coat of the veins may assist in producing dilatation; but though this is both possible and probable, it has not yet been certainly proved.

A hereditary disposition to venous dilatation, especially in certain areas, must be regarded as certain. Sometimes venous dilatations—congenital or acquired—are found without evident cause to account for them.

Venous dilatations are of very common occurrence in middle life, both in men and in women; in children and old people they are not so frequent.

Symptoms.

Dilatation of the veins is generally slowly developed, so that the vessels, as we can readily see in the cuticular veins of the lower extremities, only gradually, by long continuance of the standing posture, attain an abnormally great diameter. They then appear as more or less tortuous, knotted cords, which disappear on pressure or in certain positions of the body, but which reappear or are more largely developed when the onward flow of the venous blood is obstructed. These vessels appear of a

normal or bluish color, according to the thickness of their coats and of the overlying skin.

In the part of the body affected (the lower extremities, the testicles, etc.), the patients have a sensation of weight and fulness, and it may be swollen or even cyanotic when the smaller veins are turgid, but always cool. The neighboring organs, such as the testicles, may be atrophied by the venous pressure. Even the venous coats themselves may yield to the continuous distention, and at last give way on some trifling occasion, thus giving rise to hemorrhage, either externally or into the connective tissue.

Finally, thrombosis to a varying extent may occur, by which the vein is changed to a hard cord. In this condition the local and general symptoms vary according to the degree and character of the concomitant inflammation.

Should the venous dilatation subsist for a long time under the influence of a persistent cause, serous transudation into the connective tissue at last occurs, which by long continuance leads to induration and favors the occurrence of accidental inflammation. The mucous membranes lying within the range of such veins are subject to chronic catarrh.

Of all the various venous areas within the body, that of the vena cava superior is much less frequently affected with dilatation than that of the vena cava inferior. For the most part in it the dilatation begins and is greatest close to the heart, gradually decreasing towards the periphery. The *jugular veins* in such cases are seen in the neck like round cords, the external jugulars being as thick as a finger, while the internal jugulars may reach the size of an infant's arm ; they are more or less tortuous, often visible quite up to the cranium, and becoming turgid at each expiration or paroxysm of coughing. The bulb of the internal jugular vein is usually specially visible just before and behind the origin of the sterno-cleido-mastoid muscle. The venous swelling is usually better marked on the right than on the left side, but not always. When the dilatation is considerable, the venous valves become insufficient, so that regurgitation is caused by the expiratory movements or by the cardiac contractions. The venous pulsations can then be recognized, some-

times as presystolic, synchronous with the auricular pulsation, at others as systolic, synchronous with the ventricular contraction, and at still others both phases may be recognized in the form of an anadicrotic elevation. Which of these forms of pulsation is most evident depends upon the intensity of the contractile power of the individual cavities of the heart, on the presence of tricuspid insufficience, or on the momentary degree of tension within the vein. Sometimes, when the venous valves are insufficient, a stenotic murmur is audible synchronous with the venous pulse or with forcible expiration ; a thrill is often also perceptible.

Those veins which are surrounded by denser tissues, such as the *subclavian veins* and their branches, are less frequently dilated than the veins of the neck, and their valves are less commonly insufficient. The causes of this dilatation are : defective emptying of the right ventricle, diminution of the negative pressure within the thorax by disease of the lungs, etc., tumors which compress the anonymous veins, or, finally, the long continuance of violent paroxysms of coughing.

The *thyroideal veins* are often dilated to cords the size of a finger (their coats often concomitantly hypertrophied) in the various forms of struma. This is most remarkable in struma vasculosa, in which there is a kind of hypertrophy of all the vessels of the thyroid gland ; it is less evident, though always considerable, in struma hyperplastica, in which the vessels become enlarged from the persistent increase of the blood flow through the thyroid gland.

In *the face*, dilatation of the smaller veins of the cheek and nose are seen in disease of the heart and lungs, in alcoholismus, and in acne rosacea. Small varices occur on the eyelids, the lips, and the tongue, as well as in the mucous membrane of the fauces in chronic catarrh.

The *meningeal veins* are dilated and tortuous in general venous congestion and in atrophy of the brain (*ex vacuo*).

Meschede[1] has recorded a saccular and laterally situated varix of the superior longitudinal sinus, with a corresponding atrophy of the skull.

[1] Virch. Arch. Bd. 57. S. 525. 1873.

Venous dilatation in *the upper extremities* is observed when the main trunks are compressed by a tumor; in rare cases it is congenital.

Dilatation of the veins within the area of the *vena cava inferior* are more frequent and more important. On the trunk of this vein and the first large branches given off, the *hepatic* and *renal veins*, the dilatation is usually caused by disease of the heart or lungs; within these venous areas it generally extends even into the capillaries, and results in serous soaking, with induration of the kidneys and liver; in the case of the latter organ the long continuance of the pressure of the dilated capillaries leads indeed to atrophy of the hepatic parenchyma, and contraction of the organ (atrophic nutmeg-liver).

Of all the veins within the area of the inferior cava those most frequently dilated are the *veins of the lower extremities*, especially of the legs; they are also usually varicose. They give us by far the best opportunity of studying the pathology of venous dilatation generally. Occupations necessitating continuous standing (less frequently those requiring walking), the pressure of the pregnant uterus, of fæcal masses or of other tumors upon the vena cava, the pressure of tumors or of garters on the extremities, are its most frequent causes.

The great cuticular trunks of the two saphenous veins are often seen as thick cords; should the dilatation affect these trunks alone or principally, the inconvenience may be but trifling. But the more numerous are the branches implicated, so much the greater is the tendency to œdema, cuticular inflammations (eczema, erysipelas, boils), and to sclerosis of the subcuticular connective tissue—conditions which, in their turn, favor the progress of the venous dilatation. Ulcers are readily caused, but heal with difficulty, and may by erosion of a vein give rise to considerable hemorrhage. Sometimes concomitantly with the cuticular veins, at others independently, the deep-lying veins of the leg are also dilated and varicose; the patients have then a sensation of weight, fatigue, spasmodic tension, and numbness in the leg when they walk or stand. Sometimes a deep-seated, diffuse hard swelling can be felt.

Dilatation of the veins of the spermatic cord, *varicocele*, also

belongs to the domain of surgery. This is a very frequent ailment, almost exclusively occurring on the left side, apparently because the current of the venous blood is liable to be obstructed by the pressure of the sigmoid flexure of the colon, and because it terminates in the renal vein, and not directly in the vena cava, as the right spermatic vein does.

The varicocele is accompanied by dragging pains in the testicle and cord, and sometimes with involuntary emissions; it may ultimately lead to atrophy of the testicle.

The cuticular *veins of the abdominal walls* are somewhat dilated whenever the abdomen is distended from any cause (such as gravidity, ascites, etc.). The dilatation is very considerable whenever these veins are involved in promoting a collateral circulation (through the epigastric, the mammary, the intercostal, the azygos, and the hemiazygos veins) in cases of compression of the inferior cava or the portal vein.

In the case just referred to, the small cuticular veins around the navel are often dilated as a vascular corona (caput medusæ), by the blood conveyed to them from the portal vein by the still pervious umbilical vein, or by the parumbilical vein. In such a case the portal vein is dilated up to the contracted part (of its trunk or of its branches within the liver, as in cirrhosis).

The veins of the pelvic organs communicate so freely with each other by means of plexuses and anastomoses that their dilatation will be more conveniently dealt with under the head of dilatation of the hemorrhoidal veins, as this most frequently is the point of origin of venous dilatation within the pelvis. The veins of the female sexual organs are, however, not unfrequently independently dilated in advanced life, as the result of repeated pregnancies or of inflammatory conditions of the uterus and its connections.

Treatment.

The treatment of venous dilatation mostly belongs to the department of surgery, but particular attention must be paid to the causal phenomena (such as cardiac and pulmonary disease, compressing tumors, constipation, etc.).

When the dilated veins are still pervious, as in the extremities or in the scrotum, then we must seek to support their walls externally by enveloping the parts affected in simple or elastic bandages, and to restore their tone by cold bathing and friction.

The effort has also been made to attain this by injecting ergotine into the surrounding cellular tissue; yet, as has been already mentioned when speaking of a similar treatment of aneurism, the results observed are more probably produced by the influence of compression and inflammatory irritation.

As in aneurism, extreme measures have been resorted to, and thrombosis and obliteration of the vessel by coagula have been sought to be produced by injections, electro-puncture, etc.; indeed, quite recently, several inches of a dilated saphenous vein have been cut out.

The treatment recently recommended by Ravoth is of great theoretical as well as practical interest; he employs central compression of the dilated venous trunks by means of a ball of feathers (hernial truss). *A priori*, one would rather expect a further increase of the dilatation from such an obstruction to the onward circulation. R. explains the result as arising from a quickening of the circulation through the veins (?) and a partial relief to the tension of their walls. (It seems more probable that, as the result of the compression, collateral veins are gradually dilated, and, as these continue to convey a large part of the blood even after the removal of the compress, the blood-pressure is permanently diminished in the veins originally dilated.) According to C. O. Weber, Colles had previously employed a similar treatment.

In

DILATATION OF THE HEMORRHOIDAL VEINS,

The uniformly cylindrical form of dilatation is combined with a varicose condition. The plexus of veins lying in the submucous tissue of the lower part of the rectum, and in the adjoining subcutaneous connective tissue, are those chiefly affected in this form of dilatation; but it is also very common for the venous radicles in the mucous membrane itself, the perirectal plexus, and the adjoining venous plexuses of the bladder, uterus, vagina, and the sacral canal, to be also implicated in this dilatation.

Isolated, strongly distended varices bulge outwards the mucous membrane or the delicate skin of the anal margin in a saccular form; in other parts the pressure of several distended

veins upon one another produces local absorption of the venous coats, and a system of communicating blood sacs is thus originated.

This venous dilatation does not leave unchanged either the mucous membrane or the submucous tissue; capillary venous hyperæmia, serous transudation, hyperplasia of the connective tissue, and catarrhal swelling of the mucous membrane are associated with it, while in other places these tissues atrophy under the influence of the pressure of the varices. The natural rugosities of the rectal mucous membrane become permanently thickened and inflamed, polypous growths are formed and associated with the more or less pedunculated tumors produced by the varices.

These *hemorrhoidal tumors* range from the size of a pea to that of a pigeon's egg, and vary exceedingly in volume, according to the amount of venous turgescence; sometimes they are small, relaxed, and corrugated, at others large, turgid, and shining. According as they are situated within or without the anal margin, they are termed *internal* or *external* hemorrhoids.

Like all other venous dilatations, these also undergo many secondary changes and degenerations; and they are, from their situation, specially exposed to mechanical injuries of the most manifold character. The consequences of these are, rupture with hemorrhage either external or into the connective tissue, inflammation of the coats of the vein, or thrombosis of a hemorrhoidal tumor resulting in obliteration or suppuration. The submucous and perirectal connective tissue is also liable to become sclerosed or purulent as the result of inflammation, and the inflamed catarrhal mucous membrane of the rectum, or the skin surrounding the anus, may be eroded and ulcerated. Prolapsed varices and rugosities of the mucous membrane may be strangulated and necrosed by the pressure of the sphincter.

Etiology.

The most common cause of dilatation of the hemorrhoidal veins is obstruction to the onward flow of the blood, especially by habitual accumulation of fæces in the rectum, and also by

other tumors within the true pelvis; since the larger portion of the venous blood from the rectum is conveyed to the portal vein, by means of the superior hemorrhoidal vein, any permanent or repeated turgescence of the portal system, from congestion or frequent and copious meals, contributes to the production of hemorrhoids (consequently also any disease of the liver, such as hyperæmia or cirrhosis); disease of the heart and lungs may also act similarly through the vena cava itself or intermediately through the portal system. Abdominal tumors, the pregnant uterus, or great accumulation of fat in the omentum may also give rise to portal congestion.

As in other mucous membranes, so also in that of the rectum, persistent chronic catarrh seems to favor the production of venous dilatation.

Dilatation of the rectal veins is an extremely frequent condition in all those who suffer from chronic constipation as the result of their diet or habitual occupation, consequently in all those who live freely and take much vegetable food, who lead sedentary lives and take little exercise, such as clerks and similar officials. Much horse exercise also disposes to the formation of hemorrhoids (partly from the constipation thus induced, partly from the direct irritation of the part).

From the manifold character of the causes just narrated, it is easy to see that in individual cases several of these may often occur together, and that their effects may be cumulative, indeed that in the production of hemorrhoids, catarrh of the rectum and constipation may prove a circulus vitiosus, the individual elements of which may serve mutually to induce and aggravate each other.

It is probable that the hindrance to the respiratory movements induced by constipation, accumulation of fat within the abdomen, and sedentary habits, etc., may be an important adjuvant cause in the production of venous dilatation, since the pressure variations induced by the respiratory movements, both in the abdominal and in the thoracic cavities, are assuredly an important means of promoting the circulation within the abdomen.

Even when all the appropriate conditions are present, they by no means always give rise to dilatation of the hemorrhoidal

veins, and still less frequently to its obvious (objective) occurrence. Predisposition, probably often hereditary, plays in regard to this a most important part.

In middle life and in the male sex this affection is most common. It is very rare in childhood.

Symptoms.

A moderate dilatation of the hemorrhoidal veins often gives rise during life to no symptoms whatever ; it is either altogether unnoticed, or the individual affected may accidentally discover by touching his anus that small tumors exist there.

Even when subjective disturbances are present, these only occur periodically, when with or without any evident cause (most frequently constipation) the veins become congested, and turgescence, hyperæmia, or even inflammation of the adjacent parts may occur.

A sensation of burning, itching, and fulness in the anus is felt, especially when it is irritated by the passage of hard fæcal masses, and the sphincter thus excited in a reflex manner to more vigorous contraction. Objective examination brings to light in the neighborhood of the anus more or less tense elastic tumors, pedunculated or with broad bases, which, according to the thickness of the skin covering them, are either of a bluish-black or reddish-white color.

When submucous varices are present, these may be felt by the finger, passed through the anus, as tumors the size of a strawberry or grape ; more rarely these are prolapsed, and by strangulation by the sphincter become turgid and very painful.

The burning and itching are greater the more the surrounding skin becomes erythematous, inflamed, or excoriated ; when catarrh of the rectum is simultaneously present, this is accompanied by violent tenesmus and the escape of a glairy mucus, which by and by becomes purulent. The anus is contracted by the tumefied mucous membrane, and the latter is not unfrequently prolapsed. The pain in the anus is intense, stinging, and radiating towards the bladder and sacral region. Fissures of

the mucous membrane in the neighborhood of the sphincter are specially painful.

All these sufferings are most severe during defecation, walking, standing, or sitting, but are lessened by the recumbent posture.

Should the local inflammation be intense, fever, loss of appetite, and disturbance of the general health may occur.

Blood frequently escapes during defecation, and this either comes from the capillaries and excoriations of the inflamed mucous membrane, or—more rarely—from a ruptured varix, and in the latter case it may escape externally. The effused blood in such cases is recent, not mixed with the fæces, and unaltered by the gastric juice.

Usually the hemorrhage is trifling, and caused by the passage of the fæces. Sometimes after a somewhat copious hemorrhage —such as a few tablespoonfuls—there occurs a remission of all the sufferings, from diminution of the swelling of the veins and of the mucous membrane. Hemorrhoidal hemorrhage is very rarely injurious from its amount, and only so in weakly individuals. Moreover, it seldom occurs in those who are anæmic, and therefore it commonly ceases in advanced life.

These periodical attacks, the so-called fits of the piles, occur in greater or less violence and alternate with long periods, during which either no disturbances at all or only very trifling ones occur.

However troublesome the attacks may be, they are seldom dangerous. When the inflammation is acute, or when a nodule is strangulated, thrombosis may occur. As the result of this the varix may be obliterated, or inflammation may occur in its neighborhood, and this may lead to suppuration, to proctitic or periproctitic abscesses, and the formation of fistula; in severe cases purulent phlebitis, with pyæmia and metastatic abscesses, may occur, especially when the tumors have prolapsed, and have become strangulated and gangrenous.

As already mentioned, the other venous plexuses within the pelvis are not unfrequently implicated in the dilatation affecting the hemorrhoidal veins. When the vesical and prostatic plexuses are affected, there is present a frequent desire to micturate, and

pain in the act of micturition. The mucous membrane of the bladder is liable to catarrh, and blood is often mixed with the urine.

Dilatation of the sacral plexus is revealed by pain and a feeling of weight in the sacral region; when the communicating plexus of the spinal canal is affected, it may, by compressing the roots of the nerves, give rise to sensations of weight, numbness, formication, and pain in the lower extremities, or even in the lumbar region, so as to simulate sciatica, or a lesion of the cord itself.

Catarrhal affections of the female genital organs are kept up by dilatation of the veins, and tumefaction and inflammation of the uterus itself are thereby favored. Menstruation is often copious; it however serves to unload the veins at regular intervals, and thus it happens that dilatation of the hemorrhoidal veins is first fully developed in women after the cessation of the menses. On the other hand, the hemorrhoidal veins often become turgid coetaneously with the uterine menstrual flux.

When numerous concomitant phenomena are associated with the results of simple dilatation of the veins, the totality of the symptoms is often made to appear extremely complex by the presence of other morbid phenomena, which are either themselves the prime agents in the production of this venous dilatation, or which originate with it in a common cause. Thus we see diseases of the heart or lungs give rise concomitantly to the most manifold symptoms of liver disease, and of anomalies in the digestive and nutritive processes, with all their various consequences. And thus it is easily explicable why in earlier times such extravagant importance was attached to the presence of hemorrhoids, one symptom arising from very various conditions having been regarded as the essence of the disease.

The *diagnosis* is simple, as soon as we examine the patient objectively; but from the subjective phenomena alone this affection might easily be mistaken for syphilis, carcinoma, or a simple polyp of the rectum. For the treatment it is further of importance to determine what are the predisposing and proximate causes.

The general *prognosis* depends upon these, for actual danger

can only exceptionally arise from a purely local affection. A perfect cure is, however, very rare.

Treatment.

The treatment of dilatation of the hemorrhoidal veins must be directed against its causes, such as diseases of the liver and heart, and disturbances of the digestive process, etc. Even where constipation has not been a proximate cause of the disease, regulation of the bowels is nevertheless an important means of diminishing present and preventing future troubles. The food must, therefore, be moderate, unirritating, leaving but little fæcal refuse, and its composition must as far as possible be of such a nature as to favor secretion and the peristaltic movement of the intestines (such as fruits, certain vegetables, etc.); the regulated use of cold water and regular exercise also assist in producing the same end. Often enough such a system of diet is sufficient to reduce the trouble to a minimum; when this is not the case, mild saline laxatives, mineral waters, or rhubarb, may be employed as adjuvants.

Aloes or colocynth in small doses are also perfectly applicable, either with or without the addition of extract of belladonna. The curative use of Marienbad, Kissingen, and other mineral waters, of grapes, and the juices of various herbs, is not only useful symptomatically, but often fulfils causal indications.

Cold washing, clysters, and sitz-baths are also important remedies to keep the rectum and anus free and clean, and to prevent inflammation.

Special attention must be paid to the regulation of the diet and bowels during an attack of piles; clysters and the internal use of castor-oil are to be employed to keep the rectum free from fæcal accumulation, and to disembarrass the circulation; the horizontal position must be preserved, and the covering must be light and cool. Local inflammations are to be treated by cool sitz-baths and fomentations, or, if more severe, leeches may be applied. Ointments, suppositories, or injections of tannin, salts of lead, etc., are useful in more trifling irritation, and in the

concomitant erythema and rectal catarrh. Narcotics internally and locally are indispensable when the pain is violent.

A moderate amount of hemorrhage often gives relief and requires no treatment; even should it be more considerable, it still is usually easily restrained by the application of cold fomentations, clysters, or sitz-baths. At other times astringent fluids (liquor ferri perchloridi), and, in extreme cases, plugging the rectum, or the hot iron may be necessary.

Thrombosed nodules must as much as possible be guarded from violence so that they may become organized. Purulent inflammations and their results are to be treated upon general principles.

Large external varices may be removed by the hot iron.

Narrowing and Obliteration of the Veins.

A uniform narrowing of the veins over any considerable extent is of rare occurrence, and happens in parts which are becoming atrophied, as the result of the diminution of the blood stream.

J. Gay[1] has described cases of what he calls "hypovenosity," in which there is a diminution in the size and number of the veins in the area of the saphena, and in which (probably as the result of the pressure of the collaterally dilated deep veins) atrophy and fatty degeneration of the muscles are developed. The limb is wasted, but tense, sinewy, and painful; movement is difficult, and the skin dark-colored.

Improvement of the nutrition by exercise, friction, and warm baths are said to be useful.

Local narrowing or occlusion of the veins is of much more common occurrence. Apart from thrombosis, presently to be referred to, which is the most common cause of these narrowings, they are produced by the pressure exerted from without on the veins by tumors, organs which are inflammatorily swollen, or by cicatrices. Concomitant chronic inflammation of the walls and clot-formation often assist in producing this narrowing. This

[1] Lancet. Nov. 1871.

condition may lead at last to complete occlusion of the vein ; the walls of the vein, pressed one upon the other, grow together and are changed into a simple cord of connective tissue.

The area of the radicles of the narrowed vein exhibit all the signs of venous congestion (in so far as this is not efficiently relieved by the establishment of collateral dilatation) : such as dilatation of its affluent branches, down to the capillaries, cyanotic discoloration, a diminution of temperature, serous transudation, etc.,—phenomena which will be more particularly referred to when speaking of thrombosis, and which are all the more prominent, the more rapidly the narrowing is brought about, and the shorter the time afforded for the development of a collateral circulation.[1]

Rupture and Wounds of Veins.

Rupture or wounds of veins may be produced—

1. *Traumatically*, by cutting instruments, accidentally or operatively (venesection), usually only in superficial veins ; also by crushing or tearing, by falls from a considerable height ; or the large thoracic or abdominal veins may be ruptured when the body is driven over by any carriage, or the veins of the extremities may be torn across by violent muscular exertion or by direct blows.

Coester[2] saw perforation of the vena cava inferior by a sharp-pointed piece of bone which had caught in a diverticulum of the œsophagus. Death resulted from hemorrhage into the right pleural cavity and the stomach.

2. *By increased pressure* within a vein, such as exists in cases of congestion of a central origin, or when a communication with an artery has been made. Thus the pulmonary veins are sometimes ruptured in cases of disease of the mitral valve.

3. *By disease of the venous coats.* This, however, can but seldom be the cause of rupture, since inflammation usually very

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[1] Vide *Déstord*, Des Tumeurs cancéreuses du médiastin avec compression ou oblitération de la veine cave supérieure. Thèse de Paris, 1866.

[2] Berliner klin. Wochenschr. 1870. No. 43.

early leads to coagulation of the blood. Atrophy of the walls, such as occurs in dilatation of the veins, or fatty degeneration (as in one case of rupture of the portal vein related by Frerichs [1]) is more likely to lead to rupture.

4. *By ulcerative destruction* of the venous coats, which occurs in suppurative phlebitis of a primary or secondary character (as, for instance, in deeply penetrating ulcerations). Rupture may also result from the pressure of an adjoining tumor, for example, an aneurism, or by the vein becoming implicated in its growth (as in the case of a carcinoma).

Rupture occurs, on the whole, more frequently in veins than in arteries, since from the thinness of their walls and the trifling degree of elasticity they possess, the former are less capable of resistance than the latter.

Symptoms.—In wounds or lacerations of the peripheral veins, when the blood stream can readily find its way through collateral vessels and the blood pressure is thus not very great, coagulation of the blood readily occurs at the part wounded, which is thus closed. At other times the blood flows externally or into the adjacent cellular tissue until the blood pressure within the veins is diminished, or is exceeded by that external to them. The blood effused into the cellular tissue causes swelling and pain, sometimes even inflammation of the part affected, or it may be reabsorbed without the occurrence of any important local or general disturbance.

Should one of the large veins in the chest or belly be torn, the hemorrhage takes place into loose cellular tissue or into one of the serous cavities, and the patient dies with all the symptoms of internal hemorrhage in a few hours, rarely surviving for a few days. Sometimes such patients have had a sensation as if something within them had been torn. Occasionally the blood can be detected within the serous cavity before death.

Instead of or along with the escape of blood from the veins, foreign matters may gain an entrance into the injured vein ; in suppurative destruction of the venous coats, pus, or ichor, or cancer-juice may get into the veins, and thus lead to septicæmia,

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[1] Leberkrankheiten. II. S. 382. New Syd. Soc. Transl. II. p. 404.

embolic inflammations, or the development of secondary cancers, provided the venous lumen has not been previously occluded by a coagulum.

The *prognosis* depends upon the position and size of the vein injured.

In rupture of any of the large veins within the cavities of the body, *treatment* is of no avail. When the position of the vessel is such as to permit it, the part ruptured must be elevated, local compression made use of, and cold applied.

The introduction of foreign matter into the veins of inflamed parts will be best prevented by diminishing the tension of the inflamed part and promoting the flow of the pus externally (position, incisions).

DISEASES OF THE LYMPHATICS.

Vide the handbooks of pathological anatomy by *Cruveilhier*, *Förster*, *Rokitansky*, etc.; also the handbooks of special pathology and of diseases of the vascular system by *Wunderlich*, *Lebert*, *Duchek*.

Andral, Arch. gén. de méd. 1824. Vol. 6. p. 502.—*Assalini*, Essai médical sur les vaisseaux lymphatiques. Turin. 1787.—*Attenhofer*, Lymphatologie. Wien. 1808. —*Cohn*, Embolische Gefässkrankheiten. S. 110 and 659.—*Cruikshank u. Mascagni*, Gesch. und Beschreibung d. Saugadern, 1789.—*H. Emminghaus*, Physiologisches und Pathologisches über die Absonderung und Bewegung der Lymphe. Arch. d. Heilk. 1874. Bd. 15.—*Genersich*, *Lesser*, *Pasehutin*, *Emminghaus*, Ueber Lymphabsonderung in den Ber. über die Arb. d. physiol. Instituts zu Leipzig. 1870-73.—*Goodlad*, A practical essay on the diseases of the vessels and glands of the absorbent system. London, 1814.—*Rayer*, Dictionn. de médec. Art. Hydropisie.—*Salmade*, Précis d'observations sur les maladies de la lymphe. Paris. 1803.—*Soemmering*, De morb. vasor. absorbent. Frankf. 1795.—*van Swieten*, Comment. IV. p. 116.—*Teichmann*, Das Saugadersystem.—*Velpeau*, Arch. gén. 1835. Vol. VIII. pp. 129 and 308; 1836. Vol. X. p. 5.—*O. Weber*, in Billroth-Pitha's Handb. d. Chirurgie. II. 2. S. 63. 1865.—*Wrisberg*, De syst. vas. abs. morbos excit. et san. comment. soc. reg. Gotting. 1789. Bd. IX.

Diseases of the lymphatics, just as much as those of the blood-vessels, are most intimately connected in various ways with the most diverse organic diseases; perhaps this connection is even more close, since the capillary lymphatics, according to many authors, are in open communication with the spaces of the tissues. For evident reasons, we have been obliged in the following chapter to omit all consideration of the diseases of these lymphatic capillaries, as was also done in regard to the blood capillaries.

It will readily be seen, from the contents of the following paragraphs, that our knowledge of the pathological conditions of the lymphatic vessels is as yet very imperfect. The trans-

parency of their contents, its capacity for imbibition, the thinness of the walls, the trifling rate of movement, and low degree of tension of the lymph-stream, are all to be regarded as phenomena which obstruct any attempt at anatomical investigation of the subject; indeed, any accurate knowledge of the anatomical and physiological relations of the lymphatic system dates, for the most part, from quite recent times.

It is possible that many obscure pathological conditions owe their origin to disturbances of the lymph-stream; even van Swieten referred the occurrence of dropsy to this, partly directly and partly indirectly, by means of the venous blood-stream, and, according to our present knowledge, it is not improbable that certain cases of ascites, of pleuritic effusion, and of hydrocephalus, not connected with obstruction to the circulation of the blood, may depend upon embarrassment of the lymph-stream. The dependence of elephantiasis upon disease of the lymphatic vessels is to be regarded as almost certainly proved, as will be presently shown.

Inflammation and simple thrombosis of the lymphatics cannot as yet, on the basis of our present knowledge, be clearly separated from one another, and must, therefore, be treated of together.

Inflammation and Thrombosis of the Lymphatics.

Lymphangitis, Angioleucitis.

Allard, De l'inflammation des vaisseaux absorbens. Paris. 1824.—*Bouvel-Roncière*, Les lymphangites primitives de Rio de Janeiro. Arch. de méd. navale. Mai, 1873. p. 355.—*Breschet*, Du syst. lymphat. Thèse de Concours. 1836.—*Browne Cheston*, Philos. Transactions. 1780. Vol. LXX. p. 323.—*Cooper*, Med. records and researches selected from the papers of a private med. assoc. Lond. 1798. I. p. 28.—*Duplay*, Arch. gén. 1835. Vol. VII. p. 10. De la suppuration des vaisseaux lymph. de l'utérus.—*Fetzer*, Ein Fall von eigenthüml. Erkrankung der Lymphgefässe. Arch. f. physiol. Heilk. 1849. Bd. VIII. S. 128.—*Kiwisch*, Klin. Vorträge. I. S. 632.—*Leopold*, Die Lymphgefässe des normalen nicht schwangern Uterus. Arch. f. Gynækol. Bd. VI. S. 1. 1873.—*Ludwig*, Wien. med. Jahrb. 1863. XIX. 2.—*Oppolzer*, Allg. Wiener med. Zeitung. 1861. No. 19. —*Tonnelé*, Arch. gén. 1830. Vol. XXII. p. 354.—*Turner*, Edinb. Med. Journ.

1859, May (2 cases of occlusion of the thoracic duct by aneurisms).—*Virchow*, Arch. Bd. XXIII. 1862. S. 415.—*O. Weber*, In Billroth-Pitha's Chirurgie. II. 2. S. 63.—*Weitenweber*, Beitr. Bd. IV. H. 2.—*J. Worms*, De l'inflammation du canal thoracique. Gaz. Hebdom. 1859. p. 280.

Anatomy.

Inflammation of the lymph-vessels, as of the veins, seems chiefly to affect the external portion of their coats. The adventitia and the surrounding connective tissue appear injected, swollen, cloudy, and infiltrated with cells; the intima is opaque, uneven, and stripped of its epithelium. The lymphatic glands connected with the part affected are swollen or inflamed. The inflamed lymph-vessels are often dilated, partly from relaxation of their walls, partly because the onward flow of their contents is hindered. Instead of a normal, transparent lymph, they contain a thick, opaque fluid, full of cells, which may even be puriform, and may coagulate, and thus occlude the vessel; yet such coagula are rarer, not so readily formed, and less firm than the thrombi in the blood-vessels. They are white, pale, red, or yellowish, like pus, and exhibit under the microscope mainly a finely granular mass with lymph corpuscles sparingly scattered through it (O. Weber). Such thrombi are most apt to be formed in the immediate neighborhood of the valves. The results of the inflammation, as well as the further changes in the thrombi, are precisely analogous to those occurring in the blood-vessels in similar circumstances. When the inflammation is trifling, the new-formed cells disintegrate, and the exudation is absorbed; but relaxation and dilatation of the lymphatic vessels is a very frequent result. In other cases the inflammation leads to hyperplasia of the connective tissue; thickening of the wall of the vessel, sclerosis of the surrounding connective tissue, and narrowing or obliteration of the lymph-vessel are the consequences. Finally, the inflammation of the lymphatics may lead to suppuration of the walls of the vessels and of the surrounding connective tissue. It is characteristic of all forms of inflammation of the lymphatic vessels that the cellular tissue is wont to be implicated in a very much greater degree and over a greater extent than in

inflammations of the blood-vessels; it is possible that this may depend upon the comparative thinness of the walls of the lymph-vessels and their less distinctly defined separation from the tissue spaces. The coagulated contents of the lymph-vessels undergoes changes analogous to those of thrombi within the blood-vessels; it may be absorbed, may break down into pus, may become organized or calcified. Sometimes we find the contents of the lymph-vessels coagulated, opaque, rich in cells, or even purulent, without there being any very evident disease of the coats of the vessel; in such cases the contents of the lymphatics seem first of all to be altered by the entrance of foreign matter, or by the primary disease of the organs in which these vessels originate, and this alteration of the contents seems to give rise to a secondary affection of the coats of the vessel.

From the narrowness of the vascular lumen, only fluids, or very minute bodies suspended in them, can be conveyed along with the lymph-stream; very rarely are thrombi swept along with the current, as happens in the blood-vessels. Such solid particles seem not to pass beyond the nearest lymphatic gland; yet the inflammation of the lymphatics may be propagated beyond these glands, the irritating matter being conveyed beyond them, either in solution or in the interior of the lymph cells.

Moreover, I may remark that our knowledge of the anatomy of inflammation of the lymphatic vessels is extremely defective, and this depends partly on the fact of the inconstancy of the course of even large lymphatic trunks, as well as on the thinness of the walls, and narrowness of the calibre of these vessels, and partly because this inflammation seldom occurs by itself, but is usually secondary and accompanied by implication of the surrounding tissues.

What has just been said of the larger lymphatic trunks is even more applicable to the lesser lymph vessels, because the variations in regard to their distribution and normal anatomical structure are even more manifold. In parenchymatous inflammations these are undoubtedly very often coetaneously diseased, and convey the products of morbid tissue change. The proof of this conveyance of morbid products is to be found in the frequent occurrence of consecutive disease of the lymphatic glands, in

which these products seem to accumulate, whilst the lymphatic vessels, through which these products pass often for considerable distances, are not themselves inflamed.

Etiology.

Lymphangitis occurs very rarely spontaneously, but is almost always secondary to disease of the area in which the radicles of the lymphatic vessel affected originate. The products of the morbid tissue change, which are conveyed by the lymphatics, appear to be the active agents in inducing this form of disease. It appears to depend upon the nature of these products, as well as upon the nature of the inflammatory process itself, whether the lymphatics are themselves implicated in this morbid process or not; possibly this may also partly depend upon whether the lymphatic radicles in the part primarily diseased remain open or are closed by clots or pressure.

Sometimes an independent lymphangitis is simulated, when the primary disease from its trifling character remains unnoted. This is specially the case in slight external injuries, pricks, scratches, and abrasions of the skin, which lead to lymphangitis, not from their own intrinsic character, but by giving entrance to injurious matters, such as decomposing or putrid organic matters, and the secretions and parenchymatous fluids of inflamed organs; for this reason anatomists and medical men are specially apt to be affected by lymphangitis. While the flow of blood and of the inflammatory secretions readily cleanse the surfaces of large gaping wounds from any foreign substances which may adhere to them, this is not the case in trifling pricks and scratches; and to this source of danger we must add the slight attention paid to such injuries. The local inflammation is often slight, or may be entirely absent, while the emerging lymphatics may be actively inflamed. Lymphangitis has also been observed to follow the stings of insects, the bites of our indigenous (but little hurtful) poisonous snakes, etc.

Further, we see this disease originate in the most various parts of the body as the result of inflammation, suppuration, or the formation of ichor in the connective tissue, whether these pro-

cesses have actually occurred in the area of the radicles of the lymph vessels affected, or whether the disease has spread by contiguity from the neighboring tissues. Lymphangitis is of very frequent occurrence when the pelvic connective tissue is inflamed during childbed.

Symptoms.

The clinical phenomena attending inflammation of the lymphatic vessels are best known as they occur within the domain of surgery in external parts, particularly the extremities, and most frequently as the result of poisonous wounds. But a few hours after the receipt of such a wound, the superficial lymphatics of the skin appear as pale red lines ; these sometimes form an anastomosing network, and run together; they are painful on pressure, and may be felt as hard, knotted cords, which are not usually sharply limited. These lines and cords cannot always be traced directly to the injured part. This part itself, as well as the whole area from which the inflamed lymphatics arise, is swollen, by reason of the defective removal of the lymph, and this swelling is greater, the more extensive the lymphangitis is, and the fewer collateral routes therefore remain open. Should the deep-lying lymphatics be inflamed, the redness may be absent, or only punctiformly indicated ; the pain is violent and less circumscribed, the swelling considerable, and generally diffuse.

The corresponding lymphatic glands are invariably concomitantly affected, painful, and swollen, often in several positions simultaneously (as, for example, the cubital and axillary glands); between the painful glands and the inflamed lymphatic vessels, there is not unfrequently a part of the latter which is apparently quite unaffected.

Fever is usually present ; often it is of considerable intensity, and commences with a rigor. Should the disease run a favorable course, it commences to abate in from a few days to a week ; the local symptoms disappear (the œdema being the last to do so), and a cure follows.

At other times, suppuration—originating either in the con-

nective tissue, or in the lymphatics themselves—extends along the inflamed vessels; in these cases a cure may at last result, accompanied by cicatrization and destruction of the vessels, or the fever may assume a typhoid character, and death from pyæmia may follow.

The lymphatic glands are generally implicated in the changes which take place in the afferent vessels, but they more readily suppurate than the latter. Should any part of the inflamed lymphatics become destroyed or narrowed, the affected tissues sometimes remain œdematous, and this subsequently leads to sclerosis and permanent hypertrophy of the connective tissue, which may be either diffuse or in isolated lumps or masses. Many cases of elephantiasis, both indigenous and tropical, seem to owe their origin to repeated inflammations of the lymphatics (in connection with erysipelas and other skin affections. On the other hand, Wernher assumes that the lymphatics are simply dilated. *Vide postea*, p. 528)

Many cases of puerperal phlegmasia alba dolens, of sclerema (hidebound disease of new-born children), and of epidemic angina parotidea, have been referred to inflammation of the lymphatic vessels, but without any sufficient proof having been given that this has been really the actual and primary cause of the disease.

Bouvel-Roncière describes an idiopathic lymphangitis malariosa (popularly known as the Rio-de-Janeiro erysipelas), which affects the superficial or deep lymphatics of the whole body, often in several places simultaneously, and in which the surrounding connective tissue is also implicated. Should the disease be limited, convalescence occurs in a few days; in severer cases, those affected die from adynamia or from suppuration. The treatment consists mainly in the use of quinine, besides local applications.

The

Prognosis

of lymphangitis varies with the extent of the disease and the nature of its proximate cause. Sometimes, even when the disease is of but limited extent, the general symptoms are very severe, and the disease terminates unfavorably after assuming the form of a septic fever; this is ascribed to the virulence of the infecting substance, of which we have no more accurate knowledge, while its very existence is only proved by the severity of the infection. In general, the prognosis is all the more favorable the nearer the

periphery the part affected lies, since the interpolated glands often set a limit to the centripetal progress of the inflammation.

The

Treatment

must be chiefly directed against the proximate cause; therefore, recent wounds must be dilated, cleansed, cauterized, or disinfected by fluid or diffluent remedies (such as acetic acid, caustic potash, chloride of zinc, carbolic acid, etc.), and afterwards carefully treated like other primary inflammations, according to the fundamental principles of surgery. The diseased part must be kept at rest. Leeches are but seldom required, and only when great pain is present over a limited area. Locally, cold is to be applied at first, and subsequently poultices and tepid baths. Envelopment in raw cotton and inunction with blue ointment have also been much recommended. Abscesses must be opened as early as possible.

Internally we may employ vegetable and mineral acids, quinine, alcohol, and other remedies usually prescribed in infective processes. Residuary œdema and indurations are to be treated by appropriate position of the part, compression, warm baths, painting with iodine, oily inunctions, and methodic shampooing. Passive movements, the alternate flexion and extension of the part, seems also a rational mode of treatment, now that Genersich, Lesser, and Paschutin have taught us how very much the flow of the lymph is favored by such manipulations.

Inflammation and thrombosis of the lymphatics is also very frequently met with in the uterus and in the surrounding pelvic cellular tissue. Generally it is the puerperal injury to the inner surface of the uterus, more rarely other diseases of this organ, which, apparently from infection *ab externo*, forms the starting-point of the lymphangitis. In these cases the lymphatics are found to be dilated and filled with an opaque, thick, purulent, or ichorous matter. The wall of the vessel, according to Duplay, Virchow, and others, is at first normal, but may subsequently inflame and suppurate, which the surrounding connective tissue may also do. The lymphatics are specially apt to be diseased

in the posterior and lateral parts of the uterus, as well as in the broad ligaments of the uterus; those of the false pelvis, the inguinal and femoral regions, are more rarely affected.

Peri-uterine lymphangitis cannot be regarded as an idiopathic disease, since it is only a somewhat inconstant concomitant of the most diverse forms of puerperal disease, such as inflammatory parametritis, septic processes, and thrombosis of the uterine veins. The pathological and prognostic significance of this, as well as of other diseases of the lymphatics, does not so much depend upon its existence merely as upon the nature of the process in which it originates, and this also determines the nature of the treatment to be adopted.

We know but little about inflammations of the thoracic duct, or of its morbid affections generally—partly, indeed, because but little attention is paid to it at post-mortem examinations. Andral examined the thoracic duct with great care in three hundred bodies, and found it only five times pathologically altered.

In one case thrombosis of its upper part was found, propagated by continuity from thromboses of the subclavian and jugular veins; the wall of the vessel was somewhat thickened, the lower part of the duct dilated (Oppolzer). In other cases the wall of the vessel was inflamed, its contents purulent, or thick, cheesy, tuberculous (Andral, Velpeau, Worms, Gendrin, Cooper); the valves are in such cases thickened or adherent, the lumen often dilated.

Calcification of the wall of the duct (Wrisberg, Mascagni) or of the contained thrombus (Browne Cheston) has been also observed.

Finally, closure of the duct by adhesion throughout its entire length has also been seen as the result of inflammation.

But little is known of the phenomena presented during life in inflammation of the thoracic duct. *À priori*, one would expect at one time narrowing or occlusion of the duct (*vide infra*), at another the possible extension of the thrombosis into the subclavian vein, etc. In the case related by Worms, which seems to have been a primary inflammation of the thoracic duct, the disease ran its course in fourteen days; it commenced with violent pains in the body and fever, followed by swelling of the left arm,

thrombosis of the subclavian vein, pyæmic fever, icterus, and death.

Degenerations and Neoplasms affecting the Lymphatics.

Andral, Arch. gén. 1824. Tom. VI. p. 507.—*Armauer-Hansen*, Beitr. z. Anat. d. Lymphdr. 1871.—*Aufrecht*, Med. Centralbl. 1869. No. 28.—*Bastian*, Edinb. Med. Jour. 1867. p. 815.—*Cruveilhier*, Anat. Pathol. II. Livr. 27. Pl. 2.—*Klebs*, Virch. Arch. Bd. XLIV. S. 256.—*Köster*, Virch. Arch. 1867. Bd. XL. S. 468. Die Entwickelung der Carcinome. 1869.—*Th. Langhans*, Die Lymphgefässe d. Brustdrüse und ihre Beziehungen zum Krebse. Arch. f. Gynäk. Bd. VIII. S. 1. 1875.—*Pagenstecher*, Virch. Arch. Bd. XLV. 1869. etc.—*Recklinghausen*, Arch. f. Ophthalm. 1864.—*Rindfleisch*, Patholog. Gewebelehre. S. 99.—*E. Wagner*, Krebs d. Lymphgefässe d. Pleura. u. d. Lungen. Arch. d. Heilk. IV. S. 538.

It has already been stated that thickening of the coats of the lymphatics and hyperplasia of the connective tissue have been observed as the results of inflammation. Calcification of the walls of the thoracic duct has been also seen. No special reference is to be found to any other form of degeneration.

The lymphatics are frequently found to be implicated in certain pathological neoplasms, such as cancer and tubercle. In both of these instances it happens in this way: cells, tissue juice, or cheesily-degenerated particles of the neoplasm are taken up directly by the smaller lymphatics, carried on some distance with the lymph-stream till they adhere to the walls of the vessel or are arrested within a gland, and here, partly directly from the tissue elements which have been floated off, and partly, secondarily, from the infected wall of the vessel, there occurs a further and fresh development of cancer or tubercle. Where glands interrupt the course of the lymph, the germs of the neoplasm seem not to be propagated in a centripetal direction until the gland itself has become diseased.

Thus in tuberculosis of the epididymis, or of the intestines, the lymphatics of the spermatic cord and of the mesentery are by no means unfrequently filled with cheesy matter; Andral saw the lymphatics on the surface of tubercular lungs filled with similar contents; and even the thoracic duct has been found filled with cheesy material in similar conditions, in tuberculosis of the mesenteric and lumbar glands (Cooper).

In cases of tubercular ulceration of the intestines, Klebs saw yellow and gray tubercles within the lumen of the lymphatics, and could trace a series of these tubercles up to the nearest gland. On the other hand, Armauer-Hansen declares that these tubercles are always external to the lymphatics of the serous covering of the intestine and of the mesentery.

Similar and often more striking observations are often made on the lymphatics of the peritoneum, of the diaphragm, and of the pleura, in cases of carcinoma of the intestinal tract, of the liver, and of the uterus. In such cases the lymphatics appear as extremely beautiful nodular cords and networks, which are completely filled with cancerous material. Rokitansky found the receptaculum chyli filled with cancerous matter in a case of carcinoma of the retroperitoneal glands; Cruveilhier and Andral found the thoracic duct filled in the one case with cancerous matter, and in the other with cancerous matter and pus, in cases of uterine sarcoma.

Cancer occasionally but much more rarely develops itself primarily from the walls of the lymphatics. E. Wagner observed an instance of this in the lymphatics of the pleura and lungs.

According to recent authors, neoplasms frequently originate in the lymphatics. Klebs, Aufrecht, Rindfleisch, Bastian and others adopt this view in regard to tubercle, while Recklinghausen and others, particularly Köster, look upon carcinoma (of the skin and of the stomach) as originating in this way. We cannot now enter more fully into these observations, which only relate to the most minute lymphatics, and possess mainly an anatomical interest. But we must point out that Langhans is opposed to this view, and has not only shown the incongruence between the form of the normal lymphatic network and the network of cancer cells in the localities specified, but has also, on careful examination of the carcinoma mammæ, been unable to recognize the lymphatics as the point of origin of the disease.

During life it is impossible to recognize either tubercular or carcinomatous disease of the lymphatics. It may however be suspected whenever, in the course of any such organic disease, the lymphatic glands become swollen or the serous cavities diseased.

The therapeutics of such an affection must be confined to prophylaxis.

Narrowing, Occlusion, and Dilatation of the Lymphatics.

Alter, Generalsanitätsber. f. Schlesien. v. J. 1834. Breslau. 1836.—*Anger*, Des tumeurs érectiles lymphatiques. Paris. 1867.—*Demarquay*, L'Union médicale. 1851.—*Desjardins*, Gaz. méd. 1854. Nos. 24, 27, 30, 34.—*Hugier*, Bull. de la Soc. de Chir. T. II.—*Labbé*, Dilatation variqueuse des lymphatiques. Gaz. des Hôp. 1867. p. 421.—*Laënnec*, Traité de l'auscult. méd. 3d édit. III. p. 439. —Casuistische Angaben bei *Lebert*, S. 608.—*Levin*, Lymphangiectasis submucosa percurrens. Nord. Med. Ark. VII. 2. 1874.—*Magendie*, Handb. d. Physiol. übers. v. Heusinger. II. S. 160. Eisenach. 1836.—*Morton*, Phthisiologiæ. Lib. I. Cap. 10. p. 21. in *van Swieten*, Comment IV. p. 189.—*Oppolzer*, Allg. Wiener med. Zeitg. 1861. No. 19.—*R. Paterson*, Congenital lymphatic varix. Edinb. Med. Journ. May, 1871.—*Petters*, Prager Vierteljahrschr. 1861. Bd. 4. S. 146. —*Rokitansky*, Pathol. Anatomie. Bd. II.—*T. Grainger Stewart*, On dilatation of the lacteals. Edinb. Med. Journ. 1863. p. 448.—*Trélat*, Gaz. des Hôpitaux. 1869. No. 136.—*Virchow*, Arch. Bd. VII. S. 130. Ueber Makroglossie.— *Wernher*, Beitr. z. Kenntniss d. Elephantiasis Arabum. Deutsche Zeitschr. f. Chirurgie. Bd. V. S. 394. 1875. (Contains many references and extracts from relative literature.)— *Wutzer*, Müller's Arch. 1834. S. 311.[1]

Etiology.

Very little is known in regard to abnormal uniform narrowness or dilatation of the lymphatics, either congenital or arising during development, and extending over a greater or less area.

Alter observed a congenital ektasia of the jugular plexus: Paterson, one of congenital ektasia and varicosity of the superficial lymphatics of one leg. In the latter case there was swelling of the leg, condensation of the connective tissue over and around Poupart's ligament, and lymphorrhagia. Probably there was in this case a want of anastomotic connection between the superficial and deep lymphatics.

The individual variations in the calibre of the lymphatics must be even greater than those of the blood-vessels, because of the little constancy there is in regard to even their coarser topographical relations; but the collateral equalization of the lymph circulation is certainly more easily brought about. It is probable that the persistence of such abnormalities in the vascular lumen of the lymphatics is not without influence upon

[1] Vide Lymphorrhagia. *Infra*, p. 532.

the nutrition of the area from which they spring. Narrowing of the ductus thoracicus and of the lacteals may prevent the sufficient absorption of nutritive material, and may thus (besides disposing to disease of the intestines) give rise to permanently defective nutrition and a stunted development.

Of all pathological processes it is chiefly inflammation and thrombosis which give rise to circumscribed or more extensive narrowing, and frequently to complete occlusion of the lymphatics: by thickening of their coats, adhesion of their internal surfaces, transformation of the vessel to a cord of connective tissue, or calcification of the coats of the vessel or of the thrombus. The lymphatics may also become narrowed or occluded by particles of tubercular or cancerous material, which, when floated off from their original site, may either themselves undergo further development or may occasion a similar morbid affection of the coats of the vessels. External compression from the cicatrization following inflammation of the connective tissue, or from tumors (such as aneurisms, cancerous masses, or glandular swellings, which may compress the thoracic duct), act also in a similar manner. If the compression be permanent and complete, inflammatory adhesion of the internal surfaces of the vessel results, the centripetal portion of the vessel becomes narrower up to the nearest collateral branch, the glands connected with the occluded vessel become small, hard, and dense; within the entire area from which the narrowed or occluded vessel originates, there is lymph stasis, dilatation of the lymphatic trunk, and œdema of the tissues.

Dilatation of the lymphatics also occurs, not only as the result of narrowing of the central trunks, but also within the area of diseased glands, through which the passage of the lymph is thus hindered. Under this head we may mention neoplasms, chronic inflammation with hyperplasia of the connective tissue framework, or resulting in caseation. Stasis of the blood in the large veins, the result of cardiac disease (Petters, Oppolzer), must also be considered in connection with the thoracic duct, inasmuch as lymph stasis may thus be produced. In peripheral parts of the body, dilatation of the lymphatics is not unfrequently the result of repeated attacks of inflammations (erysipelas, inflammations of the connective tissue); excessive

exercise of function from repeated excessive flow of lymph, perhaps also slight inflammatory attacks of thrombosis of the vessels, seem to be the active agents in this case.

In a number of recorded cases of dilatation of the lymphatics, no efficient cause has been discovered; it is possible that some mechanical obstruction to the lymph stream was present and was overlooked. Yet we must not quite disregard the possibility of an idiopathic dilatation, which may be produced by pathological alterations in the walls of the lymphatics themselves, just as happens in the blood-vessels. Even a paralysis of the lymphatics, such as has been hypothetically assumed by Emmert and others, seems to be not a very improbable cause of dilatation, since unstriped muscular fibres have been observed in the walls of the lymphatics, and rhythmical contractions of these vessels have been observed in certain parts to assist the onward movement of the lymph-stream.

Apart from the lymphatic hearts of the frog, which have been long known, Heller has observed contractions in the lymphatics of the mesentery of the guinea-pig, which, commencing close to the valves, forced the contents onwards, and had a rhythm of their own quite independent from that of every other movement.

Anatomy.

The dilated lymphatics are seen as cylindrical, usually tortuous cords, which present nodular swellings in the position of the numerous valves, and often form a network. Their diameter may be double that of the normal vessels, and their varices may be as large as an apple. According to Rokitansky, their coats are sometimes thickened, and at others attenuated; consequently rupture not unfrequently takes place, lymph is effused into the connective tissue or exernally, effused chyle forms easily recognizable white extravasations in the intestinal mucous membrane between the layers of the mesentery or in the peritoneal cavity.

Within the area of dilatation the glands are somewhat enlarged, their lymph ducts distended, often so much so as to produce large, locular, cystiform tumors (Lymphadenectasiæ).

The contents of the dilated vessels is sometimes a clear,

watery, feebly opalescent, or turbid milky fluid; the turbidity, as in normal chyle, is caused by minute fatty granules; moreover, this milky fluid is not only found within the area of the intestinal lacteals, but also in other parts (as the scrotum, thigh, etc.)—probably by means of dilated collateral anastomotic branches which reach these parts. Sometimes small quantities of blood are mingled with the fluid. When the stagnation has lasted some time, the lymph may become thickish or unctuous. Further, the dilated vessel may be partially or wholly filled with coagulated lymph, either recent or in various stages of metamorphosis, or it may be filled with pus, cancerous or tubercular material.

Symptoms.

Both narrowing and dilatation of the lymphatics very often present no symptoms during the life of the patient, either from the occult position in which they are situated, or because the disturbances produced by them are compensated by the dilatation of collateral vessels. Where narrowing or occlusion of a lymphatic trunk is brought to light, this happens through this dilatation of the collateral vessels or from the congestion of the area whence the radicles of the narrowed vessel spring. This congestion sometimes causes dilatation chiefly of the nearest-lying larger vessels (*vide infra*), at others the radicles themselves are affected, and within their area we have œdema and hypertrophy (sclerosis) of the connective tissue produced, just as has been already noted when speaking of the results of lymphangitis. Under these circumstances the venous current seems, at least to some extent, to replace vicariously the interrupted lymph current. According to Emminghaus, so long as the venous current remains unimpeded, the formation of lymph is very trifling—at least within the cutis and subcutaneous layers of fat.

Of all dilated lymphatics, those best known, and most easily observed, are those which run their course superficially. The most frequent seat of this alteration is the lower half of the body, particularly the thigh, the inguinal region, as well as the scrotum and penis, and its usual cause seems to be disease of the inguinal glands.

Quite a remarkable number of such patients have lived, at least for a time, within the tropics; in the case of many of them the glandular swelling was of syphilitic origin; one patient labored under caries of the bones of the foot; another had suffered from typhus two years previously; a woman, whose case is narrated by Buchanan, suffered for years from repeated attacks of fever, combined with inflammation of the leg (phlebitis or lymphangitis?).

In such cases we see lying, just beneath the unaltered or slightly discolored skin, cylindrical or varicose cords from one to several millimetres in diameter; these are usually specially distinct upon the penis, where they form a coronet surrounding the corona glandis and one or two cords extending upwards along the back of the penis. Moreover, particularly on the thigh and lower part of the abdomen, we find a considerable number of vesicles the size of sago-grains, often arranged in rows, which originate in the enlarged cuticular papillæ, whose circumference is sometimes slightly swollen; when these vesicles are pricked, or should they burst spontaneously, which often happens, an opalescent, sometimes opaque, milky fluid escapes from them. A similar fluid is also sometimes discharged from scarcely visible capillary openings in the cutis. These originate in dilatation and rupture of the superficial lymphatic network of the cutis. A copious lymphorrhagia sets in when the larger dilatations are punctured intentionally or from an erroneous diagnosis. Nodular varicosities of the lymphatics, the size of a filbert or apple, or masses of tortuous cords are also seen, particularly in the inguinal region. These are sometimes tense and full, at others softer, usually painless and capable of being diminished by pressure; the lymphatic glands in this situation seem to assist in their formation. They may be confounded with femoral herniæ, abscesses, or other morbid affections of the lymphatic glands.

Such patients have no subjective morbid phenomena; at the most they have a feeling of tension or slight stinging pain in the region of the swelling, which is diminished on the occurrence of a rupture. Slight weakness of the corresponding extremity is also felt, especially when the lymphorrhagia is profuse.

In one case of cavernous lymphangioma of the thigh, which was incised, Gjorgevic observed a fibrinous layer on the coats of the vessels, also redness and infiltrations of the neighborhood, as often as the flow of lymph became obstructed.

Similar dilatations have been observed on the abdominal walls (Fetzer), on the anterior surface of the chest (Coley), and on the neck (congenital, Alter). In all cases they were slowly developed; a declension of the phenomena or a cure was never observed. Dilatation of the lymphatics is not dangerous to life; but should they become injured or ulcerated, inflammation and septic infection of the organism may very readily occur.

The diagnosis is all the more easily made, the more superficial the vessels are. They are chiefly to be distinguished from dilated veins by their form, position, and color; varices in the inguinal region are distinguishable from herniæ by their consistence and by their never containing air; corded lymphatics are also usually to be detected in the neighborhood of these varices. Varices may also be mistaken for tumefied lymphatic glands, but they are softer than simply swollen glands; they are also to be distinguished from abscesses by their slow development and painless character.

The diagnosis is much simplified by the formation of vesicles and the occurrence of lymphorrhagia.

The *treatment* must be partly directed to the prevention of any increase of the dilatation, by uniform pressure, by means of appropriate bandages (elastic—or lacing—stockings, suspensory bandages, etc.), and partly to the production of thrombosis or occlusion of the dilated vessels. With the latter intent, caustics may be employed, or threads soaked in alcohol may be drawn through the vessel. This procedure can only be considered rational when we have only to do with a simple dilatation and when there are sufficient collateral means of removing the lymph. When however the dilatation is the result of a central obstruction to the lymph stream, such measures can only increase the lymphatic congestion, or, at the most, cause the dilatation to be transferred to some other trunk.

Circumscribed dilatations and cavernous lymphatic tumors have been successfully extirpated.

Wernher regards a dilatation of the lymphatic capillaries of the corium as the actual cause of elephantiasis (Arabum). Extravasations of lymph and dilatations of the larger lymphatics are also present. Teichmann could trace, by means of injections, this lymphatic dilatation into the hypertrophied cuticular papillæ.

Gaëtani was able to express sixty per cent. by weight of lymph from a piece of excised skin.—W. distinguishes this true lymphatic elephantiasis from those pachydermatous and connective-tissue hyperplasiæ which are prone to accompany varicosity of the veins, and which resemble it in external appearance. The cause of this dilatation, according to W., can only very rarely be proved to depend upon lymphatic congestion, the result of inflammation of the glands, and the obliteration of the larger trunks. Gravity, as in dilatation of the veins, seems to exert an important adjuvant influence. Next to the lower extremities, the scrotum and mammæ are most frequently affected; hydrocele is not an unfrequent concomitant. The inflammations which attack parts affected with elephantiasis, according to W., are to be regarded as results rather than causes of the lymphatic congestion.

For treatment, compression and ligature of the main artery of the limb have been recommended. Instead of these, W. recommends the less serious measure of methodic compression; but neither of these treatments is said to have any permanent result. Partial excisions and incisions of the skin would seem to be more useful, as by their cicatrization the lymphatics are partly occluded.

Levin observed several times in himself a colossal swelling of the right half of his upper lip, which developed spontaneously in fifteen minutes, and disappeared in half an hour. He fancied this to be a temporary dilatation of the submucous lymphatics (possibly a rupture (?)—Q).

Virchow and Textor saw an enlarged tongue (makroglossia) in a girl two years of age, produced by dilatation of the lingual lymphatics; these had given rise to a series of cavernous spaces.

The existence of narrowing or dilatation of the deeper lying lymphatics can be only suspected, when there is at the same time dilatation of the superficial lymphatics, when the lymphatic glands over a considerable area or when main lymphatic trunks are diseased, or when œdema occurs, which is not explicable by any obstruction to the venous blood-stream.

NARROWING AND DILATATION OF THE DUCTUS THORACICUS.

Narrowing or occlusion of the ductus thoracicus is brought about by inflammation of its coats, by blocking of its lumen with masses of a simply cancerous or tubercular thrombus, by the compression of cicatrices, aneurisms (Laënnec, Bennet), cancerous tumors (Virchow), or tubercular glands within the mediastinum (Otto, Morton). The narrowing sometimes affects only a limited portion of the duct; at others this is obliterated throughout its whole extent.

The duct is often dilated above the narrowed portion; but this is by no means constant, and is all the more likely to be absent the more gradually the obstruction has occurred. In such circumstances a collateral circulation is carried on by one of those re-entering duplications of the thoracic duct (such as Astley Cooper observed in a case of its occlusion), or by one of the hepatic or thoracic lymphatics.

Even ligature of the thoracic duct, which was repeatedly carried out in horses by Dupuytren, is not always, as Magendie reports, followed by the death of the animal from obstruction to the onward flow of the chyle, but is often compensated by the establishment of a collateral flow. Röhrig also observed the flow of chyle to pass collaterally through the neck and thoracic walls in dogs who had their thoracic ducts ligatured. Varieties also occur in regard to the situation in which the lymphatics enter the veins, as Tiedemann and Fohmann tell us. Wutzer saw the thoracic duct terminate by two parallel branches in the vena azygos on the hither side of the occluded part.

Dilatation of the thoracic duct is also brought about, apart from the causes specified, in certain cases of cardiac disease, by the resulting venous congestion (Rokitansky, Petters), as well as by unknown causes. Rokitansky found it once in the body of a lunatic who had been tormented with insatiable hunger. Andral found the thoracic duct occasionally dilated on the thither or central side of a contracted part, just where the collateral vessels entered.

This dilatation usually extends as far as the receptaculum chyli and the mesenteric lymphatics; sometimes also the latter alone are dilated, and only in certain parts of the mesentery; the smaller vessels of the intestines and of the peritoneum may then be dilated in the form of cords and vesicles, and extravasations of chyle may occur within the mesentery and in the walls of the intestines; chylous fluid in considerable quantity has also been observed free within the peritoneal cavity. The chyle within the dilated vessels is usually inspissated or coagulated.

Rokitansky has described a typical case, a woman aged sixty-two, with subcutaneous œdema and very considerable milky effusion into the sacs of both pleuræ and of the peritoneum, with dilatation and hypertrophy of the heart, thickening and shortening of the mitral valve, thickening of the pyloric half of the coats of the stomach, the intestinal villi turgid with a whitish fluid. The subpleural

lymphatics of the lungs were distended, the lacteals and the thoracic duct in the following condition: "They were for the most part, especially from the intestine to the nearest range of glands, mostly nodularly dilated, and stuffed full of a whitish, soapy, unctuous mass, which broke down uniformly in water, to which it communicated a slight opalescent cloudiness. This mass consisted of an agglomeration of fatty granules, crystals of margarine, oil globules, and a few cells, some of which contained nuclei of considerable size. In isolated spots, particularly at the varicose nodosities, the mass was of a more yellowish color, and adhered to the walls of the vessels. At these parts the vessel was covered with a network, and was here and there quite occluded. The meshes of this network contained agglomerations of fatty granules. The glands were not much enlarged, and were here and there speckled with white." The receptaculum chyli and vessels entering it were dilated, their coats thickened; the former was lined internally with a grayish-red reticulated stratum, from which a nodular excrescence here and there protruded, or a filamentous bridge extended. The thoracic duct was occluded by a similar soapy material, partly filled with indurated material, and quite blocked up.

In one case observed by Dr. Fetscherin and myself, the occlusion of the lacteals, when they passed from the intestine into the mesentery, was caused by inflammatory thickening of the latter (Langhans); yet in this case the congestion of the lacteals and the extravasation of the chyle into the coats of the intestine and into the peritoneal cavity were very complete. The right pleural cavity was filled with a milky fluid; elephantiasis of one leg was also present. The latter ailment had occurred several times previously in the family of this young woman, who was thirty years of age.

Symptoms.

Narrowing and occlusion of the ductus thoracicus, like dilatation, often give rise to no symptoms whatever during life, especially in those cases of slow occurrence, in which, as already narrated, both lymph and chyle readily find collateral routes. In other cases, the results of the lymphatic congestion come to light even during life, especially within the area of the lacteals. Ascitic fluid, often to an enormous amount, is effused, and, in spite of efficient removal by paracentesis, it is rapidly renewed. The fluid drawn off is sometimes clear like lymph (Petters), at others turbid from suspended fatty molecules (Oppolzer), or milky (Morton, Marshall Hughes). Both appetite and nutrition were defective as the result of the obstructed absorption; the urine was scanty, partly from the same reason, partly from the presence of the ascites. In one case, related by Roki-

tansky, not only the peritoneal, but also the pleural cavities were filled with chylous effusion. Death resulted partly from the original disease (valvular cardiac disease and venous congestion), and partly from the mechanical results of the effusions mentioned.

Œdema of the lower extremities does not seem to be a necessary result of occlusion of the thoracic duct, at least no mention of it is made in the solitary uncomplicated case related by Morton. For these parts, therefore, other lymph routes, or even the veins, seem to act vicariously.

The treatment can only be palliative; where there is general venous congestion, the cardiac force must be regulated. Any considerable amount of ascites necessitates tapping, which must indeed be generally repeated in a very short time, on account of rapid reaccumulation.

Rupture of the Lymphatics. Lymphorrhagia.

Bergeret, De l'ascite huileuse. Jour. d'anat. et de phys. de Robin. Nov. 1873; Lyon médical. 1874. p. 305.—*Bonet*, Sepulcretum. IV. Sect. VII. Obser. XXIV. p. 360.—*Bourdon*, Journal de Savants. 5 Juin, 1874; Schmidt's Jahrb. 123. S. 274.—*Buchanan*, Escape of chylous fluid from the thigh. Med. Chir. Transactions. XLVI. p. 57. 1863.—*Canton*, Edinb. Med. Jour. Jan. 1860. p. 690.—*Carter*, The British Med. Jour. 1862. March 25; Med. Chir. Trans. XLV. p. 189. 1862.—*W. H. Day*, Lymphorrhœa. Med. Times and Gaz. April, 1869.—*Demarquay*, Mémoire sur un cas de lymphorrhagia. Mém de la Soc. de Chir. de Paris. T. III. p. 139.—*Desjardins*, Gaz. méd. de Paris. 1854. No. 24. Lymphatic fistula.—*Durand Fardel*, Traité des maladies des vieillards. 1854.—*Emminghaus*, Physiologisches und Pathologisches über die Absonderung und Bewegung der Lymphe. Arch. d. Heilk. 1874. S. 308 u. S. 329. Notices of the literature on the subject.—*Friedel*, Beitr. zur Kenntniss des Klimas und der Krankheiten in Ostasien. Berlin. 1863. S. 123.—*Gjorgevic*, Ueber Lymphorroe und Lymphangiome. Langenbeck's Arch. Bd. XII. S. 641. 1870. Literature of the subject.—*Golgi*, Virch. Arch. Bd. 51. S. 568.—*Sigismund Grass*, Ephemerides Germaniæ decur. I. ann. IX. u. X. Vratislav. et Bregae. 1680.—*Guiffart*, Bei Bartholin. Oper. Nov. p. 490.—*Hensen*, Ueber die Zusammensetzung einer als Chylus aufzufassenden Entleerung aus der Lymphfistel eines Knaben. Pflüger's Archiv. X. 1875. S. 95.—*Hilton*, Lancet. 1866. II. p. 37.—*Hoffmann*. Opera Suppl. II. Pars II. 1704. p. 460.—*Hoppe-Seyler*, Pflüger's Arch. VII. 1873, S. 407.—*Marshall-Hughes*, A remarkable case of abdominal effusion (chylous) resulting from

mesenteric tumor. Guy's Hosp. Rep. 1841. p. 297.—*Munson*, The Medical Record. 1 May, 1873.—*Munro, Donald*, An essay on the dropsy and its different species. London. 1756.—*Odenius*, Fall von Lymphorroe, Pachydermia lymphorrhagica. Nord. Medic. Ark. VII. 2. 1874.—*Petters*, Ueber Lymphorrhagia.—*Klebs*, Ueber Lymphangiectasie. Prager Vierteljahrschr. 125. Bd.—*Quincke*, Deutsch. Arch. f. klin. Medic. 1875. Bd. 16. S. 121.—*Rommel*, Ephemerid. Germaniæ decur. II. ann. VIII.—*Rudolphi*, Ueber die Tödlichkeit der Wunden des Brustganges. Casper's Wochenschr. f. d. Heilk. 1835. Nos. 41–43.—*Sattler*, Lymphextravasate. Zeitschr. f. Wundärtze und Geburtshelfer. Heft 2 u. 3. 1871.—*Scholz*, Wiener med. Wochenschr. 1868. No. 63.—*Wienkowski*, Lymphgefässfistel. Wiener. med. Wochensch. 1871. No. 33.

Laceration of the *smaller lymphatics*, as well as of the blood-vessels, is sure to occur in every variety of injury, surgical operation, contusion, etc., although it may not be recognized, on account of the transparency of the lymph; very probably effusion of lymph contributes not a little to the swelling of the tissues following an injury, and the secretions of wounds themselves may also be largely composed of lymph.

From the thinness of the walls of the small lymphatics, we may also readily assume that their rupture may, at least occasionally, be brought about by occlusion of the larger trunks by lymphangitis. The results of this are of no importance, as the lymph effused into the tissue spaces is speedily taken up by other lymphatics.

Even the œdema which occurs, especially in the lower extremities, in hydræmia and general venous congestion, may also be accompanied by rupture of the smaller lymphatics. Possibly it is nothing else than this that occurs when the overstretched skin spontaneously bursts and serous fluid escapes externally.

It is, however, no doubt true that the œdematous fluid which thus spontaneously escapes, or is obtained by puncturing the subcutaneous cellular tissue, is much more watery than the lymph obtained from larger lymphatic fistulæ (*vide infra*). O. Weber, for example, found only 1.9% of solid contents in it. The *larger lymphatics* rupture either as the result of wounds, previous disease, or, more frequently, after they have been from any cause dilated and their coats thinned. Only six cases of rupture of the thoracic duct have been recorded. It was caused by perforation of the left side of the chest with a knife, the

opening of an abscess (Hoffmann), gunshot wound of the spine (Bonet), or the being run over by a carriage (Quincke).

The symptoms of lesion of the thoracic duct were somewhat obscured by the other results of the injury. In three of the cases a white chylous fluid escaped from the wound externally.

In the case related by Hoffmann, this fluid was observed to be thicker or thinner, according as the patient had ingested solid or fluid nutriment. In Bonet's case the escape of the yellowish white fluid commenced fourteen days subsequent to the receipt of the gunshot wound, and lasted, with the exception of an intermission of two weeks, till the death of the patient, which was partly occasioned by fever and exhaustion, and partly apparently as the result of injury to the spinal cord (epileptic attacks and hemiplegia).

In the cases related by Monro and the author, the chyle was effused into the pleural cavity. In the second of these cases, the right pleural cavity was so distended by the effusion that it had to be repeatedly tapped. Nevertheless, the effusion was so rapidly renewed that the patient died on the twenty-first day from interference with the respiratory and cardiac movements. There never was any fever or pain in the side. At the dissection seven litres of chylous fluid were found in the pleural cavity, which was otherwise perfectly healthy. The fatal termination had been indubitably hastened by the withdrawal of such a large quantity of nutritive fluid from the circulation, part of it also having escaped externally. In the case observed by Guiffart, lymph and chyle were effused into the mediastinum, and caused death by suffocation.

According to these cases, the diagnosis of injury to the thoracic duct is most uncertain. It attains probability when pleuritic effusion is developed without inflammatory phenomena or fever subsequent to an appropriate etiological cause, but it only becomes certain when chylous fluid escapes from a wound or is withdrawn by tapping.

The prognosis is usually fatal, yet, according to the second case mentioned above, the continuance of life seems to be possible, and even a cure does not seem to be absolutely impossible, since, from the trifling intravascular pressure, a small wound of the wall may very readily close up.

The treatment can only be symptomatic.

Rupture of the lacteals within the abdominal cavity and effusion of chyle into it has been observed a few times (Munson, Hoppe-Seyler) ; in one of these cases the cause was obscure, and the discovery of the rupture was only made accidentally at

the dissection; in the other there was congestion of the lymphatics from the pressure of a tumor. Precisely similar results, as those arising from the rupture of one of the large lymphatic trunks, may be produced by the rupture of many lymphatic capillaries, which may be caused by congestion of the lacteals by closure of the thoracic duct (Morton, Rokitansky) of one of the larger lacteals (Marshall Hughes), or of several of the smaller lacteals within the mesentery (Langhans; the author). In all these cases there was effusion into the peritoneal cavity, which from its amount produced considerable disturbance, and which after repeated tappings always reaccumulated with considerable rapidity.

The fatal result was partly due to the original disease and partly to exhaustion and the evil results produced mechanically by the ascitic effusion; in several cases the affection lasted from five to six months, and as many as ten tappings were made. Where there is no congestion, or where the ruptured vessel is small, it may heal without producing any marked symptoms; Monro even saw a wound of the receptaculum chyli, in a pig, close very rapidly by the formation of a clot.

For the peritoneum as well as for the pleura the chyle is a perfectly indifferent fluid, which never produces the slightest inflammatory irritation. A part of the effused chyle is, moreover, taken up and removed not only by the blood-vessels, but also by the lymphatics of the peritoneum; at least this seems to have occurred in one case in which (the thoracic duct being perfectly pervious) traces of chyle were also found in the pleural cavity.

All these cases are of considerable physiological interest, inasmuch as they afforded an opportunity of examining human chyle, pure or mixed with some serous transudation. The specific gravity varied from 1007 to 1016, the fatty contents from 0.5 to 1.8 per cent. The fluid also contained a quantity of albumen, traces of peptones (but no traces of any albumen decomposing ferment, Hoppe-Seyler), small quantities of fibrine, cholesterine, diastatic ferment, and only a few lymph globules. On standing, a layer of cream is formed; it resists decomposition for a remarkably long time.

Solutions of continuity in the large peripheral lymphatics, especially on the surface of the body, are very much more fre-

quently observed than ruptures of the lymphatics of the thorax or abdomen. Sometimes they are accidentally injured in performing some trifling surgical operation (venesection, opening of a bubo or of an abscess); at other times dilated lymphatics are incised or punctured from a mistaken diagnosis; and at still other times the coats of the vessel are opened into by ulceration. Superficial thin-walled and dilated lymphatics may be also burst by muscular exertion or an unimportant blow.

Rupture of the lymphatics is most frequently observed in the inguinal region, because dilatation of the lymphatics is most commonly observed in that region.

According to the size of the ruptured vessel, the lymph escapes drop by drop or in a thin stream; in the latter way specially when pressure is made upon a turgid lymphatic. Movement of the limb affected considerably increases the flow of lymph, as in a physiological experiment. In the upright posture the flow of lymph has been frequently observed to be more copious than in the recumbent. But it may also undergo variations from other causes—may even dry up for weeks, and again recur, often very copiously. The effused fluid was sometimes serous, yellowish, slightly opalescent, at others of a milky whiteness. Usually it coagulated spontaneously, though loosely; a few times it was observed to redden on exposure to the air. On microscopic examination it was found to contain lymph globules and fat in fine molecular globules in various quantities, sometimes red corpuscles. The reaction was alkaline, the specific gravity very various.

In 100 parts of the fluid there were:

	Fetzer's Case.	*Desjardin's Case.*
Water	93.68	93.99
Albumen	4.73	4.275
Fibrine	....	0.56
Fat	0.029	0.382
Salts	0.95	1.3
Extractives	0.83	....
Ashes of the alcohol extractives	....	0.73

Traces of iron and sugar.

In Desjardin's case the lymphorrhagia was particularly copious, lasted forty-eight hours, and produced eleven pounds of lymph. In other cases only one pint escaped in a few hours. This depends upon the degree of vascular turgidity and the freedom of anastomosis with the injured vessel; when the outflow is copious, the neighboring tissues or the swollen limb may evidently diminish in size.

The copious fatty contents of the fluid in many of these cases permits the assumption that it contained chyle. And this was specially evident in the case narrated by Hensen of a ten-year-old Brazilian boy afflicted with a scrotal fistula. In it the fatty contents of the fluid could be shown to vary with the amount of fat ingested.

The amount of Fat observed varied from	0.28	to	3.96	per cent.
Albumen.....	" 1.7	to	3.9	"
Cholesterine.....	" 0.018	to	0.102	"
Salts.....	" 0.643	to	1.09	"
Water.....	" 91.	to	96.3	"

Also, small quantities of sugar and fatty acids.

It remains unexplained how in this case the chyle found its way into the peripheral lymphatics; even on the supposition of considerable dilatation of the anastomosing branches, there must have been in part of them a lymph current in a direction contrary to the normal, which could only be made possible by insufficiency of the valves, and which was probably caused by gravity.

After ligature of the thoracic duct in dogs, Röhrig[1] could frequently trace chylous congestion into the lymphatics of the kidneys and muscles, especially in aged animals.

Lymphorrhœa only influences the general health when it lasts for a very long time and is very profuse; it debilitates like every other loss of the vital fluids; and the patient has a feeling of weakness, especially in the limb affected. Evil results from inspissation of the blood, such as Lesser observed when experi-

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[1] Ber. d. k. sächs. Ges. d. Wissenschaft. 1874.

menting upon animals, can hardly occur in men who can eat and drink.

The *prognosis* in regard to life is therefore generally favorable. Should the discharge come from a recent wound in a normal vessel, a cure is generally possible; but should it come from a vessel already dilated, a permanent fistula usually results, because from the persistent obstruction to the onward flow the lymph escapes under considerable pressure, and its constant flow hinders the closure of the opening.

The *treatment* in recent cases must be confined to elevating the part affected, and keeping it as much as possible at rest, at the same time compressing the wound. When this is not sufficient, a variety of caustics have been applied to the wound. A similar procedure has been adopted in regard to the fistulæ of dilated lymphatics. These measures have been adopted with the view of producing the complete occlusion of the affected vessel.

Large losses of lymph necessitate the use of appropriate nutritives, ultimately of iron and bitter tonics.

CHYLURIA.

Golding-Bird, Lond. Med. Gaz. Oct. 1843.—*Carter*, On the connection between a local affection of the lymph-system and chylous urine. Med. Chir. Transactions. XLV. 1862, p. 289, contains notices of literature.—*Eggel*, Deutsch. Archiv für klin. Med. XI. S. 540. 1873. Literary notices here also. S. 427.—*Pandurang Gopal*, Med. Times and Gaz. 1873. June, p. 651.—*Amyot*, Ibid. 1873, July.—*F. H. Welch*, Lancet. I. 1873.—*Cunningham*, Lancet. I. 1873.—*T. R. Lewis*, On a hæmotozoon inhabiting human blood; its relation to chyluria and other diseases. Calcutta. 1872: quoted in Med. Times and Gaz. March 22d, 1873.—*Do.*, The pathological significance of nematode hæmatozoa. Calcutta. 1874. Med. Times and Gaz. 1875. I. p. 173.—*W. Oehme*, Fall von intermittirender Chylurie. Deutsch. Arch. für klin. Med. XIV. S. 262. 1874.—*J. Crevaux*, Journal de l'Anat. et de la Physiol. XI. 2. p. 173. 1875.—*Wm. Roberts*, On renal diseases. London. 1872. Bibliography.

In connection with lymphorrhagia I have now to speak of *chyluria*, inasmuch as it most probably is nothing else than an escape of chyle into the urinary passages. In this affection the urine has all the appearance of milk, and, like chyle, on standing it throws up a layer of cream. Very often a loose coagulum

separates on standing, and this is sometimes somewhat colored by red corpuscles. In one case, related by Carter, the white or pale red coagulum is said to have become distinctly reddened on exposure to the air.

Microscopically we find an innumerable number of excessively minute (fat) globules as in chyle, sometimes red, more rarely white blood corpuscles, no renal cylinders. The reaction of the recent urine is acid.

Besides the normal constituents of the urine, we find also the following substances (the figures are taken as a sample from the case described by Eggel).

Albumen coagulable by heat..... 0.32—0.63%

Fat..... } 0.2—0.69.
Cholesterine..... }

Traces of lecithin; fibrine; no sugar.

When shaken with ether, the urine is freed of fat, and becomes quite clear.

Quite recently Lewis has found in the urine the microscopic animalcule presently to be described, which he has named the "filaria sanguinis hominis."

Etiology.

This disease occurs endemically in certain tropical districts (Brazil, Mauritius, Isle of Bourbon, Bombay, and the West Indies) and also in South Carolina and Queensland, often side by side with the hæmaturia, which also prevails endemically in these districts; moreover, the patients in these parts pay but little attention to it. In Europe also it has been (with the exception of a case related by Prout, one quoted by Golding Bird, another by Cubitt, narrated by Beale, a fourth recorded by Roberts, and a fifth by Oehme) exclusively observed in those individuals who have passed at least a portion of their lives in the tropics. It is very remarkable that it has occasionally been first developed a long time (even so long as eighteen years) after leaving the tropics. It occurs chiefly in middle life, rarely in children or in advanced life [of thirty cases collected by Roberts three were

under twenty, seven between twenty and thirty, eleven between thirty and forty, six between forty and fifty, and three over fifty. The youngest case is mentioned by Prout—a male infant of eighteen months ;—the oldest by Quevenne and Crevaux, in which the patients were respectively seventy-eight and eighty.—*Tr.*]. Men and women seem to be nearly equally affected [of the thirty cases above referred to, nineteen were males and eleven females. When the disorder is endemic, it is said to be more common among women than among men.—*Tr.*]. According to Crevaux, the disease is most apt to occur during the hot season of the year.

Symptoms.

Apart from the condition of the urine, the morbid symptoms are generally trifling. A few patients complained of pain in the region of the kidneys, generally on both sides, more rarely on one side only, sometimes radiating towards the scrotum and upper part of the thigh. Dysuria is also sometimes produced by the spontaneous coagulation of the urine in the bladder.

It is only at the commencement of the disease that the general health is somewhat disturbed (bodily weakness and mental depression, the urine then also usually contains more blood); subsequently the organism seems to get used to the loss of the vital fluids.

The variations in the conditions of the urine are remarkable, as in the space of a few hours it may become clear and again milky. Bodily movement and the ingestion of food favor the occurrence of chylous urine; whilst, by fasting and continuance in the horizontal posture, it becomes clear.

For this reason the urine passed during the day is usually milky, and that passed during night normal; in Oehme's case this was reversed, and continued to remain so even when the patient kept his bed for forty-eight hours continuously.

Quite independent of these daily variations, the disease also varies within yet longer intervals of time, so that it may disappear for months or years, and again suddenly reappear. Crevaux quotes the case of a lady of the Isle de Bourbon, aged eighty,

who for fifty years had suffered from chyluria and hæmaturia. Usually the disease at last ceases spontaneously.

According to Lewis, deafness, diarrhœa, chronic conjunctivitis or more serious affection of the eyes, sometimes transitory swellings of the face and of the extremities, occur in connection with chyluria. Patients affected with chyluria often suffer simultaneously from elephantiasis of the lower extremities and of the scrotum, or from dilatation of the lymphatics and lymphorrhagia in the inguinal region. In one case of Carter's, chyluria sometimes alternated with lymphorrhagia or with swelling of the lymphatic glands.

Niemeyer and Eggel found in one case fatty molecules (from five to ten times more numerous than the red blood globules) in blood removed by cupping-glasses ; a French observer (Guibourt) has made a similar observation, but not in every case.

Lewis drew blood from the fingers by pricking with a needle, and found in it precisely the same little worms as in the urine.

Nature of the Disease.

Up to quite lately we were all in the dark as to the true cause of this disease, which is so rare with us, and in its course is so "capricious," as Rayer has termed it. The absence of any perturbation of the nutrition made any general disturbance of the interchange of material very problematical, and the occasionally perfectly normal character of the urine permitted the exclusion of any serious disease of the renal parenchyma. In both respects also the rare post-mortem examinations gave only negative results. The most probable explanation of the phenomena seemed to be the assumption of a lymphorrhagia into the urinary passages, because the condition of the urine would be thus efficiently explained, and because other lymphorrhagias intermit in a similar manner, and are also similarly influenced by corporeal position, movement, and the ingestion of food. Anatomical proof of such a supposition was certainly wanting, and it was uncertain whether the admixture of chyle took place in the kidneys or lower down in the urinary passages.

A perfectly new light was thrown upon this disease by the discovery in such patients, by T. R. Lewis, of the embryo of a round worm, which he named the *filaria sanguinis hominum.* This is 0.35 mm. long, and 0.0075 broad, is contractile, finely striated transversely, and transparent, with a flat ribbon-like appendix at each end ; these animals are sexually immature. They exist in numbers in the blood of chyluric patients during life ; Lewis estimates the total number in the blood of one patient to be 140,000. The same animals are also very frequently found in the urine, inclosed within coagula ; they have been occasionally found in the (cholera) stools, and in the milky secretion in conjunctivitis. On dissection they were also found in the kidneys, in the supra-renal capsules, in the parenchymatous tissues, as well as in all the arteries and veins. The renal pyramids had a tallowy appearance, and along the urinary canals there were visible somewhat varicose tubuli with an oily lustre, which remained unaltered on the addition of warm ether, and seemed to be occluded lymph or blood vessels.

According to Lewis, chyluria and many of its accompanying symptoms, probably also the endemic elephantiasis of the tropics, are caused by the presence of this parasite. Further inquiry must however determine the special manner in which this is brought about ; it seems possible that the great numbers of these animals circulating in the blood occlude the blood-vessels and lymphatics, or that the encapsuled animals (as in dogs, etc.) form small tumors lying along the blood-vessels, and rupture of the capillaries occurs as the result of mechanical congestion.

The development and migration of these animals has not yet been investigated. In the blood of the Paria dogs, according to Lewis and others, there are similar filaria embryos, which he and Welch at first supposed to be identical with those of man, whilst Cunningham, and subsequently Lewis himself, regarded them as of a different species. The mature specimens of this canine filaria are 25–90 mm. long (the females twice as long as the males), and they are found in tumors the size of a pea or walnut lying along the œsophagus and the thoracic aorta; they are also found within the coats of the aorta, in small tumors like tubercles, which now and then lead to bulging and ulceration of the intima; finally, they are also found in the lymphatic glands at the base of the heart. The females contained only ova, never free embryos. [Dr. Cobbold regards these nematoid hæmatozoa as identical with Bilharzia.—Vide Brit. Med. Journ. June, 1876, p. 780.—*Tr.*]

Though, according to these investigations, it seems almost certain that most cases of chyluria are caused by this hæmatozoon, yet it is always possible that in isolated cases, particularly in indigenous cases (such as that of Oehme, etc.), there may exist other causes of lymphorrhagia into the urinary passages, just as we also know that elephantiasis and lymphorrhœa may certainly occur quite independently of the presence of these parasites.

Treatment.

Before all things, it is needful to supply appropriate nourishment to replace the loss of the nutritive fluids. In opposition to this, Bouchardat, who supposes that there is an abnormal amount of fatty matter in the blood, recommends a diet poor in fat, carbohydrates, and alcohol. Such a regimen is said to be commonly employed in the Isle de Bourbon. There also cold bathing is employed, and a change of climate recommended as soon as possible.

In regard to medicaments, gallic acid has been employed with good results by Bence-Jones, in quantities of from one to two drachms per day. Besides this, other astringents, such as quinine and preparations of iron have also been used. Rayer gave tincture of cantharides in doses of from six to ten drops in the day. Harley recommends the internal use of iodide of potassium, and also its injection into the bladder, fifteen grains to three ounces of water, to kill the worms; oleoresin of male fern has also been used as an injection. Salesse employed copaiba.

DISEASES

OF

THE PERICARDIUM.

BAUER.

DISEASES OF THE PERICARDIUM.

History.

Anatomical changes in the pericardium, especially pericardial exudations, were recognized long before [1] the diseases of the heart itself were systematically described. Galen described pericardial effusions which he found in animals and suspected in men. Sal. Diversus, Forestus, and Rondelet saw and wrote about the pathological changes in pericarditis; Guarinoni and Jacutus Lusitanus observed them associated with pleuritis. The statements, however, concerning the frequency of these diseases and the idiopathic inflammatory nature of the effusions, were incomplete, and the description of the disease associated with these changes does not altogether correspond with them. Thus Rondelet described the disease with symptoms of fever, dyspnœa, pain under the sternum, and attacks of syncope. So, likewise, Riolan described it as follows: "Pericardium ipsum particeps esse potest inflammationis dolorificæ et valde periculosæ quia vicinum cordi, ac proinde patitur frequentes syncopas, tumque pulsus celerior, febris auctior, sitis vehementior quam in pleuritide aut inflammatione pulmonum. Sæpe in eo copiosus humor colligitur qui suffocationem adfert et cor obruit. Si non possis exhaurire istud per hydragogen, licetne terebra sternum aperire intervallo pollicis a cartilagine xiphoide?" [2]

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[1] The fables about the occurrence of hairy hearts in men distinguished for bravery and daring, robbers, etc., such as Leonidas, Lysander, Aristomenes, and others, are doubtless based on the discovery of stringy fibrin deposited on the heart.—*Haller*, Elem. Phys. Tom. I.

[2] Ench. Anatom. Lib. III. c. 4.

In the eighteenth century, when the knowledge concerning heart diseases made such rapid strides, we find, in the works of the authors on that topic, numerous anatomical observations of diseases of the pericardium as well. Vieussens often met with adhesion of the heart to the pericardium at autopsies, and assigned certain functional disturbances during life to the existence of this condition; whereas, in earlier times, it had repeatedly been considered to be a congenital defect.

Albertini rightly appreciated the difficulties of a symptomatological recognition of pericardial effusions; Morgagni arrived at the same conclusions, both by reviewing the work of others and by his own personal investigations, and believed, on account of the difficulty of diagnosis, that the day was yet remote when we should have recourse to the puncture of the pericardium recommended by Riolan. Senac also could not overcome the difficulties in diagnosis, which the obscure and inconstant symptomatology of these diseases made apparently insurmountable.

In hydro-pericardium, Senac thought that he recognized an undulatory movement between the third and fifth ribs; and although this sign proved to be erroneous, yet it was the commencement of the study of objective signs. Corvisart, who indeed did not see the above sign, but states that he felt it, first made the distinction between inflammatory exudations and dropsical effusions, which before his time had been generally confounded even by Senac and Morgagni, but he was unable to lay down any fixed rules for diagnosis. Auenbrügger first gave some accurate physical signs, such as bulging of the præcordial region and also increase of percussion dulness. Nevertheless Laënnec doubted the possibility of diagnosing pericarditis with certainty. These difficulties in diagnosis disappeared after the discovery of the pericardial friction sound by Collin (1824), and, thanks to the numerous works which followed close upon this, pericarditis at the present day can be classed among those diseases whose physical diagnosis is most complete and sure. They are mostly investigators in the field of heart diseases in general to whom we are indebted, and above all we should mention Louis, Bouillaud, Mayne, Latham, Hache, Gendrin, Graves, Stokes, and Skoda. In most modern times we have gained numerous facts

which aid in the recognition of pericardial diseases and especially in the study of adherent pericardium ; yet these advances belong not to history, but to the present day.

Absence of the Pericardium.

Baillie, Transact. of a Societ. for the Improv. of Med. and Chir. Knowl. Lond. 1793. I.—*Breschet*, Rep. d'anat. et de phys. path. I.— *Wolf*, Rust's Mag. Bd. 23. —*Curling*, Med. chir. trans. Vol. 22.—*Otto*, Selt. Beobachtg. Bd. II.—*Baly*, Lond. Med. Gaz. 1851.—*Rokitansky*, Handb. 2. Bd.—*Powell*, *R. Douglass*, Case of pneumothorax with congenital opening in the peric. Trans. of the Path. Soc. XX. p. 29.—*P. Bert*, Insuffisance du peric. Gaz. de Paris. 1866. No. 33. Theilweiser Defect bei einem Hunde.—*A. Weissbach*, Angeborner Defect des Herzbeutels. Wiener med. Wochenschr. Aug. 26. 1868.—*Virchow-Hirsch*, Jahrb. f. 1869. I. S. 168.

Absence of the pericardium occurs in ectopia cordis and is then generally only partial, and we find in that anomalous position of the heart an opening or fissure in the sac through which the heart protrudes. In other cases, the heart and the left lung may both lie in a single serous sac common to both ; in such cases the heart is covered by the visceral layer of the pericardium, and at the origin of the great vessels there are usually found rudimentary portions of the parietal layer in the shape of fringe-like reduplications. The latter condition gives rise to no symptoms during life, as the case of Baly shows. Slight deficiencies in the pericardial sac are occasionally observed at autopsies.

Formation of Diverticuli.

Cruveilhier, Anatom. path. Livr. 20. pl. 2.—*Hart*, Dubl. Journ. of Med. Sc. 1837, July.—*Rokitansky*, Handb. II. S. 232.— *Luschka*, Die Structur der serösen. Häute. S. 73.—*Bristowe*, Divertic. from the peric. Trans. of the Path. Soc. XX. p. 101.

The formation of diverticuli is rarely observed ; the development of such hernia-like sacs is due to the outward pressure of fluid within the pericardium : portions of the fibrous layer yield and separate or become thin and allow the serous layer to pro-

trude. Most of these cases have a chronic development. In a case related by Cruveilhier the bulging was occasioned by the distention of the pericardial sac with blood, in consequence of rupture of the left ventricle. The size of the diverticuli which have been observed was usually small, yet one in a case related by Hart contained from three to four ounces of fluid. The opening of communication with the pericardial sac may be wide or narrow. Usually the formation of diverticuli is unrecognized, and only the pericardial effusion which caused it is made out. Yet we might conceive it to be possible that a large diverticulum, capable of containing three or four ounces if in a favorable position, might be accessible to physical investigation, even though it escape diagnosis.

Tendinous Spots (Milk Spots).

Bizot, Rech. sur le cœur etc. Mém. de la soc. med. d'observ. de Paris. Tom. I. 1836. p. 347.—*J. Reid*, Cyclop. of Anat. and Physiol. Vol. II. London. 1839. Art. "Heart" and *R. B. Todd*, ibid. Art. "Abnormal conditions of the heart." —*James Paget*, On white spots on the surface of the heart. Med.-chirurg. Transact. II. S. V. Vol. London. 1840.—*Hasse*, Anat. Beschreibung der Krankheiten der Circulations- u. Respirationsorg. Leipzig. 1841. S. 142.—See, in addition, the various text-books on Path. Anat. and on Dis. of the Heart, especially that of *Friedreich*, Krankh des Herzens. Virchow's Path. u. Therapie. V. 2.

We use the term tendinous spots or milk spots on the pericardium (maculæ tendineæ or lacteæ, also insulæ) to describe the circumscribed whitish cloudy appearances and thickenings of the pericardium, which are so frequently observed in the autopsies on adults, that Baillie, Soemmering, and others believed that they were not of pathological origin. These are most frequently met with in people of advanced age, while in youth they are less frequently seen, and in children very rarely. They are oftener found in men than in women.

Bizot has made a collection of autopsies in one hundred and fifty-six individuals, which in regard to age and sex gave the following proportions:

Men.

From the 1st	to the	17th	year in	16	persons	0	cases.
" "	"	18th	"	39th	"	24	"	8	"
" "	"	40th	"	79th	"	32	"	23	"

Women.

From the 1st	to the	22d	year in	31	persons	0	cases.
" "	"	23d	"	39th	"	23	"	5	"
" "	"	40th	"	89th	"	30	"	9	"

Accordingly in one hundred and fifty-six individuals tendinous spots were found forty-five times. If an estimate were made from autopsies of adults only, the proportion would naturally be much greater.

Bizot denies their occurrence in children altogether, which certainly is incorrect (Foerster, Hodgkin).

The tendinous spots are formed only exceptionally on the parietal layer of the pericardium; most frequently they are on the visceral layer, and in by far the greater number of cases on the anterior face of the right ventricle, along the coronary arteries. Yet they do occur on other parts of the heart, and at the origin of the great vessels, either singly or in groups. These tendinous spots have no clinical importance whatever, since they are accompanied by no functional disturbance, and their presence cannot be recognized by any symptom during life.[1] There is a certain interest attached to them, however, since some authors have regarded them as the remains of inflammatory processes, traces of antecedent pericarditis (Paget, Rokitansky, and others). Others again deny their inflammatory origin, and claim that they are simply fibrous thickenings, such as occur on serous membranes elsewhere, especially on the arachnoid.

Bizot distinguishes two forms of tendinous spots, and considers one only the result of changes accompanying age; this form is by far the most frequent. The other rarer form is of inflammatory origin, and consists of granular or flat patches, opaque, whitish, and of a firm consistency, which may be pulled off, without tearing, from the serous layer, with which they are loosely connected.

It cannot be denied that a circumscribed pericarditis with

[1] It is alleged, however, that tendinous spots sometimes occasion a friction murmur; but we could hardly believe that most tendinous spots were capable of producing a friction sound, and observation supports that belief.

surrounding thickening of the serous coat may terminate in a "milk spot," but, in the majority of cases, these changes are to be considered not as inflammatory, but rather as circumscribed hyperplasiæ of connective tissue and scleroses. Thus, the change consists less in a new growth of connective-tissue fibres than in a consolidation of those already existing. It is not possible to lay down sharp lines of division between these processes and that which is truly inflammatory,[1] but in a clinical point of view it is important to remember that the formation of most of these spots is the result simply of a chronic hyperplastic process. It may be true, as Friedreich asserts, that these tendinous spots arise from a continual mechanical irritation of the surface of the heart, and accordingly are most frequently found on those parts of the heart which, uncovered by lung tissue, are continually brought into contact with the more resisting portion of the thoracic wall.

As opposed to the idea of the inflammatory origin of these milk spots, we might mention the fact that they are rarely accompanied by adhesions.

Chambers found in one hundred and sixty such cases that only three times did adhesions exist simultaneously ; and Friedreich confirms these proportions, as opposed to Paget.

Inflammation of the Pericardium

J. B. Morgagni, De sed. et causis morb. per anat. indag. lib. II. cp. 16. 17. and in other parts of the work.—*Riolanus*, Ench. anat. path. l. III. c. 4.—*Haller*, Element. phys. Vol. I. p. 285 and Add. ad elem phys. p. 128.—*Senac*, De la struct., de l'act. et des malad. du cœur. Paris. 1749.—*De Haen*, Rat. med. Tom. XIV. p. 30.—*Trécourt*, Chirurg. Abhandl. u. Wahrnehm. aus dem Französ. Leipzig. 1777.—*Romero*, Dict. des. sc. méd. Tom. XI. 1819.—*Stoerk*, Annal. med. Vol. II. p. 232, 264.— *Watson*, Philos. Trans. 1777.—*Savary*, Sur la péricard. aigue. Diss. Paris. 1819.—*J. G. Walter*, Observ. anat. p. 63 ; Mus. anat. Vol. I. p. 148.—*Portal*, Mém. sur plus. mal. Tom IV. u. Cours d'anatomie méd. Tom. III. p. 24.—*Lemazurier*, D. sur la péricardite. Paris. 1810.—*J. C. Boullier*, D. sur la difficulté du diagnostic de la péricard. Paris. 1812.—*Roux*, Collect. quaedam de cardit. exsud. Lips. 1819.—*Hubert*, Dict. de méd. Ed. 2. Art.

[1] See *Rindfleisch*, Patholog. Gewebelehre.

Pericarditis.—*C. C. Diergardt*, De pericard. acut. diag. Bonn. 1828.—*Collin*, De divers. méth. d'exploration de la poit. Deutsch von *Bourel*. Köln. 1828. —*Louis*, Mém. sur la péric. Révue méd. Janvier, 1824; Rech. anat. path. Paris, 1826.—*Andral*, Clin. méd. Tom. I. u. Tom. III.—*J. Abercrombie*, Trans. of the med. chir. Soc. of Edinb. Vol. I.—*Bouillaud*, Art. "Péricardite" in Dict. de Med. et Chirurg. 1834; Rech. sur le rhum. artic. aiguë. Paris. 1836.— *Sander*, Hufeland's Journ. Bd. 51. 1820.—*Seidlitz*, Ueber Pericard. exsud. sang. Hecker's Ann. II. 1835.—*Désclaux*, Ess. sur la péric. aiguë. Thèse. Paris. 1835. Arch. gén. de méd. 1836.—*J. P. Latham*, Lond. Med. Gaz. Vol. III. p. 209.— *Adams*, Dubl. Hosp. Rep. Vol. IV.—*Hache*, Mém sur la péric. Arch. gén. de méd. II. Ser. Tom. IX. Paris, 1835.—*Brissault*, Ess. sur la péric. etc. Strassbourg 1826.—*Stokes*, Lond. Med. and Surg. Journ. Sept. Oct. Dec. 1833.—*Stiebel*, Monog. card. et pericard. acut. etc. Franc. ad M. 1828.—*R. Mayne*, Dubl. Journ. Vol. VII. 1835. May u. Schmidt's Jahrb. Suppl. I.—*Rayer*, Arch. gén. de méd. Tom. I. p. 521.—*Hughes*, Guy's Hosp. Rep. 1836. No. 1; Schmidt's Jahrb. Bd. XVI. Lond. Med. Gaz. Vol. XIX. Schmidt's Jahrb. Bd. XVII.—*J. Watson*, Lond. Med. Gaz. 1836. July.—*Roots*, St. Thomas's Hosp. Rep. No. 4. June, 1836. Lond. Med. Gaz. 1836. Nov.—*R. W. Smith*, Dubl. Journ. Vol. IX. p. 418.— *Heyfelder*, Heidelb. klin. Annal. X. 1834. Schmidt's Jahrb. Suppl. I. Stud. im Gebiete der Heilwissensch. Bd. I. S. 208.—*Richter*, Preuss. Vereinsz. 1834. No. 47.—*G. Corfe*, Lond. Med. Gaz. 1835. June.—*Maisonneuve*, Arch. gén. 1834. Apr.—*Ebers*, Hufeland's Journ. St. 7. 1837.—*Bright*, Cases of spasmodic disease accompanying affect. of the pericard. Med. chirurg. trans. Vol. XXII. Lond. 1839.—*Karawajeff*. Preuss. Vereinszeit. No. 52. 1840.—*Taylor*, Lond. med. surg. trans. Vol. XXVIII. 1845. Med. Tim. Vol. XXI. 1850.—*Aran*, Arch. gén. 1844. Avril u. Gaz. des Hôpit. No. 38. 1858.—*Brockmann*, Holscher's Annal. Jahr. V. H. 3. 4.—*Roger*, Arch. für phys. Heilk. V. Bd. 1846.—*Kyber*, Bemerkungen über den Morb. cardiac. Med. Zeitg. Russlands. 1847. Nos. 20–25.— *Schwank*, De haemoperic. scorbut. Diss. Dorpat. 1847.—*Sibson*, On pericarditis. Lond. Med. Journ. Oct. 1849.—*Chambers*, Med. chirurg. Rev. Oct. 1853.—*Lalor*, Dubl. Quart. Journ. of Med. Sc. Vol. XIII. Sept. 1852.—*Eisenmann*, Die Familie rheuma. Bd. III. S. 72.—*Civati*, D. de pericard. Ticin. 1841.—*Skoda*, Oesterr. medic. Jahrb. 1841. März.—*Graves*, A syst. of clinic. med. Dubl. 1843. Clinic. lectures. Lond. u. Dubl. 1848.—*Skoda* und *Kolletschka*, Ueber Pericarditis in path. und diagnostischer Beziehung. Oesterr. med. Jahrb. N. F. XIX. 1839.—*King Wilkinson*, Lancet. 1845. Nov.—*Barthez* et *Rilliet*, Mal. des enf. I. —*Baillie*, Engravings, fasc. 1. pl. 1.—*Albers*, Atlas III. t. 1. 2; Erläuterungen III.—*Cruveilhier*, Anat. path. livr. 16. pl. 2. livr. 30. pl. 4. livr. 40. pl. 4.— *Froriep*, Klin. Kupfertafel. t. 61.—*Virchow*, Acute Fettmetamorphose des Herzfl. bei Pericarditis, dessen Arch. XIII. 1858.—*Gerhardt*, Zur Casuistik der Herzkrankh. Würzb. med. Zeitschr. II. 1861. S. 136. und Ueber einige Formen der Herzdämpfung. Prager. Viertelj. Bd. 84. 1864.—*Duchek*, Zur Aetiologie der Pericard. Wien. med. Wochenschr. No. 15. 1859. und Klinische Vorträge über

Herzkrankh. Allg. Wien. med. Zeitung. Nos. 24–32. 1862.—*Ch. Hirsch*, Klinische Fragm. 2. Abth. Königsberg. 1858.—*Gairdner*, On pericarditis. Edinb. Med Journ. April, 1859. p. 904. Febr. 1860. p. 736. January, 1861. p. 626.—*Oppolzer*, Ueber Pericarditis. Allgem. Wien. med. Zeitung. No. 44. et seq. 1861. und Spitalzeitung No. 19. 1862.—*Leudet*, Recherch. anat. path. et cliniques sur les péric. second. Arch. gén. de méd. Juil. 1862.—*Kirkes*, On peric. conseq. on pyaemia. Med. Tim. and Gaz. Oct. Nov. 1862.—*Kirby*, Rep. of a fatal case, etc. Lancet. Jan. 1860.—*F. Roth*, Zur Casuistik der Herzbeutelentzündung. Würzb. med. Zeitschr. III. 1. 1863.—*Kaulich*, Krankh. der Kreislaufsorgane. Beob. auf der Klinik von Prof. *Jaksch* zu Prag, während 1857–59. Prager Viertelj. LXXIII. 1862.—*Cejka*, Aus dessen lit. Nachlasse. Prag. Viertelj. 1. 1863.—*W. D. Moore*, Sur un cas sing. de péric. Gaz. méd. de Paris. 1863. No. 31. u. Dubl. Med. Press. 1862.—*Kerschensteiner*, Ueber Pericard im kindl. Alter. Bayr. ärztl. Intelligenzbl. 2. 1863.—*Radcliffe*, Extens. pyoperic. and emp. etc. Lancet. Aug. 1863.—*Ormerod*, On rheumatic and non-rheumatic pericarditis. Med. chirurg. trans. Vol. XXXVI. Lond. 1853. and Med. Tim. and Gaz. Aug. 1864.—*Trousseau* et *Lassègue*, De la paracentèse du péric. Arch. gén. de méd. Nov. 1854.—*Günzburg*, Dessen Zeitschr. VI. H. 2 u. 3. 1855.—*Bamberger*, Beitr. zur Phys. u. Path. des Herzens. Virch. Arch. IX. 1856. S. 348.—*Vernay*, Sur la ponct. du péric. Gaz. hebd. No. 45. 1856.—*Hamernjk*, Grundzüge der Phys. u. Path. des Herzbeutels. Prag. 1864.—*Mettenheimer*, Ueber pericard. Reibungsgeräusche ohne Pericarditis. Arch. für wissensch. Heilk. 1865. No. VI.—*Guéneau de Mussy*, De cert. sign. de la péric. Gaz. des Hôp. 1865. No. 49.—*Armand Debest de Lacrousille*, De la péric. hemorrhg. Union méd. 1865. 1.—*Traube*, Ges. Beiträge. II.—*Thore*, Arch. génér. 1856. Fevr.—*Petters*, Prager Viertelj. 50. Bd.—*Law*, Dubl. Quart. Journ. 1856. Aug.—*H. Kennedy*, Edinb. Med. Journ. No. 1858.—*Page*, Haemotoperic. and compl. fatty degen. of the heart, sudden death. Lancet. 1863.—*R. E. Thompson*, On rheumat. pericard. St. George's Hosp. Rep. IV. pp. 31–44.—*Hambursin*, Bull. de l'acad. de méd. Belg. 1870. IV. p. 990. Ibid. p. 930.—Of the different treatises and text-books on Diseases of the Heart, consult, in particular, *Bamberger*, *Duchek*, *Friedreich*, l. c.

Pathogenesis and Etiology.

Inflammation of the pericardium may occur both on the visceral and on the parietal layer, and throughout its whole extent, or the process may attack only single portions of the surface of the heart. Accordingly we distinguish two varieties, viz., *diffuse* and *circumscribed* pericarditis.

In most cases the whole pericardium is affected. The existence of the opposite condition is proved, however, if we include the "milk spots" amongst inflammatory

processes. Still it will be difficult to lay down an accurate numerical proportion between the two forms, since during life an opinion concerning the extent of the inflammation in many cases will only be an approximate one, and since a circumscribed pericarditis may run its course without any symptoms. Thompson found in rheumatic pericarditis a proportion of 94 to 38.

The circumscribed inflammation may occur on any part of the heart; its seat of election, however, is at the base, at the origin of the great vessels.

As regards the course of the disease, we can divide them into *acute* and *chronic* cases, without, however, making a sharp boundary line, or a decided distinction between the two categories. A further subdivision is based on the quantity and character of the exudation; thus, according to its nature we speak of a pericarditis as *fibrinous*, *sero-fibrinous*, *hemorrhagic*, or *purulent*.

Inflammation of the pericardium rarely attacks a man in previous good health in a *primary idiopathic* manner, and even amongst those cases which are apparently idiopathic doubtless many, as von Bamberger has likewise remarked, are secondary, and the original disorder has escaped observation. Perhaps there are sometimes pathological changes in the muscular tissue of the heart itself which lead to a secondary pericarditis.

Here we might readily be deceived. Inflammations of traumatic origin are generally reckoned amongst the idiopathic cases, and of these numerous examples are found in the literature of the subject, of the most various kinds, according to the character of the injury received. Thus, cases are found depending upon a blow or jar against the breast, upon crushing of the thorax, following upon penetrating wounds, bullet wounds, and the penetration of various foreign bodies.

A wonderful case of this kind was observed by Buist, and is cited by Friedreich: a pericarditis was caused by swallowing a set of false teeth, which remained fast in the œsophagus, and the gold rim had bored its way into the right portion of the pericardium posteriorly.

With regard to the other causes of primary pericarditis we have no accurate information. Catching cold is sometimes as-

signed as a cause, still we can hardly satisfy ourselves with this explanation. Some of the patients of this kind whom I have observed were decided drinkers, others were living under circumstances of much privation and exhaustion.

All statements agree as to the infrequency of primary pericarditis. For example, out of 89 cases of pericarditis, Duchek observed only one case; Bamberger found only 5 out of 63 cases, and of these one was traumatic. Out of about 3,000 patients, which is the usual number in each year in the medical division at Munich, I have seen yearly two or three cases of spontaneous pericarditis.

The *secondary* affection, on the other hand, as Corvisart taught us long ago, is a very frequent disease. Of the diseased conditions with which pericarditis is most frequently associated, *polyarthritis rheumatica* certainly holds the first place, although up to the present time we have not been able to make the connection clear between inflammation of the pericardium and articular rheumatism.

The dependence of pericarditis upon rheumatism has been recognized since the time of Pitcairn (1788). The statements regarding the frequency of this complication of articular rheumatism differ within very wide limits (see Senator's article in one of the later volumes of this cyclopædia). Certainly the figures of Bouillaud, Williams, and others concerning the frequency of heart affections, and particularly pericarditis, in the course of this disease are far too high, for, according to them, pericarditis occurs in half the cases, or even more. These exaggerated statements depend partly upon erroneous diagnoses. It cannot be denied that the frequency of the occurrence of pericarditis in the course of rheumatism is not always the same, but I think we should be near the truth if we should say that from 16 to 20 per cent. of the cases are complicated by pericarditis (Leudet, Chambers, Duchek, Thompson, Lebert, and others).

The relative percentages of cases of pericarditis of rheumatic origin, and those in which the pericarditis was due to other causes, naturally vary very greatly; thus Ormerod observed 71.7 per cent., and Chambers only 13 per cent of cases of pericarditis associated with rheumatism, and the statistics of others hold a middle place between these two extremes; von Bamberger asserts that about 30 per cent. are of rheumatic origin. I believe that we cannot lay down any percentage which will hold everywhere, since rheumatism in a severe form depends, in its frequency, upon local causes.

Wherein the cases of articular rheumatism, complicated by pericarditis, differ otherwise from cases without such complication, we do not certainly know. This complication certainly oc-

curs more frequently in the course of severe cases; and yet this is only the rule, and the pericardium may be affected in those which are only very moderately severe. On the other hand, the number of joints affected bears no relation to the frequency of pericarditis, and much less does the affection of certain joints, particularly those of the upper extremities, have any influence. So, likewise, the effect which repeated attacks of rheumatism will have upon the tendency to pericardial affections is not surely established. According to Thompson, the first attack is the one most frequently accompanied by pericarditis. Pericardial inflammation does not occur in chronic articular rheumatism, nor in rheumatic inflammation of a single joint, nor in muscular rheumatism, nor in gout.

The occurrence of pericarditis has been observed most frequently between the sixth and fourteenth days, or between the fourth and tenth, yet it may occur as well later as earlier in the disease, and there are well-authenticated cases in which the pericardial affection preceded the articular trouble. A marked predisposition to secondary pericarditis is an accompaniment of chronic diseases of the kidneys, and specially of fatty degeneration of these organs, and stands in the same class with the increased tendency to peritonitis and pleuritis, which are sequelæ of these affections. For the causal connection between these secondary troubles and the primary disorder we must refer to what has been said elsewhere concerning the inflammation of serous membranes as a sequel of Bright's disease (see the chapters on that subject in this cyclopædia). On this point, also, the statistics of various observers are greatly at variance, which may here be explained by local variations and partly also by insufficient accuracy in the conception of the meaning of Bright's disease.

The frequency of the occurrence of chronic renal affections is doubtless very different in different localities, and accordingly the number of cases of pericarditis secondary to such diseases must also be very varied. Moreover, it is stated that, in a given number of cases, the tendency to pericarditis is not everywhere the same. Frerichs observed pericarditis thirteen times in 292 cases, and Rosenstein eight times in 114 cases.[1]

[1] *Rosenstein*, Krankheiten der Nieren, p. 197.

Pericarditis also occurs quite frequently in the course of pyæmic processes and especially in puerperal pyæmia; yet in these diseases pericarditis falls far behind pleuritis in the frequency of its development. The method of its development is the same as in the inflammations of other serous tissues which so frequently follow pyæmic infection. The connection described by Kirkes, between pyæmic pericarditis and pyæmic abscesses in the myocardium, doubtless holds chiefly for diphtheritic endocarditis.

Pericarditis occurs occasionally also in the course of other acute infective diseases, as in scarlatina—according to Gendrin especially at the time of desquamation[1]—in measles, variola, exanthematous typhus, also in severe malarial fevers, in cholera, etc.; very rarely in typhus abdominalis (typhoid), erysipelas, and others.

Among chronic constitutional diseases, those with the so-called hemorrhagic diathesis are quite frequently complicated by pericardial inflammations; for instance, the *morbus maculosus*, especially scorbutus, as was accurately recognized by Seidlitz and Kyber, in which the exudations have a specially hemorrhagic appearance (pericarditis exsudatoria sanguinolenta, pericarditis scorbutica). *Tuberculosis* may be accompanied by pericardial inflammations of various kinds; it may be simply fibrinous, or serofibrinous, hemorrhagic or tubercular in character; exceptionally it may happen that a cavern in the lung perforates the pericardium and pours its contents into the sac. Pericarditis, however, is not a frequent accompaniment of tuberculosis. Rarely pericarditis occurs as a sequel of cancerous cachexia; and here it may be either a simple inflammatory process or development of the new growth upon the serous membrane. In tuberculosis of the lungs we must explain some of the secondary pericardial inflammations, by believing that, from the foci of inflammation in the neighborhood, inflammatory products and irritants find their way through the lymph channels into the pericardium. In the same way all other inflammatory processes in the vicinity of the pericardium

[1] Snow believed that the pericarditis in scarlatina was caused by the trouble with the kidneys. In two cases of pericarditis accompanying scarlatina which I saw, there were rheumatic inflammations of the joints at the same time, and in one case, before the disappearance of the eruption, without any accompanying trouble in the kidneys.

may involve it by sympathy. This is true of acute and chronic pneumonias and pleurisies, especially when the exudation is purulent; effusions on the left side more frequently lead to pericarditis, but those on the right also cause it; pneumonia and pleurisy, after polyarthritis rheumatica, are doubtless the most frequent of the diseases giving rise to pericarditis. In rare cases empyema breaks through into the pericardial sac.

The earlier observers, as Senac, Morgagni, and others, had seen most of the cases of pericarditis occurring as a sequel of pleuritis. Since the time of Bouillaud acute articular rheumatism has been considered the most frequent cause. Duchek clings to the old impression; but this sequence will be sustained only, as I believe, by statistics of the fatal cases of pericarditis alone.

The processes which are comparatively rare in the thorax, such as ulcerations and new growths in the œsophagus, the bronchial glands, the mediastinum, and the lungs, caries of the vertebræ and the ribs, may occasionally cause inflammation of the pericardium, either by the extension of the inflammatory process or by ulceration of the pericardium—in the latter case with the formation of a so-called internal fistula of the pericardium. Processes going on below the diaphragm, such as peritoneal inflammations (aside from the infective form), encapsulated abdominal exudations, perihepatitis, abscesses in the liver and spleen, echinococci, gastric ulcers, and new growths in the abdomen, may lead to pericarditis, with or without perforation of the diaphragm. All serous membranes are intimately connected with the organs which they surround, and take part in the diseased processes which affect them. This is true also of the pericardium, and all the various diseases of the heart may give rise to pericarditis; more especially is this true, however, of myocarditis and the degeneration of the muscular tissue which is observed particularly in hypertrophied hearts, next of abscess of the muscular tissue, endocarditis, and aneurisms, especially when they lie within the pericardium or press against it.

With the list already given, we have not exhausted all the diseases which at times are complicated by pericarditis; but cases arising otherwise than as above are exceptional or have no direct dependence upon the original disease.

If we consider the absolute frequency of pericarditis, it will be clear that the numerical relation to the total number of individuals sick with diseases of all kinds and to the total mortality from all causes cannot everywhere and at all times be the same, and this variation will be occasioned by the varying frequency of the chief primary diseases on which it depends.

The accounts of an endemic pericarditis, such as are given by Trécourt, Hubert, and Lalor, are to be explained by the increased frequency, which sometimes is seen, of those diseases to which it is secondary. Thus, in the first two of the writers above mentioned who observed it, doubtless the primary disease was pleuro-pneumonia, which frequently appeared in the troops acting as a garrison for fortifications, and with it was associated a purulent pericarditis.

Just as in acute rheumatism, so in these other diseases it is difficult to recognize any conditions which will explain the occurrence of pericarditis in one series of cases, while in other cases, under apparently similar circumstances, it does not occur. Pericarditis occurs most frequently in middle life, and oftener in the male than in the female sex, and for the reason that the primary disorders which give rise to it show the same preference. In children inflammation of the pericardium is rare, and, according to Virchow, most frequently associated with pneumonia. Although the greatest number of cases of pericarditis occurs in middle life, yet the statement of Willigk is not at variance with this fact, viz., that the disposition to this disease amongst very old people is considerable, when we compare them with a similar number of individuals at other periods. This statistical observation of Willigk has certainly been called in question, but has not been disproved.

The greatest frequency of pericarditis during the coldest months in the year is due likewise to the great prevalence at that time of the primary disorders.

Formerly and up to the close of the last century pericarditis was considered a rare disease ; since then it has been proved that it is of quite frequent occurrence. The absolute numerical proportion to the total number of cases of sickness or to the total mortality cannot be easily established, since its frequency is due to circumstances which are not everywhere the same, as has already been remarked.

In giving the statistics of deaths the proportion will be varied also according as the "milk spots" are included in the cases of pericarditis or not. Usually they have been included. Duchek found 15.1 per cent.; Willigk, 14.1 per cent.; Chambers, 16.2 per cent.; Taylor, 12.5 per cent.

Pathological Anatomy.

The anatomical processes which accompany pericarditis are essentially the same as are observed in inflammation of other serous membranes, especially that of the pleura. The visceral and parietal layers of the pericardium are both affected by the inflammatory changes; but frequently the process is more pronounced on the visceral than on the parietal portion, and the degree of the morbid process is not the same in all parts.

The characteristics of the inflammation are hyperæmia of the serous layer and sub-serous tissue, with parenchymatous swelling, formation of a fibrinous exudation on the surface of the membrane, and the outpouring of a fluid effusion into the pericardial sac, varying in quantity and appearance.

In the lower grades of the hyperæmia the injected vessels form a fine network, and in the higher grades the membrane is of a uniform dark-red color. With the hyperæmia, small spots are frequently found where blood has escaped, especially round the newly formed vessels. In the pericarditis of cachectic individuals and those of advanced age, the hyperæmia is ordinarily less intense. In the further course of the process the hyperæmia generally subsides, or at least is covered by the fibrinous exudation. In the first stage of the hyperæmic condition the dull sheen of the serous coat is replaced by a cloudy and velvety appearance.

The hyperæmic stage does not last long, perhaps only a few hours, and then begins the fibrinous exudation upon the free surfaces, and most frequently at the origin of the great vessels.

This exudation varies greatly in quantity and external appearances, in different cases, according to the duration of the inflammatory process. It may be a delicate, thin covering, like a bloom, only at single places on the surface, especially on the visceral portion, or it may cover the whole heart, and the parietal layer as well, as a pretty thick network or shaggy bark. Frequently

long papillæ stretch across from the visceral to the parietal layer, and may form firm partitions in the cavity.

A perfectly smooth surface on the fibrinous exudation, like croup membrane, is rarely observed; most frequently it has an areolar texture. This varied appearance of the irregular surface has given rise to various figurative names, such as cor villosum, or tomentosum and hirsutum, and, in the German, Zottenherz, Mantelherz, etc. Occasionally the comparison made by Laënnec is very striking, who said it looked as if two flat surfaces had been spread with butter and pressed together, and then pulled apart.

The fibrinous exudation has usually a whitish yellow or reddish yellow color, is of pretty firm consistency, and on pulling it apart we find it very elastic. Frequently we find numerous hemorrhagic points in the layers of exudation, and after a short time it is with difficulty that they can be separated from the serous membrane, for the new-formation of vessels takes place rapidly.

The membranes consist of fibrine and numerous cell elements. The upper layer of a fresh fibrinous membrane is usually almost lacking in cell elements; but deeper, near the serous layer, the cells are very numerous. We must consider them as wanderers from the endothelium, or, as having escaped from the vessels.

In other cases the exudation is less firm, sometimes infiltrated with serum, and occasionally it is soft, boggy, or crumbling, or may have the consistency of jelly. This results from the admixture of a great number of cell elements (pus corpuscles) and from the molecular destruction of the exudation.

The quantity of fluid which is poured out into the sac is very varied; it may be so small that the name dry, or fibrinous pericarditis, though not actually, is comparatively correct; in cases of this kind we frequently find the layers of the pericardium loosely adherent. Occasionally the connection is formed by a network scaffolding of fibrinous strings, the meshes of which are filled with serum. In extreme cases the fluid may amount to a quart or more. As the body lies at an autopsy, we usually find most of the fluid at the anterior and upper part of the sac, since the heart, which is the heavier, falls backwards and downwards. This position naturally may be variously changed by adhesions.

As a result of the exudation, the complementary space in the sac will first be filled with the fluid ;[1] in more extensive effusions the sac will be more enormously distended, and, indeed, there may be thinning of its walls, but more frequently in inflammatory effusions there is thickening and increase in volume, for the texture of the pericardium is involved in the inflammatory processes on its free surface. When the disease has a longer duration, numerous connective tissue corpuscles are formed in the parenchyma of the serous layer, and more abundantly the closer you come to the free surface of the same. At the same time the whole tissue is swollen, loosened, and thickened.

The color of the fluid exuded is bright yellow and transparent or greenish; in other cases it is brownish in various shades, deepening actually to a hemorrhagic appearance, or finally to that of pure blood. In consequence of these hemorrhages, which generally occur from the newly-formed vessels, the fibrinous layers are stained of a brown or reddish color. While a slight admixture of blood is not rare and has no particular significance, distinct hemorrhagic exudations, as a rule, are found only in individuals with some constitutional dyscrasia, as in tuberculosis, and also in the course of acute general diseases. Yet hemorrhagic pericarditis does occur in healthy people, especially in the chronic forms, and I have seen most beautiful examples of it in individuals suffering from chronic alcohol intoxication. Hemorrhagic exudations are particularly marked in those diseases which cause hemorrhages elsewhere in the body, such as scurvy and morbus maculosus. In regions where scurvy prevails, these cases are often seen associated with extensive hemorrhagic exudations, and have been described by Seidlitz as pericarditis exsudatoria sanguinolenta, and by Kyber as pericarditis scorbutica.

If the effused fluid does not contain blood, it is either entirely clear or little flakes of fibrine are seen suspended in it, of the same appearance as the layers which line the pericardium. In other

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[1] According to *Rüdinger* (Topograph. Anatomie, Abth. 1 u. 2 S. 49) the sac is larger than its contents require. Accordingly, there is a complementary space into which the heart doubtless falls at first, while the fluid collects at the upper part near the base.

cases the fluid is opalescent from its richness in cell elements. When they are still more abundant the fluid becomes cloudy, opaque, and of all grades of density up to a marked purulent character.

While in other serous membranes, particularly in the peritoneum, the fluid often has a purulent character from the first; in the pericardium this, doubtless, is not the case—here the pus is always secondary.

The causes which in some cases lead to the formation of pus are probably manifold, and are not easily defined. Yet certain conditions should be mentioned to which a certain influence may be ascribed in the more abundant cell growth which is sometimes observed. Among these may be reckoned the constitution of the patient, the nature of the primary causal disease, and also the deposit of inflammatory products upon the surface of the pericardium. In the last-named condition the extensive formation of new blood-vessels in the pseudo-membrane doubtless favors the development of pus.

The formation of pus does not ordinarily lead to a purulent destruction of the serous membrane, but its surface becomes like that of the granulating surface of a wound (Rindfleisch).

Yet in exceptional cases purulent destruction and loss of substance do occur, and it is by means of these processes that perforation of the pericardium has been observed. A case of this kind was described by Wyss, in which, after wearing away the rib, a fistulous opening was established and remained patent for years, until finally the patient was carried off by an attack of pericarditis.

A foul and ichorous condition of the exudation in the pericardium is very rarely seen; cases of this kind are generally associated with destruction by gangrenous or carcinomatous processes going on in the neighboring parts, *e. g.*, proceeding from the œsophagus; entrance of air into the pericardial sac may also cause a foul condition of its contents. This condition has also been observed in cases where no local cause could be found sufficient to explain it, and when it had to be referred to a general septic condition of the system. We might well doubt whether in the absence of any of these or any similar causes we could attribute it to spontaneous decomposition.

From what has been said above, we may divide the exudations in pericarditis into those of a *serous*, *sero-fibrinous*, *hemorrhagic*, and *purulent* (and in rare cases *ichorous*) character.

The methods of progress toward cure are analogous to those in inflammations of other serous membranes. Thin, serous effusions are absorbed. Doubtless, also, fibrinous exudations may be completely absorbed, so that there is an entire "restitutio ad integrum," or there remains at most a thickening, of various degrees, of the tissue of the pericardium. This favorable result depends upon the thickness and extent of the fibrinous deposit, and especially upon the length of duration of the process. In those cases in which the fibrinous deposit is only partial and not very abundant, as we must believe it to be in many cases of pericarditis secondary to polyarthritis rheumatica, we find at the autopsy only very slight remains, or none at all, of the pericarditis which has occurred. The absorption of the fibrinous exudation takes place through a fatty molecular destruction.

Moreover, it is probable that loose adhesions, by means of connective tissue, may subsequently be broken up, for the continual movements of the heart pull and stretch the newly-formed membranes, and, moreover, beside this mechanical violence, the circulation of the blood in the newly-formed vessels must be considerably interfered with by the movements of the heart.

Just this constant and yielding friction of the parts is the chief reason why permanent firm adhesions in the pericardium are more rare than in other serous membranes.

In those cases also in which, from the inflammatory process, new growth of connective tissue and adhesions of the pericardial layers take place, the fibrinous exudation takes no part in the process, but is absorbed after fatty degeneration.

The organization of connective tissue results from the cells present in the exudation, and in very severe forms of inflammation the tissue of the pericardium itself assists in the growth.

The new-formation of connective tissue may occur in very various extent and arrangement. Besides superficial fibrous thickenings,[1] we find irregular, knob-like projections or peduncu-

[1] As a rule, the fibrous thickenings due to previous inflammation may be distinguished from tendinous spots, especially by their greater thickness and extent, by their irregular distribution, and by the usual coexistence of adhesions. Often, however, the distinction is scarcely possible.

late outgrowths, which may even be found lying free in the pericardial sac; also bands, either stretched to a thready thinness or of considerable thickness, up to a finger's-breadth, extending from one layer to the other. In other instances we find the two layers adherent in circumscribed spots, or throughout their whole extent, by means of connective tissue, which is sometimes loose and at other times very dense. In the latter event we find the heart grown fast within a dense fibrous case, the two original layers of which can no longer be distinguished or separated. Calcification also occurs in the form of isolated concrements, and in rare cases the whole heart may become enclosed in a very hard calcareous capsule.

Even hemorrhagic exudations may be absorbed, and the reason why such cases commonly prove fatal does not lie in the admixture of blood in the pericardial effusion, but must be sought for in those constitutional conditions which usually determine the hemorrhagic character of the exudation. Only in cases in which the admixture of blood is very abundant, so that the fluid looks like pure blood, can this occurrence of itself occasion death.

Purulent exudations may likewise, in exceptional cases, end in recovery, the purulent effusion becoming thickened and the cells undergoing fatty metamorphosis, while the serous layers become adherent. It may then happen that these thickened masses become perfectly encapsulated. We find spaces between the adherent layers filled with a yellowish-white paste, made up of fatty or, together with these, calcareous molecules.[1]

The changes just described usually follow a more or less acute course to a definitive result. Cases are observed, however, in which an acute stage passes into *chronic inflammation*, but it is rare that we can assign to a simple pericarditis a chronic character from the beginning. Cases of the last-named sort, which are occasionally to be observed in aged persons, are now and then distinguished by the excessively serous or even blood-tinged character of the effusion, and by the slight amount of fibrous

[1] See the representation in *Thierfelder*, Atl. der path. Histolog. 4 Lfg., T. XX. Fig. 3.

exudation. Very likely there was in these cases primarily a transudation, to which inflammation was subsequently added.

When chronic pericarditis follows an acute attack, we find thickenings and adhesions, whilst the fluid effusion is not all or only imperfectly absorbed. The effusion shows fluctuations, its amount increasing and diminishing by turns, fresh deposits of fibrine take place, hemorrhagic admixtures occur, or the exudation takes on a purulent character. Such cases generally end in death.

Circumscribed pericarditis is found, as a rule, upon the visceral layer, and particularly at the origin of the aorta and pulmonary artery. It sometimes extends to the coats of these vessels, as far as they are covered by the pericardium, and gives rise to callosities, which even involve the middle coat. In this way pericarditis may be the cause, or at least a predisposing cause, of aortic aneurism.

The *changes in the muscular structure*, which very commonly occur in the course of acute and chronic pericarditis, and which must be looked upon as directly connected therewith, are of great importance. There are but few thorough observations in regard to the frequency with which pericarditis gives rise to such lesions; according to Wagner, fatty change in the cardiac substance was found in seventeen out of thirty-five cases.[1] Very likely this proportion is too low; but certainly if we take into account not only fatty degeneration, but the inflammatory and degenerative processes of all sorts which may affect the cardiac muscle in the course of pericarditis, we find them, in various degrees of extent and intensity, in a great number of cases. The intensity and area of the inflammation of the pericardium, the character of the exudation, and, above all, the duration of the disease, are essential elements in the causation of changes in the muscular tissue. Myocarditic lesions are more frequently met with in hemorrhagic and purulent than in other conditions of the exudation, and in chronic cases they are probably always present. Virchow believes that the occurrence of

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[1] Compare *Schroetter*, Myocarditis and Fatty Degeneration, p. 224 et seq., of this volume.

muscular affections is favored by the *strain*, or increased work thrown upon the heart, particularly by the high fever. The view has also been expressed, that the pressure of the exudation impedes the normal distribution of blood to the substance of the heart, and that by means of this disturbance of the circulation the occurrence of degenerative changes is occasioned.

In view of the importance of the serous membranes to those organs which they enclose, there is nothing surprising in the frequent occurrence of inflammatory and degenerative changes in the muscular structure of the heart in consequence of inflammations of the pericardium.

The influence exerted by affections of the muscular structure of the heart upon the symptoms and course of pericarditis was pointed out by Stokes. Virchow has shown, by several examples, how high a degree of fatty degeneration may occur, even in cases of a wholly acute character.

The changes may affect the cardiac muscle as a whole, but, as a rule, they are most clearly marked in those layers which are in immediate contact with the pericardium, so that therefore an advance of the process from without inwards is most strikingly manifest.

The condition of fatty degeneration of the heart in connection with pericarditis has been thus described by Virchow: The surface of the pericardium was very rough with layers of fibrine. The substance of the heart throughout seemed flabby, pale, and somewhat spotted. Beneath the whole extent of the pericardium the outermost muscular layer had assumed a cloudy, pale-yellow appearance. This layer was from one to two lines in thickness, and within it were groups of primitive bundles in such an extreme condition of fatty metamorphosis that their internal structure could no longer be made out, even in the slightest degree. In the deeper layers this metamorphosis gradually decreased, but in no part of the muscular substance of the heart was the interior of the primitive bundles entirely free from fat granules. Towards the surface, layers of proliferating connective tissue were met with, which extended into the thickened pericardium and the visible fibrinous layer, and which were thickly strewn with numerous masses of nuclei and cells, closely crowded together and in the act of subdividing.

The best-marked cases of fatty degeneration answer to this description of Virchow's.

Besides fatty degeneration, the other well-known acute and chronic disturbances of nutrition may affect the cardiac muscle in consequence of pericarditis. This is in part due immediately to anatomical conditions, and in part to sequelæ. Thus, a peri-

carditis may result in dilatation with consecutive hypertrophy of the whole heart or of an individual part, and this takes place without any obstruction to the current of blood, but merely by reason of the diminished impelling power and capacity for resistance on the part of the cardiac muscle as the result of preceding myocarditic changes. The so-called abscesses or purulent softening of the muscular substance are rare.[1]

Some of these changes in the heart muscle are capable of repair; they may end with hypertrophy, but they may also assume a chronic course, with further extension. The latter is seen in adhesions of the pericardial layers, in which event the muscular substance becomes highly shrunken and atrophic, what remains of it being in a condition of connective-tissue or fatty degeneration.

From the pericardium the inflammatory process may be propagated to the endocardium, as Desclaux has shown by experiment; acute endocarditis was developed in animals within a short time after an artificially induced pericarditis. As a matter of course, it is in but few cases that we can determine the dependence of an endocarditis upon a pre-existing pericarditis, since generally one and the same cause is capable of producing inflammation of both membranes.

As a rule, the fibrous layer of the parietal pericardium does not share in the inflammatory process, but there are cases, particularly of chronic pericarditis, in which the fibrous pericardium also takes part in the inflammation. Gendrin has assumed a special form, fibro-pericarditis, but this term expresses a limitation of the inflammation to this layer of tissue, such as scarcely ever exists. Even the mediastinal connective tissue may be involved in the process, leading to the development of indurated bands and tough adhesions. In rare cases of purulent pericarditis we find little collections of pus in the fibrous layer of the pericardium.

Not uncommonly, but by no means constantly, we find signs of inflammation upon the pleura pericardii, whereby agglutinations and adhesions may occur with the adjacent portions of the

[1] A case of the sort is contributed by *Th. Salter*. See *Virchow*, op. cit.

lungs, or with the costal pleura, if the lungs have previously been pushed aside.

The remaining visceral lesions, which may occur simultaneously with inflammations of the pericardium, belong partly to the primary diseases, and are in part referable to the same causes which produced the pericarditis. Only a few consecutive phenomena find their explanation in pericarditis, and they are precisely the symptoms and results of the stasis which is very likely to occur from a high degree of concomitant affection of the muscular substance of the heart.

Pathology.

General Picture of the Disease.

If, as happens in the great majority of cases, the pericarditis comes on secondarily in the course of an acute or chronic disease, the phenomena of the fundamental affection are, as a rule, not essentially, or at least not suddenly, modified. Moreover, on an analysis of cases of idiopathic pericarditis, those phenomena which can be ascertained without physical examination are not sufficiently definite and constant to form the ground of a positive diagnosis.

The *subjective symptoms* which occur in acute diffuse pericarditis are not usually of much significance,[1] consisting for the most part of a pressure-like pain or a dull sense of oppression in the præcordial region. Commonly, also, there is tenderness of the epigastrium, particularly on external pressure. Sometimes patients complain of difficulty of breathing and of palpitation. In idiopathic cases, or in those occurring in the course of *chronic* disease, the general condition may suffer but slight disturbance, especially in impassive persons, so that patients of this sort continue to go about. Sooner or later, as a rule, a feeling of languor and indisposition to bodily exertion sets in

[1] It is probable that idiosyncrasy has an influence in this matter. Thus Baeumler has published cases in which there was the peculiarity of a tolerably characteristic assemblage of symptoms. Possibly such cases may be more often met with amongst the well-to-do classes, in private practice, than in hospital patients.

All these phenomena may be wanting, or, on the other hand, the picture may be one in which the wildest disturbances figure, whilst extreme dyspnœa, orthopnœa, and sleeplessness, fainting-fits, etc., justify the portrayal which the older physicians made of pericarditis. Under such circumstances, the phenomena essentially resemble those caused by a myocarditis or muscular degeneration, or a valvular affection without compensation.

The course of *the fever* in pericarditis is not at all characteristic; as a rule, the elevation of temperature is slight, and, if fever existed already before the outbreak of the pericarditis, the newly developed inflammation does not always exert a perceptible influence upon the febrile temperature.

The only sure signs of pericarditis, whether secondary or idiopathic, are the physical changes. Not only do these render the diagnosis possible, but they also give information as to the course of the disease, at least as regards the exudation. On account of fibrinous deposits upon the layers of the pericardium, *friction sounds* are produced by the rubbing of these upon each other. These sounds may decrease or disappear upon the occurrence of a larger amount of exudation into the pericardium, and so, too, agglutinations and adhesions may cause them to cease. If the fluid in the pericardium becomes diminished, the friction sound thereupon reappears or becomes increased in intensity, to disappear finally after a varying length of time.

In proportion as the fluid distends the pericardium, the *cardiac dulness* of the anterior thoracic wall increases in *intensity* and *area*, and at the same time, in the majority of cases, the area of dulness shows a characteristic form, whereby we are enabled to recognize enlarged pericardial dulness as such. The shape is that of a *truncated triangle*, with the apex turned upwards and the base downwards. When the effusion is absorbed, the dulness diminishes again, and may return to a perfectly normal extent.

The behavior of the *apex-beat* furnishes very valuable information. At first, and for some time, it may be normal or even increased in force, but, in proportion as the fluid exudation forces the heart's apex away from the chest wall, it becomes weaker, and is at last wholly extinguished.

Very abundant exudations may visibly alter *the shape of the thorax*, compress the lungs, and, by pressure upon the neighboring organs, effect a change in their position. The pressure of the fluid upon the heart and the great vascular trunks may interfere with the circulation in variable degree. Other symptoms of pressure and compression occasionally occur on the part of the distended pericardium, although more rarely.

The occurrence of exudation in the pericardium, the existence of a friction-sound, and increase of the cardiac dulness constitute the essential features of every case of pericarditis, which always repeat themselves. But, so soon as we go a step further, we come upon a great multiplicity in the objective symptoms also. We must seek for definite causes of this variability, and here it appears that *the quantity and character* of the exudation, the *condition of the cardiac muscle, and the character of the fundamental disease* have a most important influence upon the remaining complex of symptoms of pericarditis. It is of significance, furthermore, whether the affection arises and follows its course *acutely or chronically*. Very commonly, too, other diseases coexist with the inflammation of the pericardium, and if these chance to involve impediments to the circulation and to respiration—as is the case, for example, with a pneumonia or a pleurisy—a much severer assemblage of symptoms will, as a matter of course, result from this combination than from a pericarditis alone.

In conformity with the variability of the symptoms, the importance and gravity of the disease are also various. In order to a thorough elucidation of this multiplicity of the phenomena and of the causes thereof, some authors, such as W. Stokes, have attempted to make a sort of division of pericarditis, according to the severity of the symptoms. Of course, there are no strict grounds or rules for such a distinction, which, however, tends to facilitate a general survey of the rather complicated conditions.

Stokes has distinguished three grades of intensity in pericarditis, using as distinctive marks the amount of fluid effusion on the one hand, and on the other hand the implication of the muscular structure. In accordance with this plan, we may refer

to a *first group* all those numerous cases which remain *circumscribed*, as well as all those which, while indeed they affect the whole pericardium, run their course *without a great effusion of fluid* and *without involving the muscular substance in an acute* manner.

Although under this arrangement pericarditis does not figure as the final phenomenon of a fatal fundamental disease, and may perhaps even give rise to a purulent exudation; and although it may occur under circumstances in which a slight additional functional disturbance may prove unbearable, we may yet characterize as benign those cases which, as a rule, occasion no very serious illness.

Even in the presence of manifold complications a pericarditis of this sort gives rise to no great increase of the disturbances and dangers, and may proceed to a favorable result. Spontaneous pericarditis may behave thus. It is very common to see inflammation of the pericardium behave thus in rheumatic polyarthritis, as well as in pleurisy and pneumonia, valvular lesions, etc.

Baeumler[1] has recently called attention to the short duration of many cases of *idiopathic pericarditis*, especially when circumscribed, so that so transitory an illness may well remain unobserved, and we cannot wonder if the traces of recent pericarditis are found at autopsies, where during life this disease had been entirely overlooked. Baeumler had an opportunity of making thorough observations on himself. In another example of the sort the whole process lasted four days, but the symptoms were very well marked; slight fever was present. The patient became suddenly ill, with pain in the epigastrium, which was increased by each inspiration and by any movement, particularly forwards; the aspect was that of a severe affection, but for a short time only. At the first examination nothing was found but tenderness of the epigastrium on pressure; somewhat later a circumscribed friction-sound appeared, which disappeared after twenty-four hours, without any change having occurred in the cardiac dulness. Besides the pain in the epigastrium, there were similar pains in the region of the left shoulder, radiating thence towards the left ear and the left arm. At the end of four days, perfect health, both objective and subjective, was regained.

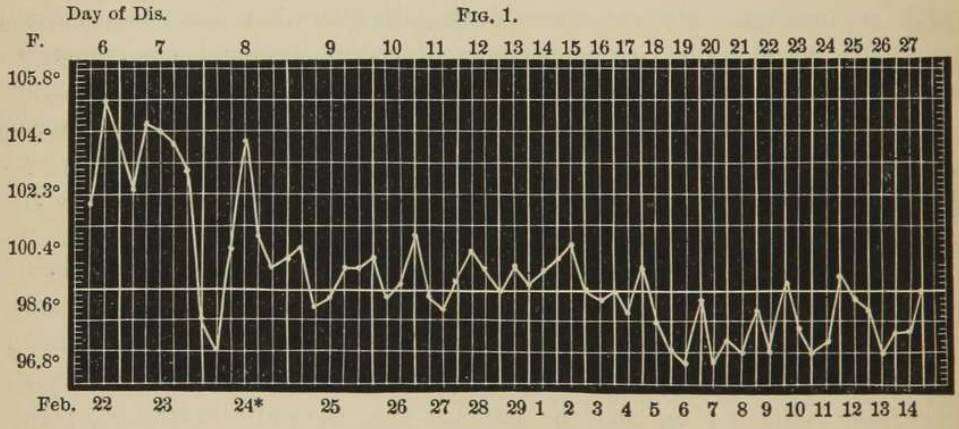
So well-defined an assemblage of symptoms, especially of the subjective ones, seems chiefly to be peculiar only on account of the acute idiopathic pericarditis, whilst secondary cases, analogous in other respects, may appear with very trifling symptoms. The following case from v. Ziemssen's Clinic is an example of the sort:

Mitral stenosis, croupous pneumonia, fibrous pericarditis, endocarditis with aortic insufficiency. K. A., a butcher's wife, forty-seven years old, had been brought to the clinic in the year 1874, with rheumatic polyarthritis, was treated with plaster-of-Paris bandages, and in the course of that affection had acquired the valvular disease first mentioned. On February 16, 1876, she fell ill with a smart chill, fol-

[1] Transact. of the Clinical Soc. V. 1872.

lowed the next day by pain in the left side, shortness of breath, cough, and bloody expectoration. On February 22, a pleuropneumonia was found, with hepatization of the left lower lobe, the progress of which was shown by the fact that up to March 15, long after the fall of temperature, fresh sputa highly tinged with blood were ejected. Resolution took place very slowly, a loud pleuritic friction sound was to be heard for a long time over the left lateral region. On March 5th a rough murmur, following the systole, appeared over the origin of the aorta, whilst up to that time only the presystolic murmurs at the apex, with a faint whirring, and marked accentuation of the second pulmonic sound, had been present, with the sounds otherwise normal. The pericarditis produced no recognizable fluid exudation, the apex-beat and area of dulness remained unchanged, although the friction-sound became diffused during the following days, occurring with two intermissions. The accession of the pericarditis was not announced by any new subjective symptom; the frequency and character of the pulse, from sixty to seventy-six beats, as well as the temperature remained unaltered. Moreover, the occurrence, on March 7, of a new soft diastolic murmur, of a humming character, along the course of the aorta, produced no change. On March 14 the pericardial friction had entirely disappeared; the new diastolic aortic murmur remained constant. The patient left the institution in good condition.

FIG. 1.



* Two drachms of salicylate of soda.

Observations, at first, every second hour during the day, then thrice and twice a day, in the axilla.

To a *second group* belong chiefly those cases which are accompanied *with abundant fluid exudation, without the muscular substance undergoing much change.* Such cases may be *primary* or *secondary*, they may occur with slight or with very severe general phenomena, and the quantity of the exudation may give rise to severe symptoms.

In such cases, if no aggravating constitutional conditions be present, if the patient be not advanced in years, and if the fundamental disease involve no unfavorable

presumption, the termination is generally a favorable one, either with perfect restitutio ad integrum, or with adhesions. However, the amount of the exudation may cause death.

At most, only serous or sero-fibrinous exudations are here considered. Hemorrhagic effusions generally derive their unfavorable character from previous constitutional troubles. Purulent exudations seldom prove fatal by their quantity, but in consequence of the primary and original change, or by retardation of absorption and by the muscular degeneration which finally results therefrom.

The following case, from v. Ziemssen's Clinic, may serve as an example of a pericarditis with large effusion and a favorable result. *Rheumatic polyarthritis; sero-fibrinous pericarditis; endocarditis; double exudative pleurisy; catarrhal icterus.* A man-servant, thirty-five years old, a moderate drinker, previously always healthy, fell ill with acute articular rheumatism, with an initial chill, after having already had subacute inflammation of several joints. On admission, numerous joints were seen to be affected; no pain in the chest. Apex-beat faintly perceptible in the normal locality. First sound feeble and indistinct at the apex, and equally so over the pulmonalis, where the second sound was somewhat accentuated. Pulse 104, bounding, rather tense, regular, and out of proportion to the weak heart-sounds. At the upper part of the left side of the chest, in front, below the second rib, the percussion note was less intense and higher in pitch than on the right side, passing abruptly into the area of comparative cardiac dulness. Respiration moderately frequent. Urine free from albumen, with a specific gravity of 1031.

On the following day, November 8, abatement of the articular pains, a feeling of pressure and distention in the pit of the stomach, somewhat increased by external pressure. Skin very dry and hot. Pulse very tense, 88 in the morning, 126 in the evening. Apex-beat in the fourth intercostal space, three ctm. externally to the nipple-line. *Absolute* cardiac dulness increased only a little towards the right, and not at all upwards; the *relative* cardiac dulness begins above at the upper border of the third costal cartilage, and extends towards the right as far as the right side of the sternum. Heart-sounds indistinct at the apex, accompanied by a soft grating murmur; in the third intercostal space, near the left border of the sternum, a loud, sharp, rasping friction murmur is audible and distinctly perceptible to the touch. Carotid sounds clear. On November 9 the epigastric pressure had entirely disappeared; patient had a very restless night, the joint pains being somewhat increased. Apex-beat still present, but feeble; slight increase of the absolute cardiac dulness towards the right as far as the middle of the sternum; over the mitral, a systolic blowing murmur; over almost the whole heart, a loud, harsh friction-sound, in two intermissions with the systole and diastole, but no longer to be felt. The relative cardiac dulness extends to the lower border of the second costal cartilage, *reaches to the left about four ctm. beyond the site of the apex-beat*, and to the right somewhat beyond the sternal line. Pulse 104 in the morning, 144 in the evening, regular, very tense.

November 10.—Articular pains less severe again. Murmurs unchanged, to be felt slightly again on firm pressure in the third intercostal space. No pain in the

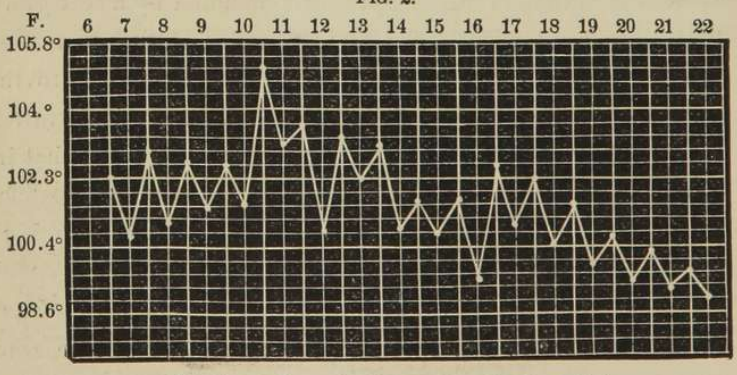
chest or epigastrium. Pleuritic effusion on the left side. Pulse, 88 in the morning, 104 in the evening, regular, full, and tense; respirations 36. No further change in the absolute cardiac dulness, the base of which measures 11 ctm., the right side 8 ctm., the diagonal, from the summit to the site of the apex-beat, which is still feebly perceptible, 11 ctm. The relative cardiac dulness has still further extended, and surrounds the absolute dulness with a belt of about two finger-breadths, which is materially increased when the patient sits upright.

November 11.—Double pleuritic effusion; additional joints affected; apex-beat felt feebly in the fourth intercostal space, somewhat within the nipple-line. Absolute and relative cardiac dulness perceptibly smaller; second pulmonic sound moderately accentuated; heart-sounds more easily distinguished than hitherto; a loud friction-sound at the left border of the sternum. Moderate general icterus, liver not enlarged and not tender. Pulse, 120 in the morning, 144 in the evening, rather jerky. The area of cardiac dulness changes little with the upright position; the friction sound becomes softer.

November 12.—Icterus somewhat less; no pain in the chest; joints free, with the exception of a little swelling of the knees. Apex-beat very feebly perceptible in the fourth intercostal space, about 2 ctm. inside the nipple-line. The friction-sound at the left border of the sternum is very soft; a newly-developed friction-murmur is found in the region of the tricuspid. Carotid sounds clear. The cardiac dulness now extends only to the lower border of the third left costal cartilage; skin moist in the morning, dry and hot in the evening; pulse 100 morning and evening, slightly jerky.

November 13.—A restless night, with renewed articular pains. Heart-sounds clearer; friction-sound still soft. Pleuritic effusion unchanged. A stitch-like pain on inspiration in the region of the heart. Apex-beat still weak; the relative dulness no longer extends beyond it, but reaches to the right beyond the right border of the sternum. The absolute cardiac dulness is nearly normal.

FIG. 2.



Morning and evening temperature (in the axilla).

November 14.—Articular pains not much changed. Friction-sound has almost entirely disappeared; a soft murmur lags after the second aortic sound. At the apex of the heart a systolic blowing murmur and an accentuation of the second

pulmonic sound. The relative cardiac dulness is scarcely increased in breadth, reaching to the right border of the sternum. The icterus is gone, as well as the pain in the region of the heart. Apex-beat again plainly to be felt in the fourth intercostal space, 2 ctm. to the inside of the nipple-line. Pleuritic effusion still present.

From this time on the friction-murmur disappeared; the pericarditis had run its course. The polyarthritis, with the endocarditis and pleurisy, lasted until November 20, and left a slight valvular deficiency.

To a *third group* belong those cases in which *disease of the cardiac muscle* appears among the symptoms, or constitutes the chief feature. The phenomena depending thereon may be present in very various degrees of intensity. To this category belong also many cases of *purulent pericarditis*, as well as all *chronic inflammations of the pericardium*, almost without exception.

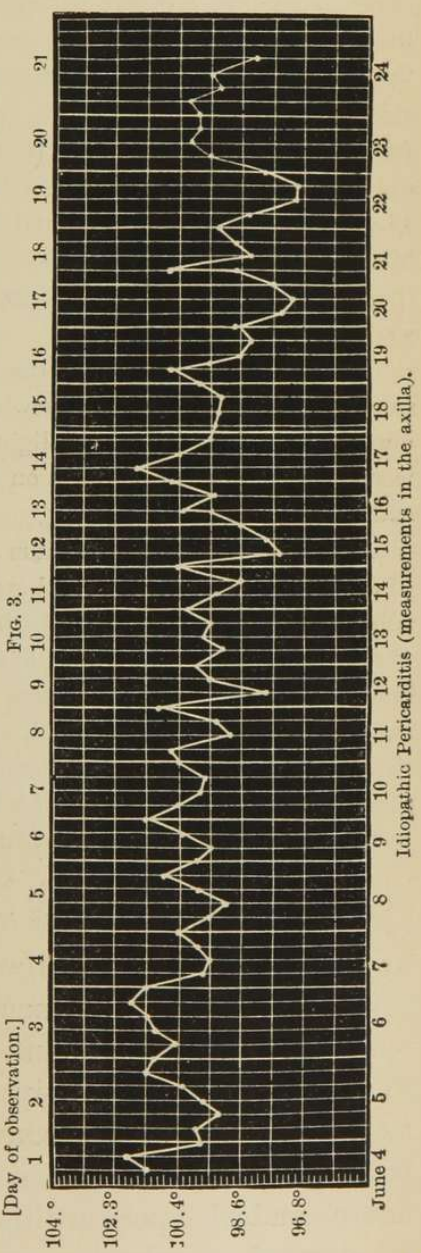


FIG. 3.

Paralysis of the heart may occur in an acute way in consequence of acute myocarditis, and under such circumstances death may take place speedily and quite suddenly; or a degeneration of the substance of the heart may develop gradually, generally by layers; the impelling power constantly diminishes, stases occur, general dropsy, and finally death. We then find the heart more or less degenerated, atrophied, or, if the fluid effusion have been absorbed in the meantime, the cavities dilated.

The acute, as well as the more chronic changes, may remain stationary, ending in hypertrophy preceded by dilatation.

Idiopathic pericarditis, with *hemorrhagic exudation* and *muscular degeneration; death.*—A man fifty-two years old, always before healthy, was living under favorable conditions. He dated his ill-health five weeks back, when he fell ill with stitch-like pains in the chest, could no longer lie on the left side, and suffered much from cough, without expectoration. The pains in the side lasted only

two days, and then the appetite and strength gradually failed. Bowels irregular; sleep good; occasional dyspnœa; powerfully built and well nourished, apparently a drinker. Radial artery very small; pulse uncommonly faint, and rendered more so on inspiration, without wholly disappearing; 104, regular. Moderate emphysema. The apex of the right lung lies somewhat deeper than the left, and is less resonant on percussion. Expiratory sound somewhat prolonged.

The cardiac area is enormously enlarged laterally and upwards, of a triangular shape; apex-beat nowhere perceptible, but intestinal resonance immediately below the sixth rib. On standing, the cardiac area becomes strikingly enlarged, especially towards the right—about 3 ctm. On lying down, the base measures 19 ctm., the right side 16 ctm., the left side 17 ctm.; from the apex to the base 13 ctm. A soft, scraping friction-sound is heard over the sternum; heart sounds clear, but very feeble; hepatic dulness somewhat diminished in breadth, and the left lobe appears small. Spleen 13 × 8 ctm. Slight distention and undulation in the veins of the neck. Considerable cough, with frothy, mucous sputum. Scrotum large and lax, without cheesy deposits. Urine of a dusky brown, without sediment or albumen; 400 cc.; specific gravity 1021. No dropsy.

During the progress of the case the area of cardiac dulness became perceptibly smaller, but the outline was somewhat irregular, and the inspiratory movement of the border of the lung was very slight. But the condition of the pulse became still worse. It disappeared entirely on inspiration. Extreme cyanosis was developed, and dropsy, ending in death.

At the autopsy the pericardium was found enormously distended by an effusion of a faintly bloody tinge. The borders of the lungs were moderately distended and adherent to the pericardium. In both lungs there were scattered cirrhotic nodules. *Pericardium* thickened, with villous, tufted deposits on the inner surface of both the visceral and the parietal layers, single fibrinous bands passing from one layer to the other. The heart is enlarged, cuts easily, and shows quite a layer of fat beneath the exudation; muscular substance pale and very fragile; valves healthy; liver somewhat diminished in size, of a nutmeg color. Kidneys rather small; granular and scarred on the surface. (v. Ziemssen's Clinic.)

Hemorrhagic pericarditis is met with under various conditions, and does not always give rise to a peculiar train of symptoms. Signs of anæmia and its consequences occur only when there is an abundant effusion of blood into the pericardial sac. This is preëminently the case in pericarditis due to scurvy, as we learn from the accounts of it as occurring in the northern maritime portions of Russia. Together with the symptoms of an acute exudative pericarditis, marked prostration suddenly occurs, accelerated respiration, with great anxiety, swelling of the jugular veins, imperceptible pulse, cyanosis, coldness of promi-

nent parts, and dilatation of the pupils. Consciousness remains unimpaired until death, which generally occurs suddenly.

In chronic cases of scorbutic pericarditis the cardiac affection is preceded by so-called rheumatic fever symptoms. The effusion takes place more gradually, and accordingly the phenomena of collapse appear with less severity and suddenness, and the danger to life is not so immediate.[1]

A special interest attaches to the *chronic* inflammations of the pericardium, whether they assume the chronic character, by repeated extensions of inflammation following an acute beginning, or whether they creep on insidiously, without local symptoms and often also without fever, attention not being directed to an examination of the heart until difficulty of breathing, changes in the pulse, etc., supervene. In such cases the effusion is commonly hemorrhagic, and the duration of the illness often extends over many weeks or months. The result is generally fatal by secondary degeneration of the heart and atrophy, and indeed in favorable cases obliteration of the pericardial sac, dilatation of the ventricles, etc., always occur.

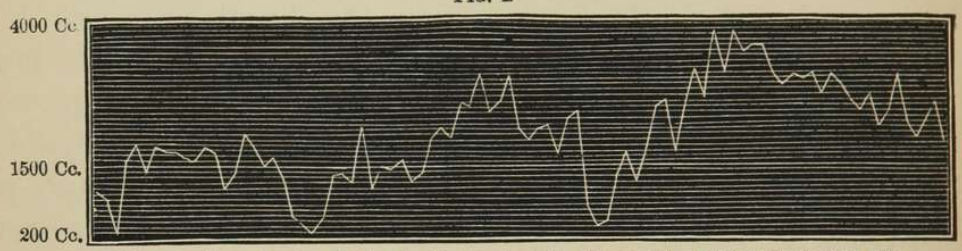
The following case from v. Ziemssen's Clinic affords a very striking example:

Chronic pericarditis, degeneration of the cardiac muscle.—A man, thirty years old, a confirmed drinker, very corpulent, had always been healthy, except for a contused wound which he had received in the Franco-Prussian war, until Easter of 1874, when he first fell ill with rheumatic pains, and then with a croupous pneumonia of the left side, which ran its course with marked adynamic symptoms and with a great amount of dilatation of the right ventricle. After this he again felt perfectly well and strong until the end of May, when he was taken ill, first with gastric symptoms, then with stitch-like pains in the chest, dyspnœa, and oppression at the epigastrium. Moderate œdema, face somewhat bloated, complexion yellowish and bluish. Abdomen swollen with ascites. Subjective and objective dyspnœa, respiration forty. Pulse tremulous, scarcely perceptible in the radial, whilst in the carotid it was so irregular that its frequency could not be determined. Right external jugular vein tense and swollen, not pulsating; the same, less marked, on the left side. Liver markedly swollen, without pulsation. No apex-beat in the region of the heart, nothing more than a diffused tremor being observable between the acts of respiration. The cardiac dulness reaches from the right parasternal line to the line of the anterior boundary of the left axilla, and upwards to the jugular fossa, forming a

[1] *Kyber's* article, Med. Zeitg. Russl. 1847. I have not been able to get access to it in the original; so I have taken these statements from *Friedreich*, loc. cit.

triangle truncated superiorly. The breadth of the area, at the level of the nipple, measures twenty-five ctm.; the height, at the left border of the sternum, eighteen ctm. The area is somewhat larger still in the standing position. Heart sounds clear everywhere, but very weak and distant. The upper lobe of the left lung is very much compressed, that of the lower lobe less so. A moderate amount of free movable fluid is found in the right side of the thorax. His sleep is restless and interrupted; occasional orthopnœa. The appetite is poor; bowels irregular, sometimes diarrhœa. Temperature elevated, but not constant.

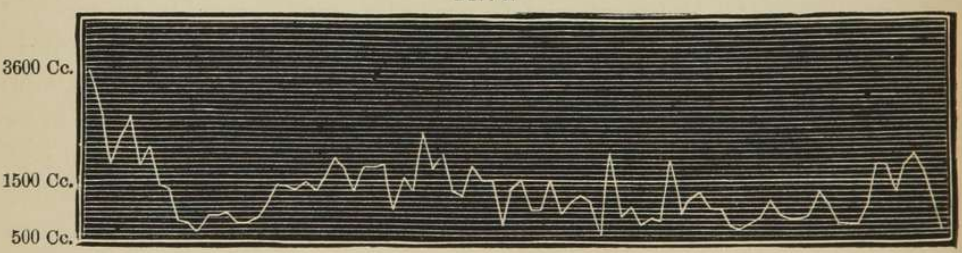
FIG. 4.



Daily quantities of urine during the first part of the disease, May 20th to September 4th.

His condition improved constantly, although with many fluctuations. The pericardial effusion was partially absorbed, a friction sound occurred, and the dulness diminished in extent; the state of the pulse improved, and the dropsy disappeared entirely, so that the patient left the institution after the lapse of four months. The variations in the amount of water excreted by the kidneys are of

FIG. 5.



Daily quantities of urine during the second part of the disease, December 14 to April 19.

interest, since the means which served to strengthen the blood-pressure caused a decided increase in the quantity of urine.

The patient left the institution at the beginning of October, and returned December 14, in a very sad condition. Without any essential change in the state of the heart, the area of cardiac dulness had constantly enlarged, and the outlines had become somewhat irregular. The phenomena of obstructed circulation and the cardiac weakness constantly increased, the dropsy was very considerable, and the patient died April 19.

Autopsy.—Cyanotic colorations; very great dropsy; a great deal of serous fluid in the pleural cavities and in the abdomen. The anterior borders of the lungs were still adherent in many places. The whole pericardium was thickened and indu-

rated; its cavity contained a moderate amount of hemorrhagic effusion, and its serous surfaces were covered with fine tufts. The apex of the heart, together with both auricles, was drawn backwards by a band as thick as the finger. The visceral layer of the pericardium was also thickened, the muscle thinned, the cavities narrowed, and all the valves normal. The fibrillæ of the cardiac muscle were found to have undergone thorough fatty degeneration; the bundles were strewn with large oil globules. There was also a slight degree of connective-tissue new formation and hyaline degeneration. For the rest, there were only the signs of long-standing stasis.

We must still mention briefly the *pericarditis of children.* According to the statements of Rilliet and Barthez, this disease is extraordinarily rare in children under six years of age, but it has been known to occur in the earliest periods of life, and even during the fœtal state. In childhood, too, most cases are of a secondary nature. Kerchensteiner has published a case of idiopathic pericarditis in a girl eleven months old.

Circumscribed pericarditis in children remains latent, as a rule. Even the diffuse variety shows no characteristic phenomena, since the pain is absent or not to be discovered; and the objective signs are commonly very ill marked—the effusion being often very scanty, not more than a few tea-spoonfuls. In other instances, however, the amount of effusion is very considerable. Large effusions appear to affect the functional activity of the heart more rapidly in children than in adults, and to occasion the earlier occurrence of signs of disturbance of the circulation, even although, as in idiopathic cases, there are no complications present. Chronic pericarditis also occurs in children.

In early infancy diffuse pericarditis is certainly a very dangerous disease, generally leading to speedy death. In later childhood its course and result are subject to the same influences as in adults.

Physical Signs.

Inspection.—In rather moderate effusions into the pericardial sac, the thorax may, as a result, suffer a visible alteration of form, provided the chest wall be sufficiently yielding, as is particularly the case in young persons and in the female sex. On

the other hand, an unyielding state of the thorax, shrinking of the lung, and extensive pleuritic adhesions tend to prevent a perceptible change of configuration.

With these provisos, the region of the heart becomes somewhat more bulging and prominent, in consequence of an abundant pericardial effusion, and the left nipple may be situated somewhat higher than the right one. We often see, too, an increased distention of the corresponding intercostal spaces and a diminished respiratory movement of the same. We should not forget, however, that, even in considerable enlargements of the heart itself, an increased prominence of the cardiac region may also take place.

According to Duchek, in pericardial effusions we have a uniform dilatation of the left half of the thorax, the same as in pleuritic effusions; while, in consequence of the increased pressure in the left side of the chest, the ribs remain in the inspiratory position. Duchek is in accord with Corvisart, Philipp, and Gendrin, whilst nearly all the more recent authors speak only of an increased prominence of the region of the heart. Inasmuch as the pressure in the left side of the chest is actually increased by a large pericardial effusion, we must agree with Duchek to this extent, that the left side of the thorax may undergo a certain degree of enlargement from this cause. But the enlargement is not uniform, as in pleuritic effusion, but the particular region of the heart is the most bulged out, because the pressure of the pericardium filled with fluid cannot be exerted equally in all directions, but manifestly must be directed chiefly from behind forwards. The costal cartilages are thus arched forwards, and at the same time the ribs raised *in toto*. By this feature the dilatation of the thorax from pleuritic effusion is distinguished from that of pericardial effusion.

In the opinion of some observers, such as Gendrin, the enlargement referred to is partly due to inflammatory paralysis of the intercostal muscles. Although this theory is somewhat fanciful, I have yet seen in many patients with pericarditis excessive tenderness of the intercostal spaces without concomitant pleurisy, and we must thence infer that in these cases the intercostal muscles participate in the inflammation.

In excessive pericardial effusions the epigastrium is also sometimes seen to be highly swollen, as was first observed by Auenbrugger. This phenomenon is partly due to a forcing downwards of the diaphragm and displacement of the liver, and partly to swelling of this organ on account of stasis.

It has been stated, too, that the skin of the præcordial region

occasionally shows an œdematous condition in pericardial effusions. In regard to this, Bamberger remarks that he has seen such an œdema only in connection with accompanying general dropsy; and such, indeed, will always be the case.

An undulating motion, visible in several intercostal spaces, has been described by many authors, and first by Senac, and referred to the fluid being set in undulatory motion by the movements of the heart. This view arises from confounding such motion with visible contractions of the heart itself. On the contrary, a visible undulation of this sort upon the thorax tends to show the non-existence of a large pericardial effusion (Cejka).

Under some circumstances *palpation* gives evidence of important changes. This is especially true of a careful examination of the apex-beat. If the fibrinous element preponderate in the exudation, any alterations of the apex-beat which may be present possess nothing characteristic, and serve only to confirm such abnormalities as are partly recognized by the pulse, such as irregularity, acceleration, and strengthening of the heart's action; in the latter case the apex-beat may also be strengthened and somewhat diffused.

The pericardium is distended by the fluid effusion, and the heart thus becomes endowed with greater mobility than it possesses under normal conditions. Accordingly, we are sometimes enabled to make out a greater capacity for change of position on the part of the apex-beat in conformity with changes in the patient's position than was the case before.

It was formerly considered a sign of pericardial effusion if, on changing the patient's position, an alteration took place in the site of the apex-beat, apart from the degree of mobility of the apex. But a variation in the locality of the apex-beat is common likewise in health; and, according to numerous observations by Gerhardt, in very many healthy persons, upon lying on the left side, the apex-beat moves about 2 ctm. further towards the left.

As we have already mentioned, the cavity of the pericardium is not entirely filled by the heart, so that a certain mobility of the latter is allowed on change of posture. In regard to the degree of this mobility, pretty wide individual differences prevail.

Apart from a readier mobility of the apex-beat on change of the patient's posture—as a sign of increased mobility of the heart—its position is rather frequently changed in such a manner

that it is pushed somewhat further outwards (to the left), and occasionally also a little lower down. Inasmuch as the effusion into the pericardium first collects about the base, the latter is turned downwards, the heart lies rather more horizontal, and therefore the apex must turn outwards. It is only when the diaphragm is pressed down by the effusion that the heart's apex descends.

In proportion as the fluid fills the whole pericardial sac and surrounds the heart on all sides, the apex-beat grows weaker, and may finally wholly disappear, because the fluid presses the apex away from the chest-wall, and thus prevents the beating of the apex against it. Occasionally, indeed, according to Hammernik, a slight sinking in occurs with the systole in place of the previous bulging. The weaker the apex-beat was at the commencement, the earlier, of course, will it disappear, since the energy of the heart's action diminishes with the progress of the exudation. *Vice versa*, a much thicker layer of exudation will be required to produce a disappearance of the apex-beat in case the heart's action is very powerful, and especially if hypertrophy be already present. If, in consequence of old (circumscribed) adhesions, or also as the mere result of agglutinations between the cardiac and the parietal layers, the crowding down of the heart's apex away from the anterior thoracic wall has been rendered impossible by the imprisoned fluid, the apex-beat may continue to be perceptible.

Under any circumstances, the disappearance of the apex-beat is an important symptom, all the more valuable if a disproportionately full and tense pulse show that it cannot be owing to weakened power of the heart.

We not unfrequently observe that the apex-beat, which the effusion had caused to disappear in the dorsal decubitus, becomes apparent again on bending the body forwards in the sitting or standing posture, because the heart falls further forwards and forces the fluid back.

The friction of rough pericardial surfaces is but comparatively seldom to be felt, as it requires a striking intensity of the friction sound; in such cases we feel, with the flat hand or with the fingers singly, a more or less diffused, multiple

and strong scratching, shoving, or creaking. It is not generally difficult for a skilled observer to distinguish a rubbing from a purring. In many cases it is possible to intensify the perceptible rubbing sensation by pressure on separate intercostal spaces, or to first render it perceptible in this way.

A feeling of fluctuation in those intercostal spaces against which the pericardium, filled with fluid, lies, is indeed mentioned by some, but is denied by authoritative observers, such as Bamberger. *À priori*, it is, in truth, very unlikely that under the conditions existing a fluctuation should be perceptible.

Moreover, Friedreich justly rejects Zehetmayer's statement, that with his outspread hand he had obtained a sensation as if the heart were beating in several places at once, so that it was impossible to distinguish at what point the true apex-beat took place, and where he felt a movement communicated to the fluid.

Percussion.—The cardiac dulness can undergo no change until a certain amount of fluid has been effused into the cavity of the pericardium. The pericardium becomes distended by the exudation, and as a consequence the cardiac dulness is extended and increased, and its figure, as mapped out on the anterior thoracic wall, shows, in the majority of cases, a characteristic form. How great a quantity of fluid is necessary, to be recognizable by percussion, can be only approximatively stated; as a general thing, we must not expect notable changes of the cardiac dulness if the quantity be less than 100 cubic centimetres. But many circumstances may conspire to render this approximative measurement worthless.

As a rule, pericardial effusion enlarges the area of relative as well as that of absolute cardiac dulness. Observers are still at variance as to whether it is more correct to lay the greater stress in diagnosis upon the extent of perfect lack of resonance or upon that of diminished resonance—*i. e.*, upon absolute or relative cardiac dulness. Bearing in mind the various statements in literature seeming to show the absence of any change in the cardiac dulness in a considerable number of cases, which statements refer altogether to the absolute cardiac dulness, and taking counsel of our own experience, we shall reach the conclusion, that a knowledge *of the relative cardiac dulness* is of the greater value in the diagnosis of pericardial effusions, since indeed the absolute

cardiac dulness not uncommonly, in cases of well-marked effusion, shows little or no alteration.

The increased intensity of the relative dulness is very noteworthy, and by its aid we are the more easily enabled to mark out with perfect clearness and thoroughness the limits of the dulness.

The enlargement of the area of relative dulness is due to the fact that the distended pericardium compresses the lungs forward to a greater or less degree. The absolute cardiac dulness enlarges almost in exact proportion as the borders of the lungs are pressed backwards entirely away from the anterior chest wall by the distended pericardium, which does not always take place, and never uniformly. It occurs especially when the pleura pericardii (lamina mediastinalis) is agglutinated to the pleura costalis; therefore all statements which assert the importance of such agglutination in the diagnosis of pericarditis are of value only in so far as they refer to enlargement of the area of absolute cardiac dulness.

Besides the quantity of exudation in the pericardium, the intensity and extent of the dulness really depend upon some other factors. Foremost among these is the degree of elasticity of the lungs, since the stiffer and more inelastic is their tissue, the greater resistance do they offer to compression. Such lungs, particularly if emphysematous, are not so easily forced away from the chest wall and compressed, and, moreover, they are more voluminous, and less of the pericardium comes in contact with the anterior wall of the chest. If the borders of the lungs be firmly adherent they cannot retreat, but can only be compressed. Partial adhesions of this sort may cause the dulness to assume an irregular outline; this happens to a still greater extent in consequence of old partial adhesions of the pericardial layers to each other, since in that case the distention of the pericardium takes place in whatever direction is rendered possible by the particular sort of adhesion.

It is the different degrees of elasticity of the lung tissue which, above all else, determines the individual varieties of the state of the absolute and relative cardiac dulness in exudations of like amount. Hammernik sought to explain these differences by assuming a superficial (normal) and a deep position of the pericardium.

It is true that in certain persons we find the heart more or less movable, more or less covered with lung, but the folding of the pericardium upon the anterior chest wall and its fixation there by pulmonary tension, assumed by Hammernik, do not exist. According to him, on lying down, and as the result of exudations, the pericardium is raised from its firm position and at the same time displaced. The bulk and the degree of elasticity of the lungs are sufficient to produce the differences which have been mentioned in the dulness in pericardial effusions.

The increased intensity and area of the cardiac dulness are all the more significant when suddenly developed under the eye of the observer. If the change occurs slowly, it will certainly be very difficult on some occasions to distinguish small effusions, without a friction-sound, from actual or apparent enlargement of the heart. It is only in rare cases that large effusions present difficulties, on account of the extent as well as the form of the dulness.

The enlarged and more intense cardiac dulness of pericardial effusions may be confounded with enlargement of the heart's volume, as well as with denudation of the organ or retraction, fixation, and diminished size of the lungs, allowing a larger portion of the anterior surface of the heart to come into contact with the chest wall. Error may arise, too, from thickening of the anterior border of one or both lungs, from sacculated pleuritic effusion, from tumors and neoplasms. By calling *all* the facts to our aid, a sure diagnosis is possible, as a rule.

The enlargement of the area of relative cardiac dulness takes place in the direction of the heart's length as well as in that of its breadth, and it not uncommonly happens that, together with the other signs of pericarditis, a suddenly developed increase in the breadth of the cardiac dulness, to the extent of one or two finger-breadths, may suffice to show the probability of a trifling effusion. As a rule, however, the increase in the direction of the long axis preponderates.

In larger effusions it may happen that the apex-beat is still to be felt, and that the left boundary-line of the area of relative dulness extends towards the left a considerable distance beyond the site of the apex-beat, because the pericardium is lifted away from the heart by the fluid (Traube, Gerhardt). This is the only condition under which the cardiac dulness can extend beyond the situation of the apex-beat towards the left, and therefore the presence of this symptom is a sure sign of pericardial effusion.

The outline of the enlarged pericardial dulness upon the anterior thoracic wall constitutes in the majority of cases a *triangle*, the apex of which is directed upwards and its base downwards.

Von Stoffela (Oppolzer's Lectures) believes that at the beginning of the effusion the dulness forms a similar triangle with the base directed upwards, and that this is reversed only with the increase of the fluid. This view does not accord with observation, and rests upon the erroneous conception, that at first the effusion accumulates at the base of the heart, and distends the pericardium in its breadth; whereas this takes place rather lengthwise, the base of the heart sinking down lower at the same time.

The triangular shape of the cardiac dulness is owing to the fluid collecting at first around the base of the heart, and thus distending the pericardium in an upward direction, while the base of the heart sinks as far downwards as its attachments will allow. Now, with a further increase of the effusion, the position of the fluid is governed by the weight and the distensibility of the pericardium, the base of which is independently directed downwards, and thus the form is determined, and not, as Duchek believes, by the separation and recession of the borders of the lungs, since in that case any increase of the heart's volume would necessarily cause a triangular dulness.

As a rule, this triangular area of dulness in pericardial effusions is considerably enlarged, particularly in its whole upper segment, immediately the patient is brought from the dorsal decubitus to the upright position of the trunk, and to a still greater degree if the body be bent forwards (Gerhardt), since in these postures more of the fluid sinks forwards and impinges upon the chest wall. We find this indeed in enlargement of the heart also,[1] because a heavy heart sinks forwards, but not in the same degree, and with the same regularity, as in pericardial effusions.

As regards the size of the triangle, it is of course proportionate to the amount of fluid effusion in the pericardium, but not in so strict a manner that we can estimate the quantity of

[1] I have had an opportunity of observing this state of things in a great number of cases at *v. Ziemssen's* Clinic, and the same statement may be found in Niemeyer and Seitz.

the fluid from the size of the area. The reasons for this include nothing more than has been said in regard to the varying degrees of elasticity of the lungs.

In effusions of moderate amount, the apex of the triangle usually reaches to the third or second costal cartilage of the left side; but in very large effusions the sternum may be dull on percussion throughout its whole extent.

The left border of the triangle, in effusions of slight amount, usually runs above the left nipple, or somewhat towards the left from it downwards and outwards. In copious effusions the left border of the triangle may even cut the anterior axillary line, so that the dulness may extend into the axilla itself.

The right leg of the triangle is, as a rule, more nearly vertical and less divergent than the left, following the course of the border of the right lung, and reaches to the right border of the sternum, or only a little further to the right. In very large effusions, however, it may even overstep the right mammillary line, so that a large portion of the anterior half of the thorax is taken up by the outline of the heart.

The lower limit of the dulness, corresponding to the base of the triangle, is commonly found at the level of the sixth and seventh ribs. We can only partially determine the lower limit directly by percussion, on account of the contiguous left lobe of the liver; we therefore project the line, from that portion which we have ascertained, towards the right side of the thorax to the point where the hepatic and cardiac areas of dulness come together at an obtuse angle.

More rarely still is the diaphragm pushed down on the left side by pericardial effusions—as a rule, only in those of unusual amount or very long standing, so that the dulness reaches down to the eighth rib, and an evident displacement of the left lobe of the liver coexists.

As a result of this mobility of the effusion, in obedience to gravitation, we also observe a manifest capacity in the dulness to change its position laterally, so that, for instance, in lying on the right side the dulness extends further to the right. At Lindwurm's Clinic I saw a young girl with polyarthritis and pericarditis, in whom, when lying on the right side, the fluid sank so

far over to the right that the outline of dulness reached nearly 4 ctm. further to the right than when she was lying on her back. This phenomenon, too, is seen in great enlargement of the heart, but not generally to a very striking extent.

On percussing the left subclavicular region, in large pericardial effusions, we find that the pulmonary resonance differs considerably, as a rule, from that of the right side ; it is higher in pitch and duller or more or less decidedly tympanitic, in consequence of the compression sustained by those portions of lung which rest upon the pericardium. Graves observed also in one case a hernia-like projection of the apex of the left lung above the clavicle. Behind, also, it is not rare to find the lower portions of the lungs deficient in air, on account of compression, and they may even be perfectly destitute of air, although very rarely, it is true.

Usually there is no difficulty in distinguishing a compression of this kind from a coexisting pleuritic effusion, the presence of the latter being shown by the greater intensity of the dulness, by the linear course of the dulness descending from behind forwards, and by the diminished vocal fremitus, which is generally increased in cases of compression of the lung.

In accordance with the importance formerly ascribed to the adhesion of the pleura pericardii to the anterior wall of the chest, the immobility of the borders of the lung during respiration came to be regarded as an important diagnostic sign of pericarditis. This sign is, however, not unfrequently absent in pericarditis ; in fact it is found, as a rule, only in chronic inflammations with extensive adhesions of the borders of the lung, resulting from adhesive pleuritis and other causes. For the diagnosis, therefore, of pericarditis the symptom has no special value. The adhesion of the pericardial pleura to the anterior chest wall, even when the lesion is admittedly extensive, is obviously incapable of rendering the borders of the lung entirely immovable. Of course the adhesions act as a restraint upon the excursions of the pulmonary borders, but the resistance is overcome in part by the act of respiration, which forces these portions of the lung against the adherent pleura. This supposed fixation of the borders of the lung from pericarditis is regarded by Traube as of less significance than the presence of a vesicular murmur surrounding the line of

dulness, indicating that the lung is capable of expansion, notwithstanding some compression and retraction.

In proportion as the fluid exudation becomes absorbed, the areas of absolute and relative cardiac dulness in the majority of cases return to their normal limits, often very rapidly. If the adhesions referred to were as common and as important as has been supposed, the absolute cardiac dulness would necessarily remain enlarged in a considerable proportion of cases.

The most constant and most trustworthy physical sign of pericarditis is the *pericardial friction sound.* This is produced by the roughening of the smooth surface of the serosa by the deposits of fibrin, usually upon both surfaces, but friction may occur even when only one is roughened. Roughness and a resulting friction sound may be occasioned also by concretions, neoplasms, etc., as well as by inflammatory deposits.

Collin and Walsh have expressed the opinion that a friction murmur might be excited by dryness of the serous surfaces. This view has been confirmed by the observation of Pleischl, that the marked viscosity of the serous membranes in cholera can give rise to pericardial friction. Moreover, Mettenheimer has heard friction in a case of extravasation into the muscular substance of the heart without pericarditis. These observations have, to be sure, been doubted by many writers, but without good reason. On the other hand, Gendrin is doubtless mistaken in his opinion that friction may occur simply as a result of violent action of the heart.

The friction sound usually occurs very early in the disease, continues for several days or weeks, then becomes gradually feebler, and at last disappears ; or it may cease very suddenly from the occurrence of copious effusion or adhesions. The latter may after a while give way, and then the sound reappears.

In many cases the occurrence of friction is the first indication of the onset of the disease—in fact, it is generally the only symptom ; but it may also be preceded for some time by inflammatory symptoms, such as pain in the cardiac region, fever, etc.[1] When considerable pericardial effusion occurs, the friction sound often becomes weaker or disappears entirely ; still this is by no means always the case, the friction being sometimes heard and felt where as much as a quart of fluid is present (Cejka). When the

[1] See the cases of Mayne, Traube, and others.

separated serous surfaces again come in contact with each other, in consequence of absorption of the fluid, the sounds frequently become clear again. It is rare for the friction sound to be absent during the entire course of the disease. This may happen, however, where there is a large effusion poor in fibrin, or where the action of the heart is very feeble, but the cases are exceptional where general adhesions take place so early that the murmur cannot at some time be detected.

Frequently the friction sounds are heard first and loudest over the base of the heart, near the origin of the large vessels, and here also the sign may disappear last, because in many cases the deposit of fibrin commences at this point, and here the serous surfaces remain longest in contact when effusion takes place. Still the rule is not invariable; friction may be heard first and most distinctly over any part of the heart, while over the base it is absent altogether.

The friction murmurs differ very much in quality and intensity. Sometimes they sound like a slight grazing of rough or viscous surfaces, sometimes like a faint grating, at other times like a loud scraping or creaking, such as is produced by bending new leather or by walking on half-softened snow.

Attention was first called to the new-leather murmur (bruit de cuir neuf), as a sign of pericarditis, by Collin. Bouillaud distinguished three varieties of pericardial friction sounds: the grazing sound, the new-leather sound, and the grating sound, and he thought that the conditions for the origin of each of these varieties could be accurately determined. Observation, however, teaches that the quality of the friction murmur depends upon other causes than the character of the deposits and false membranes—*e. g.*, on the quantity and position of the serous exudation and the force of the cardiac movements.

These are certainly the most common varieties of friction sounds in the pericardium, and their pericardial origin is best determined by these characteristics. But modifications of the most various kinds may occur, which it is impossible to describe clearly or to render intelligible by comparison with familiar sounds. Thus very peculiar combined sounds may be produced by the union of pericardial with endocardial murmurs. In many cases, to be sure, the character of the pericardial friction sound is so striking that its true nature can at once be deter-

mined with a considerable degree of certainty. In other cases, however, the distinction from endocardial murmurs by this method alone is very difficult or impossible. This is especially the case with the rough systolic pulmonary murmurs. In such cases the distinction must be made by other means.

The pericardial friction sounds, being produced by the movements of the pericardial surfaces over each other, must evidently be heard through the whole period of the heart's action, and are not, like the endocardial murmurs, connected exactly with the moment of the closure of the valves, that is, with the systole and diastole. The friction sounds not only accompany the heart sounds, but are prolonged beyond them, are interposed, as it were, between them (Skoda), and may occupy the whole duration of the cardiac movement. In the latter case, the ear often receives at first quite a confused impression, but intermissions can be detected, which remind one of the rhythm of the footfall of a galloping horse. Each side of the heart can produce a systolic and diastolic murmur of different durations, so that each beat of the heart may be accompanied by four murmurs. Most frequently three are heard, one presystolic, belonging to the systole of the auricles, and two longer sounds, corresponding to the systole and diastole of the ventricles.[1]

Gerhardt has noticed a rare pericardial friction sound, which he describes as *divided into several parts*. It was heard by him in a case of complicated valvular disease, where the pulse had sunk to forty. Multipartite undulations were also observed in the jugular veins, and, synchronous with the undulations, a multipartite friction sound was heard to the outside of the aorta. The undulations and the friction murmur were supposed to be produced by the right auricle undergoing multipartite contractions[2] before a ventricular systole occurred.

When the friction murmur occurs only at the time of the systole or of the diastole, it is generally prolonged beyond the cardiac sounds; if it be exactly synchronous with these sounds it is usually very difficult to determine absolutely its pericardial character.

The pericardial friction sounds, moreover, frequently present

[1] Traube, Ges. Abhandlungen. 2. Bde.

[2] Lehrb. der Auscultation und Percussion.

the peculiarity of being altered by the respiratory movements, there being almost always an increase of the sound during inspiration. A similar result occurs also in endocardial murmurs, but very rarely; these usually remain unchanged or decrease in intensity. The inspiratory increase of the pericardial sounds is probable due to the greater friction between the opposed surfaces of the pericardium, occasioned by the contraction of the diaphragm.

Another important feature of these sounds is the effect produced by changing the position of the patient, this procedure, where the lesion is circumscribed, causing their appearance or disappearance. When, in consequence of effusion, the sound has been lost, it will frequently become audible again if the patient be made to sit upright or to bend his body forwards. Sometimes also, if the chest be elastic, forcible pressure with the stethoscope will develop or increase the sound,[1] but if the pressure be too forcible, the movements of the heart, and at the same time the friction, are enfeebled (Friedreich).

According to Gerhardt, the abstraction of blood sometimes increases the intensity of the friction sounds, sometimes diminishes it (through absorption of the exudation, or weakening the cardiac movements ?).

Under some circumstances the sounds seem very superficial, as if heard directly underneath the ear; but this is the case only when they originate in the superficial cardiac region, (where the heart is not covered by lung tissue), and then the impression results rather from the strength than from the proximity of the sound. On the other hand, friction murmurs, particularly the more feeble ones, are often confined to a very limited space, while the endocardial murmurs, except perhaps the presystolic mitral murmur, are generally audible over a larger surface.

Sometimes, though rarely, a strong friction murmur can be felt. Occasionally it is so loud that the endocardial sounds can no longer be recognized, and are completely concealed. In such cases it is important to auscultate the carotid, where the transmitted aortic and pulmonary sounds can usually be heard.

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[1] This fact was doubted by Skoda, but without good reason.

Not only is pericarditis frequently combined with endocarditis and valvular affections; but aside from this connection, it may itself give rise to endocardial murmurs.[1] Thus adventitious systolic murmurs are sometimes observed at the mitral orifice, and still more frequently at the origins of the vessels, particularly the pulmonary artery. Generally these vascular murmurs are due to the usual cause, but it is probable that in some cases they are produced by the pressure of the effusion upon the walls of the vessels.

Cejka has noticed a disappearance of the first aortic sound, and explains it by an inflammatory relaxation of the portion of the arterial wall covered by the pericardium.

A division of the second vascular sound, as frequently observed by Skoda, has no diagnostic significance.

The equalization of the intervals between the cardiac sounds—the so-called pendulum rhythm, as Roger calls it—is at least of infrequent occurrence.

When a copious effusion into the pericardium has taken place, the cardiac sounds and endocardial murmurs may become feeble or entirely disappear. Sometimes this is the case only when the patient is lying down, the sounds returning in the upright position, or on bending so as to throw the heart forwards. Usually the sounds are merely weakened; a complete disappearance is very rare. Under such circumstances the force of the pulse is often found to be disproportioned to the feeble sounds of the heart.

If the separation of the opposing pericardial surfaces (anteriorly) be slight, or wholly prevented by the occurrence of agglutinations or adhesions, the cardiac sounds will still continue to be audible over this region.

The pericardial friction sound may be confounded not only with endocardial murmurs, but also with the *pleural* friction sound produced on the external surface of the pericardium by roughness of the pericardial pleura or of the opposing pleuritic surface. This sound is called the *extra-pericardial, pneumo-pericardial, or pleuro-pericardial friction sound.*

[1] As has been observed by Latham and Hughes.

A. Ferber has recently, in his work on Pleuritis,[1] carefully investigated this form of friction sound. According to his observations, it is heard most frequently in the neighborhood of the apex of the heart, that is, near the tongue-shaped lobe of the lung and the almost triangular complementary fold of the pleura lying in front of the heart; more rarely it is heard along the sternum, as high as the third or second rib, where the median borders of both lungs are situated. Usually in such cases two sounds can be distinguished, one connected with the respiration and the other with the movements of the heart. The friction sound produced by the cardiac movements is modified in several different ways by the respiration, becoming more distinct on superficial breathing, while the portion of the sound connected with the inspiration is frequently increased by moderately strong respiration. When the breath is held, the sound disappears immediately or after a few beats of the heart; but it may also continue during the entire respiratory pause, or, after a partial disappearance, return. A. Ferber thinks the sound may be produced in two ways, according to the situation of the roughness, either by the friction of the roughened pleura against the pericardium or by the friction of the roughened pericardial pleura against the opposing pleural surface. The influence of the suspension of breathing depends upon the action of agglutinations, which prevent the systolic impulse of the heart from producing any rubbing until a strong respiration breaks up the adhesion.[2]

The extra-pericardial nature of the friction sound is indicated with certainty when a suspension of breathing has the effect of diminishing the intensity or of immediately arresting the sound. If it continues unaltered through the respiratory pause, a distinction is impossible.

Some other respiratory sounds are liable to be confounded with endocardial,[3] but not with pericardial murmurs. I recollect, however, one case in which there was a dry, crackling systolic râle near the apex of the heart, sounding very much like a pericardial friction sound.

The *frequency* and *quality* of the pulse during the course of pericarditis are modified by so many causes, particularly by the occurrence of complications, that the pulse cannot be said to present any distinctive character. In fact, rules are impossible. There are probable indications, however, which it may be well to

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[1] Die physicalischen Symptome der Pleuritis exsudat. Marburg. 1875.

[2] For particulars in regard to this form of friction sound, see article on Pleuritis; also A. Ferber, l. c.

[3] See *Kuessner*, Beitr. zur Kentniss d. accid. Herzgeräusche. Deutsch. Archiv f. klin. Med. XVI. 1. 19.

consider, because in many respects, especially as regards the condition of the heart, the examination of the pulse affords information of the greatest importance.

It is not uncommon for pericarditis to run its course without any considerable acceleration of the pulse. Thus, we often see pericarditis occurring in connection with articular rheumatism, without any further increase of the pulse; but where the patient has been much reduced beforehand, the complication, as a rule, exerts an accelerating influence.

During the first few days of the disease the pulse is often *temporarily* accelerated, so as to run up to one hundred or one hundred and twenty; but this initial acceleration usually subsides within a short time. It is commonly ascribed to an irritability of the heart, induced by the inflammation of its serous coat; that is, the inflammation exerts an accelerating influence upon the cardiac muscle or the excito-motor ganglia. Very frequently it is noticed that a slight bodily exertion is sufficient to produce a considerable increase in the frequency of the pulse.

A *continuous* acceleration may be due to quite a variety of causes, among the most important of which are myocarditis and degeneration of the muscles, lesions which usually induce other changes in the pulse besides an increased frequency. The acceleration of the pulse may depend also upon elevation of the temperature and upon respiratory and circulatory disturbances.

The *rhythm* may also be changed, but this occurs less frequently than some former observers have stated. Thus, Gendrin has asserted that he has observed alterations of rhythm in eleven out of fourteen cases, and Louis in about half; according to Bamberger, however, this symptom is not so frequent, and usually occurs only temporarily at the outset of the disease, and the same opinion is expressed by Nothnagel.[1] The alteration may consist either of an irregularity or of a simple intermittence of the pulse. When the symptom occurs during the latter part of the disease, it frequently depends upon disease of the cardiac muscle. In other cases, and particularly in the initial stage of the disease, the irregularity may be occasioned by the excitation

[1] Ueber arhythmische Herzthätigkeit. Deutsches Arch. für klin. Med. XVII. 2 u. 3.

of the inhibitory nerves induced by the inflammation. In a particular case, however, it is hardly possible to decide whether the chief share is taken by the muscular elements, by the musculo-motor ganglia, or by the inhibitory fibres. A special form of pulse, the *pulsus paradoxus*, will be considered somewhat more fully at the conclusion of this chapter.

Great differences prevail also in regard to *the tension of the arterial system* and *the height of the pulse-waves*. In many cases no change is noticed. At the outset of the disease, corresponding to the more forcible action of the heart, the tension may be increased, and the pulse extraordinarily full. If the filling of the ventricle be interfered with by the occurrence of copious effusion, or if the muscle have undergone important changes, the tension falls and the pulse-waves become very low. In the first instance, if the apex-beat can still be felt, a striking disproportion is often noticed between the action of the heart and the pulse-waves ; in the latter case the pulse is frequently also irregular, and over the heart itself we meet with the changes which belong to degeneration of the organ (feebleness of the apex-beat and cardiac sounds, systolic murmurs, etc.).

Retardation of the pulse occurs but rarely during the course of pericarditis ; now and then, however, it occurs as an after-symptom, and is then generally due to exhaustion of the cardiac muscle. That such a fatigue may occur without any considerable lesion of the muscular substance, and may of itself induce paralysis, cannot at present be denied, although no proof on this point can be obtained during life. Recovery is possible even in inflammatory and degenerative processes, and is no evidence therefore that the condition was one of simple exhaustion.

Traube has recently called attention to the fact that in some cases of copious pericardial effusion the left carotid and radial arteries are smaller and pulsate less forcibly than the corresponding arteries on the right side. He is unable to give a full explanation of this symptom, but he regards it as certain that it must be ascribed to the effusion.[1]

The effusion exerts upon the external surface of the heart a pressure proportioned to the amount of fluid and to the lateral

[1] Charité-Annal. 1874.

resistance resulting from the tension of the pericardium. The share taken by each of these two factors will of course differ considerably in particular cases, according to the varying quantity of effusion, and to the thickness, thinness, or elasticity of the pericardium.[1] The systole of the auricles and ventricles is not restrained by the effusion in the pericardium.[2] During the contraction of the heart the intrapericardial pressure must be diminished. The pressure weighing upon the heart may, however, interfere with the diastolic movement, and thus lessen the flow of blood into the cavities. This mechanical influence of the effusion is felt chiefly upon those portions of the heart which, in consequence of their membranous character, are less resistant to pressure, viz., the auricles.

The diastolic relaxation of the cardiac cavities, particularly the auricles, and the filling of the same with blood, are assisted normally by the *vis a tergo* of the venous blood as it pours into the auricles, and by the elastic traction of the lungs. These two forces are directly opposed by the pressure of the pericardial effusion. Moreover, the traction of the elastic and tense lungs upon the walls of the heart is diminished in cases of pericardial effusion, because the lungs are themselves compressed, and therefore lose a part of their tension.

A large effusion may also exert pressure upon the large vessels where they lie within the pericardial sac, and thus interfere with the filling of the aorta and pulmonary artery.

Inflammation of the pericardial layer of the large vessels within the pericardial sac seems to be a rare occurrence. When it does occur, it may lead ultimately to such lesions as thickening, diminished contractility, and even dilatation.

When the veins are unable to discharge their blood into the auricles in the normal manner, over-distention of the veins in the general, as well as the pulmonary, circulation naturally re-

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[1] The lateral pressure of the effusion in the pericardium can, to be sure, be estimated manometrically in the cadaver, but the conditions are so much altered by death that the figures obtained in this way would be of little direct service.

[2] In Traube's opinion the *systolic motion* of the heart is greater in pericardial effusions than under normal circumstances, because the fluid opposes less resistance to this movement than do the membranes where they fit close to the heart in the normal manner. Berl. klin. Wochenschr. 1872. 7. 78.

sults, while the filling and the pressure in the aortic system is diminished. Under such circumstances the veins in the neck are found to be swollen, and to present undulatory movements; in high degrees of stasis the distention is very marked, and sometimes a true venous pulse is noticed (Stokes, Friedreich, and others). This venous congestion may lead also to passive hyperæmia as well as to catarrh and œdema of the lungs, to more or less cyanosis, and finally to œdema of the extremities and general dropsy. Other results also make their appearance, such as headache, vertigo, enlargement of the liver, gastric and intestinal derangements, and of course also various symptoms due to a defective supply of oxygen to the tissues.

The swelling of the cervical veins is a comparatively frequent symptom, and is, as a rule, accompanied by multipartite undulatory movements. The occurrence of a true venous pulse, without tricuspid insufficiency, was demonstrated by Friedreich in one case of pericarditis. This observer, as is well known, distinguished two forms of venous pulse, one a more marked form, associated with tricuspid insufficiency, and the other a slighter variety, in which the faulty closure occurs with the valves of the veins and not with the tricuspid.

According to Skoda, the venous pulse in large pericardial effusions is produced in the following way: the motion communicated to the fluid by the ventricular systole is transferred to the auricle as the most yielding part, while the expansion of the auricle is hindered by the effusion. This explanation is, however, unsatisfactory; the venous pulse is formed here just as it is under other circumstances.

The highest degrees of stasis and derangements of circulation may be produced by large pericardial effusions, but in most cases they are due chiefly to some affection of the cardiac muscle itself, while the effusion serves to increase still more the results of the imperfect force of the heart. The occurrence of derangements in the nutrition of the cardiac muscle may manifest itself by the diminished fulness of the pulse—the pulse-waves becoming small, and not unfrequently also irregular and intermittent, and the increase of tension at the time of the ventricular systole becoming less marked.

In the presence of cardiac changes like those described, it is usually noticed that the cavities of the heart become dilated. This result is frequently absent, however, when the pericardial effusion is abundant; on the contrary, the heart is found to be strikingly small, notwithstanding the marked degeneration of the muscle, obviously

on account of the pressure of the fluid upon the wall of the heart and the imperfect filling during the diastole. This condition cannot be recognized by physical signs during life on account of the fluid accumulation.

Sometimes the symptoms of weakness of the heart make their appearance very rapidly; attacks of sudden loss of power in the heart may also occur, with an almost imperceptible pulse, and death may ensue in an attack of this kind. These symptoms of cardiac exhaustion are of course not peculiar to pericarditis; they occur in like manner in diseases of the cardiac muscle arising from other causes.

The derangements of *respiration*, which are sometimes noticed during the course of pericarditis, are not to be regarded as always directly due to the pericardial inflammation. Severe pains in the chest and epigastrium may induce an acceleration of the breathing. On the other hand, the mechanical effect of the effusion, as well as disease of the cardiac muscle during the course of a pericarditis, may give rise to serious respiratory disturbances, and in fact to the highest degrees of subjective and objective dyspnœa.

Although the effects of the pressure of the pericardial effusion upon the heart have, no doubt, been frequently exaggerated, yet there can be no question that under favorable circumstances the circulation may be considerably deranged in this way, and that the interference thereby produced in the exchange of gases, may even lead to severe dyspnœa. Still another agency in the production of dyspnœa is the compression of the lungs, or of the left bronchus, by the pericardial effusion. In such cases the respirations are frequent, deep, and markedly dyspnoic, the accessory respiratory muscles are tense, the alæ nasi play, the patient becomes very cyanotic, cold, and collapsed, and the skin is covered with a clammy sweat. Very often there is exquisite orthopnœa. The difficulty of breathing is usually most intense in the recumbent posture, and it is easier for the patient to lie on the left side than on the right. Zehetmayer saw one patient, with a large pericardial effusion, who was able to breathe only when supported on his hands and knees. Patients also experience great distress and restlessness, and suffer unspeakably from a sense of oppression and craving for air.

The orthopnœa, in cases of pericardial effusion, is specially due

—in addition to the usual causes—to the fact that the mobile fluid lies more unfavorably in the horizontal position of the body than in the sitting posture. In the latter position, especially if at the same time the body be bent forwards, the effusion sinks forwards and downwards; while in the recumbent posture it presses upwards and backwards, so as to exert considerable pressure upon the auricles and origins of the large vessels. Moreover, the pressure upon the lungs is greater in this position, and, for the purpose of avoiding this effect upon the right lung, the patient prefers to lie upon his left rather than upon the right side.

In many cases the respiratory disturbances are still further increased by the presence of a dry cough, with a mucous, frothy expectoration.

Sometimes there is said to be a paralysis of the diaphragm, producing a retraction of the epigastrium on inspiration. This has been explained partly by the pressure of the distended pericardium upon the diaphragm, and partly by the extension of the inflammation.

The general condition of the patient is also of importance as regards the appearance of dyspnoic symptoms, for in the anæmic the dyspnœa is less severe than it is in those who are well nourished; the same is also true of the rapidity with which the exudation and consequent obstruction appear, according to the well-known saying, that the more acutely a disturbance begins the more severe will it be.[1]

If the heart itself undergoes a considerable disturbance of nutrition in consequence of the inflammation of its serous envelope, this myocarditis or myodegeneration, with the consequent weakening of the power of propulsion, and the disturbance of the circulation, may lead to intense dyspnœa, and if, in the course of an uncomplicated pericarditis, severe dyspnœa and diminution of the aëration of the blood appear, they are much oftener due to an affection of the heart itself than to the mechanical results of the exudation. The disturbances of respir-

[1] This is analogous to the well-known experiment, according to which an animal dies in an atmosphere containing a certain amount of carbonic acid, if it is suddenly compelled to breathe in it, while it will survive if the air is brought gradually to contain the same amount of the gas.

ation which arise in such a way behave exactly like those which occur in the course of other heart diseases; a more abundant exudation into the pericardial sac would naturally tend to add to the symptoms.

In many cases the dyspnœa, anxiety, and oppression seem to be purely nervous manifestations, due to irritation of the branches of the pneumogastric. Irregularity, also, acceleration of the movements of the heart, stenocarditic attacks, are thought to be due to the influence of the inflammation upon the regulating nerves of the heart in their course.

During the attacks of dyspnœa violent vomiting sometimes occurs, together with tormenting, painful singultus. The latter symptom might be explained by an inflammation of the phrenic nerve passing down over the pericardium. In rare cases the patients complain of a painful and convulsive feeling in the throat and œsophagus, and of dysphagia. The dysphagia may be caused by the direct pressure of a large exudation upon the œsophagus; in other cases it seems likewise to be a symptom of irritation, for it has also been found when the exudation was small.

Double paralysis of the vocal cords, occasioned by the pressure of a large pericardial exudation upon both recurrent nerves, was observed by Baeumler. The voice returned after the exudation was absorbed. On account of the rarity of this symptom Baeumler[1] thinks that it may require the coöperation of several forces, and that the great venous congestion in his case may have had some influence, especially upon the paralysis on the right side.

Some nervous attacks, especially fainting-fits, also convulsions, maniacal attacks, delirium, sopor, which were regarded by the older physicians as frequent and characteristic symptoms of pericarditis, are due not to it but to disturbances of the circulation in the brain, cerebral œdema, or other complications. Only seldom is there such a concurrence of circumstances in the course of a pericarditis that symptoms of this kind are observed.

Corvisart mentioned the purulent breaking down of the bulb of the eye, sometimes appearing suddenly, as a symptom of pericarditis. Friedreich adds to this observation that the symptom is due not to pericarditis, but to endocarditis and embolism of the arteria ophthalmica.

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[1] Deutsches Arch. f. klin. Med. Bd. II. S. 550. 1867.

Bouillaud expressed the opinion that no painful symptoms accompanied pericarditis, and that when such were present they were due to pleuritic complication. This view is decidedly incorrect, for there are cases, especially of spontaneous pericarditis, in which a very characteristic group of abnormal sensations is present.

There are, however, many cases in which all pain is lacking, and others in which it is very slight, or promptly disappears again, so that the patients do not complain of it. A few transient stitches may be felt in the region of the heart. Many patients complain only of a dull pressure upon the breast, or of the sensation of weight and tension, of a load over the heart. In many cases a moderately intense stabbing or pressing pain is felt for some time in the neighborhood of the heart, under the sternum, and may be increased, especially by the respiratory movements, but also by those of the body, by pressure over the heart and by percussion. A notable sensitiveness to pressure in the intercostal spaces occurs rarely.

According to the observations of Baeumler and Guéneau de Mussy, pain is experienced in the epigastrium more frequently than in the region of the heart, and this pain is spontaneous and occurs also upon pressure; the latter is indeed the more common. The epigastric pain can also be increased by respiration and movements of the body, so that dyspnœa and inability to walk result; the movements of the heart can also increase it.

According to Guéneau de Mussy, sensibility to pressure in the epigastrium is found especially at the angle between the ribs and the ensiform process, sometimes on the left, sometimes on the right side.

Pain between the lower insertions of the left sterno-cleido-mastoid muscle—sensibility of the trunk of the phrenic nerve which is involved in the inflammation in consequence of its anatomical relations with the pericardium—is less frequently present at the same time with that in the epigastrium. The pain may radiate from that point, especially towards the left shoulder, but also along the whole of the left arm and towards the left ear. Baeumler also noticed painful sensibility of the left side of the larynx increased by every movement of the heart.

The pains about the heart and in the epigastrium may also radiate in different directions, to the right side of the thorax, the back, and the abdomen.[1]

These subjective symptoms appear in many acute cases at the beginning of the inflammation, and precede the objective changes; in other cases the latter appear first after a friction murmur has existed for some time.

The feeling of palpitation of the heart is not unfrequently complained of, and in fact, under certain circumstances, painful palpitation may be felt. The friction murmur is comparatively seldom perceived by the patient.

Sleep is often disturbed, especially when the above-mentioned painful sensations are present. The head is usually free or but slightly affected. Severe brain symptoms do not belong to pericarditis, but to consecutive or complicating circumstances.

The ultimate changes which the secretion of urine undergoes in consequence of pericarditis depend upon the height of the temperature of the body, upon the amount of liquid exudation into the pericardium, and upon the effect which in some cases the pericarditis has upon the aortic pressure.

Moreover, the fundamental disease must of course be taken into consideration, whether it is combined with high fever, abundant perspiration, exudations elsewhere, albuminuria, etc.

In the majority of cases the amount passed in twenty-four hours is considerably below the average, the specific gravity is increased, it is rich in coloring matters (relatively, and also absolutely in many cases), and a brick-dust deposit is often thrown down on cooling; for at the temperature of the body, the urine being diminished in quantity, becomes saturated with the urate

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[1] The pericardium normally possesses only a slight degree of sensibility; compare Zuckerkandl, Sitzungsber. der Wiener Akad. 1870. LXII. 1. 151. et seq. This author found branches of the pneumogastric of considerable size, varying in number and points of origin, which came from the œsophageal or posterior pulmonary plexus, and were distributed mainly to the posterior wall of the pericardium. The pericardial nerves arise, first, from the pneumogastric; second, from the phrenic, especially those of the anterior and lateral walls; third, from the sympathetic, with fibres from both inferior cervical ganglia.

of soda. Nothing definite is known as to whether the different products of secretion suffer any qualitative or quantitative change, but, according to F. Heller, it is thought that the chlorine compounds, as in other exudative processes, are greatly diminished; as the previous conditions, however, were not exactly known, these investigations cannot be relied upon.

Of the known factors which contribute to the reduction of the secretion of water through the kidneys, several, as a rule, act together. The lessening of the pressure in the aorta, in consequence of the diminished power of the heart, has the greatest influence. The obstruction to the flow of the blood which results leads to the development of congested kidney, and therewith to albuminuria. Even without pronounced congestion, albumen may appear in the urine, rarely as a febrile symptom, more often as the expression of an already existing or complicating disease of the kidneys.

General rules cannot be laid down with reference to the behavior of the fever, for in most cases it is secondary. If the primary disease is an acute febrile one, a further rise of temperature may or may not be associated with the appearance of pericarditis. If the pericarditis first appears while the fever of the primary affection is going down, it is often the case that a new rise of temperature does not occur. Inflammation of the pericardium occurring in the course of chronic affections sometimes begins very acutely with a chill and high temperature; in other and very numerous cases it comes on insidiously, without any rise of temperature. Spontaneous pericarditis, also, simulating an acute affection of the chest, may show high fever with an initial chill, or, still more frequently, without any; this is especially the case when strong, previously healthy, individuals are suddenly attacked by pericarditis. On the other hand, when the disease is developed slowly, in feeble and old people, it often happens that its entire course is free from any rise of temperature. If fever is present it often disappears when the exudation has reached its maximum, and perhaps reappears, during absorption, as a slight increase of temperature. When the exudation is purulent, fever is always present, at least temporarily.

Diagnosis.

To distinguish inflammatory effusion into the pericardium from hydro-pericardium, we have a number of distinctive signs which, in the majority of cases, render it possible to discriminate the one from the other. There is no difference on percussion, or at the most, it may be remarked that the distention of the pericardial sac by a serous exudation may become enormous, greater than it is in most cases of pericarditis. On the other hand, a friction murmur is never present with a serous exudation. Furthermore, all inflammatory and febrile symptoms are lacking, while they are present, frequently, at least, in pericarditis. The consideration of the etiological conditions, which usually declare plainly for one or the other, is very important.

The diagnosis of the character of the exudation is very difficult and generally is only a probable one. There are no certain differences in the physical signs [1] and we can do little more in this way than determine approximately the quantity of liquid. The nature of the primary affection and the individuality of the patient furnish some useful means for determining whether the exudation is sero-fibrinous, or hemorrhagic, or purulent.

Experience teaches that the pericardial exudation in feeble and cachectic individuals, especially with the so-called hemorrhagic diathesis, is very often hemorrhagic; this is especially true of scurvy, morbus maculosus, variola, and scarlatina hemorrhagica. There is the more reason to suppose a hemorrhagic effusion if with its appearance the patient becomes suddenly and visibly anæmic, which always presupposes a considerable loss of blood. In tuberculous and carcinomatous individuals also the exudation is not unfrequently hemorrhagic, but hemorrhages may also take place into the effusion in individuals who were previously quite healthy, and without the aid of any severe

[1] An intense, widespread friction murmur lasting for a long time with slight increase of dulness is certainly to be expected with a fibrinous exudation, and, on the other hand, we may look for a large area of dulness and a slight temporary friction murmur, when the effusion is mainly serous. The fever also is often quite high when the exudation is more fibrinous. These diagnostic signs are very uncertain, although theoretically they appear correct; but manifold complications do not permit so simple a distinction.

general disease. As a rule, however, a sure diagnosis is not possible.

The diagnosis of purulent exudation usually offers great difficulty. It may be suspected in pyæmic affections, for there the pericardial exudation is generally purulent; in other grave general diseases it occurs not unfrequently. Leaving out of consideration those processes whose course is marked by violent symptoms, and in which the appearance of pericarditis usually hastens the catastrophe, there are purulent processes, in the neighborhood especially, which set up inflammation of the pericardium with formation of a purulent exudation. Furthermore, we often find purulent exudations in those cases which take on a chronic character, and in which, temporarily at least, febrile symptoms of a moderate degree usually appear. In such cases the symptoms of disease of the muscular tissue of the heart also appear with especial frequency and severity.

The opinion, that the friction murmur is often absent in purulent pericarditis, seems to me to be erroneous. The murmur often disappears, but only after it has lasted for a long time, and in consequence of the adhesion of the pericardial surfaces to each other.

Ichorous exudations occur rarely, and can only be suspected when there are neighboring abscesses or general septic processes, such as endocarditis diphtheritica, and others. A process of this kind is indicated by no local symptom; only in exceedingly rare cases do we have a pyopneumo-pericardium. The severe general symptoms in such cases, the high fever, the frequent thready pulse, delirium, and rapid collapse belong to septicæmia.

As for the physical signs of pericarditis, similar physical changes under other circumstances may of course lead to mistakes. The increased dulness of the pericardium may be confounded with enlargement of the heart depending upon dilatation and hypertrophy. The shape of the area of dulness is characteristic enough, at least if the exudation is abundant. The disproportion also between the increased area of dulness and the feeble apex-beat, which is entirely lost when the patient lies upon his back, and is also perhaps overlapped by the dulness on the left, together with the slight heart sounds and the absence of valvular murmur, indicates a pericardial effusion.

The spreading and increase of the area of pericardial dulness under the eyes of the observer is very significant. It is true that this may occur rapidly in consequence of dilatation of the ventricles, and thus be mistaken for a moderate pericardial effusion. But acute enlargement of the ventricles most frequently affects the right side of the heart, and consequently the area of dulness seems only to be broader. Such a dilatation of the ventricle must also have a definite cause, which could scarcely escape detection.

Uncovering of the heart by retraction and collapse of the lungs would be made clear by the shape of the area of dulness, by the elevation of the diaphragm, and by the effect upon the dulness of the shifting of the edges of the lungs during inspiration. If, on the other hand, the uncovering is due to connective tissue adhesions, the area of dulness is but slightly changed by inspiration, and at the same time the area of relative dulness does not show an increase corresponding to that of absolute dulness. On the other hand, the edges of the lungs may be so fixed that even when there is a pericardial effusion they cannot be pressed back. In such cases the area of absolute dulness is not increased, but that of relative dulness is. The diagnosis becomes much more difficult when there is emphysema of the lungs. The presence at the same time of an effusion in the left pleural cavity may add some difficulties to the diagnosis, especially if the pleuritic effusion fills the anterior portion of the thorax so that the upper and left outlines of the heart cannot be distinguished, and the organ itself is shifted to the right. In such cases the distinctness of the heart sounds on the right of the sternum is important to this extent, that it indicates displacement of the heart.

It certainly happens very seldom that the edges of the lungs are empty of air at such points and to such a degree that the area of dulness resembles that of a pericardial effusion. This may be caused somewhat more often by encapsulated pleuritic effusions or by pleuritic membranes. Mediastinitis also can cause a similar dulness by the deposit of an exudation on the posterior surface of the anterior wall of the thorax. In all these cases the vesicular respiratory murmur, which is present with

pericardial effusion, is absent over the area of dulness. Fremitus and the movability of the edges of the lungs all furnish important distinctions. Mediastinitis is very rare, and is usually the result of a traumatism, of disease of the sternum, or of the ribs, or of the burrowing of pus downwards from the neck. With aneurism of the aorta or tumors of the mediastinum, the dulness may exceptionally have the form of the pericardial triangle, and as in both cases other characteristic signs may be absent, a mistake may easily be made.

The following case, from v. Ziemssen's Clinic, is an example of the kind:

A man, forty-nine years old, came on the 18th January, 1876, to the surgical clinic to be treated for a tumor on the left forearm, which had first been noticed in 1870 as a small knot, and which in the subsequent interval had grown to the size it then had. The patient, in consequence of loss of strength and severe dyspnœa, was in a condition which made an operation impracticable, and he was therefore sent to the medical clinic. The difficulty in breathing began early in December, 1875.

A tumor on the left forearm, chiefly on the flexor side, extending from the upper third to the wrist, increasing anteriorly, and largest at the wrist, covered with a glistening skin traversed by large veins. A few knot-like prominences of soft fluctuating consistency. The consistency of the rest of the tumor was firmer. The muscles were involved in the tumor. Flexion and extension quite feeble. The upper arm and hand are unaffected. The cubital, axillary, and sub-clavicular glands are not swollen. Appearance anæmic, voice weak, considerable emaciation. The patient cannot lie at all upon the left side, and for only a short time upon the right; pronounced orthopnœa. Respiration frequent, costal and very dyspnoic; the epigastrium scarcely moved at all; no cyanosis, no swelling of the veins in the neck. An area of dulness upon the anterior thoracic wall, of the shape of the distended pericardium; the blunt angle of the triangle reaches to the clavicle, and is quite broad, the left border extends down from the left axilla, the right one runs along the right side of the sternum, and unites at the third rib with another area of dulness. In the right anterior region, relative dulness begins at the lower edge of the third rib, and absolute dulness at the lower edge of the fourth rib, extending towards the axilla and down to the normal boundaries. These limits change but slightly on inspiration, and the same is also true of most of the points of the triangular area of dulness. On changing from the recumbent to the sitting posture and bending the upper part of the trunk, the absolute dulness on the right side shifts a very little upwards. The triangular area also is slightly affected by the change of position; the right border alone is somewhat nearer the sternum when the patient is lying down. Posteriorly on both sides is a hand's breadth of dulness, which can be somewhat displaced. The apex of the left lung slightly dull, especially posteriorly towards the vertebral column. On the right

side, in front (R. A.), harsh, puerile respiration; on the left side, anteriorly (L. A.), undefined breathing with rhonchi, deep respiration slightly bronchial; left posterior superior region (L. P. S.) also undefined breathing; R. P. I. and L. P. I. feeble breathing, with many dull râles. R. A. in the region of the dulness the breathing is almost absent, and the same condition exists over the triangle. R. P. I. and L. P. I. the vocal fremitus very weak, and is likewise very much diminished in L. P. S.; in front and on the left side, it is stronger at the apex than on the right side; in L. A. in the region of the dulness, it is very weak; and on R. A. it is normal to the edge of the dulness, where it entirely disappears.

Apex-beat nowhere to be felt, heart sounds very weak, and still more difficult to hear on account of the labored respiration. Spleen not enlarged. The lower border of the liver lies three finger-breadths below the ribs. Purulent sputum in considerable quantity, some of it brownish-red and mixed with blood. Microscopical examination showed pus, blood, and some alveolar epithelium. Urine scanty, large deposit, very acid, no albumen. Temperature normal. The following diagnosis was made: effusion into the pericardium, perhaps due to new growths; a tumor in the right pleural cavity arising by metastasis from the one on the left forearm; and also a double pleuritic effusion. The patient died on the 24th of January.

Autopsy.—Body large, skin pale; a tumor on the left forearm, largest near the wrist. Both lungs adherent, especially at their anterior edges. An effusion of blood between the two pleural surfaces in the side of and behind the left lung. Scattered through both lungs encephaloid nodules, varying in size from a walnut to a child's fist, and containing extravasations of blood. The neoplasm crowds the mediastinum to the left, away from the lower and anterior border of the right lung, and causes it to cover the heart. The pericardial sac contains very little liquid. The surface of the heart covered with abundant stringy flakes; otherwise the heart shows no change. Liver normal in size, full of blood, evenly colored; the kidneys also full of blood, firm. The spleen flaccid, pale, measuring 10½ by 8 ctm. The mucous membrane of the stomach somewhat reddened, and showing small hemorrhages. The intestinal mucous membrane somewhat injected in places. Mesenteric glands unaltered. The tumor proved to be a sarcoma.

As for the auscultatory signs of pericarditis, it was said, when speaking of the symptomatology, that in the majority of cases pericardial could be distinguished from endocardial murmurs. Often the character of the murmurs and the moment at which they are heard are sufficient; the pericardial sounds are frequently delayed, and if they constitute a pure systolic-diastolic double murmur, the systole and diastole give the same impression of friction, while in the endocardial double murmur the systole and diastole are different. The point also at which the murmur is heard is of importance: the pericardial murmurs

are often heard at the origin of the great vessels and over the right ventricle, and then are not unfrequently quite circumscribed. Change of posture, too, has much more effect upon the pericardial than upon the endocardial murmurs; a murmur which can be heard only when the patient is in the upright position or lying upon his side is always pericardial (Gerhardt).

The possible means of distinguishing intrapericardial from extrapericardial friction have also been already mentioned.

The diagnosis, whether and to what extent the muscular tissue of the heart is affected by the inflammation of the pericardium, can be based only upon such symptoms as are caused by myocarditis and myodegeneration, feebleness of the heart, weakness of the circulation and stasis, irregularity of the movements of the heart. It must, however, be remarked that not every irregularity in the action of the heart which occurs during pericarditis, justifies the diagnosis of disease of the muscular tissue.

Course—Terminations—Prognosis.

As the intensity of the symptoms and the fundamental cause in pericarditis show great differences, so, too, is its course very irregular, and no rules can be laid down for the duration of the process. The whole process may terminate completely within a few days, as the cases of Baeumler and others show, though it is true that this can only happen when the effusion is not great. Even considerable sero-fibrinous exudations may be completely absorbed in the course of a few days, during which there is a notable increase in the amount of urine, but the entire duration of the disease in those favorable cases is ordinarily from eight to ten days; this is also usually the case in the course of polyarthritis rheumatica, pneumonia, and pleurisy, and in many idiopathic cases—much more rarely when preceded by primary disease of the heart. But even under these circumstances a longer course, one of from three to six weeks, is by no means an exception. The arrest of the inflammation, or rather of the exudation, is sometimes made known by a febrile attack; if then absorption goes on rapidly, a slight elevation of temperature may be associated with it.

A fatal termination is very seldom the exclusive and immediate consequence of such a pericarditis, and happens only when the effusion is very large and when there are symptoms of disturbed respiration and circulation, dyspnœa, cyanosis, venous congestion, reduction of the arterial pressure. But as soon as we have to deal with feeble or old people, the conditions are changed and more unfavorable.

In complicated cases, the kind of complication is of course of the greatest moment, since it is often more important than the pericarditis itself. Implication of the muscular tissue of the heart must be considered not merely as a complication, but rather as a result of the pericarditis. It may either appear acutely and cause death in the course of a few days, or it may lead to a chronic wearisome illness.

On the other hand, death is caused often and promptly—within twenty-four hours—by scorbutic or hemorrhagic pericarditis, in which the effusion offers almost the characters of pure blood and may be very abundant, so that the loss of blood has much to do with the suddenness of the fatal termination. Thus a short time ago I saw a middle-aged man succumb to an idiopathic pericarditis, in which the exudation had the appearance of pure blood and was very abundant.

In contrast with sero-fibrinous pericarditis purulent inflammation may lead to death very rapidly before the exudation has become large. This is most frequently the case in grave general affections, such as pyæmia, and then it is always a question how much is to be ascribed to the pericarditis. In other cases, in which the purulent condition of the exudation is dependent upon chronic constitutional or local causes, the course may be a very long one, but death is almost certain to result sooner or later.

The importance and course of pericarditis due to chronic affections of the lungs, kidneys, and also the heart itself, may differ greatly ; every possible form of exudation and course may present itself. In general we can only say that in such cases every form of pericarditis may appear as the exclusive and direct cause of death.

The gradual change into a chronic condition must be men-

tioned as a termination of acute pericarditis. This change is rare in rheumatic pericarditis, but more frequent in the idiopathic form, especially in chronic diseases of the heart and in chronic constitutional affections, in Bright's disease of the kidneys. After an acute period [1] the exudation remains stationary, or it may be in part absorbed, and then after a while poured out again, and thus the process drags along.

Chronic pericarditis may grow out of an acute attack, or it may have this character from the beginning; recovery seldom takes place. Death usually results—sometimes suddenly, and in consequence of secondary myodegeneration, which is the almost inevitable result of chronic pericarditis. If life is preserved, absorption of the exudation goes on slowly; there remain portions [2] encapsulated between the pericardial layers; the abundant vegetations and the impermeability of the lymphatics interfere with the absorption of the exudation. In such cases permanent lesions of the heart almost always result,—partial or general adhesion of the surfaces of the pericardium, degeneration and atrophy, dilatation and hypertrophy.

After severe attacks and long duration of the disease, the signs of exudation may entirely disappear, while restoration of the muscular tissue of the heart follows slowly, and the symptoms from that quarter still continue. This is more likely to be the case when the general nutrition has suffered during the disease and anæmia has been developed. Every bodily exertion causes oppression and palpitation, a condition which only gradually wears away.

It is hard to say in how many cases, and under what circumstances in the various forms of pericarditis, complete restitutio ad integrum ensues, and in how many cases lasting changes remain. We can only compare the frequency of pericarditis during life with the number of autopsies in which pathological changes of that nature are found. We can also arrive at some conclusion from the appearances presented at autopsies on the bodies of persons in whom during life pericarditis is known to have

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[1] If the exudation is purulent, a cheesy collection and afterwards tuberculosis may result.

[2] The term chronic, as applied to pericarditis, depends less upon the duration of the disease than upon the permanence of the symptoms or upon the appearance of inflammatory relapses.

occurred, but from which they have recovered, and subsequently died from other causes. But we should be careful to exclude those cases of milk spots which appear to have arisen without any discoverable inflammatory cause.

We have already remarked (p. 565), that doubtless even connective-tissue bands may subsequently be ruptured and absorbed. Loose spongy adhesions of this kind possibly may frequently be the result of a pericarditis, but firm and lasting adhesions of considerable extent are not often found as the result of a simple sero-fibrinous inflammation;[1] they usually arise where the course of the disease is protracted, and absorption is delayed. More frequently, instead of adhesions, we find fibrous thickenings of the serous layers as the remains of a pericarditis, which however cause no functional disturbances.

The *prognosis* of pericarditis must have been much more unfavorable in early times, when they were only able to recognize the more severe cases, and especially those associated with affections of the heart itself, than now, when even the slightest cases come into the computation. Even Corvisart, Hope, Gendrin, and others considered pericarditis a very dangerous disease; but Hache, and especially Louis, showed that this view was incorrect.

In one hundred autopsies of persons affected with pericarditis, Willigk found a cure had resulted in seventy-three; but here the milk spots were counted also. Duchek, in fifty-six cases of general pericarditis, found twenty-seven cases (48.2 per cent.). Bamberger, in sixty-three cases, found thirty-seven (58.7 per cent.). Louis, in one hundred and six cases, found seventy (66.1).[2]

Naturally the prognosis varies greatly, according to the sequence of events and the peculiarities which we have described under the "Course of the Disease;" but yet we are able to give a more accurate opinion in pericarditis, in some cases, than in many other forms of disease. First of all, we must consider the underlying cause of the pericarditis; thus the pericarditis arising in the course of polyarthritis rheumatica has a very favorable prognosis. Von Bamberger had not a single fatal case out of seventeen such, which he observed; and out of a great number of cases which I have seen, I have never yet seen one with a directly fatal result. So also those cases arising during attacks of pleuritis and pneumonia have a favorable prognosis, according to von

[1] Duchek asserts that adhesions of the pericardial layers are found in ten per cent. of the cases in which recovery has occurred.

[2] See *Duchek*, loc. cit. p. 37.

Bamberger. For idiopathic cases we cannot lay down any general rules, and the prognosis will be very varied. When there is heart disease already existing, a pericarditis will be regarded with suspicion. In all other cases the prognosis will be more or less unfavorable, according to the character of the primary disease.[1] Beside the primary disease, existing complications will have more or less importance; thus pleuritis, endocarditis, or even pneumonitis does not make the prognosis unfavorable, but simultaneous disease of the heart-substance renders it highly unfavorable: accordingly all symptoms which suggest such a condition should be carefully considered in forming a prognosis.

The extent of the inflammatory process is also of importance, and a limited inflammation gives cause for anxiety only in cases where it complicates abscesses of the heart-substance, aneurisms, etc., and here on account of the primary disease, to which it possibly points. Moreover, the extent and character of the exudation is of weight, for large exudations cause much more danger than slighter ones. The prognosis in sero-fibrinous exudations is generally favorable, while in purulent it is bad, and in hemorrhagic effusions the general constitutional symptoms and the quantity of the blood effused decide the question. So, likewise, the onset and course of the disease influence the decision; for nearly all cases which terminate in a cure run an acute course, while chronic cases offer very little encouragement.

The age of the patient influences the prognosis very decidedly; infants of a year old usually die (Gendrin); in the later years of childhood the mortality is less; and young adults show the least mortality. Beyond middle-life and in old people the prognosis again is unfavorable. The constitution also must be considered, for weak and broken-down patients succumb more frequently than those who are strong and well nourished. Duchek is inclined, according to his statistics, to lay some stress on the sex of the individual, for the females showed a worse ratio of cures than the males.

The prognosis will generally be rendered more difficult only in cases where pathological changes take place in the heart itself,

[1] According to Latham, of patients with non-rheumatic pericarditis, ninety-one per cent. died.

for these arise very acutely, but very insidiously, so that we may be suddenly and unexpectedly surprised by a failure of the heart's action.

Whether or not the cure will be complete, we can only say approximately in the lighter cases, but not in the severe ones; indeed, some of the sequelæ we cannot count upon, even after the cure of the disease; these may occur later or not at all. (Such are hypertrophy, many cases of adhesion, etc.)

Treatment.

The objects to be attained in the treatment of pericarditis must be very varied, according to the character of the case, and quite opposite indications will arise; for one case will require only a symptomatic and expectant treatment, while another must be attacked very energetically.

Considering pericarditis in general as an inflammation of a serous membrane, our first indication would be to limit as far as possible the extent and amount of the inflammatory process. Of the antiphlogistic measures to accomplish this end, general blood-letting certainly is useless and at the same time dangerous, and it has been generally abandoned. So, also, the employment of mercurial preparations, especially in the form of inunctions, although much extolled still by many physicians, has scarcely any influence that we can recognize on the course of pericarditis. We must at the outset confess that the end to be sought in our treatment is not a direct and immediate limitation of the inflammatory process, which we cannot accomplish, but rather the control and arrest of important conditions which accompany it. First among these are derangements of the heart's function, and next pain and fever. To control the early increase in rapidity and force of the heart's action and also the fever, digitalis in large doses has proved itself a sovereign remedy.[1] But a

[1] The dose must be varied according to the strength of the preparation which is employed, which is not the same in all localities. Here in Munich we need to give from thirty to forty-five grains to develop the full influence of the digitalis. It is given in powder, from fifteen to thirty grains divided over the twenty-four hours, or as an infusion with nitrate of potash or soda.

careful and strict watch is necessary on the part of the physician that he may at once stop the remedy as soon as the digitalis shows its full effect on the body and regularity of the pulse. The advantages to be gained in pericarditis by getting a quieter action of the heart are greater than would appear at first sight, and possibly the inflammatory process itself is thereby indirectly affected.

The application of an ice-bag over the heart at the same time has been found to be very advantageous, whether in quieting the tumultuous action of the heart together with the sensation of palpitation, or allaying the pain which accompanies the disease. If the application is kept up uninterruptedly, it will most wonderfully allay the pain, and is gladly used by most patients, and recognized as efficacious. At the same time the influence of cold upon the heart itself is advantageous. The employment of cold has been proved by others, as by Friedreich, to be useful, so that nowadays the moist and warm applications and poultices of former times, for this stage of the disease at least, are no longer used. If the pain is very severe, so that respiration is impeded, then a small dose of morphine, either hypodermically or by the mouth, is clearly indicated, and is to be recommended in preference to local blood-letting; blisters we cannot recommend. To control the occasional sleeplessness, we may order morphine, or a Dover's powder, or a dose of chloral, with benefit.

As regards the treatment of the patient in other respects, above all we must secure complete rest of mind and body, and he should not be allowed to leave his bed. The giving of nourishing food in liberal quantities is usually prevented by a loss of appetite, or even disgust for food; but still there is here no imperative demand to avoid the loss of bodily strength by crowding the nourishment from the very outset. Accordingly, in the beginning, we might order a purely liquid diet, and in addition some acid drink—lemonade (drinks containing carbonic acid gas are by many physicians considered injurious).

The bowels will be best regulated by enemata, or by some mild mineral waters.

When we reach that stage where the inflammation is arrested, and the effusion remains at a stand-still, we must change our

treatment. If the effusion is not great and resorption begins properly, then no more active treatment is required. We lay aside the ice-bags, give more strengthening nourishment, and simply order the patient to lie quiet in bed for a time. If, on the other hand, the effusion is great, and does not begin to subside for several days after reaching its highest point, then we must endeavor to hasten its resorption. For this purpose we employ the various diuretics, *digitalis* in small doses to increase the arterial pressure, solution of acetate of potash, etc.[1]

Concerning the value of iodide of potassium for this purpose, I have no accurate information.[2]

In this stage of the disease good nourishment is very essential.

If possible, the patient should not now lose any more of the elements which make up the body, but rather add to them, for we know how absorption of effusion is promoted by increased nutrition. The demand for albumen must generally be met by easily digested animal food, such as eggs, milk, and the like. The demand for non-nitrogenous food is probably less than normal, and we must give it in a variety of forms as it may be required. The patient may be allowed beer or light wine. Unfortunately the improvement of the nutrition cannot always be accomplished, for various reasons which we need not here discuss, and moreover our knowledge of the quantitative interchange of materials under these circumstances is very deficient.

Sometimes the exhibition of preparations of iron and quinine may serve to assist in the improvement of nutrition, and also after the effusion has been absorbed, these remedies may be employed to combat the existing anæmia.

Besides causing diuresis, we may hasten the removal of the effused fluid by other measures—by derivation to the skin, exciting diaphoresis, and likewise by derivation to the intestines. Physicians are divided as to how far we should rely on one or other of these methods, but at all events derivation to the skin is the one usually employed—flying blisters, and warmth and moisture (Bamberger), or, when the fluid has become purulent,

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[1] Of digitalis, for this purpose, we give doses of from eight to fifteen grains; of the salts which cause diuresis by their affinity for water, acetate of potash is, according to my experience, the most efficacious. Of this I am in the habit of giving from sixteen to fifty-six grains, and as a rule the quantity of urine is thereby decidedly increased.

[2] Warring Curran recommended veratrine, which he said caused a diminution of the fever, lessened the dyspnœa, and increased the quantity of urine.

continuous vesication, inunction with iodine ointment, or painting with the tincture of iodine, etc. The method by diaphoresis has in later days been somewhat neglected; not wisely altogether, as it seems to me, for, in appropriate cases and with proper precautions, I have found the use of hot baths and steam baths, with subsequent packing, of great advantage.

When reabsorption of the effusion has occurred, and when the patient must be considered as really cured, we must advise careful and prudent living for some time yet, and, above all things, that he should shun severe bodily exertions and all things which call forth increased action in the heart (excessive use of alcohol, tobacco, etc.). If the circumstances of the patient permit, he should for the time have favorable surroundings (a trip to the country, etc.).

The general principles for the treatment of an acute pericarditis must often be variously changed or supplemented in given cases, according to the character of the primary disease, the presence of complications or specially dangerous symptoms, the nature of the material exuded, the course of the disease, and, finally, the peculiarities of the individual.

In many cases of secondary pericarditis the conditions are such that we do not feel called upon to administer remedies for this morbid process; at most, we endeavor to arouse by stimulants the more rapidly failing power of the heart. Such a state of things is encountered in acute general diseases, and also in patients with phthisis or Bright's disease.

From other, and indeed opposite reasons, many cases of articular rheumatism with pericarditis will not call for any change in treatment on account of this complication, since there are no subjective sufferings, and no important symptoms appear.[1] In other cases, on the contrary, especially with larger

[1] In the use of salicylate of soda, in the treatment of articular rheumatism, a remedy has been introduced which certainly, as regards promptness of action, must be considered an acquisition. As regards its influence upon the pericarditis, it cannot be doubted that, by completely cutting short the rheumatic attack, the occurrence of pericarditis will be prevented. But whether a pericardial inflammation once existing will be substantially affected thereby, seems to me doubtful at least, with our present knowledge, even though the use of salicylate of soda may control the affection of the joints.

effusions, I do not consider a thoroughly indifferent treatment warrantable, though I readily allow that even by that means we may obtain favorable results. The same holds true for the pericarditis, which complicates a pneumonia or a pleurisy, and especially for that which arises in the course of an already existing heart disease. Moreover, it is to be remembered that in all cases in which, from the existence of a pneumonia, pleurisy, etc., the resistance to the circulation is very great, the utmost attention must be given from the outset to sustaining the powers of the heart, and every treatment calculated to weaken it must be avoided.

Of the numerous individual symptoms, weakness of the heart in its various forms should be carefully considered. So soon as any evidences of it appear, either in an acute or chronic form, the treatment must be essentially a strengthening one. The acute attacks of insufficiency of the heart, associated with very great anxiety, a feeling of oppression in the chest, and cold surface and extremities, call for the strongest stimulants: camphor internally or subcutaneously, then ether, musk, and, above all, alcohol (brandy in the form of punch or champagne).

If we want to accomplish anything under such circumstances, we must use our stimulants energetically and continuously.

The only object in the use of stimulants is to sustain life during the momentary danger, and to support and improve the circulation for the time; the heart itself will not be affected thereby. Permanent improvement must be sought first in nourishment, and especially in that containing large amounts of albumen. With this, digitalis, carefully given in small doses, will greatly assist, and also a continued use of alcohol (in the form of wine, not beer) will be found of service.

If the process of degeneration of the heart has come on in a more chronic form and led to dropsy, the same principles of treatment hold good; but here diuresis may be promoted and assisted by other measures, and in such cases hot baths and steam baths show excellent results.

If patients of this kind suffer from distress for breath and are entirely sleepless, the use of chloral or morphine is, in my opinion, imperatively demanded; also, the occurrence of hic-

cough or frequent vomiting may sometimes call for the use of narcotics.

The nature of the exudation may call for careful consideration; this is especially so in the distinctly hemorrhagic form, as this declares itself by other special symptoms. Aside from the fact that in many cases of this kind the constitution demands a very strengthening treatment, we should make the attempt to arrest the bleeding by the application of ice-bags and the use of ergotin. In the acute scorbutic forms Kyber has occasionally found excellent results from the use of large doses of quinine.[1]

The treatment of purulent effusions must in many cases be addressed to the primary disease; aside from this, our object will be to sustain the strength of the patient, and particularly to prevent any loss of power in the heart and to control the exacerbations of fever. Inasmuch as almost every treatment shows bad results, the question arises whether we should not attempt to procure an artificial emptying of the purulent exudation by an operative procedure; and here we may discuss the question whether the operation is permissible and what is its value in pericardial exudations generally.

The operation, first proposed by Riolan the younger, was subsequently recommended by Senac, and also more recently by van Swieten, but was rejected by Morgagni and Corvisart—partly on account of the danger of the operation, and partly on account of the uncertainty of the diagnosis. The operation was first performed by Romero, in Barcelona, and with success (1819).[2] Later, repeated attempts were made at paracentesis pericardii by Skoda and Schuh, and also by Kyber in scorbutic cases, and many times with favorable results. Trousseau likewise recommended the operation. Aran carried out the proposal of Laënnec, to throw in some irritating material into the pericardial sac, after puncture, to hasten the adhesion of the surfaces, and with favorable results, although the operation had to be repeated several times, on account of a renewal of the accumulation, in consequence of which air entered into the pericardial sac. Yet physicians were not wanting to condemn the operation, declaring it to be dangerous and not proportionately productive of good. It did not come into general use, so that we cannot give a verdict based on practice. Very recently various physicians have repeated the operation, and especially in England it seems to have gained a firm foothold, where T. Clifford Albutt has particularly interested himself in it. T. H. Bartlett

[1] M. Friedreich, loc. cit.

[2] Friedreich, loc. cit.

has also published a case which was operated upon with favorable results, in which the aspirator was employed (drawing off fourteen ounces of bloody serum).[1]

Experience thus far has taught us that the removal of pericardial effusions by an operation is not attended by any great danger, if ordinary precautions are taken. The results in accidental injuries of the pericardium support this view of the matter. The question therefore remains, how far does the operation accomplish its object, judged by results? and how should it best be performed? Much that applies to the emptying of pleuritic effusions would be in place here.

The number of cases operated on is as yet too small to be able to give a verdict as to the results of this method of treatment, and accordingly the indication for the operation is now confined to a narrower field than it perhaps will be later, when our experience shall have been enlarged. These indications are: *immediate danger to life from the amount of the effusion*, and the certainty of an unfavorable result from a stationary and particularly a *purulent effusion.*

As regards the method of performing the operation, this, in the course of time, has been as varied as that of thoracentesis. Riolan and also Laënnec proposed trephining the sternum. Probably we must here make a distinction between serofibrinous and purulent effusions, and we should doubtless select a capillary trocar and aspirator with exclusion of air (Potain) for the former, while in the latter the simple removal of the fluid would scarcely suffice for a permanent cure of the trouble. In the purulent form we shall have to establish an open wound with the opportunity of washing out the cavity, as has already been done. In the simpler operation with the capillary trocar and exclusion of air, we need not be very anxious about the exact spot where the puncture is made. We should insert the instrument in the fourth, or better, the fifth intercostal space, close to the edge of the sternum, and, in order surely to avoid wounding the heart, the operation should be performed with the patient lying on his back. We can easily avoid wounding the mammary artery.

When the position of the parts is normal, it is scarcely possible to puncture the pericardium near the left edge of the sternum without opening the pleural sac at the same time, as was shown by Hammernik, and as we learn from the position of the parts observed on frozen sections of the body. But this injury of the pleural sac is of trifling importance when the capillary trocar is used with exclusion of air;

[1] Lancet. Dec. 19, 1874.

on the other hand, we need not anticipate wounding the edge of the lung, since the latter is pressed aside by the distended pericardium.

If we are considering the question of a radical operation for pyopericardium, we should first endeavor, to the best of our ability, to assure ourselves that the pericardial and costal pleuræ are adherent, which in long standing, purulent effusions, we certainly may expect to find is the case.

For this reason, the advice given by Larrey and Desault, to enter between the xiphoid cartilage and the seventh left costal cartilage, though anatomically justifiable, should not in most cases be followed, for here other difficulties are added to the operation.

Whether the result of the operation is simply palliative, *i. e.*, whether the emptied sac refills or not, would, with a larger experience, probably receive the same answer as in the operative treatment of pleuritic exudations.

So far the results are favorable; that is, after the operation the emptied sac collapses, and permits the lungs to expand; nevertheless a better result is attained by emptying the sac partially and at intervals rather than all at once.

At all events, where life is threatened it is justifiable to operate, and it is also allowable to make an exploratory puncture with a Pravaz's syringe for the purpose of ascertaining whether the exudation is purulent or not.

Tubercular Pericarditis.

Acute miliary tuberculosis of the pericardium is very rare, and is only seen associated with general acute miliary tuberculosis, especially affecting the serous membranes. In the course of general acute miliary tuberculosis, the eruption on the pericardium is destitute of symptoms—at all events, we are not yet sure whether miliary tubercles on the pericardium give rise to friction murmurs or not, although it is quite probable they do so.

Tubercular pericarditis is most frequently found in persons suffering at the same time from tuberculosis in other organs, and especially in the lungs, and either is developed from the outset in this manner, or else a simple chronic pericarditis finally assumes a tubercular form. It is rare that the latter occurs in

individuals who give no evidence of tuberculosis elsewhere in the body; moreover, we must remember that, even in cases of marked pulmonary tuberculosis, a simple pericarditis is more common than the tubercular form.

We may learn how rare tubercular pericarditis is even in tuberculous patients from the records of Willigk, who in one thousand three hundred and seventeen autopsies on tuberculous individuals only found the pericardium affected eleven times. Eppinger found tubercular pericarditis somewhat more frequently.[1]

We find in such cases a more or less abundant exudation in the pericardial sac, which is almost always markedly hemorrhagic in character. The pericardium is in a state of chronic inflammation, is thickened in bands, and the two coats are here and there adherent and covered with both old and recent deposits of lymph. Together with the inflammatory products, numerous gray and yellow miliary tubercles are found distributed over the serous coat, and between the bands and pseudo-membranes, and sometimes, wedged in between the muscular tissues of the heart, are seen yellow, cheesy nodules, from the size of a bean to that of a hazel-nut.

The symptoms of tubercular pericarditis resemble exactly those of a simple chronic inflammation of the pericardium. In the course of the disease the watery portion of the exudation may be partially reabsorbed—a commencement of a cure, just as occurs in tubercular pericarditis. As the physical signs give no clear indication for the diagnosis of tubercular pericarditis, we can only suspect it when a chronic pericarditis occurs as a complication of tubercular disease in the lungs, peritoneum, etc. In these complicated conditions we can naturally arrive at no certain conclusion from the nature of the fever, etc. The diagnosis will be all the more uncertain from the fact that, as we have already said, even in tuberculous patients, the pericarditis, as a rule, has the character of a simple inflammation, and that, on the other hand, cases are unknown where the tubercular inflammation confined itself entirely to the pericardium.

Our treatment can only be the same as that for a chronic

[1] Prag. Vierteljahrschrift. 1872. 113. p. 13.

pericarditis—endeavoring to sustain the heart, allay the fever, and support the general health. When there is existing tubercular disease of the lung, we must avoid all depressing treatment as well as all operative procedures.

The occurrence of a pericarditis of this nature will have no special influence on the treatment of the primary trouble in the lung, but it will serve to hasten the surely fatal termination of that disease.

Carcinomatous Pericarditis.

Carcinoma very rarely occurs in the pericardial sac, and when it does, it is as an extension of the disease from neighboring parts, particularly from the mediastinum, sternum, or œsophagus, on to the pericardium. Much more rarely do we find nodules on the pericardium, secondary to acute miliary carcinoma in other serous membranes.

Köhler found cancer of the pericardium six times in 9118 autopsies; Günsburg once in 1700; and Willigk, in 477 autopsies on patients dying of cancer, found the pericardium affected seven times.[1]

When the new growth extends to the pericardium from neighboring parts, the parietal layer shows, as a rule, a diffuse infiltration of cancer, or else the mass grows from a single limited spot and projects in nodules into the sac. Associated with it, there is almost always a fluid effusion into the cavity. The fluid is frequently hemorrhagic in character, sometimes purulent and ichorous, and again in other cases it has more the appearance of serum.

The symptoms may be the same as those of chronic pericarditis or of a serous effusion. When cancer exists in the neighborhood, the development of an increased dulness over the heart, of a triangular shape, will cause us to suspect that the pericardium has become involved in the new growths, although even in such cases a simple pericarditis or a serous effusion is more common. We have alluded to the fact that under such

[1] In *Duchek*, loc. cit.

circumstances the extension of the new growth outside the pericardium may easily simulate enlargement of the pericardium itself as regards the signs on percussion.

Sometimes when the new growth extends on to the pericardium we may have the signs of an acute pericarditis ; this occurrence will also cause us to suspect cancer of the pericardium when it exists in the immediate neighborhood.

Adhesions between the Heart and Pericardium.

Meckel, Observ. sur les malad. du cœur. Hist. de l'acad. royale des scienc. Tom. XI. Berlin. 1875. p. 56.—*Haller*, Element. phys. Tom. I. Lausannæ. 1757. p. 289.—*Morgagni*, De sedibus et causis morb. Tom. II. lib. II. ep. 17-24.—*Lieutaud*, Hist. anat. med. Tom. II. lib. II. Paris. 1767.—*Pohl*, De pericardio cordi adherente ejusque motum turb. Progr. Lips. 1775.—*Nebel*, Progr. de pericard. cum cordo concreto. Giessen. 1778.—*Senac*, Traité de la struct. du cœur. etc. Tom. II. Paris. 1783.—*Corvisart*, Ess. sur les mal. et les lés. org. etc. Paris. 1818. p. 37.—*Kreysig*, Die Krankh. des Herzens. II. 2. S. 623.—*J. Hope*, Krankh. des Herzens und der grossen Gefässe. übers. Berlin. 1833. S. 333.—*Dundas*, On a peculiar disease of the heart. Med. chir. trans. Vol. I. 1815. p. 37.—*Müller*, De concret. morbos. cordis cum pericard. Diss. Bonn. 1825.—*Skoda* und *Kolletschka*, Ueber Pericarditis in pathol. und diag. Beziehung. Oesterr. med. Jahrb. 28. Bd. Wien. 1839. S. 419.—*Aran*, Recherch. sur les adhér. génér. du péricarde. Arch. gén. de méd. Avr. 1844. p. 466.—*Gairdner*, On the favorable terminations of pericarditis. etc. Edin. Month. Jour. of Med. Sc. 1851.—*Skoda*, Ueber die Erscheinungen der Verwachsung des Herzens mit dem Herzbeutel. Zeitschr. der Wiener Aerzte. April, 1852. Sitzungsberichte der k. k. Akademie der Wissensch. Nov. 1851.—*Körner*, Casuistische Beiträge zur Lehre der Erscheinungen der Verwachsung des Herzens mit dem Herzbeutel. Wochenbl. d. Zeitschr. d. k. k. Gesellschaft d. Aerzte zu Wien. Nos. 2 u. 5. 1855.—*Williams*, Lectures on diseases of the chest. Deutsch von Behrend. 1841.—*Francis Sipson*, On the changes, etc. Worcester. 1844.—*Cejka*, Drei Beobachtungen. Prag. Viertelj. 1855. 2. Bd.—*Law*, Obs. on pericard. Dubl. Quart. Journ. of Med. Sc. Aug. 1856.—*Potain*, Bullet. de la soc. anat. de Paris. Août, 1856.—*Bertin*, Pericard. adhés. avec caillots hémorrhg. enkyst. dans les fauss. memb. Bull. de la Soc. anat. Juil. 1857.—*Traube*, Zur Lehre von der Verwachsung des Herzens mit dem Herzbeutel. Med. Zeitung. hrsg. vom Vereine für Heilk. in Preussen. 11. 1858.—*Breithaupt*, Vollständige Verwachsung des Herzbeutels mit dem Herzen. Ibid. No. 14. 1858.—*Mauriac*, Des adhérences du péric. et de la ponct. de cette cavité séreuse dans la péricardite. Gaz. des Hôp. 38. 1858.—*Henry Kennedy*, On adherent pericard., its diagnosis and its results. Edin. Med. Journ. May. 1858.—*Gairdner*, On the results of adherent pericard.

Edinb. Med. Journ. June, 1858.—Rechtseitiges pleuritisches Exsudat, mit Pyopneumothorax, Pericarditis und theilweiser Verwachsung des Pericards mit dem Herzen. Oesterreichische Zeitsch. für pract. Heilk. V. 41. 1859. 691.—*Hinds*, Does the pericard. become universally adherent to the heart after acute pericarditis and recov. Brit. Med. Journ. 16. March. 1861.—*P. Bosisio*, Not. clinich. int. ad un caso di aderenza totale de l'peric. Ann. univers. di Med. Milano. Nov. 1861.—*Skoda*, Zur Diagnose der Verwachsung des Herzens mit dem Herzbeutel. Wien. allg. med. Zeitsch. 1863. 36.—*Henrie-Marie Fournier*, Des adherences du pericarde. Thèse, Strassbourg. 1863.—*Abelles*, Deutsche Klinik. No. 31. 1859.—*Geist*, Klinik der Greisenkrankheit. Erlangen. 1857–60.—*Oppolzer*, Spitalzeitung. Nos. 10, 13, 14. 1861.—*Jaccoud*, Gaz. hebd. VIII. 1861.—*Moore*, Gaz. méd. de Paris. 31. 1863.—*Betz*, Auscult. Erscheinungen bei pericard. Verwachsungen. Memorab. IV. 8. 1859, und XI. 1866.—*N. Friedreich*, Zur Diagnose der Herzbeutelverwachsung. Virch. Arch. 29. Bd. 1864.—*E. Archer*, Two spec. of extensive calc. depos. in the peric. Trans. of Path. Soc. XX. p. 191.—*S. Wilks*, Adherent peric. as a cause of cardiac disease. Guy's hosp. rep. XVI. pp. 196–208.—*E. Galvagni*, Stud. clinic., Sulla sinfise card. e sul. rientram sist. Riv. clinic. di Bolog. 1873. Nov.—*A. Kussmaul*, Ueber schwielige Mediast. pericarditis und den paradoxen Puls. Berl. klin. Wochenschr. 37–39. 1873.—*E. Cerf*, Die Verwachsung des Herzbeutels. Diss. Zürich. 1875 (contains a number of bibliographical references).—*Webb*, A case of a most extensive pericardial adh. Philadelph. Med. Times. April 15, 1872.

After the erroneous supposition, that obliteration of the pericardial sac was a congenital defect, had been abandoned, Vieussens, Lieutaud, Morgagni, and others began to investigate the matter, both clinically and from a diagnostic point of view. Morgagni found, by the comparison of a series of forty-five cases, that the most frequent sign was absence of a visible apex beat. By various observers, as by Testa and others, severe palpitation was considered an important sign; while Senac and likewise Corvisart declared that irregular and tumultuous movements of the heart and the feeling of tearing in the cardiac region were essential symptoms.

Heim and Kreysig asserted that they could recognize an adherent pericardium by a depression formed at each systole of the heart, at the left of the scrobiculum cordis, close under the false ribs. Sanders stated that he had observed an undulatory movement at the same place. Laënnec, Bouillaud, and Piorry, however, could not satisfy themselves of the accuracy of these last statements. J. Hope described various symptoms which he

believed to be characteristic of adherent pericardium ; but none of these proved trustworthy. According to Williams' observations, the movements of the heart can be more distinctly felt ; the intercostal spaces are retracted ; the dull sound, on percussion in the pericardial region, remains the same during inspiration and expiration. All these are given presupposing a simultaneous adhesion to the anterior chest wall. Aran considers the disappearance of the second sound of the heart to be an essential sign.

In spite of the efforts of these numerous investigators, the diagnosis of adhesions between the heart and pericardium had no solid foundation until Skoda gave us the exact information, that in a series of cases characteristic symptoms are always observed, which render a diagnosis possible. Recently Friedreich has added another objective sign of obliteration of the pericardial sac.

Etiology.

Adhesions of the two pericardial layers are found varying in extent and of a more or less solid consistency. At autopsies we occasionally meet with them, either as unimportant and accidental conditions, or else as forming the anatomical starting-point of disease of a special character, which may lead directly to a fatal termination. We must distinguish between loose adhesions limited in character, and those which are firm and more extensive. If the adhesion is so close and so general that we can no longer recognize the point of separation of the two layers, the condition is called *obsolescence* or *obliteration* of the pericardial sac.

Every adhesion of the layers of the pericardium is the product of an antecedent pericarditis. Not uncommonly the inflammation which caused the adhesion has been entirely latent, or at least has given rise to no violent symptoms.

Yet abundant fibrino-serous effusions with delayed reabsorption and chronic pericarditis are the most frequent causes of adhesions. In how many cases of pericarditis permanent adhesions remain, is, as we have already hinted, a difficult question to answer. Limited adhesions are much more frequently found

than those which are extensive, or those which cause total obliteration of the sac.

Chambers found adhesions in 5 per cent. of all autopsies. Leudet saw in 1,003 autopsies adhesions in 5.7 per cent., and total obliteration in 2.5 per cent. Willigk found complete adhesion sixty-eight times in 4,500 post-mortem examinations, *i. e.*, in 1.5 per cent. According to Gairdner, extensive adhesions occur in 2.3 per cent., and slight ones in 5 per cent. more. Geist, in the examination of five hundred and fourteen people of advanced age (from sixty to ninety-three years), found adhesions twenty-six times—that is, in 5 per cent. Günsburg saw it only in 1.2 per cent. of bodies examined.

How far other things, besides those enumerated, serve to develop adhesions, we do not as yet know. As examples we might mention feeble movements of the heart, continued duration of the inflammatory irritation, repeated renewals and cessation of the inflammation, etc.

Bouillaud and Ayres observed total adhesion of the pericardium in cases of new growth in the mediastinum, which had completely enclosed the heart. Barlow and Peacock saw hydatid cysts with adhesion of the pericardial layers.[1]

As regards age, the statements hold true which were made in reference to pericarditis.

E. Cerf made a collection of forty-three fatal cases with total adhesion; and, arranged in order of age, the autopsies occurred as follows.

From the 1st to 10th year	3	times.
" " 10th " 20th "	15	"
" " 20th " 30th "	8	"
" " 30th " 40th "	5	"
" " 40th " 50th "	6	"
" " 50th " 60th "	3	"
" " 60th " 70th "	3	"

The youngest of these forty-three patients was eight years old. Cases of pericardial obliteration occurring in early childhood have been reported also by Rilliet and Barthez, Wilks, Hope, and others. Bednar found adhesions in an infant of three months, and Billard and Hueter in new-born children. In the latter case there must have been pericarditis during fœtal life.

The *time* required from the beginning of the inflammation, for the development of adhesions, is very different in different cases.

[1] *Cerf*, loc. cit.

Sometimes the period cannot be marked at all during life, and in other cases not accurately.

In one case, observed in the General Hospital in Vienna, after eight days from the commencement of the disease, a retraction over the apex was observed with each systole, and this gradually and steadily increased; at the autopsy fresh adhesions were found between the apex of the heart and the pericardium.[1] E. Cerf relates a case in which symptoms of adhesion were developed after nine days. Bouillaud at an autopsy saw adhesions formed in twenty-four days, which could with difficulty be torn asunder. Similar observations have many times been reported.

Pathological Anatomy.

The connective tissue which binds the two layers together is sometimes delicate and loose, and again firm and abundantly developed. Often, owing to the constant movements of the heart, the adhesions are drawn out into strings and fibrous cords, which possess considerable thickness and firmness; and this is especially the case at the apex. In other cases both serous layers are grown fast together. Moreover, the pericardial layers, and particularly the visceral, may be more or less thickened, so that the heart seems to be surrounded and enclosed by a firm sheath or capsule of connective tissue. Fibrous thickenings of the pericardial layers of this nature are not always present, however. Sometimes we are still able carefully to separate them, and in other cases the union is so intimate that separation is impossible.

Sometimes between the adhesions we find remains of the exudation imprisoned and fully encapsulated, thickened pus, and masses like pap or cream, consisting purely of chalk molecules, arising from calcification of the pus. Sometimes larger, irregular, brittle, chalky concretions are formed in this way.

The fibrous thickenings and the connective tissue binding the two pericardial layers together may also undergo calcareous degeneration. In this way bony plates are formed, of either a rounded or an irregular, branching shape, and situated chiefly upon the visceral layer. Where the calcification is extensive,

[1] Schmidt's Jahrb. Bd. 103. S. 139.

the heart may appear to be encased in a bony shell. In such cases it is difficult to conceive how it is possible for the heart to contract at all.

Such calcifications were observed even by the older physicians. Laënnec, in one case, saw the heart entirely enclosed within a bony shell. Louis, Cheselden, Gendrin, Kreysig, report similar instances. Among eighty-five cases of adhesions between the pericardial layers, Chambers observed calcific deposits five times. In a case reported by Bouillaud a concretion on the apex of the heart had perforated the pericardium. Förster and von Bamberger have also reported instances of extensive calcific deposits. There was extensive calcification in the following case, a brief history of which I subjoin:

S. J., æt. fifty-four, a whitewasher, presented himself on the 25th of May, 1875, at the Clinic of Prof. von Ziemssen. He stated that he had formerly often suffered from shortness of breath, especially upon making any physical exertion. He had never had acute rheumatism, but had probably indulged too freely in the use of alcohol. In December, 1874, the difficulty of breathing having increased very decidedly, and the feet and abdomen having begun to swell, patient applied for admission to the hospital, and remained there under treatment six weeks, when he was discharged in an improved state of health.

His condition on the 25th of May was as follows: Marked cyanosis, becoming extreme during paroxysms of coughing; mucous membranes of a dark, livid hue; the skin of the lower extremities bluish-red and decidedly sclerotic. The external jugulars are distended with blood, and do not diminish in size even when a deep inspiration is taken; there is no visible pulsation in the veins of the neck. The eye balls are very prominent. Pulse small, soft, 104, tolerably regular, and not paradoxical. Number of respirations, twenty-four; slight dyspnœa. The apical impulse cannot be felt at any point; no murmurs; heart sounds feeble; the second pulmonary sound alone is somewhat louder. The area of cardiac dulness does not appear to be enlarged when the patient is in the recumbent position; in the sitting posture the area of relative dulness is increased, not only in length, but also in breadth. Effusion into both pleural cavities, rather more in the left than in the right; above the effusion the respiratory murmur is vesicular at all points; loud rhonchi also audible. Abdomen large, tense; its walls œdematous; abdominal cavity contains an abundant free exudation. The left lobe of the liver seems to be exceedingly small; it extends to the linea alba near the xiphoid process; its border is hard and sharp; the right lobe has diminished to a less extent. Owing to the presence of fluid in the left pleural cavity, the spleen cannot be accurately measured; it does not, however, extend as high up as to the arch of the ribs. Urine dark-brown; contains no albumen. Appetite good; slight constipation; almost entire sleeplessness without the aid of remedies; dizziness; mind acts slowly. *Diagnosis:* Degeneration of the heart muscle, connected with—in all probability—old pericarditis. Died the same day.

Autopsy: Body of powerful build; abdomen distended; cyanosis; well-marked

dropsy. Costal cartilages ossified; both pleural cavities contain large quantities of a clear yellow serum; the lungs correspondingly compressed, in most places dry and bloodless; the bronchi very red, and their contents purulent. *Pericardium* adherent to heart throughout its entire circumference; anteriorly and posteriorly, but not on the sides, the connective tissue binding the two together has undergone complete ossification; marked œdema of the subserous connective tissue of the heart. On the left side, between the two layers of the pericardium, there is an encapsulated mass of substance like thick pus; the walls of this cavity are covered with chalky scales, and the contents consist of a chalky emulsion containing no pus corpuscles. The muscular tissue of the heart is extremely thin, the left ventricle being to a very marked degree narrower than the right. The bundles of muscular fibres seem still to be pretty well preserved; transverse markings still distinct. A small osseous fragment, the size of a pea, rests upon the right aortic valve. The noduli Arantii are somewhat thickened; the valves, however, are apparently sufficient. Spleen, 9 × 15 ctm.; parenchyma tough, dark, and of a blackish-red hue. Liver somewhat diminished in size; nutmeg coloring; calcific, pea-sized spots on the surface of both lobes. Abdominal cavity contains a moderate amount of clear yellow serum. Abundant pigmentation of the mesentery and intestinal walls. Kidneys a little increased in size, cyanotic, tough. Mucous membrane of the stomach strongly pigmented and hyperæmic.

In regard to the behavior of the heart itself, after adhesions have taken place between the pericardial layers, some authors have maintained that, in addition to the complete obliteration of the pericardial sac, the organ very often undergoes hypertrophy and dilatation. The chief advocate of this view has been J. Hope, who states that he has never in a single instance failed to find these alterations. According to his view, the hypertrophy and dilatation are the necessary consequences of the adhesions, which, by furnishing increased resistance, impose additional labor on the heart. He also believed that the hypertrophy is further favored by the myocarditis which accompanies the whole process.

Stokes, among others, opposes this view and, without denying the possibility of such a consecutive hypertrophy, disputes both the necessity and the frequency of its occurrence. The statistics compiled by Beau and Kennedy showed that hypertrophy is a frequent accompaniment of pericardial adhesions. Gairdner's investigations led to the opposite result. Quite recently Duchek, Friedreich, and others have completely refuted the idea that obliteration of the pericardial sac necessarily entails

a consecutive hypertrophy of the heart. This, they declare, is a common though accidental occurrence, which either existed already before the formation of the pericardial adhesions, or followed upon valvular defects coetaneously developed.

It is an undoubted fact that hypertrophy and dilatation are very often wanting. The heart may be perfectly normal. This, it must be confessed, is rarely the case, and is encountered almost only in cases where the adhesions are limited in extent, or at least are not very firm. As a rule, the heart is found in a more or less well-marked condition of degeneration and atrophy. The bundles of muscular fibres show evidences of fatty degeneration, or even of hyaline and pigment degeneration; or the appearances are those of an interstitial chronic myocarditis with its results (true cardiac stenosis, tendinous degeneration of the papillary muscles, secondary endocarditis).

While in some cases these processes may, without doubt, have already begun during the acute stage of the preceding pericarditis, in others they are, as Friedreich has also remarked, the direct result of the pericardial adhesions. Without entering into the question whether the myocarditis is the immediate result of the pericarditis, or first originates after the adhesions have taken place, the results which follow the myocarditis may be different. Thus in one case there will be simple hypertrophy and dilatation, in another degeneration and atrophy—the results, in both instances, of the myocarditis. The atrophy is clearly due to the hyperplasia of the connective tissue, and to the pressure exerted by the stiff fibrous encasement into which the pericardium becomes converted. Under these circumstances dilatation of the cavities of the heart is rendered impossible by this same rigid envelope. If the heart substance has already become flaccid and flabby before the connective tissue has acquired sufficient strength and stiffness to offer such a degree of resistance, passive dilatation of the ventricles will follow, on account of the enfeebled contracting power of the heart. The dilatation may then lead to secondary muscular hypertrophy; this will continue, however, only up to the point where the cicatricial tissue begins to restrict the supply of nourishment to the heart substance, and then from that time forth degeneration sets in. As

a matter of course, in a few cases the hypertrophy may have already been present before the adhesions formed.

Adhesions may form not only between the pericardial layers, but also between the outer surface of the pericardium and the anterior thoracic wall, the pleural surface, and the organs situated in the posterior mediastinal space—the œsophagus, the aorta, and the spinal column.

Pathology.

General Picture of the Disease.

In earlier times physicians were divided in their opinions regarding the question, whether pericardial adhesions were capable of exerting a disturbing influence upon the normal functions of the heart and upon the general health. Thus, Laënnec and many others believed that obliteration of the pericardial sac was a matter of no very great importance, while Corvisart, in harmony with the views of the earlier observers—Lancisi, Morgagni, Vieussens and others—considered the condition as incompatible with long life. These differences of opinion may be explained by the fact that the adhesions themselves present great differences, and consequently give rise to very different consequences as regards the functions.

The pericardium may be adherent to the heart without giving rise to any disturbances whatsoever. In these cases the adhesions are either so limited in extent or so loose that the heart experiences no interference in its movements. At the same time the heart is neither atrophied nor degenerated, and consequently all those symptoms which usually accompany degeneration of the heart are wanting. These changes, it is true, may exist for a certain indefinite length of time with a perfect condition of health, and are only accidentally discovered, in most cases, at the autopsy, unless the observation, at some previous time, of a pericarditis, has led us to suspect the existence of adhesions.

Of those cases in which certain functional disturbances point to the existence of heart trouble, the majority are not distin-

guished by symptoms that are at all characteristic or peculiar. The symptoms which are observed in enfeebled muscular contractility of the heart and interference with the circulation are the same as those which are wont to accompany various other affections of the heart, even chronic pericarditis.

Often there is a very marked degree of cyanosis; the distended veins stand out prominently upon the neck; the pulse wave is extraordinarily low, and scarcely to be felt by the finger; the pulse, too, is often irregular, the individual waves, in particular, being of different heights; the frequency of the pulse is almost always increased. The apex-beat is lost, or only a slight tremor is to be felt, and the heart sounds are usually extremely feeble. The liver is more resistant than usual, and increased in size, or, on longer duration of the process, the organ may even present the appearance of the atrophied nutmeg liver. The spleen is not unfrequently enlarged. In consequence of the diminished aortic pressure the urine is considerably diminished in quantity, of high specific gravity, high colored, and may also contain albumen.

Usually the patient suffers from dyspnœa, particularly on slight bodily exertion or mental excitement, and not unfrequently also from continuous orthopnœa with præcordial distress, a tendency to faintness, and insomnia. The sensation of palpitation is experienced only occasionally.

If death do not occur from complications, or from a sudden arrest of the heart's action, general dropsy is developed, which may attain a very high degree. Should the heart temporarily regain its energy as a result of rest, improved nutrition, the use of digitalis, etc., the secretion of urine becomes considerably increased, and the dropsy declines for a time, but only to return anew. In consequence of the chronic œdema of the lower limbs, scrotum and abdominal wall, the skin of these parts becomes thickened, acquires a bluish-red color, and is very apt to be attacked with erysipelas. The often copious effusions into the serous cavities add to the difficulty of respiration, and by the sensation of fulness which they occasion render the administration of food more difficult. The result is invariably fatal, and may be produced by gradual exhaustion, by œdema of the lungs,

by hemorrhagic infarctions of these organs, or by terminal inflammations.

These symptoms either follow directly after the occurrence of a pericarditis, or they may develop successively without a marked beginning. The causal pericarditis may have remained latent, or it may have occurred a long time before, and have terminated in apparent recovery.

Only a part of the symptoms mentioned are produced by the adhesions ; the rest, and in fact the greater portion, are due to the concomitant changes in the cardiac muscle, so frequently attendant upon the presence of adhesions. Even when these adhesions are of such a nature as to produce but little interference with the movements of the heart, muscular degeneration may still ensue during the further progress of the disease.

Not unfrequently the functional disturbances do not make their appearance until the connective tissue of the adhesions has become more rigid, or even calcified—processes which require considerable time for their completion.

The stage of muscular degeneration may be preceded by a period of hypertrophy, during which the compensatory, though probably merely incidental gain in the working force of the heart, undoubtedly better enables the organ to overcome the resistance to its contractions, produced by the pericardial adhesions.

Cases of this kind present in their symptoms, particularly in their later stages, a great resemblance to each other,[1] although they may differ very much in duration.

When the cardiac movements are seriously impeded by close adhesions, symptoms ensue, which, by reason of their rare appearance, except under these conditions, are of the greatest value in the diagnosis of pericardial adhesions. These symptoms consist of an immovable position of the heart, systolic depressions of the intercostal spaces with a loss of the apex-beat, and the diastolic swelling of the cervical veins, noticed by Friedreich. Sometimes these symptoms may be observed to occur as an im-

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[1] Vide *S. Wilks*, Adherent Pericardium as a Cause of Cardiac Disease. Guy's Hosp. Report. XVI. 196-208.

mediate result of a pericarditis, the systolic depressions being noticed at a morning visit, whereas nothing of the kind could be detected the evening before. In such cases the derangement of the circulation, and the enfeebled action of the heart, must be produced immediately by the rapid occurrence of adhesions between the pericardial surfaces. As a rule, however, the signs referred to develop very gradually and imperceptibly, and are not detected until after complete adhesion has taken place.

The *firm adhesions*, which are the subject of present consideration, are scarcely ever broken up, and always prove fatal after a period varying with the firmness of the agglutinations and the progress of the muscular degeneration. The reported cases, in which the supposed signs of adhesions disappeared again, were presumably instances where the connections were so slight as to be ruptured when the heart recovered its energy.

The rapid disappearance of pericardial friction sounds during the course of pericarditis is by itself not to be regarded as an indication that adhesion has taken place, because the same sudden disappearance may be produced by several other causes. On the other hand, the friction may still continue to be audible for some time after the surfaces have become partially adherent.

Analysis of Individual Symptoms.

To describe more fully the subjective and objective symptoms occasioned by the derangement of the circulation, would be merely to repeat what is already obvious, viz., that they constitute the most striking part of the symptoms of pericardial adhesions. For historical reasons, however, the *sensation of palpitation* requires special mention, because several of the earlier observers, Vieussens, Lancisi, Lower, Baillie, Testa and others, have laid great stress upon this symptom. Palpitations, however, occur only in a minority of cases, and present no characteristic features. When they are present to a noticeable degree, it is usually in connection with hypertrophy of the heart.

The different views, which still prevail up to the present time, in regard to the occurrence of *hypertrophy of the heart as a result of pericardial adhesions*, and in regard to the way in which

this result is produced, have been already considered in the discussion of the anatomical conditions. The differences of opinion in regard to the relative frequency with which cardiac hypertrophy is encountered will be reconciled, if we avoid starting out with generalizing views of the significance and genesis of the lesion.

If hypertrophy be met with in some cases, and atrophy in others, it is evident, without determining the exact relative frequency of these opposite events, that the pericardial adhesion is not *necessarily followed by hypertrophy*, and that the opposite result, the atrophy, which is found in so many cases, must be produced by the presence of some other factor. If we examine the anatomical descriptions of the cases where marked atrophy existed, we find very frequently that special mention is made of the firmness of the adhesions, and of the presence of calcifications; and it is only fair to suppose, therefore, that the nature of the adhesions must have some influence upon the occurrence of this lesion. It is the pressure of the cicatricial tissue upon the nutrient vessels, and upon the muscular substance itself, which initiates the atrophy, while the passive dilatation of the cavities is prevented by the resistance of the enveloping capsule.

Whether the traction of the cicatricial tissue may not in many cases also operate excentrically, and thus bring about a dilatation of the ventricles, especially when the parts are fastened to the spinal column or the anterior chest wall, I shall not venture to decide. While we must concede that such an event is possible, it is certainly rare.

In the opinion of Hope and his followers, it is the increased resistance to the cardiac movements which calls forth a *compensatory hypertrophy*. But, unfortunately for this view, it is just in the cases where the adhesions are the firmest, and the resistance the greatest, that hypertrophy fails to occur. Furthermore, if we admit that the serious impairment in the nutrition of the muscle in these cases tends to prevent the occurrence of hypertrophy, it follows equally that the view, according to which the work of the heart is increased by the adhesions, must be erroneous, and that, instead of adding to the normal work, the lesion actually diminishes the capacity of the heart for its usual task.

Only in those cases where a passive dilatation of the ventricles has occurred early in the disease, before the connective tissue has exerted its concentric traction, can there have been any occasion for the performance of increased work, and in such instances hypertrophy may result, if the conditions of nutrition are favorable.

If it were demonstrated that a concentric hypertrophy of the heart does take place in cases of obliteration of the pericardial cavity, this fact would militate against the view expressed above. I have never, however, seen a case of this kind, nor do I think that even such a condition ought to be ascribed to a muscular hypertrophy induced by the additional work made necessary by adhesions.

Cardiac hypertrophy from this cause is brought about in just the same way as the muscular hypertrophy resulting from idiopathic myocarditis. In both cases the lesion may affect the entire organ or only one of its divisions.

In the further course of such cases, too, there prevails a similarity in many respects to hypertrophy of the heart as it occurs without pericardial adhesions. In consequence of the pressure of the tissue which constitutes the adhesion, a degenerative stage, with all the symptoms proper thereto, may occur within a comparatively short time. Under this head belong *relative valvular insufficiencies*. Cases of the sort are described by Jaccoud, Schützenberger, Marvaud, and others. We then observe valvular murmurs, and, in insufficiency of the tricuspid, marked venous pulsation, etc.

The hypertrophy of the heart, whether produced in this way or already present before the formation of the adhesions, may give rise in many cases to a plainly perceptible apex-beat in spite of the adhesions present, whilst otherwise *the apex-beat is wanting*, or only a feeble tremor is to be felt. In forty-five cases of obliteration of the pericardial sac Morgagni observed absence of the apex-beat thirty times.

Moreover, in obliterations of the pericardium absence of the apex-beat may depend solely upon feebleness of the heart's action, the adhesions not being firm enough to hinder the corresponding change in the heart's position. Such being the case, an apex-beat may at times be perceptible and at other times not. So soon, however, as the adhesions acquire a very

firm character, they may so hamper the mobility of the heart as to render the systolic sinking and erection of the organ impossible. Very commonly both causes—weakness of the heart's action by reason of an affection of the muscular tissue, and the hindrance due to adhesions—work together.

Skoda observed a systolic beat in the region of the base of the heart, and in one case a sort of pulse-beat in a partial aneurism of the heart.

If the pericardium be adherent to the anterior thoracic wall over a considerable area, the apex-beat, if there be one present, must necessarily remain unchangeable as regards its locality in all postures of the body. Inasmuch as this fixation hinders the downward movement of the diaphragm, the inspiratory bulging below the ribs may appear less on the left side than on the right. According to Williams, who first insisted upon these relations, we feel, in adhesions of this sort, the same close contact of the heart with the anterior chest-wall on both inspiration and expiration. Williams has pointed out also that in adhesions of the pericardium to the anterior wall of the chest, if at the same time the borders of the lungs be adherent, *the absolute cardiac dulness will remain unchanged on inspiration and expiration.*

Frequently, too, in this condition of things we find the absolute cardiac dulness somewhat enlarged, according to the retraction of the borders of the lungs as a result of the fixation. If, however, adhesions of the pericardium exist without accompanying adhesion to the anterior chest-wall, and without fixation of the pulmonary borders, the absolute cardiac dulness will, as a matter of course, be diminished in extent during inspiration. Hence, we should draw no further conclusion from unalterability of the absolute cardiac dulness than what this symptom strictly proves. It is only in connection with other data that these extrapericardial adhesions are of value in the diagnosis of obliteration of the pericardial sac.

The most important phenomenon in adhesions of the pericardial layers consists of *a systolic depression in the place of the deficient apex-beat.* Besides the systolic depression at the locality proper to the apex-beat, pittings may also be present in two or three intercostal spaces to the left of the sternum.

Systolic depressions in one or more intercostal spaces to the left of the sternum, *while at the same time the apex-beat is present*, are observed under various circumstances, without the pericardial layers being adherent. Diverse views have been advanced as to the causes to which these systolic depressions are to be attributed.

Under normal conditions they do not occur, because the heart, in its systole, presses with a uniform surface against the front wall of the chest, and because the vacuity thus produced is filled out by systolic distention of the lungs. If these normal conditions are abolished, from any cause, the intercostal spaces must sink in during systole. See the introduction to this volume.

According to Skoda's original idea, which most physicians have followed, the above-described systolic pittings can occur only when the pericardium is adherent at one and the same time to both the heart and the anterior chest-wall. But, in the first place, Traube has shown by a case that a single band of connective tissue between the heart and the pericardium may give rise to systolic indentation in the region of the heart's apex, and that an adhesion between the mediastinal and the costal pleura is in no wise necessary to its production. Traube afterwards observed a case in which, in spite of systolic pitting, there was no adhesion between the heart and the pericardium. Instead of this, an exceptionally tense fold was found along the posterior wall of the pericardium, which, arising near the opening for the emergence of the pulmonary artery, and extending downwards, was inserted into the left wall of the auricle, and could be traced along this nearly to the sulcus transversus. This anomalous duplicature was capable of hindering the systolic movement of the base of the heart forwards, downwards, and to the left. Since then further observations by Baler, and especially by Friedreich, have proved that systolic pitting at the site of the apex-beat may occur without any adhesion of the heart to the pericardium. Friedreich saw the most palpable systolic pitting at the site of the apex-beat in a patient with well-marked stenosis of the aorta and left lateral hypertrophy, but at the autopsy the pericardium was shown not to be adherent.

Friedreich's explanation of the occurrence of systolic pitting in the last-mentioned case was that the heart, in consequence of deficient backward impulse from insufficient filling of the aorta, could not perform the normal motion. This explanation seems to be perfectly correct for this case, and upon the strength of it

we may state in general terms that systolic pitting at the site of the apex-beat may be present in all cases in which the normal movement of the heart downwards and to the left, with elevation of the apex, is hindered, provided at the same time the lungs do not sufficiently approach each other, and the contraction of the heart is powerful enough to force the apex away from the chest-wall. Under such circumstances the pitting follows, as a matter of course, from atmospheric pressure.

Plainly, the causes which have just been mentioned as hindering the motion of the heart are found most frequently, but by no means exclusively, in cases of pericardial adhesions. Hence the resulting systolic pitting cannot be taken as absolute proof of the existence of pericardial adhesions.

The systolic movement of the lungs is doubtless capable of hindering the sinking-in of the intercostal spaces; but it seems to me doubtful if the borders of the lungs need be fixed in order for the occurrence of depression by atmospheric pressure. I am much more certain that the systolic movement of the lungs may be hindered by other causes.

Furthermore, I am unable to decide as to how far the resisting capacity of the intercostal muscles is to be taken into account. We may imagine that the resistances to the systolic movement of the lungs are in no wise absolute. Such being the case, may it be that systolic pitting occurs in weak, emaciated persons, and not in those possessed of muscular strength?

Apart from the influence of atmospheric pressure, if the heart be perfectly fixed and its contraction powerful, the systolic contraction of the cardiac muscle may give rise to pitting of the intercostal spaces. If, indeed, the pericardium be adherent both to the anterior chest-wall and to the vertebræ, the fixation of the heart will be so complete that its contraction must, almost of necessity, cause sinking of the intercostal spaces. It is precisely under such circumstances that systolic pitting is most commonly found, not only at the site of the apex-beat, but more widely diffused, combined at the same time with systolic retraction of the lower costal cartilages and of the lower part of the sternum.

Thus far there is no case known in which such forcible retraction has occurred in any other way than as the result of pericardial adhesions. Hence its presence must be held as con-

clusive in diagnosis. On the other hand, it is not absolutely necessary to the production of this forcible and diffused retraction that the pericardium should be fixed to both the anterior chest-wall and the vertebral column. Thus, Friedreich has observed marked systolic retraction of the cardiac region, the ribs, and the sternum without the existence of any such external adhesions of the pericardium. Friedreich assumes, on the contrary, that it is essential to the production of this pitting that the lower surface of the heart should be firmly adherent to the diaphragm, so that the heart in contracting must draw the diaphragm upwards and its point of attachment to the chest-wall inwards. In this wise, too, according to Friedreich, may we explain the fact that this pitting is more marked and more noticeable at the height of the inspiration, whilst the descent of the diaphragm operates in opposition to the heart, so that the latter, in order that its apex may execute an equally great movement upwards, must draw the chest-wall still more forcibly inwards, since at this moment the connecting band is shortened.

There are many considerations which make against this explanation of Friedreich's.[1] It may be avoided, too, without doing violence to the facts, if we remember that the natural attachments of the pericardium are of themselves sufficient to occasion pitting of the cardiac region, provided the base of the heart be fixed immovably in the pericardium. Moreover, we must take into account the action of atmospheric pressure. We may see often enough, indeed, how in patients with emphysema the lower portion of the chest-wall is pressed in on inspiration.

If the heart is to draw the intercostal spaces inwards by its diminution in size, its contractions must take place with a certain amount of force. It is therefore on account of impaired

[1] I will simply mention here that it is not yet by any means certain that the apex of the heart makes such an extensive upward movement as to stretch that portion of the diaphragm in question, out of proportion to its mobility and capacity for yielding, and thus depress its point of insertion. Furthermore, I do not believe that the fixed portion of the diaphragm can contract in such manner that the inspiratory intensification of the phenomenon can be explained by the force of the same. In point of fact, it must be referred to atmospheric pressure.

cardiac force that in many cases we see this marked retraction disappear after a time. In like manner may we explain Skoda's failure to find pitting in cases of simultaneous fixation of the pericardium to the vertebral column, the anterior chest-wall and the diaphragm. If the heart's power be sufficient, pitting may occur in spite of these manifold adhesions.

According to von Dusch, the pitting does not exactly correspond with the beginning of the systole in point of time, but is synchronous with the radial pulse, and is thus noted somewhat later than the carotid pulse.

Together with systolic pitting, a *diastolic concussion from the apex* may occur, if the apex during diastole sinks down again upon the chest-wall. This phenomenon is designated as diastolic heart-beat. In those cases in which the lower portion of the chest-wall is forcibly incurvated, the latter flies back into its original position with a jerk or shock. It is by means of its own elasticity that the chest-wall suddenly resumes its proper contour as soon as the overpowering force ceases, with the beginning of the diastole, to act. The diffuse impulse thus produced may be very plainly felt by the applied hand. According to Friedreich, the rebound of the chest-wall produces a dull sound, which may be heard on auscultation after the second sound of the heart. In this way the second sound seems split or doubled. Just this sort of doubling of the diastolic sound is frequently observed in obliteration of the pericardium. This phenomenon is therefore partly explained by vibrations of the thorax; but, if none such are present, and yet a division of the diastolic sound is heard, it is due probably to a lack of synchronism in the closure of the pulmonic and aortic valves (Skoda), which indeed is not rare under other circumstances.

Friedreich has described also peculiar phenomena pertaining to the *cervical veins*, which likewise are connected with the systolic retractions and are never present in a striking degree without them. These include, according to Friedreich, a sudden *diastolic collapse*, or subsidence *of the cervical veins* (*often tensely filled during systole*), so that they disappear from view. Even the supra-clavicular fossæ may become deepened at the same time. This subsidence takes place in entire synchronism with the dias-

tolic beat against the chest-wall, and alternates with the carotid pulse.

In a case contributed by von Dusch, decided dicrotism was perceptible together with this phenomenon, whilst the first sudden collapse was followed by a second and more complete one.

Friedreich has explained the phenomenon observed by him, and which cannot very well be confounded with a venous pulse, by the hindrance offered to the return of blood through the cervical veins by the diminution of the thoracic space during systole, whilst the sudden diastolic enlargement of the same must have an aspiratory effect. It is probable, too, that under the given conditions the diastole must take place more forcibly and quickly on account of the traction of the adhesions from without, and the descent of the raised diaphragm. Friedreich also assumes, that, in consequence of the diastolic descent of the heart, especially as caused by the action of the diaphragm, the large vascular trunks, including the superior vena cava, undergo an elongation, by means of which the downward current of blood from the cervical veins is likewise hastened. The dicrotism observed by von Dusch is probably to be referred to the diastole of the auricle and ventricle.

Diagnosis.

In the majority of cases of pericardial adhesions, great difficulties stand in the way of a sure diagnosis; in fact, it is quite often impossible to make the diagnosis. This is true, not only of those cases in which there is an entire absence of symptoms, but also in those in which it is impossible to decide whether the symptoms are due to a degeneration of the heart muscle alone, or to this condition in conjunction with pericardial adhesions. If the area of cardiac dulness is enlarged, if the borders of the lungs are bound down by adhesions, the case may be one of chronic pericarditis. In the latter case, variations are observed at times in the area of cardiac dulness, corresponding with the variations in the amount of the exudation.

In distinguishing pericardial adhesions from a primary degeneration of the heart muscle, material assistance may be obtained

by looking carefully into the previous history of the case, for the purpose of ascertaining whether the patient has or has not had a pericarditis. I do not agree with S. Wilks in the view that in individuals of mature age, where severe heart symptoms are present, without valvular murmurs, we may infer the existence of degeneration of the heart muscle, but that in youthful individuals the same state of things points rather to pericardial adhesions; my own observations show that also in youthful individuals degeneration of the heart muscle is more common than pericardial adhesions.

Among the objective symptoms, we can place no safe reliance upon the *size and shape of the area of cardiac dulness*, for in obliterations of the pericardial sac this area may be normal, or it may be either larger or smaller than normal. On the other hand, we may consider it a valuable sign if the area of cardiac dulness remain unchanged in size and shape. Absence of the apex-beat is not of very great value in differential diagnosis. I cannot admit, however, that under certain circumstances, the movements of the heart, which are felt by the hand when placed upon the chest, present, in pericardial adhesions, certain characteristics which are not observed in every case of hypertrophy of the heart—provided, of course, that there are no systolic retractions.

The character of the pulse also presents no distinctive features to aid us in the diagnosis.

We are, therefore, often compelled to refer the disturbance in the circulation to some disease of the heart without valvular defects, and to leave undetermined the question of the existence or non-existence of pericardial adhesions or disease of the heart muscle.

If a valvular defect is associated with pericardial adhesions, the difficulty of making the diagnosis is greatly increased, for the thought of an uncompensated valvular defect is very apt to divert the mind altogether from the idea of pericardial adhesions.

A tolerably sure diagnosis can only be made in those quite rare cases in which well-marked systolic retractions and Friedreich's sign are present. On the other hand, the rarity of these

cases is still further increased by the fact, that the progressive degeneration of the heart muscle soon causes these manifestations to disappear.

Course and Prognosis.

It has already been stated, that in a certain number of cases pericardial adhesions do not constitute a very serious affection, for the reason that they do not interfere in any way with the normal functions or with the continuance of life. In those cases in which functional disturbances manifest themselves, the course of the disease is not a rapid one, but extends over a period of months, or even years. The fatal termination occurs earlier or later according to the progress of the changes in the heart muscle and the nature of the adhesions. The result also depends in a measure upon individual circumstances and upon certain complications that may arise.

If the symptoms of failing heart-power are pronounced, it must not be forgotten that, under favorable circumstances, the disturbances in the circulation may still be rectified, and the existing dropsy absorbed; as a rule, however, this improvement is not of long duration.

The most unfavorable cases are those in which the disease of the heart muscle makes rapid advances, or those in which progressive atrophy of the heart renders the organ less and less capable of maintaining the circulation. On the other hand, if the heart muscle remains in a sound condition, the existing obstacles will, in the majority of instances, be overcome by compensation. This is especially true of those cases in which hypertrophy of the heart exists at the same time.

So far, therefore, as the prognosis is concerned, a strong action of the heart, and a well-marked systolic retraction of the chest-wall (provided at the same time well-marked indications of stasis are absent), are relatively favorable indications, at least for the near future. If there are reasons for believing that the heart muscle is undergoing progressive degeneration, we may then predict the probable occurrence of death within a few weeks or months.

Under all circumstances we should consider every case of pericardial adhesions which is capable of calling into existence any symptoms whatever, as constituting a serious disease of the heart, which can, in a direct manner, lead to death at the end of a shorter or longer period of time.

Treatment.

Treatment can exert no influence upon the adhesions themselves. The attempt to loosen them by exciting violent action of the heart, would certainly be in vain when they were composed of firm connective tissues, and, on account of the accompanying danger, would also be impracticable when they were recent.

The degeneration of the muscular tissue must be considered as the most important element. Consequently the treatment must be mainly directed with a view to this. The presence of adhesions does not change the principles which underlie the treatment of simple myodegeneration. The results of strengthening treatment addressed to the heart, together with rest and suitable nourishment, are very significant in many such cases. Especially does the proper and carefully watched use of digitalis correct for a time the disturbance of the circulation.

When dropsy coexists, the use of digitalis may be associated with diuretic remedies and diaphoretic measures. Puncture may be necessary to remove large serous effusions from the abdominal or pleural cavities.

Subjective troubles, loss of sleep, require narcotics. Most active stimulation must be employed if symptoms of collapse appear.

Indurated Mediastino-pericarditis and Paradoxical Pulse.

At the Tübingen clinic in 1854, Griesinger[1] observed a case in which the autopsy showed fibrinous mediastinitis together with fibrino-purulent pericarditis. Stiff stringy exudations were found in the connective tissue in which the large ves-

[1] Beitrag zur Diagnose der Mediastinitis, by *A. Widenmann*, Diss. inaug. Tübingen. 1856.

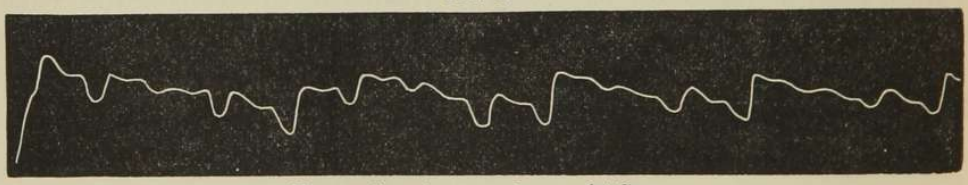
sels are imbedded after their exit from the pericardium. Some of the venous trunks were notably constricted, as was also the aorta in its ascending portion and arch; it was also indented and partly twisted about its axis. The pericardial cavity was almost obliterated by fibrinous membranes. At one point only was there found a circumscribed, encapsulated collection of pus. The anterior surface of the thickened pericardium was also covered with membranes. The heart was pale, shrunken, and flabby. The patient was a young man who had previously been healthy; the affection was probably of traumatic origin and caused death in about nine weeks. It began with stitch in the side, dyspnœa, bloody sputa, followed by cyanosis and general dropsy. Examination of the heart showed only a slight enlargement of the area of dulness, weak apex-beat, and muffled sounds.

A small irregular pulse was noticed in this case during life, while the heart beat regularly. Griesinger noticed also that the intermission of the pulse occurred at regular intervals and simultaneously with inspiration.[1]

This observation of Griesinger's entirely escaped general notice until Kussmaul brought it to light in 1873.

Kussmaul observed two similar cases in which this affection was suspected, principally on account of the character of the pulse, the *pulsus inspiratione intermittens*, or *pulsus paradoxus*. Kussmaul selected the name *indurated mediastino-pericarditis* for this affection which Gendrin has described less distinctively as fibropericarditis. (See page 569.)

FIG. 6.



Kussmaul's pulsus paradoxus. 1st Case.

Kussmaul's first case[2] was that of a youth twenty-one years old, previously healthy, who fell ill the latter part of October, 1872, with indefinite symptoms and without known cause. A sense of constriction and pain in the side then appeared and the patient took to his bed. On the 18th of December the physician found pleurisy on the left side, with fever, sweats, loss of appetite, cough with watery mucous sputa, which on one occasion contained streaks of blood. In January, 1873, the pleural exudation decreased steadily, but the dyspnœa continued, the pulse remained small, frequent and irregular, and dropsy appeared.

While the case was under observation in Kussmaul's clinic the temperature at the beginning (from the 1st to the 26th April) was always below the normal, the pulse

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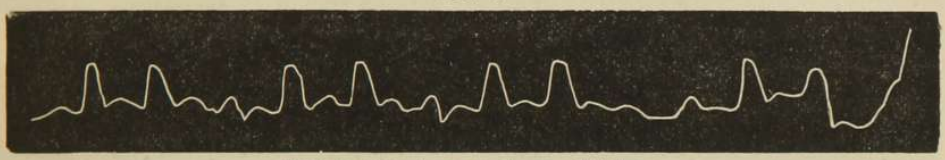
[1] *Vierordt*, Die Lehre vom Arterienpuls. Braunschweig. 1855. Taf. II. der Curv. N. XXV.

[2] Ueber schwielige Mediastino-Pericarditis und den paradoxen Puls. Berl. Klin. Wochenschr. 1873. No. 37 et seq.

never less than 100, usually from 100 to 120, small and intermitting; it disappeared with inspiration and returned with expiration. After every second beat and perhaps an incomplete third, it intermitted. The respiration was quickened, the apex-beat could not be felt. The area of pericardial dulness extended upward to the third intercostal space, passed one and a half centimetres beyond the right border of the sternum, and extended on the left nearly to the mammary line. The heart sounds were uncomplicated, muffled, and weak; their rhythm was regular, notwithstanding the intermissions of the pulse in the arteries. The boundaries of the heart altered considerably with deep inspiration, especially on the left. The veins in the neck, especially the right internal jugular, were greatly distended. The liver was swollen, the appetite small, urine scanty and reddish-yellow, with traces of bile and albumen.

Fig. 7.



Kussmaul's pulsus paradoxus. 1st Case.

Later, in the course of the disease, the temperature often rose above the normal. On the 20th of May paracentesis of the abdomen was performed and six litres of liquid removed. By the first of June the dropsy had again become very great. The jugular veins were considerably swollen, that on the right side undulated. *Their distention was slightly increased with each inspiration.* Puncture was repeated on the 10th of June, and on the 17th the skin of the right leg was incised to relieve the œdema. Phlegmonous inflammation set in about the incision and caused death on the 22d of June. The disease had lasted about eight months.

At the autopsy the right lung was found attached about its root by a number of tough fibres to the pericardium and the mediastinum above it. On the left the tongue-shaped lobe was attached by stout adhesions to the pericardium and diaphragm; the upper lobe also was closely adherent to the posterior surface of the pericardium up to the point where the large vessels are given off. In front, the pericardium was free over the region corresponding to the conus arteriosus and the limits of the left ventricle. All the rest of it was attached by strong connective tissue to the diaphragm, lungs, and anterior wall of the chest. Above the pericardium the two layers of the mediastinum formed a normally constituted membrane. The fibrous layer of the pericardium was thickened and firmly attached to the visceral layer by a recent tough fibrous membrane.

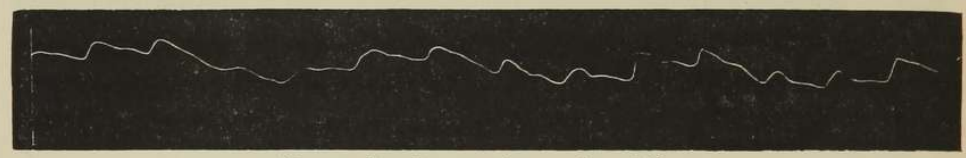
At the point of origin of the large vessels the fibrous pericardium increased in thickness. Tough bands of connective tissue, enclosing the remains of crumbling masses of fibrin, extended from the base of the heart into the cavum mediastini and accompanied the large vessels upward. These indurated bands formed loops about the arch of the aorta and the ascending portion. The arch of the aorta was

thereby drawn downwards and somewhat bent at the origin of the innominata; the ascending portion of the aorta was compressed from before backwards, so that it would scarcely admit the little finger. The trunk of the arteria pulmonalis also was compressed.

Kussmaul's second case was a woman thirty-two years old, who had suffered for years with chronic bronchitis and chronic pneumonia. The time when the mediastino-pericarditis began was not determined. When brought to Kussmaul's clinic, in the latter part of June, 1873, she presented general dropsy, cyanosis, dyspnœa, with orthopnœa at times. The urine contained no albumen and was diminished in quantity.

While the heart's action continued regularly, the pulse became smaller or imperceptible with every inspiration, and returned to its original fulness during expiration. The wave was small, the tension slight, the frequency from 104 to 140. The inspiratory intermission occurred in all the arteries that were accessible. There was

FIG. 8.



Kussmaul's pulsus paradoxus. Second Case.

also a slight increase in the area of precordial dulness, absence of apex-beat, weak, muffled heart sounds, consolidation of the left lung. Respiration was frequent, dyspnoic, the supra-clavicular fossæ distended by the bulbs of the internal jugulars, the external jugulars also distended.

The patient's temperature was usually sub-normal in the morning, normal or somewhat raised in the evening. The dyspnœa and dropsy steadily increased, and death took place on the 20th of July.

At the autopsy the pericardium was found immovably attached by strong adhesions; only a small part of the right ventricle on the left and in front was free. The thick adhesions extended to both lungs, the anterior wall of the thorax, the diaphragm, and the lower dorsal vertebræ. The pericardium, and principally its fibrous layer, was so thickened that it could not be thrown into folds. The visceral layer also was thickened and condensed. The serous surfaces were covered with crumbling false membranes, composed of fibrin and connective tissue, and were glued together.

From the point of reflection of the pericardium at the base of the heart, thick, indurated cords extended into the cava mediastini anticum and posticum, and accompanied the large vessels, constricting and shortening them by traction. Tough cords stretched especially from the base of the left ventricle forwards over the pulmonary artery, back to the arch of the aorta, which they indented slightly in several places and drew directly downwards towards the compressed arteria pulmonalis and the left ventricle. The valves of the heart presented no important lesions; the muscular tissue was pale and soft.

In a third case which Kussmaul observed during life the symptoms were identical. The pulse, frequent and small, intermitted at every deep inspiration. Both internal jugulars and the superficial veins of the neck were enormously distended, but neither undulated nor pulsated. *At every forced inspiration the bulbs of the jugulars became greatly swollen, and subsided again during expiration.*

F. Kipp[1] recorded a case in which the diagnosis of mediastino-pericarditis was based upon a paradoxical pulse. The case differed, however, essentially from the preceding one in this, that the formation of the indurated cords had not taken place upwards along the great vessels. Cases of the kind are less rare.

The same was seen in the garrison hospital at Berlin. The patient was a young man previously healthy, and it was probable that a pleurisy several years before had been the beginning and foundation of the subsequent chronic mediastino-pericarditis. While he was under observation, from the 3d of October until his death on the 16th of November, the important symptoms—cyanosis, dyspnœa, and general dropsy—were present. The area of precordial dulness was increased, and extended upwards to the third rib; the heart sounds were uncomplicated and moderately loud; the apex-beat could not be felt. The area of dulness did not change during deep inspiration. The jugulars were distended, and there was an indication of a venous pulse. *The pulse intermitted completely at every deep inspiration.* The urine contained no albumen; its quantity was diminished.

At the autopsy the sternum was found more firmly fastened down than usual by tense connective tissue of new formation. After its removal a membrane became visible which corresponded in shape to the area of dulness of the heart. Encapsulated, cheesy masses were found under the left costal cartilages. The boundary between the lungs and mediastinum could not be made out. The diaphragm was adherent to the pericardium to an unusual extent, and greatly hypertrophied. The indurations, enclosing cheesy masses, extended to the left over the lower lobe of the lung as far as the axillary line; the right lung was tightly adherent only at its lower part to the pericardium. The pericardial cavity was entirely obliterated; the entire ventricular portion of the heart was imbedded in a stiff callosity. The heart was considerably dilated and hypertrophied; the valves normal. *The formation of the callosities had not extended far above the ventricles.* The vessels leading to and from the heart were dilated. Their inner surface was polished everywhere. Connective-tissue bands, capable of drawing the vessels forward and causing indentations in them, were not found. The muscular tissue showed commencing fatty degeneration.

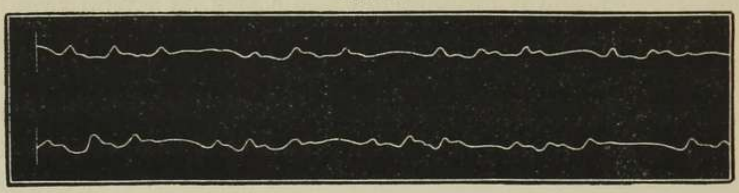
The symptoms, as they appear in the above described cases, agree essentially with those of chronic pericarditis or obliteration of the cavity of the pericardium. The intermitting pulse was peculiar to them. Since Kussmaul's publication it has been shown that this phenomenon is not the exclusive criterion of mediastinitis fibrosa. With reference to diagnosis, the symptom observed in the veins of the neck by

[1] Ein Fall von schwieliger Mediastino-pericarditis. Dissert. inaug. præs. v. Ziemssen. München. 1875.

Kussmaul is a more exact indication of the presence of an indurated mediastinitis. *The traction of the indurated bands during forced inspiration produces a visible, or even a considerable distention of the veins of the neck instead of the normal reduction.*

Kussmaul considered the occurrence of the paradoxical pulse in these cases dependent upon the fact, that the indurated cords encircled the aorta and drew upon it. According to his observations it is not necessary that the trunks of the vessels should be immediately adherent to the sternum (Griesinger). It is sufficient if the callosities extend from the pericardium to the large vessels, for the former is drawn forwards during inspiration, and thus traction is exerted.

FIG. 9.

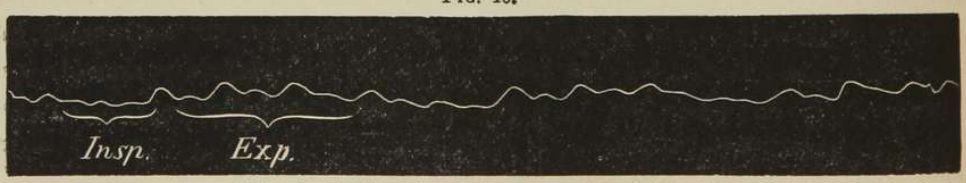


Pulsus inspiratione intermittens, according to Baeumler.

There are observations by Traube[1] and almost simultaneously by Baeumler,[2] from which it appears that the cause of the paradoxical pulse ought not to be sought exclusively in the formation of indurated bands in the mediastinum. Both observers saw the phenomenon in question occur in pericarditis exsudativa without mediastinitis. These cases differ from Kussmaul's in this, that the symptoms in the veins of the neck were absent, and the heart sounds became weaker during inspiration.

Traube thought he found a substantial explanation in an accompanying notable thickening of the pericardium, because the tension of the stiff pericardium produced by the diaphragm during inspiration would interfere with the contraction of the ventricles, especially if the heart were weak.

FIG. 10.



Pulsus inspiratione intermittens, according to Traube.

The explanation given by Traube will not do for all cases, at any rate. The stiffness of the pericardium upon which it depends was not present in Baeumler's case, and the thickening was not considerable in the following case from von Ziemssen's clinic:

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[1] Berl. Klin. Wochenschr. 1874. No. 21. – Charité-Annalen. 1876. S. 270.

[2] Deutsch. Archiv für klin. Med. Bd. XIV.

Anna E., sixty years old, could remember no previous diseases; a fortnight before admission she was taken with difficulty in breathing, tormenting cough, and pain in the left side.

Frequent, dyspnoic respiration; on making slight exertion the number of respirations doubles; frequent orthopnœa. The internal jugulars are dilated on both sides and pulsating. The veins of the neck do not become greatly distended during inspiration. The pulse very rapid, its volume small, not tense, perfectly regular during expiration and the following pause; as inspiration begins the pulse sinks to a minimum, and if the inspiration is deep it becomes imperceptible. The apex-beat can be felt distinctly nowhere, but when the breath is held a diffused jarring can be perceived. The area of precordial dulness is considerably enlarged; it reaches, when the patient is lying down, to the attachment of the second rib, and when she is sitting up, somewhat higher, and is lost on both sides in the dulness due to the bilateral pleural effusion. The heart sounds are uncomplicated, muffled, and weak, are not noticeably changed during inspiration; the second pulmonary sound is somewhat louder.

Furthermore, there is consolidation at the apices of the lungs, especially on the left side; the resistance of the liver is increased, the anterior border of the spleen is two centimetres from the curve of the ribs, the urine is very scanty, with brick-dust sediment, and without albumen. General dropsy, considerable cyanosis, temperature at times slightly raised. During the whole period of observation, from the 15th of May to the 1st of July, the pulse remained paradoxical.

At the autopsy a considerable effusion of hæmorrhagic liquid was found in both pleural cavities, the lower portions of the lungs were without adhesions, were greatly compressed on both sides, especially the left, and there were several hæmorrhagic infarcti in the right lower lobe. There was a large amount of hæmorrhagic liquid in the cavity of the pericardium, both serous surfaces were covered with fibrinous layers, especially where reflected at the origin of the large vessels. There were also partial adhesions of the heart to the pericardium at the lower portions, firm at the point, loose on the sides. The pericardium was thickened, exclusive of the fibrinous coating. The cavity of the right ventricle small, the muscle thin, the layer of fat thicker than the muscle. The left ventricle likewise diminished, its muscle thin, pale, and brittle; on examination with the microscope it showed marked pigment-degeneration. All the valves normal. No inflammation on the outer surface of the pericardium, nor in the mediastinum.

To these cases of pericarditis exsudativa may properly be added an observation by W. Graeffner.[1] In a patient with purulent pericarditis and double pneumonia the pulsation in all the arteries that could be felt became less during inspiration. If the thorax was maintained for some time in the position of inspiration the pulse remained uniformly small, and the converse took place when the expiration was prolonged. Weakening of the heart sounds and swelling of the veins of the neck were not perceptible during inspiration.

[1] Berl. klin. Wochenschr. 1876. No. 27. S. 386.

Fig. 11.

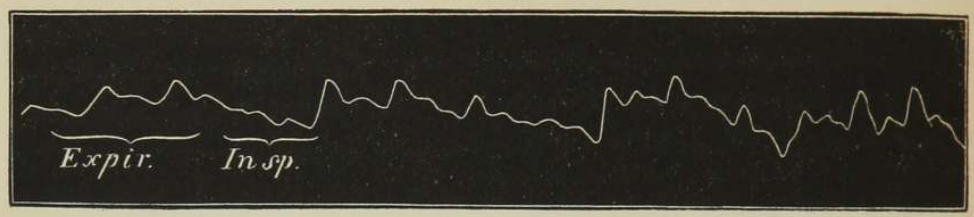
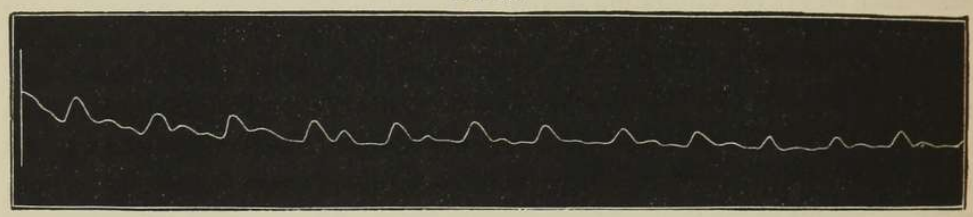


Fig. 12.



After the removal of the sternum at the autopsy the pericardium was found entirely free. The edges of the lungs touched each other nowhere, and were adherent to the pericardial pleura on both sides at the points corresponding to the ends of the costal cartilages. The pericardial sac contained about three hundred grammes of purulent exudation. Both layers of the pericardium were covered with a thick tufted coating, as were also the aorta and pulmonary artery, the former only to the point where it turned. The aorta was also bound down by circular adhesions which, short and tense, ran over it from the pericardium.

Graeffner considers the paradoxical pulse in this case the result of the firm attachment of the pericardium to the pleura on each side, by which the inspiratory traction upon the pericardium was transmitted to the bands encircling the aorta and exerting their tension from the point where it turned to the inner surface of the parietal layer. The consequence was indentation of the vessel and diminution of its lumen.

I add further that one of our colleagues shows this peculiarity of the pulse exquisitely. His radial pulse, which is perfectly normal, disappears completely at every deep inspiration, and remains imperceptible as long as the thorax is kept in the position of inspiration; in the carotid and femoral the wave becomes much smaller, the heart sounds continue uniformly. The individual in question enjoys very good health and is very well nourished. In his childhood he had pleurisy upon the left side. I may also mention that in a case of large effusion into the left pleura, observed at von Ziemssen's clinic also for some time, while the exudation filled the cavity entirely, and caused considerable displacements, the radial pulse disappeared almost entirely during inspiration.

In all these cases the phenomenon of the pulsus inspiratione intermittens must be connected with a factor due to the alteration of the thorax during inspiration. I cannot assent to the idea expressed by Baeumler. He believes that the *pressure which is rendered positive* by the exudation within the thorax hinders the aspiration of blood during the act of expiration to such an extent that when the systole of the

left ventricle occurs simultaneously with the act of inspiration, there is but a small amount of blood to be expelled. This condition cannot cause the paradoxical pulse, because the latter beats regularly during prolonged expiration, and always stops regularly during a pause in the position of inspiration, even if the pause is kept up as long as possible. In my opinion the inspiratory displacement of the thorax may produce, under different conditions, mechanical obstructions which result in the expulsion of a less amount of blood; these may occur in the aorta, perhaps also in the subclavian, or at the origin of the vessels, and then the heart sounds are unchanged; or they may be in the heart itself, and then we find at the same time a diminution of its action. The production is favored by a weak, degenerated heart. Besides these mechanical hindrances in the circulatory system, caused by the act of inspiration, the phenomenon of the pulsus inspiratione intermittens may also be produced *by obstruction to the entrance of air* into the lungs, for the inspiratory augmentation of the negative pressure (suction) within the thorax opposes the supply of a proper amount to the aortic system (see Baeumler, l. c.). In this case there is an exaggeration of a normal relation, for even under normal conditions the sphygmograph shows that the negative pressure within the thorax during inspiration can cause a slight diminution of the wave, as Riegel and Sommerbrodt have shown.[1]

Pneumopericardium.

Voigtel, Hanab. der path. Anat. 2 Bd. Halle. 1804.—*Bricheteau*, Obs. d'hydropneumopéricarde. Arch. gén. de Med. Tom. IV. 1844. p. 334.—*Horst*, Hufeland's Journ. Jan. 1844.— *W. Stokes*, Dis. of the heart and the aorta. Dublin. 1854. p. 21.—*Chambers*, Lond. Journ. July, 1852.—*Feine*, Diss. pericardii læsi cas. rar. sist. conatum cum simil. etc. Lips. 1854.—*Sorauer*, De Hydropneumopericardio diss. Berol. 1858.—*O. Wyss*, De fistula pericard. comment. Vratisl. 1866.—*Demarquay*, Essai de Pneumatologie médicale. Paris. 1866. p. 363.—*Tütel*, Fall von Pneumopericardium. Deutsch. klinik 37. 1860.—*J. Beckers*, De Pneumopericard. Diss. Greifswald. 1860.—*Bodenheimer*, Ein Fall von Pyopneumopericard aus der klinik des Prof. Dr. Munk in Bern. Berl. klin. Wochenschr. 1865. 35.—*Morel-Lavallée*, Rupt. du péricarde; bruit de roue hydraulique, bruit de moulin. Gaz. Méd. de Paris. 1864. No. 46.—*C. Eisenlohr*, Ein Fall von Pyopneumoperic. Berl. klin. Wochenschr. No. 40. 1873.—*Saexinger*, Pneumopericard. bedingt durch Perforation eines runden Majengeschw. Prag. med. Wochenschr. 1865.—*Fetzer*, Ein Fall von Pneumopericard. Wurtemb. Medic. Corresp.-Bl. 1874. No. 40. See also handbooks and textbooks on diseases of the heart.

The presence of air in the pericardial sac is an exceedingly

[1] Berl. klin Wochenschr. 1876. No. 26. S. 369; and Ein neuer Sphygmogr. Bresl. 1876.

rare occurrence. The older physicians considered pneumopericardium, except the cases due to a traumatism, as an idiopathic affection, or they explained the accumulation of gas by the supposition of spontaneous decomposition of a pericardial effusion.

The possibility that pneumopericardium could arise by the development of gas in a decomposing ichorous exudation was maintained by the most trustworthy observers—Laënnec, Skoda, Stokes, Rokitansky, Traube and others. Duchek and Friedreich also defended the opinion that most cases of pneumopericardium were due to the development of gas in ichorous exudations, and in the two cases recorded and observed by them, no other cause could be found, although the search was thorough.

To express a doubt of the demonstrative value of such conscientious observations, in which another explanation scarcely seems allowable, is always somewhat questionable. But, notwithstanding its improbability, in the face of the results of the anatomical investigation, the possibility must be borne in mind that there was a connection through which the air entered from outside—from the pleural cavities into the pericardium. The reasons why such observations, apparently so convincing, must be doubted, are of course mainly theoretical, but they are substantial enough to make the production of pneumopericardium by the development of gas in a decomposing exudation appear extremely improbable.

The analysis of the gas collected in the pericardium, in those cases in which the anatomical examination makes it seem probable that it has been developed there, will determine whether this view is correct or not.

Huefner has recently examined gas taken from a pyæmic abscess in the thorax, with the following result:

Carbonic acid gas and sulphuretted hydrogen	1.05 per cent.
Oxygen	14.50 "
Nitrogen	84.45 "

This result agrees with the values found by Dressler in his analyses of gases of the same kind taken from an encapsulated peritoneal exudation, an ovarian cyst, and a cyst of the thyroid gland, to this extent that the quantity of the indifferent nitrogen was almost the same. Dressler found more carbonic acid gas and less oxygen in the gases he examined.

Huefner concludes from these results, in opposition to the opinion held by Dressler, that the presence of gas was due to the entrance of atmospheric air with subsequent oxidation. Future examination of the gas will determine whether its presence in a serous cavity can result from the decomposition of an exudation or not.

In cases in which the symptoms of a pneumopericardium appear during life, and which end in recovery, it is not justifiable to suppose there has been a development of gas in an exudation, difficult as any other explanation may be. The cases of purely spontaneous development of gas without the presence of a decomposing exudation must be considered as mistakes.

Perforation of the pericardium occurs by suppurative destruction from within outwards, when there is a purulent exudation within the pericardium, and from without inwards likewise by advancing ulceration and destruction, also by neoplasms extending from the mediastinum, from the œsophagus, or, after destruction of the diaphragm, from the abdominal viscera to the pericardium, and finally by aneurisms. If the opening establishes communication between the pericardial sac and a cavity containing air, or if it opens externally, air can enter into the pericardium. The entrance of air may be aided by positive pressure, such as is exerted upon it in the stomach, the lungs, or the pleural cavity when pneumothorax is present; it is also helped by the elastic traction of the lungs upon the pericardium, and the diminution of the heart during the systole.

In Chambers', Becker's, and Tuetel's cases ulcerative perforation took place from the œsophagus; McDowel saw a case of perforation from a cavity, Eisenlohr one from a pyopneumothorax, and Saexinger one from a gastric ulcer into the pericardium. In a case given by Graves an abscess of the liver communicated at the same time with the stomach and pericardial sac.

The pericardial sac can also be opened by injuries of all kinds, so that air can penetrate into it. According to von Bamberger's testimony the entrance of air does not necessarily take place in every case of penetrating wound of the pericardium.

Feine and Bodenheimer have recorded cases due to penetrating wounds. Thompson and Walshe saw pneumopericardium follow opening of the pericardial sac from the œsophagus by a knife that had been swallowed; Morel-Lavallée saw

it caused by penetration of a fractured rib into the lung and pericardium. Morel-Lavallée and Steiger saw air enter the pericardial sac after contusions and crushing.

The relations are, without exception, such that inflammation of the pericardium is set up at the same time. The character of the exudation for apparent reasons is purulent, hæmorrhagic, or ichorous (pyopneumopericardium). The pericardium may be more or less distended and stretched by the accumulated gas, and consequently the air escapes with a hissing sound when the sac is opened. The gas, as a result of its specific gravity, always occupies the upper portion. Retraction of the lungs and depression of the diaphragm occur to an extent corresponding to the distention of the pericardium. In rare cases pneumatosis of the pericardium occurs as a cadaveric incident, in which case the serous membrane presents a parchment-like dryness, the result of evaporation (Foerster).

The subjective symptoms in pneumopericarditis are not characteristic ; on the other hand, the objective signs are so prominent and striking that they can hardly be misinterpreted.

The patients complain usually of severe dyspnœa; cyanosis also is present ; and if the condition, as is often the case, coincides with severe symptoms of collapse, the skin is pale with a bluish tint. The pulse is small, also irregular ; delirium and fainting fits occur ; sometimes the patients complain of nothing, and are only very weak and indifferent. Usually also there is sleeplessness, caused partly perhaps by the loudness of the heart murmurs. High fever or chills are sometimes observed, which, however, like the profuse sweats, diarrhœa, etc., must be attributed to the original lesion and not directly to the pneumopericarditis. Dysphagia was observed by Eisenlohr.

The chest-wall may be noticeably prominent in the region of the heart when it is soft and the pericardium is greatly distended by air. The apex-beat varies, it may be weakened or absent; sometimes pulsation can be felt in several intercostal spaces. The crackling of air bubbles, synchronous with the movements of the heart, can be perceived by the hand laid upon the breast. In Graves's case a loud metallic ring was heard with each beat of the heart. The faintness of the apex-beat disappears as soon as the patient sits up and bends forward.

The signs obtained by percussion are very characteristic. The note over more or less of the region of the heart is purely tympanitic with metallic resonance, the height of which corresponds to the smallness of the cavity containing air. Gerhardt noticed in one case that when rapid and continuous percussion was made at one point the metallic resonance became higher and lower, following the rhythm of the heart, as the result of the changes in the shape of the body of air contained in the pericardium. According to Feine's observation the note in the region of the heart became duller with each systole, because during it the heart is situated further forward and downward against the thorax and presses back the air, and during the diastole the note again became clearer. The cracked-pot sound has been repeatedly noticed; whether this can occur in a closed pericardium without an open communication, as is claimed, appears doubtful.

The air in the pericardial sac is always found in the highest portion, the heart and the exudations that may be present always sink downwards. When the patient is seated, therefore, the clear tympanitic note is limited below by a non-resonant one, and the limits change suddenly and remarkably with change of the patient's position. In the horizontal position upon the back the portion of the pericardium containing air must be spread to the greatest extent against the anterior wall of the chest, and this extent grows constantly less as the patient rises and bends forward. In the same way the contents of the pericardium arrange themselves according to weight in the different lateral positions of the patient.

It is not certainly known, but it is to be expected à priori, that a succussion sound can be obtained by shaking the patient, as in hydro-pneumothorax.

The signs of pneumopericarditis obtained by auscultation are very surprising and peculiar, for they are remarkable for their intensity as well as their character. As a rule several different sounds can be heard, and indeed they are usually so loud [1] that

[1] I have heard very loud endocardial murmurs (naturally without metallic resonance) by chance within a short time in three cases of aortic insufficiency, and they were diastolic. These murmurs could be heard at a distance of ten steps from the bed.

they can be perceived not only by the patient, but also by those standing at some distance; thus in Stokes's case the sleep of the patient himself and of his wife was disturbed by the murmur.

The heart sounds are unusually loud and accompanied by a clear metallic ring like a chime (Friedreich). It is of course said that in many cases the heart sounds cannot be heard, but this can only be the case when other sounds conceal them.

In most cases a large amount of liquid exudation is present at the same time in the pericardial sac, and this is moved about by the heart in the space containing air, and is agitated and mixed with it. In this way there are produced loud sounds accompanied by a metallic ring, splashing and spluttering, gurgling and rattling, and consequently known as water-wheel sounds (bruit de roue hydraulique, bruit de moulin). In the case observed by Oppolzer in Pitha's clinic the sound was not unlike that produced by shaking shot in a shot-pouch. Dropping is heard as in pneumothorax. If pericardial friction is present it is also accompanied by metallic resonance.

As compared with these symptoms, those of pericarditis, which are present at the same time, retire to the background. If there is a large purulent effusion it naturally helps materially to greatly oppose the circulation and respiration, and to make the prognosis exceedingly unfavorable. The collection of air itself in the pericardium can of course exert an influence by its great tension upon the filling of the ventricle during the diastole and also upon the retraction and compression of the lungs.

What makes the progress and termination of the affection especially unfavorable is of course in most cases the original process which in itself often gives a purulent, ichorous character to the consecutive pericarditis. We can form no opinion as to the nature of those cases which have terminated favorably;—of fourteen cases collected by Friedreich ten ended fatally. Hitherto these have been usually explained as due to the development of gas in the exudation; but one would suppose that a decomposing exudation in the pericardium would certainly prove fatal. In the cases that recovered it must certainly have been atmospheric air which penetrated in some unknown way into the peri-

cardium and was there reabsorbed, as we know can be done in the course of a few days.

The *prognosis* of pneumopericarditis is consequently unfavorable; those cases must be considered certain to end fatally in which the original process produces dangerous complications. Most favorable in any case are those due to mechanical traumatisms.

The *diagnosis* of pneumopericarditis seems as a rule to offer no great difficulties. It may be confounded with a greatly distended stomach, which may produce metallic sounds in the region of the heart, but which usually are independent of its movements. In many cases of this kind, however, the action of the heart produces, by a sort of internal percussion of the stomach, systolic metallic sounds and even râles of metallic timbre (Gerhardt). The distention of the stomach, which can be easily recognized, the normal area of dulness, the apex-beat at the normal spot, or a little to the outside of it, make it easy to avoid a mistake. Metallic sounds dependent upon the movements of the heart can also be heard in cavities near the apex of the heart. It is not difficult to discriminate them: the heart occupies its normal position, rales are also produced in the cavity by respiration, and by opening and shutting the mouth the hight of the percussion note is changed (Gerhardt). Encapsulated pneumothorax near the heart is most likely to present symptoms similar to those of pneumopericarditis. Still in such cases the precordial dulness can usually be made out, even if displaced; and on auscultation, besides the metallic ring of the heart sounds, the resonant phenomena which accompany the respiratory sounds can especially be made out.

The *treatment* must be directed to the original lesion and the consecutive pericarditis. As for the latter, the principles already laid down hold good; oppose the adynamic symptoms by strengthening and stimulating measures, regulate the action of the heart by digitalis in small doses, place ice bags over the heart.

Narcotics must be used to control the pain, restlessness, and sleeplessness as well as the dyspnœa.

As soon as the distention of the pericardium becomes very

great, the question must be considered whether it is not proper to remove as much as possible of the air from the pericardium by puncture with a fine trocar. The procedure may be considered as free from danger; the precaution must only be taken to introduce the trocar while the patient lies upon his back.

If the escaping gas shows by its odor—like sulphuretted hydrogen—that processes of decomposition are going on in the pericardium, or if it is accompanied by decomposing liquid, then the performance of a radical operation and the washing out of the pericardial sac with a disinfecting liquid is decidedly indicated. But this should be determined upon only in case the fundamental lesion offers some chance for the preservation of life.

Hydropericardium.

Schellhammer, Diss. de aqua pericardii. 1694.—*Fr. Hoffmann* and *Graetz*, Diss. de hydrope pericardio rariss. Hal. 1697. v. Opp. Suppl. II. 2.—*Merker*, Diss. de hydrocardia. Ultraj. 1711.—*Landvoigt*, Diss. de hydr. pericard. diag. Hal. 1798.—*Heinecke*, Diss. de hydr. pericard. Erf. 1709.—*Modes*, Essai sur l'hydrod. du péricarde. Paris. 1808.—*Schuh*, Oesterr. méd. Jahrb. Bd. XXIV. *Schmidt's* Jahrb. XXXII. S. 196.—*Skoda*, Oesterr. méd. Jahrb. 1841.—*Philipps'* Jahresb. 1841. S. 39.—*Waschsmuth*, Virch. Arch. Bd. VII. 330. *Virchow*, Ges. Abhandl. S. 108. u. Spec. Path. Bd. I. S. 205. See also the various manuals and text-books.

In the pathology of former times *dropsy of the pericardium* played an important part, since not only was no distinction made between inflammatory effusions and serous transudations, but the affection was also said to occur as a metastasis in a great variety of acute and chronic affections, and to be the cause of their unfavorable result. In recent pathology pericardial dropsy occupies a different position, but in many places at least the old view is still the popular one, and the symptom is still regarded as an unmistakable harbinger of death.

By *hydrops pericardii* (pericardial dropsy, hydro-pericardium, hydrocardium), we are to understand an accumulation of serous fluid in the pericardium occurring independently of inflammation. These terms, however, in view of the fact that a

certain amount of fluid is met with in most autopsies, are applicable only to those cases in which the dropsical accumulation is excessive. The hypothesis has been frequently advanced that a certain amount of liquor pericardii is present even during life, for the purpose of filling the vacuum and facilitating the movements of the heart. I cannot agree with this view ; on the contrary, it seems to me that even the normal liquor pericardii is to be regarded as a transudation, which has escaped during the agony, or after death, from the vessels and from the heart itself, particularly from the frequently tensely distended right ventricle. This transudation continues for a time immediately after death, so that more fluid is found in the pericardial sac when the autopsy is made at a late period than when it is made early. The quantity of fluid depends, moreover, upon the duration of the agony, and upon the final cause of death, being increased in those affections which are accompanied by great congestion of the coronary veins, as for instance emphysema, distortion of the thorax, phthisis, death from suffocation, valvular diseases, etc.

Fluid is not found in the pericardium at every autopsy, but it occurs in most cases to the amount of from half an ounce to an ounce. Under favorable circumstances, as much as three ounces and upwards may be present without being necessarily regarded as anything more than a result of the agony and post-mortem changes. Such cases have no pathological significance unless it can be shown that the fluid was present during life (v. Bamberger). The transudations which take place during life are sometimes very copious, amounting to a pint and a half or more, and Corvisart reports the scarcely credible quantity of eight pounds.

The transudation is composed, as a rule, of a clear yellowish or greenish serum ; occasionally, however, it is somewhat turbid from the presence of desquamated epithelium, which has undergone fatty degeneration. Some of the coloring matter of the blood may also be mixed with it, giving it a reddish or brownish tinge, and this escape of hæmatin may take place after, as well as before, death. The reaction of the fluid is alkaline.

The solid ingredients of the effusion are the same as those of

the blood-serum, but in different relative proportions. The percentage of solids also varies considerably in different cases.

The fluid which is effused into the pericardial cavity after death must always be regarded as the product of a filtration, under increased pressure, from the blood-vessels and cavities of the heart; while the fluid contains less albumen than the blood-serum on account of the difficulty with which albumen passes through membranes. The percentage of albumen will depend in these cases upon the force of the pressure, the duration of the process, and the composition of the blood. When the effusion takes place during life, its composition is the result of a number of complex processes (see E. Wagner's Manual of General Pathology).[1]

The fluid probably always contains fibrogenous matter, and therefore the occurrence of spontaneous coagulation in the serous fluid does not warrant the separate classification of a lymphatic or fibrinous dropsy.

Urea is also present in the serous fluid, especially in renal diseases, together with other products of decomposition. In general icterus the coloring matter and acids of the bile are also found.

The upper surface of the pericardium presents more of the characters of inflammation, and has an opaque, pale, dull-white appearance. Formerly there was recognized also an *inflammatory dropsy* of the pericardium, which included those cases of pericarditis where the effusion was chiefly of a serous character—and partially organized products of inflammation were found upon the serous surfaces (chronic pericarditis), or, perhaps, only trifling changes of an inflammatory character are

[1] Composition of the pericardial effusion:

Wachsmuth.		Wagner.		Gorup-Besanez.	
Water	95.37–97.34	Water	96.51	Water	95.51
Solids	2.66– 4.63	*Solids*	3.49	*Solids*	4.487
Albumen	1.43– 3.01	Albumen	2.915	Fibrin	0.081
Other ingredients	1.23– 1.64			Albumen	2.468
				Extractives	1.269
				Inorganic Salts	0.948

noticed. In cases of the latter kind the original affection may have been a simple effusion, in the course of which inflammation has supervened ; but in all such instances, where inflammatory changes are noticed, the inflammation rather than the dropsy is the object of chief concern. At the same time it may be difficult in individual cases to decide which is the more important element.

The cavity of the pericardium is dilated in proportion to the amount of fluid, and its fibrous layer is thinned, or occasionally, when the dropsy has continued for a long time, thickened. In chronic cases, the subserous fat about the heart disappears, the subserous cellular tissue appears œdematous, and the endothelia are relaxed, swollen, and of a granular opacity. The muscular substance of the heart is pale and flabby.

In the more copious effusions the lungs are found compressed, and the diaphragm forced downwards.

The *causes* of pericardial dropsy are very numerous, but the affection is always secondary and never primary and idiopathic.[1] When it makes its appearance during life, and before the occurrence of the agony, it is either due to a stasis in the veins and lymphatics of the heart and pericardium, or it is merely one of the symptoms of a general dropsy.

When the reflux of the blood from the veins of the heart and pericardium is impeded by stases in the lungs, or in the heart itself, the passive congestion thus induced may give rise to a dropsy of the pericardium ; the same result may be produced by atheromatous disease of the coronary arteries. In rare cases also, the contractions of bands of connective tissue in the thorax, or neoplasms in the mediastinum or in the heart, may derange the circulation in such a way as to induce a serous effusion into the pericardium.

The hypothesis has also been advanced that fluid may be effused into the pericardial cavity to fill the vacuum produced in the chest in cases of shrinking of the lungs, atrophy of the heart, and particularly where traction results from the adhe-

[1] Duchek found dropsical effusions in the pericardium in 13 per cent. of his autopsies, Günsburg in only 7.4 per cent., and, in fact, most frequently in pulmonary tuberculosis.

sion of the pericardium to the lungs. The occurrence of such a *hydrops ex vacuo* is especially insisted upon by v. Bamberger, who advocates this view, as a result of atrophy of the heart arising from acute dyscrasic processes, such as cancer, tuberculosis, etc. Günsburg, and afterwards Friedreich and others, have shown, however, that such a genesis of pericardial dropsy is impossible, because the dilatation of the lungs, the retraction of the thorax, and the ascent of the diaphragm would naturally prevent the occurrence of a vacuum.

In the cases mentioned hitherto, the hydrops pericardii is dependent upon local conditions. It may occur, moreover, as one of the symptoms of a universal dropsy, or as the result of a watery condition of the blood, particularly in parenchymatous nephritis, also in tuberculosis, cancer, and other cachectic conditions. But, at the same time, it should be remarked that dropsical effusions do not occur so frequently in the pericardium as in other serous cavities, probably on account of the influence of the incessant muscular contractions of the heart in promoting the circulation of the blood and lymph.

The smaller quantities of serous effusion present during life no symptoms of any kind, and remain undetected until the fluid has accumulated in greater amount. The physical evidence consists chiefly of the results of percussion, and, as regards the quantity of the fluid necessary for recognition, the same rule applies as in the case of pericardial exudations. In the latter, however, the results of percussion are substantially confirmed and their significance certified by the presence of friction murmurs and subjective symptoms; whereas, *in pericardial effusions friction can never occur.* When the amount of effusion is small, therefore, there is greater danger of drawing erroneous conclusions from the percussion signs than is the case in exudations.

Percussion shows an increase of the cardiac dulness corresponding to the distention of the pericardium, the conformation of the distended sac being the same as in pericardial exudations, viz., that of a triangle with its base directed downwards. Here also we have an enlargement of the cardiac triangle and an increase of dulness on the patient's changing from a recumbent to an upright position, or on bending forwards; and this sign is particularly valuable in cases where, in consequence of a coëx-

isting pleuritic effusion, the broadening of the triangle can be detected only at the apex, which is pushed considerably upwards in the upright posture.

In serous pericardial effusions there is of course an absence of inflammatory changes upon the external surface of the pericardium, and therefore, although the borders of the lungs are forced backwards by the distended pericardium, adhesions do not occur. The respiratory mobility resulting from this fact is, however, of no importance as a means of distinguishing this condition from pericardial exudation.

The character of the apex-beat in hydropericardium is also the same as in pericardial exudations, the beat becoming more feeble, or disappearing altogether when the accumulation of fluid is large. The sounds of the heart grow duller, and in very rare cases become entirely inaudible.

Certain symptoms, such as a visible undulatory movement, fluctuation on palpation in the cardiac region, etc., have been described by some writers, particularly the older ones, as occasioned by the presence of fluid in the pericardium; but these signs are just as uncommon in effusions as in exudations. According to Hope, the apex-beat, when it can be detected in spite of a large accumulation of fluid, does not coincide with the systolic sound of the heart, on account of the resistance which the fluid opposes to the cardiac movements.

The influence of the pericardial dropsy upon the cardiac movements, the circulation and the respiration, can be estimated only in those cases in which these functions are not seriously impaired by the coëxistence of a general dropsy or by the fundamental disease. In such cases the *mechanical* influence of the fluid accumulation must manifest itself in the same way as in the case of an exudation. On the other hand, there is an absence of all the symptoms which are produced by inflammation of the serosa, among the most important of which are the acute derangements of nutrition on the part of the cardiac muscle. Where the dropsy continues for a long time it is possible that the pressure of the fluid may impair the nutrition of the cardiac muscle, and thus gradually induce atrophy and degeneration.

The mechanical effects of large pericardial effusions are mainly these: a lowered pulse-wave, a diminished filling of the arteries, a lessened secretion of urine, venous congestion, cyanosis, compression of the lungs, and depression of the diaphragm. As a result of these conditions, difficulty in breathing ensues,

which may increase to a continuous orthopnœa, with extreme dyspnœa in the horizontal position. The patient experiences a sensation, not only of anxiety and difficulty in breathing, but also of oppression and weight in the chest.

When the fundamental disease is of such a character as to itself induce symptoms of this kind, it is impossible to determine what share in their production is taken by the pericardial dropsy. On the other hand, the fundamental disease, by occasioning a high degree of anæmia, may even assist in moderating the intensity of the dyspnœa, etc. Moreover, the rapidity with which the effusion takes place will modify in an important degree the severity of the subjective symptoms.

As regards the *diagnosis* of hydropericardium, the first object is to ascertain whether the existing physical changes are in fact occasioned by a distended pericardium; and here we are to be guided by the same rules as in inflammatory pericardial effusions.

In the differential diagnosis from a pericarditis exsudativa we must inquire whether any local or general conditions can be found which might give rise to a dropsical effusion, and especially whether dropsy exists in any other part of the body. When such a condition is present, the absence of friction sounds, of fever, and of all inflammatory symptoms, is a very important indication.

The *prognosis* is very unfavorable, because the conditions which produce the dropsical effusion are almost invariably of a permanent character; in fact, we generally have to deal with an incurable fundamental disease, of which the hydropericardium is merely a comparatively unimportant consecutive symptom. Inasmuch, however, as it is not at all uncommon for the general dropsy of kidney and heart diseases, etc., to disappear temporarily, it is reasonable to suppose that when such a happy result occurs the pericardial dropsy may also be absorbed. Such an occurrence, however, will be rarely observed, because the effusion does not usually take place until a disappearance of the general dropsy is entirely out of the question. A permanent cure may be anticipated if the dropsy be dependent upon a transitory hyperæmia.

In general dropsy a large pericardial effusion will unquestionably contribute largely to the fatal result, and may even induce it directly by impeding the cardiac and respiratory movements.

The *treatment* must be directed to the fundamental disease. In many cases, therefore, the same remedies are to be used as in general dropsy, and where the latter declines under the use of diaphoretics and diuretics, the same beneficial effect will extend to the pericardial effusion. Cardiac tonics are also indicated.

In copious effusions, which involve a direct danger to life, puncture of the pericardium is entirely justifiable, and is unattended with much risk, if care be taken to exclude air.

Hemorrhage into the Pericardial Cavity.

Hæmopericardium.

This condition occurs, independently of wounds, in rupture of the heart. The quantity of blood poured out into the pericardial cavity will vary considerably, according to the manner in which the rupture takes place. In large lacerations the extravasation is not so copious as one would expect (perhaps a pint), partly because death ensues rapidly from acute anæmia of the brain or from a momentary arrest of the heart's action, and partly because the pericardium opposes more resistance to a sudden than to a gradual distention. Where the laceration is small the effusion of blood takes place more gradually, its quantity is much more considerable, and death results more tardily.

Pericardial hemorrhage may also be produced by rupture of the coronary vessels, where these have been previously diseased, and particularly where aneurisms have formed upon them. Aneurisms of the aorta may likewise burst into the pericardial sac.

The physical signs of pericardial hemorrhage consist of distention of the pericardium, together with the symptoms of acute anæmia and paralysis of the heart. Inflammation of the serosa will ensue as a complication only where the case does not terminate in sudden death.

A certain amount of extravasated blood is frequently found in exudative pericarditis ; and in some inflammations, especially the scorbutic forms, the fluid contents of the pericardium may present the appearances of pure blood.

Free Bodies in the Pericardium.

Lanzoni, Miscell. Natur. Curios. Dec. 111. Ann. VII. et VIII. Obs. 75. p. 119.—*Kussmaul*, Würzb. med. Zeitschr. V. Bd. 1864, and Steinlein, Ein Dorn im Herzfleisch und ein freier Körper im Herzbeutel. Diss. Erlangen. 1868.—*Hyrtl*, Ein freier Körper im Herzbeutel. Sitzungsbericht der K. K. Akad. der Wissensch. 51. Bd. 23. März. 1865.—*Klob*, Freier Körper im Pericard. Zeitschr. der K. K. Gesellschaft der Aertzte zu Wien. N. 49. 1860.—*Rokitansky*, l. c.

Free bodies within the pericardium have been met with on only a few occasions. Some of them were soft and smooth substances, as large as a pea or bean, while others were of a firm, fibroid consistence, sometimes stratified, and even calcified, either at the centre or *in toto*—cardiac calculi. They probably originate in the formation of precipitates or fibrinous coagulations around a foreign body, or in the formation of polypoid growths, which ultimately become detached. No characteristic symptoms have been observed during life.

Pericardial hydatids are of very rare occurrence. A case of this kind has been described by Barlow.[1]

[1] Pathological Transactions, 1855.

WHOOPING-COUGH.

(TUSSIS CONVULSIVA.)

STEFFEN.

WHOOPING-COUGH.

BIBLIOGRAPHY.

Adams, Lancet, Feb. 5, 1870.—*Ballantyne*, Lancet, June 25, 1870.—*Bisset*, Medical Essays and Observat., 1776, p. 174.—*Todd*, Med. Times and Gazette, Mar. 4, 1854.—*William Butter*, A Treatise on the Kink-cough. London, 1773.—*L. Chalmers*, An Account of the Weather and Diseases of South Carolina. Lond., 1776.—*Fl. Churchill*, The Diseases of Children, 1870, p. 270.—*William Cullen*, First Lines of the Practice of Physic. Edinburgh, 1791, Vol. III., p. 422.—*Copland's* Dictionary of Practical Medicine. London, 1858, Vol. II., p. 235.—*Thomas D. Davis*, Philadelph. Med. Times, 61, 1872.—*J. Duncan*, Dubl. Quart. Journ. of Med. Science, August, 1847.—*Th. Forbes*, Disput. de tuss. convuls., Edinb., 1754.—*Fuller*, Lancet, July, 1860.—*Gibb*, A Treatise of Whooping-cough, its Complications, Pathology, and Terminations, etc., 1854, p. 395; Brit. Med. Journ., Oct. 26, 1861.—*M. Good*, Study of Med., by Cooper and Doane, 6th American ed. New York, 1835, Vol. I., p. 268.—*M. Gregor*, Lancet, II., 1846, p. 146.—*J. Grantham*, Brit. Med. Journ., 1871, 16th Sept.—*Hewitt*, On the Pathology of Whooping-cough, 1855, p. 34.—*Hicks*, Med.-Chirurg. Review, April, 1837.—*W. B. Herapath*, Lancet, July, 1849.—*F. Home*, Princip. Med. Edinb., 1762.—*J. Huxhami*, Opp. physico-medic., 1784, Tom. I., p. 98.—*W. Hillary*, Observations on the Changes of the Air and the Concomitant Epidemical Diseases in the Island of Barbadoes, etc. London, 1766.—*Lombard*, Dublin Journ., 1838, Nov.—*William Macall*, Jahrbuch für Kinderheilkunde, 1873, p. 209.—*William Macall*, Remarks on Whooping-Cough, The Glasgow Medic. Journ.—*Maisch*, Americ. Journ. of Pharmac., 1871, p. 530.—*Murchison*, Lancet, Oct. 29, 1870, p. 596.—*W. C. Norwood*, Charleston Med. Journ. and Review, 1852.—*Okes*, Phys. and Med. Journ., 1862, Nov.—*Watt*, Treat. on the Nature, etc., of Chincough. Glasgow, 1813, p. 87.—*Pearce*, Lancet, 1857, 11th April.—*Geo. Hamilton Roe*, A Treatise of the Nature and Treatment of Whooping-cough and its Complications, 1838.—*Rigden*, Practitioner, XXVII., 1870, Sept., p. 151.—*R. C. Russel*, Journal für Kinderkrankheiten, 1862, P. 304.—*Sydenham*, Epist. respons. I. Opp. omn., 1757, p. 194 etc.—*Streeter*, London Gaz., Nov., 1844.—*C. S. Shelton*, Americ. Med. Times, July 20, 1861.—*James Whitehead*, Third Report of the Clinical

Hospital of Manchester, containing Results of Physical Development, Whooping-cough, and Transmitted Diseases.— *Walshe*, A Practic. Treatise of the Diseases of the Lungs, 1860, p. 573.— *Williams*, Diseases of the Chest, 4th edit. — *Wilde*, Practic. Observations on Aural Surgery, etc., 1853, p. 486.— *Webster*, Med. and Physic. Journ., Dec., 1822.— *Watson*, Edinb. Monthly Journ., Dec., 1849; and, On the Topical Medication of the Larynx, 1854, p. 123.— *Willis*, De morbis convulsivis. Amstelodami, 1782.— *Ch. West*, The Diseases of Infancy and Childhood, 1865.—*J. Solis Cohen*, Inhalation; its Therapeutics and Practice. Philadelphia, 1867.—*J. M. Da Costa*, Inhalations in the Treatment of Diseases of the Respiratory Passages, particularly as Effected by the Use of Atomized Fluids. Philadelphia, 1867.—*Martin*, On Treatment of Pertussis, Amer. Journ. Med. Sci., Apr., 1868.—*Prentiss*, Measles and Whooping-cough Coincident; Proceedings of Clin. Path. Soc'y of Washington, Amer. Journ. Med. Sci., Oct., 1868.—*E. Siegle*, The Treatment of Diseases of the Throat and Lungs by Inhalations, with a new Inhaling Apparatus. Translated by S. Nickles. Cincinnati, 1868.—*Felix von Niemeyer*, A Text-book of Practical Medicine, with particular reference to Physiology and Pathological Anatomy. Translated by Humphreys and Hackley. New York, 1869.—*Bedford Brown*, Observations on the Treatment of certain Complications of Whooping-cough by means of the Oil of Turpentine, Amer. Journ. Med. Sci., Jan'y, 1870,—*J. Ludlow*, The Use of Chestnut Leaves in Whooping-cough, Cincinnati Lancet and Observer, Mar., 1869.—*R. Bartholow*, Bromides, their Physiological Effects and Therapeutical Uses. Providence, 1871.—*J. Lewis Smith*, A Treatise on the Diseases of Infancy and Childhood, 2d ed. Phila., 1872.—*P. B. Porter*, Chloral-hydrate in Pertussis, New York Med. Journ., Aug., 1872.—*J. S. Hough*, How to Prevent Paroxysmal Coughs, Amer. Journ. Med. Sci., April, 1873.— *B. F. Dawson*, Quinine in Whooping-cough, Amer. Journ. of Obstet., Feb., 1873.—*Meigs* and *Pepper*, A Practical Treatise on the Diseases of Children, 5th ed. Phila., 1874.—*J. Bordley*, Hydrate of Chloral in the Treatment of Pertussis, Amer. Journ. Med. Sci., April, 1874.—*A. Flint*, A Treatise on the Principles and Practice of Medicine. Phila., 1857.—*D. Maclean*, On the Open-Air Treatment of Whooping-Cough, Glasgow Med. Jour., Nov., 1872.—*D. Carlos*, Treatment of Whooping-Cough by Carbolic Acid, Lond. Med. Record, Nov. 15, 1875.—*R. Harrison*, Treatment of Whooping-Cough with Carbolic-Acid Inhalations, Brit. Med. Jour., 1875, II., p. 489.—*R. Bell*, Treatment of Whooping-Cough by Iodide of Silver, Obstet. Journ. of Gt. Brit. and Ire., III., p. 589.—*R. J. Lee*, Treatment of Whooping-Cough with Carbolic-Acid Vapor, with Description of a Steam Draft Inhaler, Brit. Med. Journ., 1875, II., p. 425.

Ballonius, Epidem. lib. Const. æstio, 1578. Genev., 1724, I.—*Billard*, Traité des maladies des enfans, etc., 1828, p. 535.—*Blaud*, Revue med., 1831, T. I., p. 233.—*Beau*, Gaz. des hôpit., 1861, No. 48, und Archiv. génér., Sept., 1856.— *Blache*, Diction. de médec., en 30 vol., T. IX., p. 24, und Archiv. génér., 1833, Oct. et Nov.—*Blache* et *Guersent*, Union méd., 1853, p. 196.—*Bell*, Diction-

naire des études médicales, T. XIV., p. 226.—*Barrier*, Maladies de l'enfance, T. I., p. 135.—*Bouchut*, Traité des malad. des nouveau-nés, etc., 1862.—*De la Berge* et *Monneret*, Compendium de médec. pratique, T. II., p. 526, 1837.—*Barthez* et *Rilliet*, Maladies des enfants, 1843, II., p. 207.—*J. Capuron*, Abhandlungen über die Krankheiten der Kinder. Uebersetzt von B. Puchelt, 1821, S. 339.—*A. Clermont*, Employment of the Arseniate of Iron, L'Union, 15, 1875.—*Constant*, Gaz. med. de Paris, 1836, p. 532, und Bulletin thérapeutique, VI., p. 229.—*A. Dugès*, Dictionn. de médec. et de chirurg. pratiq., T. V., p. 487.—*Desruelles*, Traité de la coqueluche, 1827.—*Finaz*, Revue médic., T. II.—*Guersent*, Dictionn. de médec., en 21 vols., T. VI., 1823.—*Nat. Guillot*, Union médic., 1853, p. 184.—*Guibert*, Recherches nouv. sur le croup et la coqueluche, 1824.—*Gendrin*, Gaz. médic. de Paris, 1850, 7. Dec.—*Gallerand*, Diss. sur la toux convuls. des enfants, 1812.—*Hervieux*, Journal für Kinderkrankheiten, 1862, S. 114.—*Jacquart*, Gaz. méd. de Paris, Mars, 1862, No. 13.—*Laborde*, Gaz. des hôpit., 1865, No. 38.—*Luroth*, De la coqueluche et de son traitement, 1849.—*Laënnec*, Traité de l'auscultation, 2. edit., T. I., p. 216.—*J. Lieutaud*, Synops. univers. prax. med., 1765, T. I., p. 493.—*Mignot*, Union méd., 3. Juillet, 1862.—*Montan*, Sédillot journ. de méd., 1812, p. 384.—*Ozanam*, Archiv. génér., 1854, T. I.—*J. A. P. Ozanam*, Histoire méd. général et particul. des malad. epid., 1818, T. II., p. 140.—*Roger*, De l'emphyseme généralisé pulmonaire, mediastin. et sous-coutané, Arch. gén. de méd. Août, Sept., Oct., 1862; Bulletin de l'acad., Dec., 1852; Gaz. des hôpit., 1863, No. 3.—*Rostan*, Cours de méd. clinique, T. II., 1830.—*Trousseau*, Mémoire sur la coqueluche, Journ. de méd., Janvier, 1843; Clinique médic. de l'hôtel Dieu de Paris, T. I., 1861, p. 495.—*Triquet*, Gaz. des hôpit., 1863, No. 9.—*Fallami*, Gazz. med. ital. Venet., 1866, 5 Magg.—*Aless. Gambarini*, Schmidt's Jahrb., Bd. 83, S. 326.—*Garelli*, Gaz. med. Stat. Sard., 1851, No. 7.—*Zaniboni*, Gazz. medic. Lomb., 1864, No. 43.—*Brünniche*, Bibliothek for Laeger, Jan., 1867.—*Nic. Rosen von Rosenstein*, The Diseases of Children and their Remedies. Translated from the Swedish into English by Sparrman. London, 1776.

U. A. Aaskow, Sammlung auserlesener Abhandlungen, Bd. IV., S. 512.—*Alberti*, Diss. de tussi infant. epid. Halle, 1728.—*Aberle*, Tuss. convuls., etc., 1843.—*Archner*, Froriep's Notizen, Bd. II., S. 272, Bd. III., S. 336.—*Atenstädt*, Medic. Centralzeitung, 1856, No. 50.—*J. H. F. Autenrieth*, Versuch für die praktische Heilkunde, 1807, Bd. I., Heft 1–2.—*J. H. F. Autenrieth* und *J. G. F. v. Bohnenberger*, Tübingen, Blätter für die Naturwissenschaften und Arzneikunde, 1813, B. I., S. 23.—*Bednar*, Krankheiten der Neugebornen und Säuglinge, Bd. III., S. 48.—*Biermer*, Handbuch der spec. Pathol. und Therap. von Virchow, Bd. V., Abth. I., S. 531, with numerous references to the literature of the subject.—*Breidenbach*, Centralblatt für die medic. Wissensch., 1869, No. 34.—*Binz*, Jahrb. f. Kinderheilk., N. F., Bd. I., S. 235, und Bd. IV., S. 103.—*J. B. Burserius*, The Institutions of the Practice of Medicine. Translated from the Latin into English by Wm. Cullen Brown, in 5 vols. Edinburgh, 1802.—*Peter Camper*, Sammlung auserlesener Abhandlungen, Bd. XVIII., S. 126.—*Canstatt*,

Handbuch der med. Klinik, Bd. II., Abth. 2, S. 677, 1847.—*F. G. Danz*, Versuch einer allgemeinen Geschichte des Keuchhustens, 2. Auflage, 1802.—*Ebeling*, Dissertat. de tuss. convuls. Göttingen, 1768.—*Ettmüller*, De morbis infant, Opp. omn., P. II., p. 1, 1685.—*Ferber*, Jahrbuch für Kinderheilkunde, N. F. III., S. 229.—*Faber*, Würtemb. Correspondenzblatt, 1834, Nos. 19-20.—*C. B. Fleisch*, Handbuch über die Krankheiten der Kinder, 1804, B. II., S. 399.—*Flügel*, Schmierkur gegen Keuchhusten, Blätter für Heilwissenschaft, 16, 1873.—*Friedleben*, Beiträge zur Lehre vom Keuchhusten der Kinder, Archiv für physiol. Heilkunde, Bd. XII., Heft 3–4, 1853.—*J. Frank*, Prax. med. univ. præc., P. II., Vol. II., Sect. 1, p. 825, 1823.—*J. A. P. Gesner*, Sammlungen und Beobachtungen aus der Arzneigelehrtheit, 1771, S. 199.—*Chr. Girtanner*, Abhandlung über die Krankheiten der Kinder, etc., 1794.—*Gerhardt*, Lehrbuch der Kinderkrankheiten, 1871, S. 378.—*Gauster*, Oesterr. Zeitschrift für prakt. Heilkunde, 1857, 30.—*Gelmo*, Jahrbuch für Kinderheilkunde, 1861, Heft 2.—*Griva*, Froriep's Notizen, B. XLII., S. 48.—*E. L. Heim*, Vermischte med. Schriften, 1836, S. 213.—*Hennig*, Jahrbuch für Kinderheilkunde, III., S. 49.—*Hauke*, Jahrbuch fur Kinderheilkunde, VI., S. 75.—*Helmke*, Jen. Zeitschrift, 1866, Bd. III.—*Henke*, Ueber mikroskopische Organismen in den Sputis keuchhustenkranker Kinder und über die Wirkung der Chinin-Inhalationen bei dieser Krankheit, Deutsches Archiv für klin. Medicin, 1874, XII., Heft 6.—*Ad. Henke*, Handbuch zur Erkentniss und Heilung der Kinderkrankheiten, 1818, Bd. II., S. 181.—*Henoch*, Beiträge zur Kinderheilkunde, 1861, S. 71, und N. F., 1868, S. 231.—*Hinze*, Hufeland's Journ. der prakt. Arzneik. und Wundarzneik., Bd. V., S. 906.—*C. F. Holzhausen*, Dissert. inaug. de tuss. convuls., 1815.—*E. Horn*, Archiv für medic. Erfahrungen, 1803 und 1805.—*C. W. Hufeland*, Bemerkungen über die natürl. und geimpften Blattern zu Weimar im Jahre 1788, etc., 1793, und Journ. der prakt. Arzneik. und Wundarzneik., Bd. 4, 7, 9, 13, 15, und 20.—*Fr. Hoffmann*,. Diss. de tussi convuls., 1732.—*Fr. Jahn*, Neues System der Kinderkrankheiten, etc., 1803.—*D. F. Jahn*, Klinik der chron. Krankheiten, 1821, Bd. IV., T. II., p. 375.—*M. Jacobi*, Horn's Archiv für medic. Erfahrungen, Bd. VI., S. 47.—*J. H. W. Klinge*, Etwas über den Keuchhusten, 1792.—*J. G. Köhler*, De sede et natura tuss. convuls., 1818.—*Krukenberg*, Jahrb. der ambulat. Klinik in Halle, 1824, Bd. I., S. 288.—*Küttlinger*, Bayerisch. ärztl. Intellig.-Blatt, 1860, No. 2.—*Keller*, Jahrb. für Kinderheilk., VIII., S. 21.—*Köstlin*, Archiv für wissenschaftliche Heilkunde, II., 4–5, S. 338, 1865.—*J. F. B. Lentin*, Beiträge zur ausübenden Arzeneiwissenschaft, 1797, B. III., S. 25.—*Lesser*, Zur Behandlung des Keuchhustens, Allgem. med. Centralzeitung, 49, 1873.—*W. Sachse*, Das Wissenswürdigste über die häutige Bräune, 1810.—*L. Letzerich*, Virchow's Arch., Bd. XLIX., 1870, p. 530, and Bd. LX., 1874, p. 409.—*Lebert*, Handbuch der prakt. Medicin.—*Lersch*, Rhein. und Westphäl. Corresp.-Blatt, 1844, No. 8.—*Löbenstein-Löbel*, Ueber Erkenntniss und Heilung der häutigen Bräune, des Millar'schen Asthma und des Keuchhusten, 1811, S. 143.—*Lorey*, Jahrb. für Kinderheilk., N. F., Bd. V., S. 248.—*D. C. J. Lorinser*, Lehre von den Lungenkrank-

heiten, 1823, S. 429.—*Löschner*, aus dem Franz-Josef-Kinderspital, II., 1868, S. 159.—*A. Fr. Marcus*, Der Keuchhusten, etc., 1816.—*Matthaei*, Horn's Archiv für med. Erfahrungen, Bd. III., S. 227.—*Mellin*, Beschreibung des Keuchhustens zu Langensalza, 1770.—*Mehlhose*, Rust's Magazin, Bd. LI.—*Fr. C. Meltzer*, Abhandlung von dem Keuchhusten, 1790.—*Memminger*, Hufel. Journ., Bd. XIII., S. 189.—*Monti*, Jahrb. für Kinderheilkunde, N. F., Bd. VI., S. 103. —*Oppolzer*, Wien. med. Presse, Nos. 34–36.—*L. Paldamus*, Der Stickhusten, 1805.—*J. Panzani*, Beschreibung der Krankheiten, die in Istrien, 1786, geherrscht haben. Aus dem Italienischen von Fechner. 1801, S. 35.—*Pohl*, Progr. de analog. inter morbill. et tuss. convuls., 1789.—*A. B. Polack*, Diss. de tuss. convuls., 1829.—*Rapmund*, Das Chinin in der Kinderpraxis, etc., Deutsche Klinik, 7, 1874.—*Rehn*, Wiener med. Wochenschr., 1866, Nos. 52–53.—*C. L. Reinhard*, Spec. Nosolog. und Therap., 1834, B. I., S. 408.—*A. G. Richter*, Spec. Therap., 1821, Bd. VIII., S. 17.—*Rosenthal*, Die Athembewegungen und ihre Beziehungen zum N. vagus, 1862.—*J. Schäffer*, Hufel. Journ., Bd. VI., St. 2, and Ueber die gewöhnlichen Kinderkrankheiten, 1803, S. 359.—*Schenck*, Observ. med. rar. nov., 1600, Lib. II., p. 382.—*M. Stoll*, Ration. medendi, etc., P. II., 1779.—*C. Sprengel*, Handbuch der Pathol., Bd. III., 1797, und Versuch einer pragmat. Geschichte der Arzneikunde, Bd. III., 1794.—*Steiner*, Compend. der Kinderkrankheiten, 1873, S. 155, und Jahrb. für Kinderheilk., N. F., Bd. V., S. 392.—*Scoda*, Allgem. Wiener med. Zeitg., 1860, No. 1.—*Stelzl*, Oesterreich. Jahrb., 1844, August.—*Schott*, Württemb. Correspondenz-Blatt, Bd. IX., No. 38.—*Santlus*, Journ. für Kinderk., XXIII., 1854, Heft 4.—*A. Steffen*, über Inhalat. bei Tussis convuls., Journ. für Kinderkrankh., XLVII., S. 6, und Jahrb. f. Kinderheilk., N. F., Bd. IV., S. 227.—*J. A. Unzer*, Med. Handbuch, 1789, P. I., S. 228.—*J. Wendt*, Die Kinderkrankheiten systematisch dargestellt, 1822, S. 457.—*Wilde*, Zur Therap. des Keuchhustens, Deutsches Archiv für klinische Medicin, Bd. XIV., Heft 2.—*Wintrich*, Med. Neuigkeiten, 1858, S. 63, and 1861.—*Zitterland*, Rust's Magaz., Bd. XXII., p. 300.

Finally, I cite the Handbook of Historico-Geographical Pathology, by Hirsch. In the second volume, 1862–64, p. 103, will be found the history of whooping-cough treated of in the greatest detail, with the very abundant literature which belongs to it.

The literature of whooping-cough has already increased to such proportions, that in the foregoing list space could be found only for what was most important.

History.

It cannot be determined with certainty when whooping-cough first showed itself, or when it was first observed as a specific disease. Almost all authors agree in this, that the first well-established accounts are derived from Schenck (circa 1650) and Baillou (circa 1600), and refer to epidemics of tussis convulsiva

in the sixteenth century. All supposed previous descriptions of this disease are far from being characteristic of it, but seem to have been based upon epidemics of bronchitis or influenza. Among the works upon whooping-cough which immediately follow the above, that of Ettmüller (1685) is to be specially mentioned. The disease seems now to have spread rather rapidly. Moreover, we may judge how considerable was the prevalence of whooping-cough in certain places during the second half of the sixteenth century, from the report that in the year 1580 nine thousand children died from it in Rome.

In the course of the eighteenth century it extended over the principal part of Europe, and also over quite a number of countries in other parts of the world, and seems, from its rapid and energetic development, to have had a preference for certain regions. The consequence is that people paid closer attention to this form of disease in various quarters, and in the second half of the century, especially, several works regarding it were published in Germany, England, and Sweden, *e.g.*, by Alberti (1728), Fried. Hoffmann (1732), Sauvages (1757), N. Rosen von Rosenstein (1768), M. Stoll (1779), Butter (1773), Willis (1782), McCullen (1784), Fr. C. Meltzer (1790), C. W. Hufeland (1793), Girtanner (1794), C. Sprengel (1794 and 1797), and others. Biermer (l. c., p. 535) has compiled a synopsis of the epidemics of whooping-cough which prevailed in the eighteenth century in Europe and other countries.

It seems always to have been assumed that whooping-cough was of a contagious nature. As the ideas of a humoral pathology were the prevalent ones in the eighteenth century, authors and physicians, with few exceptions, sought for the cause of this disease in disturbances of digestion. These latter, it was supposed, exerted an irritating action upon the diaphragm and respiratory organs, and thus caused the attacks of coughing.

With the constantly increasing geographical extension of whooping-cough since the beginning of this century, the observations and treatises on it have naturally accumulated. In the first half of this period we find a large number of works published, especially in Germany, England, and France. Among the more important of this date belong, in Germany, Danz (1802), E. Horn

(1803), Fr. Jahn (1803), Paldamus (1805), A. Fr. Marcus (1816), J. Frank (1823), Kruckenberg (1824), Faber (1834), Aberle (1843), Canstatt (1847); in England, Okes (1802), Webster (1822), Hamilton Roe (1833), Copland (1842), Duncan (1847), Herapath (1849); in France, Gallerand (1812), Guersent (1825), Guibert (1824), Desruelles (1827), Blaud (1831), Blache (1833), Trousseau (1843), Barthez and Rilliet (1843). The conviction is constantly gaining ground that whooping-cough has its origin in the nervous system, the respiratory nerves and sympathetic. Some are of opinion that the cause of the seizures is to be sought in the peripheral ramifications, others in the centres of the nervous system. These views, moreover, differ as to whether tussis convulsiva is to be defined as a pure neurosis or as a catarrh of the respiratory organs, combined with an affection of the respiratory nerves.

In very recent times, since the middle of this century, a great number of works on whooping-cough have appeared, including in addition many histories of cases. Even now we are met by almost the same theories regarding it as formerly. At the present time the disease is considered by some authors as a pure neurosis, as it has been already by Copland, Webster, and others, and its origin either transferred to the nervous centres, or ascribed, as it is by Friedleben and others, to the pressure of swollen tracheal or bronchial glands upon the vagus nerve. In opposition to this Broussais[1] and Desruelles had already maintained that whooping-cough was nothing but an inflammation of the mucous membrane of the respiratory organs. The latter author, however, admits that a condition of hyperæmia and irritation of the brain and its membranes is associated with the inflammation. Marcus, Blache, Oppolzer, and especially Löschner, are most decidedly committed to defining whooping-cough as a simple bronchitis, which mainly involves the finer bronchioles and the alveoli. They would explain the accesses of coughing as a reflex irritation produced by the decomposed secretion.

Beau, comparing these accesses of coughing with those which are caused by the entrance of a foreign body into the larynx, felt compelled to assume the existence of an inflammation of

[1] Annales de la méd. physiolog., 1824, p. 471.

that part of the mucous membrane of the larynx which lies above the glottis; from this a drop of secretion would sometimes fall into the glottis, and occasion these seizures.

Some who regard the catarrh of the respiratory organs as the foundation of tussis convulsiva, can find in the paroxysms of coughing nothing which does not belong to them from the location of the trouble. On the other hand, others maintain that a definite specific cause determines this catarrh and the paroxysms, or that the latter are due to a peculiar decomposition of the secretion, which at the same time assumes a contagious character. Letzerich has endeavored to show that the explanation of whooping-cough is to be found in the inhalation of a kind of spores, which produce the paroxysms after they have undergone a rapid and considerable increase.

Finally, we must consider one more theory which was adopted by famous clinical observers like Canstatt, Lebert and others, although it has now but few adherents. It is that whooping-cough is due to a zymotic affection of the general system, and that the paroxysms only indicate that the respiratory organs are the part of the body the most involved. Some authors, as Volz,[1] J. Frank, and others, classed this so-called general affection among the acute exanthemata, particularly because epidemics of whooping-cough often precede or follow those of measles, or both occur close together in the same place or the same individual.

Definition.

The essential features of tussis convulsiva, whooping-cough, Keuchhusten, coqueluche, pertosse, kikhosta, etc., further designations of which are to be looked for in Biermer's work, consist in a catarrh of the respiratory organs; not merely of the infraglottic region of the larynx, of the trachea, bronchi, and their ramifications, but also often, especially in small children, of the nose, and this latter process is indicated by marked paroxysmal sneezing. The catarrh may attain different degrees of intensity, and be of variable duration. It occurs only through infection, and in fact the entrance of the contagious substance

[1] Haeser's Arch., Vol. VI., 3.

is effected by inhalation. The whooping-cough may be confined to this catarrh. Should the disease develop to a higher degree, there set in violent convulsive attacks of coughing, with intervals which are free from it. In consequence of the irritation of the branches of the vagus in the respiratory mucous membrane, and especially of the internal branch of the superior laryngeal nerve, by the specific catarrhal secretion of the affected organs, there occurs a spasmodic contraction of the muscles of expiration, and a spasmodic narrowing, sometimes amounting to closure of the glottis. The action of this sort of respiration is manifested by more or less violent ringing coughs following rapidly one after another, and which are generally interrupted by whistling spasmodic inspirations. During the attack the action of the heart is essentially interfered with. With the remission of the spasm some secretion is expectorated from the air-passages, although this is not a necessary feature, and the contents of the stomach also are often vomited. If the disease has been developed up to this stage, as it subsides there follows in every case a period which is characterized by a catarrh only of the respiratory organs, with more or less abundant secretion, but without these attacks of spasmodic cough. The whole disease has a duration of several weeks, or even months, and is in itself unaccompanied by fever.

Mode of Occurrence and Causation.

At the present day whooping-cough occurs everywhere in Europe, and in most countries in other quarters of the world. It has nowhere been possible to prove an independent development of this disease, although it must be said there is no decided evidence against the possibility of its originating in this way. On the other hand, the importation of the contagium from without has often been positively determined, especially in sparsely inhabited islands. The disease occurs as an epidemic much more often than sporadically, and in the former case is apt to run a severer course.

Climate seems to have no controlling influence on the development or intensity of the disease, although it does look as if

the colder, and especially swampy regions were more likely to exercise a harmful influence than those which are warm and dry. The facts cited by Hirsch (II., p. 105) prove how extensive and dangerous may be the spread of such epidemics in a cool climate. According to these, about 72,000 persons, or one-fortieth of the whole number who died, succumbed to whooping-cough in England and Wales in the period from 1848 to 1855. Wilde reports from Ireland that the disease is almost endemic, and ranks fifth in mortality among epidemic diseases. According to Rosen von Rosenstein, over 43,000 children died of this disease in Sweden between 1749 and 1764. As many as 5,832 children fell victims to it in the year 1755, while in favorable years the number scarcely reached 2,000. On the other hand, 9,000 children died in the year 1580 in Rome, situated in a warm country, although, to be sure, surrounded by marshes.

The seasons, as Hirsch has proved, have no influence on the tendency to the reception of the contagious matter of whooping-cough, or the diffusion of this disease. Earlier observers, not relying, it is true, upon a large number of cases, maintained that a more favorable soil for the development of this disease was furnished in winter and spring,—in the cold and rainy season in the tropics,—especially with sudden and considerable changes in the temperature. Only this, however, appears to be established, that in the winter single cases, whether they be sporadic or belong to an epidemic, have a tendency to run a slower course, because the patients cannot be exposed to mild out-of-door air.

According to Hirsch, no differences exist among races and nationalities in respect of liability to the attack or dissemination of whooping-cough.

As regards sex, all observers who have a considerable amount of material at command agree that the female is more readily attacked than the male.

Bouchut,	out of	33	cases,	records	12	boys and 21	girls.
Aberle,	"	356	"	"	123	" 223	"
Macall,	"	307	"	"	163	" 143	"
Ch. West,	"	100	"	"	44.7%	" 55.3%	"

With regard to age, the experience of authors is a little at variance. Barthez and Rilliet assign the period from one to five years as that in which they have observed the most cases of tussis convulsiva. They have seen one case in a new-born child whose mother had been attacked with this disease four weeks before her confinement. On the very day after birth violent seizures occurred. This disease is rare up to the end of the first half-year of life, somewhat more frequent from six months up to a year. It is rare from the fifth to the seventh year, and grows constantly less frequent from then up to puberty.

According to Wunderlich, the time of life below eight years shows the greatest tendency. Blache had 106 children under eight years of age out of 130.

According to Bouchut, a child was infected on the second day of its life, and on the eighth had a well-developed whooping-cough.

Biermer observed the majority of those suffering from this disease before the sixth year of life. It seems to occur most frequently in the first two years, though but seldom in the first six months.

According to Steiner, children contract the disease most readily between two and seven years. He has even observed cases in children of from two to three weeks, but these are rare, as is the case especially with nurslings.

According to West, by far the largest number of cases, 82.9 per cent., fall within the first five years of life. Bednar has observed the disease between the third month and the eighth year of life. Macall found 52 per cent. under two years, 84 per cent. up to four years, 3.25 per cent. over six years, 5.2 per cent. up to two months.

My own experience agrees with this as a whole, but I am obliged to make the period in which I have seen the greatest number of cases extend from the first up to the eighth year of life.

The nearer children approach to puberty the more rarely does whooping-cough attack them. Still I have observed it in some instances in persons between forty and fifty years of age. Biermer cites cases from Heberden,[1] who had seen a woman of

[1] Op. Medic. Lips., 1831.

seventy years and a man of eighty years suffering from it, and from Berger, who had seen it in a woman of fifty-seven.

Individual predisposition to be attacked by whooping-cough is as much a characteristic of this disease as it is of other infective maladies. It cannot, however, be determined on what this depends, apart from age and sex. It is maintained, to be sure, that delicate, weakly persons who suffer from chronic disturbances of nutrition have a greater tendency to it than the strong and healthy. I cannot assent to this opinion. I have often in the same family seen delicate anæmic children go through with this disease more easily, and suffer less from it, than strong and full-blooded ones. It seems as if children who are or have been suffering from the acute exanthemata—measles, for example,—show a greater tendency than some others to be attacked by tussis convulsiva. In general, many are attacked by this disease in childhood, adults but seldom, some persons never.

In common with the acute exanthemata and most infective diseases, whooping-cough has the peculiarity that persons who have once gone through it acquire a certain immunity from it. It may even be said that a second attack of whooping-cough, when both are reported independently by trustworthy observers, is of vastly rarer occurrence than that persons should be affected a second time with an acute exanthem, such as scarlatina, measles, roseola, or small-pox.

There has always been much controversy over the causation of whooping-cough. The view of the humoral pathologists of the eighteenth century which referred the irritation of the diaphragm and other respiratory muscles, as well as that which determined the paroxysms of cough, to disturbances of digestion, may be indulgently set aside.

Neither can the assumption that whooping-cough is an internal neurosis be regarded as valid. In regarding the cyanosis and œdema of the face, and the conditions discovered post-mortem, viz., hyperæmia of the brain, of its membranes, of the medulla oblongata, and the exudations connected therewith, as causes and not as results of the attacks of coughing, we make the same error as is often made in respect to spasm of the glottis. Even if these conditions have existed previously to the development of

the whooping-cough, we should still have to regard them as simply associated with it. At the end of the last century and the beginning of this, various authors, especially Hufeland, Jahn, Breschet, and others, in order to give support to the idea of a pure neurosis, maintained that a material lesion, an inflammation of the vagus nerve, lay at the bottom of whooping-cough. This nerve was said to have been found swollen and reddened. More thorough investigations at autopsies have proved this view untenable. With a similar view, Friedleben and others inferred that swelling of the tracheal and bronchial glands, by pressure upon the recurrent nerve, might occasion the seizures of tussis convulsiva. With regard to the possibility of this process I must refer to what I have stated on the subject in the section on Spasm of the Glottis. But, even if we concede its possibility, two things still attract our attention: first, that so many children exist with considerable swelling of these glands without contracting whooping-cough; secondly, that the duration of the latter does not always coincide with the duration of the swelling of the glands, but is generally much shorter.

The following facts are directly opposed to this view of tussis convulsiva: 1. Its occurrence as an epidemic. Neuroses do not spread in this way. 2. The origin and subsequent diffusion of the disease by infection. This is just as little observed in neuroses. 3. The absence of a tendency to relapse in whooping-cough, while with neuroses it is well known that such is apt to be the case.

Just as little is whooping-cough to be regarded as a zymotic disease of the general system, in which the exciting cause of the malady is specially localized in the organs of respiration. This idea has been deduced from the observation that this disease, in common with certain infective diseases, attacks the same individual but once; and further, that it seems to show a sort of connection with measles, scarlatina, and small-pox, because it has been epidemic at the same time with these diseases, or before or after them. Cases have undoubtedly happened in which, in the same person, the outbreak of whooping-cough has followed eight or ten days after the eruption of measles, which reminds us of the analogous instance of the simultaneous occurrence of

two acute exanthemata in the same individual, a process which, although rare in recent times, has still been positively established. Whooping-cough has rarely been observed to develop simultaneously with the prodromata of measles. According to Barthez and Rilliet, this cough does not generally appear until some weeks or months after the subsidence of the exanthem. Faber maintained that tussis convulsiva and measles mutually excluded each other. Equally false with this is the conclusion, to which J. Frank and others have come, that the nature of both processes is the same.

Quite apart from the consideration that the essence of these acute exanthemata is widely different from that of whooping-cough, the table prepared by Hirsch also shows that out of 416 epidemics of tussis convulsiva, in only 107 could any coincidence whatever with epidemics of these acute exanthemata be proved. The absence of fever and the free intervals argue positively against its zymotic character.

A catarrh of the respiratory organs lies at the foundation of whooping-cough. This affects principally the mucous membrane of the larynx within the glottis, of the trachea, the bronchi, and their ramifications, extending sometimes even to the alveoli. In rare cases, especially in small children, the nasal mucous membrane is also involved. This catarrh arises from infection, and consequently it, as well as its secretion, has a specific quality, which may certainly be proved, but cannot at present be more closely defined. The proof of the specific character is furnished by the infection of other individuals, as a result of which the same disease is developed in them. The mode in which the infection takes place is that the contagious substance gains access to the respiratory organs during respiration, and there sets up the specific catarrh with its secretion. We do not know of what kind this infectious substance is. It is either simply of a gaseous nature, and is thus inhaled, or there may be very minute particles contained in this gas, which are the proper vehicles of the contagium, or themselves constitute it. It is natural that various hypotheses have been broached as to the character of this infectious substance. Letzerich, especially, has sought to establish by his investigations

in the year 1870, that whooping-cough originates in a kind of fungi which are taken in during respiration, which increase rapidly, and cause the peculiar symptoms. Attractive as this view appears, it has not yet been well enough established, and has even been disputed by other experimenters of repute.

Let the contagium be what it may, it is certain that after it has gained entrance into the air-passages it sets up a catarrh which produces a secretion of specific character. The peculiarity of this latter lies not merely in the fact that it contains the infectious matter, but also that this increases with rapidity, so that this increase has a certain duration until such time as it abates, and the contagium is gradually destroyed. If the secretion has acquired a character which renders it capable of exercising a sufficient degree of irritation upon the catarrhal mucous membrane, the characteristic attacks of coughing are determined as a reflex phenomenon by the excitation of the branches concerned of the vagus nerve, or, in the larynx, of the internal twig of the superior laryngeal.

The reception of the contagium by other individuals is effected principally by the stay of such persons in an atmosphere impregnated with the infectious substance exhaled by patients. Such an atmosphere is found in a room in which patients are, or have been shortly before, and if they are in the open air, then in their neighborhood. Since, however, the contagium may not only be exhaled in a gaseous form, but is also contained in the secretion of the air-passages, we may assume that the fresh sputa of a patient with whooping-cough are capable of proving infectious from the development of gases. By some authors this quality is even ascribed to the dried expectoration. We should therefore avoid using for healthy persons handkerchiefs, towels, etc., which have been used about children suffering with whooping-cough. It seems to me exceedingly doubtful, if it be possible, for healthy persons, without being themselves infected, to convey to others any substance clinging to their clothes, so that the latter shall be attacked with whooping-cough. I at least have never observed such a train of events.

Some authors have maintained, especially in recent times

Oppolzer and Löschner, that there is no other process at the bottom of attacks of whooping-cough but a simple bronchitis, which extends into the finest subdivisions, often even into the alveoli. They claim that the secretion adhering in these places, and stagnating there, undergoes a decomposition, and on the one hand occasions the attacks of cough by the reflex irritation it develops. On the other hand, through the secretion so constituted, the possibility is acquired of its conveying a similar process to other individuals, in analogy with the communicability of acute catarrhs especially, which by means of their secretion are capable of setting up the same process in other persons. In opposition to this view it is to be said that a simple bronchitis never sets in with the characteristic symptoms of whooping-cough; that it never shows the peculiar course of the latter; that it is not contagious in the same sense as this, and that it may have quite frequent relapses, to which whooping-cough scarcely ever shows a tendency. Nor is it at all necessary that the bronchial catarrh in whooping-cough should extend into the bronchioles and alveoli, any more than that a certain amount of secretion must be present to occasion the attacks of coughing. Very respectable attacks of coughing are often observed in which hardly anything is expectorated. If they were excited by a considerable amount of secretion, they could not cease until this was removed.

Gendrin and Beau believed that it should be assumed, in analogy with the seizures of cough which take place when a foreign body enters the larynx, that a similar condition lies at the root of the attacks in tussis convulsiva. They proposed the hypothesis that in this disease a catarrhal inflammation of the entrance and upper portion of the larynx was present. If any of the secretion fell from here into the glottis, the attacks in question would be set up. It does not agree with this that at the height of the process patients are apt to cough harder and more frequently at night than by day, although the horizontal posture in the night ought to diminish the tendency of the secretion to flow into the glottis, and the erect position in the day to increase it. The post-mortem appearances may be delusive in this respect, because the hyperæmia and swelling of the mucous

membrane may have diminished after death. Now although Beau is inclined to sustain his theory by laryngoscopic investigations, similar investigations of others, particularly of Rehn, decidedly controvert it. This observer, in fact, found in adults the supra-glottic region normal, while, on the other hand, the infra-glottic region and the upper part of the trachea were reddened and swollen.

Among the predisposing causes, which may determine the attacks when whooping-cough is present, is primarily to be mentioned a sudden change of temperature, especially passing from warm to cold. In addition to this the purity of the air has an important influence. Hauke has proved by experiment that contamination of the air with carbonic acid is apt to augment the intensity and frequency of the seizures. This may be the explanation of the fact that the disease is wont to attain a higher degree at night, as well as that patients have a greater tendency to these attacks if they are in rooms, whether large or small, which are crowded with people. A brisk wind or air, rendered impure from dust, is likewise injurious. Besides this the attacks are provoked or intensified by all active movements of the larynx, such as continued speaking, crying, etc., as well as by taking food or drink, especially if anything should find its way into the larynx. Rapid movements of the body and mental emotions, as well as external pressure on the larynx, smoking, and the taking of spirituous drinks, may act as predisposing causes of the attacks. Finally, it cannot be denied, that in children even the regimen may at times contribute towards allowing the more rapid development of the seizures.

Symptoms and Course.

Simple whooping-cough manifests itself in the form of paroxysms which are followed by intervals during which the patient is free from cough. These seizures vary according to age, predisposition, and the intensity of the infection.

For the sake of greater simplicity I follow the ordinary division of this process into three stages: 1. The stadium prodromorum, which, after the infection has been received, is mani-

fested only by the symptoms of a catarrh of the air-passages, with more or less violent cough. 2. The so-called convulsive stage, which sets in with the characteristic attacks. 3. The final stage of a catarrh of the respiratory organs, with abundant secretion, but without characteristic attacks of cough.

Strictly speaking, this division does not properly exist. The separate stages often pass so imperceptibly from one into another that the dividing lines cannot be positively drawn. For the stadium prodromorum may be so briefly and imperfectly developed as to be overlooked. In rare cases it seems possible that it may be wholly absent. Finally, the first stage may run its course, and the disease terminate without the second stage having developed.

The chief characteristic, then, of the first stage is a catarrhal affection of the respiratory organs. In the majority of cases the larynx is found to be attacked from the very outset, and thence the process extends downwards to the trachea, the bronchi, and their larger branches. Occasionally the disease begins as a catarrh of the nasal mucous membrane, with smart sneezing, which is sometimes quite frequently repeated in a short space of time, and there is also considerable discharge of a muco-purulent secretion. More often the mucous membrane of the pharynx is found to be simultaneously attacked, even at the beginning. This stage sets in suddenly without premonitory signs. Children, who have been perfectly healthy up to this time, begin to cough as soon as the larynx is affected. The cough generally exhibits no special characteristics ; it is teasing and urgent, and accompanied by little or no expectoration. In certain cases, particularly if this stage is drawing to a close, and is about to pass into the spasmodic, it is found that the paroxysms are more violent, and the individual coughs are repeated more frequently, and follow more closely one upon the other. Trousseau maintains that they may often be repeated forty or fifty times in a minute, and that this violence of the cough may show itself very obstinately through a series of days. In rare cases this stage begins, especially at night, with an attack of catarrhal inflammation of the mucous membrane of the larynx, with acute swelling of the tissues, and a cough which, in consequence of the exist-

ing narrowing of the glottis, presents, both in the sound of the expirations, and in the spasmodic, whistling inspiration by which they are interrupted, the most marked resemblance to that of laryngeal croup. By the next morning these symptoms have disappeared, and the catarrhal stage of the whooping-cough has begun.

We sometimes see this stage run its course without fever, while sometimes there is a moderate degree of it, as in many acute and wide-spread catarrhs. The setting in of the fever is not indicated by an initial chill. Moreover the fever is apt to have no regular course, but is subject to oscillations, which are likewise quite irregular, and are manifested by an increase of the pyrexia after slight chilliness, alternating with some heat. The children look pale and weak, are fretful, with little appetite, and increased thirst, and sleep uneasily; their heads are somewhat affected, and the conjunctivæ moderately injected.

Nothing can be gathered from these symptoms which can be regarded as premonitory of whooping-cough. They are precisely like those of any catarrhal inflammation of the respiratory organs. In rare cases this stage is entirely absent in nurslings, or is limited to one or two days. But even here a duration of as much as one or two weeks has been observed. Berger has estimated this stage as averaging from eight to fourteen days, Lombard from four to six weeks. Wunderlich assigns from half a week to six weeks; West, from two to thirty-five days. From these notes it is perceived how variable the duration of this stage is. In young children it is apt to be the shortest, and to last longer in older ones. On the whole, however, this difference depends upon the predisposition, and the violence of the infection. In other respects there is no doubt that in very many cases it may be difficult, or even impossible, to determine with certainty the beginning of this stage, or its passage into the second.

The whole disease may come to an end with the expiration of this stage, if the predisposition to infection has been too slight, or the contagium not sufficient in intensity and amount to produce the spasmodic stage. In considerable epidemics this course

of events is quite often observed, most frequently among children at the breast.

In the majority of cases the first stage is followed by the spasmodic stage, either gradually and insensibly, or sometimes suddenly. The febrile symptoms which had accompanied the first stage diminish, and there are intervals perfectly free from fever. The attacks of coughing grow by degrees more violent and spasmodic. For some ten minutes before the onset of the attack the children become restless and anxious, call upon their attendants, run to them and seize them as if they were seeking help; older children run to the spittoon or some other vessel, so as to deposit in it, when the expected seizure comes, the sputa, and ultimately the matters vomited. The attack is sometimes preceded for some minutes by nausea. The feeling of anxiety is portrayed in the children's countenances. Older children and adults assert that sensations of tickling or scratching, or of the presence of a foreign body in the larynx, occur as premonitions of the attack. Sometimes, too, there is a sensation as if the larynx were being compressed, and as if a certain degree of insufficiency of breathing were caused thereby. This feeling of constriction of the air-passages often extends down the trachea. It is not unusual for persons standing near to hear coarse râles in the trachea and bronchi of the patient before the seizure, and these may be even more clearly perceived during sleep.

From the latter circumstances the theory has been deduced that the attacks of cough are occasioned by the specific secretion, which, being brought up from the deeper air-passages irritates the portion of the larynx situated below the glottis, already the seat of catarrh, and sensitive from it. As a reason for this it has been alleged that after the expectoration of the secretion the attack stops. It cannot be denied that this theory sounds very plausible, and may certainly serve as a partial explanation of the attacks. Still, it is to be borne in mind that there are attacks without anything being expectorated, and that the irritation of the bronchial mucous membrane may, in itself alone, suffice to excite decided coughing. One has only to call to mind the attacks of cough produced by a foreign body which has entered some ramification of the bronchi.

The attack begins with spasmodic expirations following one after another at brief intervals, and which may be of variable intensity, and the sound of which is dependent upon the degree of narrowing of the glottis existing at the time. The latter may be very slight, and then the resonant sound of the expiration presents nothing striking, while if the constriction is considerable this sound may be similar to that in croup. In the slighter cases the inspiration which is made between these expirations has no special characteristic, and is scarcely audible. The more violent the attack the more rapidly do the expirations follow; the more the glottis is narrowed the more will the inspiration be spasmodic, long-drawn, and accompanied by a whistling sound, the whoop. In the shorter attacks this inspiration is heard but once, while in those which are more violent and long-continued it may be perceived several times, so that an inspiration follows every time upon a certain number of expirations, and the attack is finally brought to an end by expirations. When this happens a viscid, tenacious secretion is generally expectorated from the air-passages. If the attacks are rather violent the contents of the stomach are vomited at the same time, especially just after taking food.

Just before the beginning of an attack the respiration and action of the heart are quickened. During the attack speech is interfered with; only a few almost inaudible words can be forced out. The expression of the patients indicates a greater or less insufficiency of breathing. Older children brace themselves with their arms so as to facilitate inspiration. Tears flow from the eyes, the nostrils move convulsively. The face, which at the beginning may be moderately suffused, becomes livid, and in violent and long-continued attacks may attain to quite a high degree of cyanosis. At the same time it is bloated from the hyperæmia due to interference with the circulation, and this condition is apt to last during this stage, and even longer, so that in some cases the expression of the child seems completely altered. The extremities become cool; sometimes a cold sweat breaks out. Moderate hemorrhages from the nose and mouth are not unusual during the attack. Often, too, the fæces and urine are expelled involuntarily. The younger the children the more easily are

they stupefied by any considerable passive congestion of the brain during the attack, and often remain in this condition for a short time afterwards.

Examinations of the larynx during an attack are not practicable either with the laryngoscope or with the stethoscope. Examination of the lungs during the spasmodic expirations, in the course of which some air is continually driven out, and that contained in the respiratory organs compressed, gives a deadened and shorter percussion note. Auscultation in the main furnishes only a negative result. The spasmodic inspiration is distinctly audible with the stethoscope, especially the older the children are, and percussion again reveals the normal resonance of the lungs. As a matter of course, while these processes are going on, the lower borders, as well as those edges of the lungs which partly cover the heart, suffer the displacement due to the existing state of affairs.

During the attack the action of the heart is essentially impeded by the passive congestion which has occurred, and partly on this account, and partly also, it may be, from the notable irritation set up in the vagus nerve, the heart's action may cease altogether momentarily. Moreover, we must assume that a transient dilatation of the heart is produced by the passive congestion, and that this has an injurious effect upon its action.

After the attack is over, the relations of the respiration and circulation gradually become normal again. The patients are for a short time languid, depressed, and irritable, preferring a recumbent position until the effects, with perhaps the exception of the swelling of the face, disappear. If the attacks are repeated, at short intervals, and especially if they are of an aggravated character, the patients may not be able to recover themselves between times. They remain depressed and apathetic, prefer to lie down constantly, their strength diminishes, they grow thin, especially when the attacks are accompanied by frequent vomiting of what has been eaten. It happens not infrequently that a second attack follows immediately upon the first, if no sputum has been expelled, and it is then generally expectorated with the second. This process likewise argues against the absolute validity of the theory that the attacks are caused the moment any of

the bronchial secretion gets into the infra-glottic region. In the cases under consideration this must be present in the deeper portions of the air-passages, for otherwise it would inevitably have been discharged with any one of the spasmodic expirations.

Physical examination instituted in the intervals always shows the symptoms of catarrhal tracheitis and bronchitis, and, according to whether the process has or has not extended to the finer bronchial ramifications, it exhibits the peculiar features belonging to them. The condition of the heart is found normal.

The duration of the attacks is from a few seconds to several minutes. Their number in the twenty-four hours may be small, or may increase to from sixty to eighty. Macall claims to have observed in a child, eight months old, the enormous number of 140. They are apt to be more frequent and violent at night than in the daytime; so that, as a rule, it may be assumed that with the remission of the nocturnal attacks this stage is tending to its close.

The more violent the attacks are, the more numerous and the more marked are the manifestations to which they give rise. These latter are mainly dependent upon disturbances of the circulation and respiration. To the former belong the rupture of small vessels and the resulting hemorrhages. Hemorrhages from the nose and mouth are nothing unusual. Sometimes, however, they are profuse, and accompany almost every attack for days and weeks, so that the life of the child may be endangered from the loss of strength. The hemorrhages from the mouth may originate in the mucous membrane of the buccal cavity and pharynx; they may also come from the larynx. Hemorrhages from the more deeply situated air-passages, viz., apoplexies of the lungs, have not as yet been observed during life. Hemorrhages into the conjunctiva, sometimes quite extensive, are not seldom observed. Generally they involve both eyes, and in like manner the extravasation of blood into the surrounding tissues, which is often quite considerable, is apt to be on both sides. Trousseau has seen blood escape from the conjunctiva and mix with the tears. Ecchymoses of the cheeks and throat are less common, and ordinarily of small compass. Hemorrhages from the ears, with perforation of the drumhead, have been several

times observed. Roger in such a case has seen the blood spirt out. Effusions of blood into the brain, the meninges, and between these are rare.

In consequence of the passive congestion the neck may be swollen during the attack, and its veins filled to distention. There may occur œdema of the brain, or transudations into the ventricles and between the membranes. The case observed by Sebregondi, in which a girl of six years is said to have become blind with every attack, must in like manner be referred to the blood stasis. A year ago I treated a girl of eight years who saw indistinctly during the attack, but also lost some of her sharpness of sight in the intervals, as long as the spasmodic stage lasted.

The more violent the expirations are in the attack, and the more rapidly they follow one another, so much the more positively is spasm of the glottis developed with narrowing of its chink. Since enough air cannot escape through the latter, in spite of the violent expirations, what remains behind is compressed to the utmost by the muscles which preside over respiration, and it may even result in injury to the vesicles of the lungs. From their being so distended by the air spasmodically forced into them, emphysema results, which, as a rule, is apt to involve only the superficies of the lungs or certain peripheral sections, and then passes off without any symptoms. If the whooping-cough does not last too long, and the elasticity of the alveoli is retained, this condition may again pass into a perfectly normal one on the subsidence of the attacks. Under opposite conditions it may continue, and if the attacks attain a very high degree, it may increase, by rupture of the alveoli, to interstitial and mediastinal emphysema. Perforation of the pleura with subpleural emphysema, and the development of pneumothorax are exceedingly rare, and, in fact, as a rule, fatal, because the fits of coughing do not allow of reabsorption of the air which has escaped, and are constantly forcing it back anew, so that the seat of rupture cannot close. More frequently, after the previous occurrence of mediastinal emphysema, the development of a general emphysema has been observed, which has involved at least the larger part of the surface of the body.

Roger, Blache, Hervieux, J. Frank, Gelmo, and others have published a number of such cases. If the point of rupture of the alveoli closes, reabsorption of the air and complete recovery may ensue.

If whooping-cough is severe, we shall not fail to find in the majority of cases an ulcer on one or both sides of the frænulum linguæ, less often on the upper surface of the tongue. These ulcers are shallow, with somewhat elevated edges, which, as well as the base, are of a grayish-yellow color. They depend upon the fact that during the attack the tongue is wounded by being thrust between the teeth at a spot where they are either very much inclined or very prominent. The ulcer disappears of itself, with the subsidence of the spasmodic stage. The first descriptions of this ulcer come from about 1840.

In attacks of a high degree of severity the increase of struma has been observed. In like manner the development of herniæ and prolapsus ani are met with, or the increase of these conditions, if they were already present. Biermer quotes a case from Schott[1] in which hydrorrhœa was established from rupture of the fœtal membranes during whooping-cough.

If the spasmodic stage runs an uncomplicated course, it usually gives rise to no serious apprehensions of any kind. Still it has been observed that in excitable persons the attacks have been the source of severe headaches, which also continued in the free intervals, and sometimes appeared to leave the issue of the disease in doubt. I have seen cases in which the headaches were constant during the whole duration of this stage. Moreover, there not infrequently occurs a morbidly increased irritability of the brain. This is characterized in its least degree by great sulkiness, which may increase to perfect indifference. In a more advanced degree, especially in connection with profuse hemorrhages, frequent vomiting and loss of appetite, delirium may set in, which is dependent upon the inanition of the body and the deficient nutrition of the brain resulting from it. In its highest development Ferber has observed acute mental aberrations at the acme of the whooping-cough.

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[1] Württemberger Correspondenz-Blatt, Vol. IX., No. 38.

As a result of profuse hemorrhages, loss of appetite, and disturbances of nutrition, after frequent vomiting, a degree of marasmus may be gradually established which may be alarming and fatal.

In very rare cases children are seen to succumb suddenly and unexpectedly during a coughing fit. This accident generally occurs to children in the first or second year of life. Their sudden death may be attributed to various causes. The spasm of the glottis which has taken place may reach so high a degree as to occasion complete persistent closure of the glottis and suffocation. Death may also result from effusion of blood into the brain, or from considerable transudation into the brain and its ventricles. Besides this, it may be the consequence of paralysis of the heart which may develop after a momentary stoppage of it. Finally, there is the possibility that the rapid development of a diffuse pneumothorax may suddenly lead to a fatal termination.

Authors are divided in opinion as to the duration of this spasmodic stage. Gerhardt fixes it at from two to ten weeks, Steiner at from three to eight weeks. Biermer assigns from four to five weeks as the medium duration, but has also seen this stage last but two weeks, and, on the other hand, several months. According to Barthez and Rilliet it varies between fifteen and sixty-five days. It is difficult to determine the duration of this stage, as it is not always possible to fix its limits exactly. So much as this, however, is certain, that the longer it lasts the more feeble and infrequent do the attacks gradually become, and that when it lasts a long time the attacks are not apt to be either numerous or very severe. If the patient has passed safely through the spasmodic stage, there always follows a so-called stadium decrementi, into which the former gradually merges. The attacks lose their violence and become less frequent, and this is particularly noticeable during the night. The spasmodic inspiration disappears entirely, and the cough assumes the character of that in a simple catarrh of the air-passages. The expirations are no longer so violent and spasmodic, and the pauses between them become longer. The feeling as if something were obstructing the breathing no longer precedes the cough. The

secretion of the air-passages is now expelled by the cough, and its character has undergone a change; it is somewhat thick, yellowish, or greenish, and consists of mucus and pus-corpuscles. The vomiting and the disturbances of the circulation, which were present during the attack, have now ceased. The blood effused into the mucous membrane or the integument is reabsorbed. The physical examination of the organs of respiration indicates the symptoms of a simple catarrhal affection merely. The appetite, if previously diminished, now speedily returns, and with the disappearance of the cough and expectoration the child recovers its strength in a longer or shorter time, if any adequate power for reaction is present. Still, children even in this stage have been seen to succumb to marasmus.

It sometimes happens that patients are believed to have a return of all their symptoms when the stadium decrementi has already been reached. After certain exciting causes the attacks of spasmodic cough are suddenly observed to recur. Still, this stage of relapse is not apt to last long.

The period occupied by the third stage is variable. In the most favorable case it may last only a few days, but it may also last for weeks. In many children who have had whooping-cough there may remain for years a tendency to this spasmodic character, even though the cough proceeds from a simple catarrh.

The picture of whooping-cough, as here presented, is that of the disease as observed in childhood. When children grow older, that is, after the tenth or twelfth year, and especially in adults, the attacks of cough gradually lose their violence, and the respiratory and circulatory symptoms that follow are less actively developed. The whole duration of whooping-cough, which in children may extend over some months, shows itself in adults decidedly shorter.

Complications.

There are certainly few diseases which may not complicate whooping-cough.

Among the most frequent complications are to be reck-

oned other diseases of the respiratory organs. Among 307 cases of whooping-cough, Macall found eighty-one complicated with other diseases of the air-passages.

In very rare cases croupous laryngitis has made its appearance in the course of the disease, and almost always with a fatal issue. In Biermer's case the whooping-cough attacks remitted on the setting-in of the croup. Blache, Gauster, and some others have observed this complication. Barthez has seen œdema glottidis with a fatal result occur in a girl of four and a half years, who had suffered from whooping-cough for three months. Tracheotomy was performed in vain.[1] A similar case was observed by Benoit.

I have once seen typical tonsillitis accompany the outbreak of whooping-cough.

In the majority of cases the bronchitis which accompanies tussis convulsiva remains limited to the larger divisions of the bronchi. If it also attacks the bronchioles and alveoli, the seizures are apt to diminish in intensity, or to become less frequent.

The indications of fever vary; a moderate degree of cyanosis is persistent, but now and then increases without attacks of cough. The variable degree of respiratory insufficiency which is present, depends upon the extent and intensity of the capillary bronchitis, and is sometimes temporarily relieved by a free expectoration of muco-purulent masses, vomiting sometimes occurring in connection with it. The children are very restless, throw themselves about, are fretful, and the appetite diminishes, while on the other hand there is great thirst. Sometimes they are afflicted with intercurrent diarrhœa, and I have also observed profuse sweats. In higher degrees of the affection children are seen to be apathetic in consequence of the stasis of blood in the brain, and its being overcharged with carbonic acid; they take no interest in anything, and rarely make any response if they are addressed or touched. Sometimes they are so stupefied that for hours they cannot be aroused, and their fæces and urine are passed unconsciously.

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[1] Journal für Kinderkrankheiten, 1869, I., p. 221.

Physical examination of the chest reveals labored respiration, bulging forward of the upper portions of the thorax, and below, the peripneumonic furrow (Trousseau); on auscultation, fine, partly sonorous râles in the regions affected by capillary bronchitis. Sometimes the process has been observed to begin in certain places, diminish, and then involve others.

This complication is of a very serious nature, the more so if the process is widely diffused. The majority of those affected in this way succumb to it, rarely suddenly, but only after the expiration of a week or weeks. If the disease passes into convalescence, the affection of the bronchioles and alveoli first diminishes; that of the larger branches may last still longer, so that a long time elapses before the patients completely recover.

Whooping-cough is found to be complicated with pneumonia just about as often as with capillary bronchitis, especially as the pneumonia develops upon the bronchitis as a foundation. Diffuse pneumonias, with exudation into the alveoli and bronchioles, are very seldom found. On the other hand, the inflammation of the alveoli and bronchioles passes over to the neighboring tissues, and forms circumscribed pneumonic foci, which extend from one to another if they are situated close together, and if they are present in large numbers may produce consolidation of considerable sections of a lobe, or of a whole lobe. The younger the children are, the more likely is this process to take place under the form of the well-known *stripe-pneumonia*,[1] in the posterior portions of the lungs. At times the lower lobe only is affected, at others the process reaches from the base to the apex. It may be found upon one or both sides; in the latter case either simultaneously or one side following the other.

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[1] I have used this term, "stripe-pneumonia," as a translation of our author's word "Streifenpneumonie," which, so far as I have been able to learn, has yet found no recognized English equivalent. In the Jahrb. für Kinderheilk., VIII., 3, p. 255, 1875, Steffen thus defines the condition referred to. "An inflammatory consolidation of the lung tissue in the form of a strip found on one or both sides, at the posterior portion of the lungs, which extends in width from the vertebral column to the angle of the ribs, and in length may include either the whole height of both lungs from base to apex, or merely a single lung or lobe." It is always a secondary affection or complication of some other disease, and occurs in connection with hypostatic congestion, atelectasis, or bronchitis.—TRANSLATOR'S NOTE.

The functional symptoms are similar to those of capillary bronchitis, especially as the latter disease is associated with the pneumonia. If neither process, however, is very extensive, the symptoms are apt not to be so severe as in general bronchitis without pneumonia. The respiratory insufficiency and cyanosis are not so noticeable, and even, if the children are apathetic in the higher degrees of the disease, the sensorium is not apt to be affected to any considerable extent. The conditions found on physical examination in general or in circumscribed pneumonia are such as are well known, and are to be found in the sections treating of these forms of disease.

All painstaking observers agree that the complication of whooping-cough with pneumonia is just as dangerous as with general capillary bronchitis. About two-thirds of those affected with it die, and this takes place the more quickly the younger the children are. Generally, the majority of fatal cases is to be ascribed to this complication. Out of twenty-seven fatal cases Charles West observed thirteen complicated with capillary bronchitis and pneumonia. Macall ascribes two-thirds of the fatal cases in whooping-cough to this complication. Still the course of the pneumonia is apt to be much more prolonged than that of the bronchitis. Suddenly fatal cases do not occur in this disease. It may, however, last for weeks or months, whether its termination be favorable, or the reverse. A complete recovery, in which no traces of the affection are left behind, is, to be sure, exceedingly rare. Usually there remains a partial emphysema, due in part to the whooping-cough, and in part to the inflammatory condensation of the lung. Moreover, there remains in those regions which were attacked by the inflammation a retraction of the interstitial tissue, and partly from this, and partly from the same law under which the emphysema develops in whooping-cough, there remain partial dilatations of the bronchi with persistent catarrhal symptoms. If the pneumonic foci fall into the retrogressive course of caseous formation and degeneration, the patients sink with the symptoms of pulmonary phthisis.

Not infrequently atelectasis is developed in the course of the capillary bronchitis of whooping-cough, particularly in younger children, in isolated points, or even involving more considerable

areas. The reason is found in the plugging of the bronchioles by secretion that cannot be expelled from them,—a result brought about by the absorption of the air contained in the affected alveoli followed by their collapse. The functional and physical symptoms render it difficult to distinguish this process from pneumonia, especially as it is generally allied with processes which are accompanied with fever. With younger children it has this additional resemblance to pneumonia, that it not seldom occurs in the stripe form in the posterior parts of the lungs. If the obstacle to the entrance of air into the alveoli is not removed, circumscribed pneumonia develops itself at these places. A sudden occurrence of consolidation in the lungs would rather lead us to infer atelectasis than pneumonia.

In many quarters, and with justice, stress is laid upon the frequent occurrence of pulmonary phthisis after whooping-cough. The connection may be of various kinds: in the first place, the degeneration of cheesy pneumonic foci may in itself cause phthisis; then again, we may have to do with a secondary development of acute or chronic tuberculosis in the lungs alone, or in other organs also. This tuberculosis may originate in the pneumonic foci indicated, or also in tracheal and bronchial glands which had entered upon the condition of hyperplasia and cheesy transformation even before the beginning of the whooping-cough. This affection of the lymphatic glands is, however, in many cases, also primarily the result of a long-continued or very violent bronchitis or pneumonia. For the development of tuberculosis may often not occur till long after these inflammations have run their course, and that too even favorably. It is well known that pulmonary phthisis occurs after chronic tuberculosis only through the secondary development of an insidious pneumonia. Finally, it is plain that chronic tuberculosis and pneumonia, when already present, may be brought into a condition of greater activity by whooping-cough.

It is known that pneumonia seldom runs its course without pleurisy; yet in this connection it is apt to be more of a parenchymatous nature and to furnish only a little free exudation. Pleurisy without pneumonia has scarcely been observed in

whooping-cough. In light cases whooping-cough may run its course without spasm of the glottis. In more severe cases this is not absent, and in intense cases the two keep pace with one another. The spasm may set in with such violence and persistency as to result in actual stupor, or sudden death from suffocation.

As a rather infrequent complication on the part of the nervous centres, are to be mentioned slight convulsive movements, at the height of the attack, in various parts of the body, especially the muscles of the face; spasmodic movements of the eyeballs are also noticed. In a boy nine years old I saw strabismus internus of the right eye occur during violent attacks, while the left eye was retained in the normal direction by tonic spasm. They are partly the result of the essentially violent excitations of the nervous system, partly of the stasis of blood in the brain and spinal cord. In coughing attacks of the greatest severity they sometimes amount to general convulsions, which, in the majority of instances, prove fatal the first time, or after some repetitions. The intervals which follow are not, as a rule, complete, but are marked by slight partial convulsions. West maintains that this complication furnishes by no means a rare cause among the fatal cases after whooping-cough. Macall estimates them at one-sixth of all the fatal issues. If the children withstand these attacks, there remains for a long time a morbidly increased irritability of the nervous system. In extremely rare cases, always as a result of centres of disease in the brain, epilepsy and chorea have been seen to follow. Rilliet reports the cure of two cases in girls of five and six years old. Ozanam has seen but one child restored to health after convulsions.

The complication of pneumonia with acute meningitis or acute miliary tuberculosis of the pia mater, and the results of these diseases, have been observed only in the most isolated instances.

Endocarditis and pericarditis are exceedingly rare in whooping-cough.

Deafness or hardness of hearing is not an infrequent result of hemorrhages into the organ of hearing, or of long-continued catarrh of the tubes or tympanum, or of purulent inflammation

of this cavity with perforation of the drum-head and chronic otorrhœa.

Among the very frequent accompaniments of whooping-cough, especially if vomiting often occurs with the attacks, belong want of appetite, impairment of digestion, and catarrh of the mucous membrane of the stomach and intestine. This is a disagreeable addition, particularly in cases where the children are delicate, because the diminished supply of nourishment on the one hand, and on the other the oft-recurring attacks of diarrhœa, may contribute essentially to wear out the strength. Heyfelder has seen cholera as a fatal complication.

I have found swelling of the liver in whooping-cough only when pronounced rachitis was present at the same time. The autopsies demonstrated the presence of increased accumulation of fat in the liver cells.

At the time of violent seizures, or shortly after them, according to my observation, albumen may be contained in the urine. Investigations are wanting as to whether admixtures of blood are also present in it.

The passive congestion, which is produced in the body by violent attacks of whooping-cough, and often by affections of the lungs also occurring at the same time, may, in connection with increasing marasmus, lead to dropsical troubles which are calculated to hasten the fatal issue. Sometimes these exudations are only partial, in the face and upper extremities, but as they increase the cavities of the body also do not escape.

If the strength has been impaired by chronic lesions of nutrition, such as rachitis or scrofula, and the foundation thereby laid for spasm of the glottis and for functional or organic disturbances of the lungs, there is likely to be a more severe development of the whooping-cough, and the way is also paved for the diseases I have described as sequelæ.

I have once seen whooping-cough develop in a girl three years old during convalescence from typhoid fever. The attacks were violent, capillary bronchitis and circumscribed pneumonia set in, and the issue was fatal.

Whooping-cough is not infrequently found to be complicated with the acute exanthemata, measles, scarlatina, and small-pox.

The first is the most frequent. This exanthem has been seen alike to precede, to follow, to develop at the same time with the whooping-cough, or in the middle of its course. The course of the exanthemata is not thus essentially modified, although in many cases where they are associated with whooping-cough, the latter is found to undergo a considerable diminution in respect to the intensity and frequency of its attacks ; it may even disappear entirely, in analogy with our experience as to a general outbreak of chronic eruptions of the skin during the subsidence of tussis convulsiva.

In the majority of cases whooping-cough follows upon the subsidence of the acute exanthemata, or is developed in the last stage of them. It seems as if children who were ill with the latter, or who have suffered from them, were specially disposed to be attacked by the former. This may not appear strange if we bethink ourselves that the contagium of these exanthemata likewise enters through the respiratory organs, and that, therefore, in such cases the infectious matter of whooping-cough finds a favorable soil for its reception in the morbidly irritated and hyperæmic mucous membrane.

Measles forms a specially unfavorable complication While this disease is subsiding, or perhaps even after it has run its course, a capillary bronchitis or a pneumonia is very likely to become developed. Now whooping-cough, coming on in a person who is convalescing from such a pulmonary affection, falls upon respiratory organs which have not yet returned to their normal condition. Besides this, too, it is certain that whooping-cough, which has developed in the course of measles, shows a greater tendency than in any other complication to set up a capillary bronchitis or a pneumonia, and in consequence of it to end fatally.

It has been maintained by many that if a febrile disease is associated with whooping-cough, the latter loses somewhat of the intensity and frequency of its seizures, and sometimes disappears altogether. Others declare that they have observed the opposite. Both sides are right ; both conditions have been observed. Especially is it the rule, that on the occurrence of capillary bronchitis or pneumonia the attacks remit, and become less and

less marked. I have had an opportunity of making the following observation with regard to a complication with varioloid.

A girl of six years came under treatment on December 24th, 1870. She had suffered for a short time from tussis convulsiva and bronchitis, with intercurrent albuminuria. On January 11th, aphthous stomatitis. On January 16th, an eruption of varioloid, with diphtheria on the tongue on the following day. On the 23d, decided diminution of the fever. As early as January 19th the attacks of whooping-cough were less in number and violence. On the 26th the number increased again, to sink on the 29th, remaining stationary then for a time at a lower figure, and then entirely disappearing. The child was completely restored to health.

Complications with acute pemphigus are very rare. I have treated in my hospital two such cases with several successive outbreaks of the eruption. They were in two boys of fourteen and fifteen months.

Pathological Anatomy.

The results of pathological anatomy in regard to whooping-cough are rather negative. The constant occurrence, as laid down by Beau and Gendrin, of catarrhal inflammation at the entrance of the larynx and in the supraglottic region has been proved to be erroneous. On the contrary, in the majority of cases, as it appears, a catarrhal inflammation is found in the infraglottic region. In one case I have seen the mucous membrane of the epiglottis and arytenoid cartilages thickened but pale.

Breschet and Autenrieth[1] maintained that the vagus nerve was reddened and inflamed in whooping-cough. The investigations of Krukenberg, Guersent, Constant, and others have proved the contrary.

In every case the presence of a catarrhal inflammation of the air-passages can be established. The mucous membrane of the nose and pharynx is less often attacked. On the other hand, the signs of a catarrhal inflammation from the glottis downwards are present in the majority of cases. Still this is not always the rule. The process often even begins in the large bronchi; farther up, the mucous membrane is pale and not swollen. I have once, in a girl of a year and nine months, seen several shallow ulcera-

[1] Tübinger Blätter, Vol. I., p. 23, 1815.

tions of small circumference in the reddened and swollen mucous membrane of the trachea. The mucous membrane of the bronchi and their ramifications is swollen, either pale or deep red, and more or less covered with tenacious muco-purulent masses. The calibre of the larger air-passages is often filled with these masses, which have acquired a frothy character from the admixture of air. If the bronchioles are affected their calibre is occupied by a thickish, muco-purulent secretion. If the alveoli are also involved in this process, their yellowish-white contents may simulate the presence of tubercles, especially when they lie immediately under the pleura. By puncturing them and pressing out the contents we may guard against this delusion. If a general or circumscribed pneumonia, or atelectasis have occurred, or if the retrograde products of inflammations are present, such as retraction and thickening of the interstitial tissue, and dilatations of the bronchi, then the appearances indicate the features peculiar to these processes, the discussion of which belongs in the section which treats of them. The younger the children the more frequently is stripe-pneumonia found.

If there has been pleurisy, the pleura in the inflamed region is found thickened and dulled, and overlaid with more or less fibrinous exudation. It is not unusual to see numerous ecchymoses on the pleura, especially on the posterior surfaces of the lungs. The pericardium is also sometimes the seat of such effusions of blood.

Emphysema will seldom be found absent in the lungs of a child that has died from whooping-cough and its results. Its seat is generally marginal and peripheral. Less frequently it is found to have extended to the interstitial tissue of the lungs and the cellular tissue of the mediastinum. If the air has thence been diffused into the subcutaneous cellular tissue of the surface of the body, we cannot fail to recognize this in the elastic tumor which is presented, yielding on pressure, and giving rise to a peculiar sound. If pneumothorax has resulted from subpleural emphysema the half of the thorax affected is found to be enlarged, and the intercostal spaces distended. If no adhesions of the surfaces of the pleura already exist, the lungs are found to be pressed inwards, backwards, and upwards by the air that has

escaped. Pleurisy need not necessarily have followed a pneumothorax arising in this way.

In some cases I have found one or more circumscribed apoplexies in the lungs which had been evidenced by no symptom whatever during life.

The subjects of œdema, chronic pneumonia, acute and chronic miliary tuberculosis, endocarditis, pericarditis, meningitis, acute tuberculosis of the pia mater, croupous laryngitis, and œdema of the glottis must be sought for in the sections devoted to them. Letzerich[1] claims to have proved the existence of masses of whooping-cough fungi in the dilated alveoli of the lungs.

Hyperplasia of the tracheal and bronchial glands, often with cheesy degeneration and breaking down in the centre, is to be demonstrated in almost every corpse after tussis convulsiva. It is not unusual for a like condition of the mesenteric and retroperitoneal glands to be associated with it.

If the children have died before the disease has passed into the third stage, in the great majority of bodies the ulcer on the under surface of the tongue will not be absent. I have once seen diphtheria of the tongue.

In the brain, in its membranes, and between these may be found effusions of blood of varying size. Œdema of the brain is almost constant, as is also exudation into the ventricles, and between the membranes of the brain and also those of the spinal cord. If general dropsy has taken place, there are effusions of variable amounts into the pleural cavities, the pericardium, and the peritoneal cavity.

If the patients were not extremely emaciated, the liver is more or less hyperæmic. Isolated peripheral foci of liver-cells laden with fat are rarely absent, especially in young children. Less frequently, although this is the rule in rachitis and chronic tuberculosis, this accumulation of fat is general, the liver is considerably enlarged, and incisions through the yellowish-gray organ leave the blade of the knife covered with fat. The bile in these cases is generally scanty and clear.

I have constantly found the mucous membrane of the œso-

[1] Virchow's Archiv, Vol. 49.

phagus pale, and not swollen. The mucous membrane of the stomach, however, is often the seat of catarrhal inflammation, reddened and swollen. Inflammation of the follicles of the intestine, as is very apt to be found in all diseases of childhood, is not unusual in this disease. Sometimes I have seen considerable follicular ulcerations at the ileo-cæcal valve. It has as yet been impossible to establish the existence of any pathological process involving the kidneys.

Conditions depending on general lesions of nutrition, such as rachitis and scrofula, and on the acute exanthemata, are referred to in the sections treating of them.

Diagnosis.

It is not generally easy to confound whooping-cough with any other disease. The first stage may, it is true, be taken for a simple catarrhal bronchitis, especially if no epidemic of whooping-cough is prevailing, and if it is not known that infection has been rendered possible. Sometimes the violence of the fits of coughing attracts attention. In like manner the third stage may simulate a catarrhal bronchitis, which is subsiding, if it is not known that spasmodic attacks have previously occurred, and if the ulcer which may have been present on the under surface of the tongue has already healed.

The spasmodic stage is sufficiently characterized by the aura which precedes the attack, and which is evidenced by tickling in the throat and a feeling of oppression in the chest; by the convulsive expirations, with the whistling inspirations that take place between them; by the extreme cyanosis, often by hemorrhages; by the expectoration, especially toward the close of the attack, of bronchial secretion, and by the vomiting; by the absence of fever, and by the ulcer of the tongue, which is present in the majority of cases. In the free intervals, the swelling and pale color of the face betray the attacks which have gone before.

The attacks caused by the presence of a foreign body in the larynx or deeper air-passages show the greatest resemblance to those of whooping-cough. The sudden onset of the attacks, without an antecedent catarrhal stage, points to this process.

The laryngoscope gives us information of the retention of the foreign body in the larynx. In such a case, too, the attacks of coughing are of the most violent character. If the body is movable in the trachea or large bronchi, this fact can be determined by auscultation. If it has plugged a smaller bronchial twig, this can be proved by the sudden cessation of the respiratory murmur in the affected region of the lungs.

I have never observed convulsive attacks of coughing in children in consequence of elongation of the uvula. As whooping-cough is rare in adults, there will scarcely be an opportunity given of confounding these two processes. Examination of the mouth, moreover, would save us from this error.

Barthez and Rilliet assert that tracheo-bronchitis in the evening, especially in very young children, and suffocative bronchitis may have a delusive resemblance to whooping-cough. It may happen that the crying of small children with cough makes it appear spasmodic. Further than this, in suffocative bronchitis a considerable cyanosis may be present, and more or less marked obscuration of the sensorium with seizures of cough occurring spasmodically. Both processes, however, are accompanied by fever, which generally has evening exacerbations. The long-drawn whistling inspirations between the spasmodic expirations are wanting. There are no free intervals ; on the contrary, a certain degree of insufficiency of breathing is constantly present. The attacks do not end with vomiting. Physical examination makes known what is going on in the air-passages, and if it is associated with tussis convulsiva, the characteristic seizures of the latter are apt to become milder.

The same authors assert that whooping-cough may be confounded with the stage of resolution of some pneumonias, if their progress is marked by abundant moist râles, or with tuberculosis of the bronchial glands and lungs. It will be difficult for any one to take the stage of resolution of pneumonia for whooping-cough. The cough which occurs with the former is not to be compared with the attacks of the latter. A thorough physical examination of the respiratory organs, which cannot be made too often nor too carefully in whooping-cough, will secure us, moreover, against this error.

Tuberculosis of the bronchial glands cannot be diagnosticated during life, and therefore cannot be confounded with whooping-cough. Just as little are we to assume the existence of hyperplasia and cheesy degeneration of these glands, unless the tracheal or cervical glands are similarly affected. Moreover, spasmodic attacks of cough, as in whooping-cough, have never yet been established as depending on disease of the bronchial glands.

As regards tuberculosis of the lungs, this, as is well known, cannot be determined by physical examination, for the simple reason that miliary tubercles of the lungs stand in no direct relation to the respiratory passages, and therefore can cause no alteration of the respiratory murmur. Even tubercles situated in the mucous membrane of the trachea, bronchi, or their ramifications are not to be diagnosticated by physical examination. Acute miliary tuberculosis of the lungs, unless the pia mater is involved, and its symptoms are overwhelming, may in the vast majority of cases be determined with certainty by the excessive frequency of breathing, if no other reasons for this are present, and if in general other diseases can be completely excluded. Chronic tuberculosis diffused throughout the body may be recognized, when other pathological processes are excluded, by the constantly increasing emaciation. Chronic tuberculosis of the lungs occasions a change in the physical signs belonging thereto only when chronic pneumonia has been secondarily developed. In that case, however, the pathological conditions found on physical examination belong to this process and not to the tuberculosis. The cough in acute tuberculosis of the lungs may be teasing and painful from the secondary bronchial catarrh, but it has no characters common to it with whooping-cough. Neither can the cough of chronic pneumonia after tuberculosis be in any way confounded with whooping-cough. Besides, the processes mentioned are accompanied by more or less active fever, they have no free intervals, but a greater or less degree of respiratory insufficiency is constantly present. In chronic tuberculosis, the liver is usually so swollen from a general excess of fat that its increase in size can be distinctly demonstrated. This swelling of the liver will be found in tussis

convulsiva only when pronounced rachitis is present at the same time.

Spasmodic cough, as it occurs especially in hysterical women, cannot be mistaken for whooping-cough. It likewise runs its course without fever, but shows itself as an almost constant cough, which cannot be repressed. Thus a great number of such attacks of coughing may occur in twenty-four hours, and in fact this is apt to be the case at night to a greater degree than during the day. Spasmodic whistling inspirations between the expirations are wholly wanting. There is no vomiting; expectoration is generally scanty; the disease is not contagious.

The differential diagnosis of the complications of whooping-cough belongs in the sections where the diseases concerned are treated of.

Prognosis.

The prognosis is determined by the age and sex of the patients, by individual predisposition, by the strength, the mode of life, the intensity of the infection, and the complications.

The vast majority of patients recover their health completely after whooping-cough. Still at various times and places this disease has claimed many victims. In the period from 1838 to 1853 (Biermer, p. 578), of the general mortality of the whole population of London 3.4 per cent. were from whooping-cough, and from 1821 to 1835 inclusive, according to Roe, about 3.3 per cent. By others from 3 to 10 per cent. of the fatal cases in childhood have been attributed to whooping-cough. C. West compiles the following table of thirty-five cases ending fatally.

None died under six months.

5	"	between	6	and	12	months.
6	"	"	1	"	2	years.
8	"	"	2	"	3	"
4	"	"	3	"	4	"
6	"	"	4	"	5	"
1	"	"	5	"	6	"
3	"	"	6	"	7	"
1	"	"	7	"	8	"
1	"	"	10	"	11	"

According to Gibb and Friedleben, by far the largest percentage of mortality falls in the first two years of life, and it diminishes rapidly in the third. K. Majer[1] reports that 96 or 97 per cent. of all the fatal cases of tussis convulsiva occur under five years. In the first year the percentage reached 58. Macall estimates the mortality in the first year at 13.25 per cent., after the third year at 2.38 per cent.

In another table (Fifth Report of the Registrar-General) West gives the ratio per cent. of children dying of whooping-cough to the mortality of the whole population, as follows :

Under	1	year.....			5.6	per	cent.
Between	1	and	3	years	10.6	"	
"	3	"	5	"	10.2	"	
"	5	"	10	"	5.0	"	
"	10	"	15	"	0.8	"	

According to Löschner, in 700 cases of whooping-cough the mortality was as 1 to from 27 to 30.

Biermer has brought together the statistics of mortality as given by various authors. The results vary between 2.7 per cent. (Küttlinger), and 15 per cent. (Whitehead), thus averaging 7.6 per cent.

The younger the children the more dangerous is the disease. The largest number of deaths are caused by suffocation as a result of extreme spasm of the glottis. The next most frequent causes are effusions of blood and acute exudations into the nerve-centres and their membranous envelopes. In many cases, especially among the poorer classes, owing to their unfavorable surroundings, children lose their strength and sink under marasmus, sometimes with general dropsy.

The older the children are, especially if they have passed their fifth year, the better are they able to endure the disturbances of their health caused by whooping-cough. They much more rarely succumb to those processes which carry off younger children. Still they, as well as the latter, may be seized with convulsions

[1] On Epidemic Diseases of Children, in Bayer. Journ. für Kinderkrankheiten, 1871, I., p. 223.

in consequence of morbid irritability of the nervous system, and a high degree of passive congestion of the brain. This condition is, with rare exceptions, fatal, particularly if the patients are still in their first year. Profuse and repeated hemorrhages from the nose and mouth may likewise endanger life from progressive marasmus. A fatal case of whooping-cough has but seldom been observed after the age of puberty.

The female sex shows a larger number of fatal cases than the male. Among thirty-five deaths West counts twenty-one girls and fourteen boys. Tables on a large scale, which have been compiled with reference to the mortality in whooping-cough, prove a like proportion with few exceptions. Apart from the fact that many more girls than boys are attacked by whooping-cough, no explanation can be found for the excessive mortality of the former.

Miserable weak individuals suffering from chronic lesions of nutrition, such as rachitis or scrofula, and also the periods of weaning from the breast would, in general, call for an unfavorable prognosis, particularly when the children, from bad circumstances, are obliged to pass their lives in impure air, with deficient clothing and unsuitable food. This is the reason why the poorer classes furnish the largest proportion of fatal cases.

The intensity of the infection is not alike in all epidemics. In some the mortality is very great, especially from the complications, while in others very few of the patients die. If the disease occurs sporadically, the cases are apt to run a milder course than in epidemics. In like manner we may venture on a better prognosis in warm weather than in winter, or the beginning of spring. Biermer quotes from Witsell, of Charleston, the statement that the negro children in the Southern States of the Union present a very considerable mortality. Among other circumstances the conditions of life would here be of essential influence.

The first stage of whooping-cough furnishes a very small contingent of fatal cases. It chiefly concerns only very young and ill-conditioned children, who may succumb to marasmus before the second stage has developed, for the constant cough and the fever that accompanies the catarrhal bronchitis deprive them of

sleep and appetite. In very rare cases sudden death may take place even in the first stage, especially in connection with rachitis or scrofula, from effusions of blood, or transudations into the nerve-centres.

The spasmodic stage furnishes the largest number of fatal cases. The reasons for this have been already spoken of.

In the third stage a child does not readily fall a victim to this disease, provided it runs a simple course. Should death take place it is because the strength is gradually spent, and it cannot bear up against the progressive emaciation.

Taken altogether, in most epidemics the prognosis of uncomplicated whooping-cough is decidedly favorable. Quite a sure indication for a favorable prognosis is to be found in the circumstance of the intervals between the attacks being absolute, for the children are then bright, and retain their appetite.

So soon as whooping-cough is complicated by other diseases the prognosis becomes more unfavorable because of the greater depression of the strength.

When complicated with croupous laryngitis and œdema glottidis, no child has yet been known to survive.

Capillary bronchitis and pneumonia form a very serious addition. From a full half to two-thirds of those attacked may be lost. In the course of these complications the more the symptoms of whooping-cough recede, the more severe does the inflammation of the respiratory organs become, and the more unfavorable should be the prognosis. Increasing apathy, and above all the occurrence of somnolence are very bad signs.

Atelectasis may be recovered from if the readmission of air into the alveoli is rendered possible, and then its injurious effects disappear. If this does not happen, circumscribed pneumonia is developed, and the prognosis becomes unfavorable.

Marginal and peripheral emphysema may disappear, or may continue without special injury. Interstitial, mediastinal, and general emphysema render the prognosis doubtful, while perforation of subpleural air-sacs and pneumothorax may be regarded as always fatal.

On the occurrence of œdema of the lungs death must be looked for with certainty.

The prognosis in chronic pneumonia is generally unfavorable. In rare cases this process is recovered from, with contraction of the connective tissue and the development of dilatations of the bronchi; but the individuals remain feeble through life and predisposed to diseases of the respiratory organs. We generally see this complication run into pulmonary phthisis.

The pleurisy which accompanies these pneumonias has no special importance. If the patients survive, it gets well, with permanent thickening and adhesion of the pleuræ.

Whooping-cough, when complicated with pericarditis, endocarditis, and meningitis, runs a fatal course, as also when complicated with chronic or acute tuberculosis.

Disturbances of digestion, such as catarrh of the stomach and bowels, in themselves involve no danger. In feeble subjects, however, the loss of appetite and the profuse evacuations may become of importance from the loss of strength they cause. We are particularly glad to see vomiting, because it brings the attack to an end. Besides this, it is useful in capillary bronchitis, by preventing the accumulation of the bronchial secretion, which might lead to the development of atelectasis or circumscribed pneumonia. Too frequent vomiting may act injuriously, both by the act and by the rejection of food.

Marked swelling of the liver is a sign of rachitis or chronic tuberculosis, and therefore of unfavorable prognosis.

Complications with the acute exanthemata influence the prognosis unfavorably. This is especially true in regard to measles, the more so as capillary bronchitis and pneumonia are apt to show a special tendency to develop on these two processes as a foundation.

Hemorrhages into the organ of hearing and chronic catarrh of the Eustachian tubes or of the cavity of the tympanum may result in persistent hardness of hearing or deafness.

Treatment.

There are not many articles in the materia medica which have not been advised and employed against this disease.

As we possess no specific which would be capable of putting

an end to whooping-cough, except in a few instances, we must pay more attention to prophylaxis than in many other circumstances. If this disease is of spontaneous origin, we are unable to exercise any influence upon it. But, since it is of a contagious nature, we must exert ourselves to prevent any infection. With this object, it is of the first importance to prevent persons who are suffering from whooping-cough from meeting those who are well. It is therefore necessary to isolate such patients in private life as well as in hospitals, and not to allow well people to enter the rooms in which patients have stayed. We must be the more prudent in these respects if we have to deal with persons who would be likely to have the disease in a severe form. Among such may be classed children in their first year and delicate, feeble persons, especially such as have suffered from chronic disturbances of nutrition or have been exposed to changes in their nutrition owing to weaning ; also children who are suffering or have recently suffered from the acute exanthemata, whose respiratory organs are diseased or have been impaired by disease ; and, finally, such subjects in particular as have been weakened by any pathological process whatever and have not strength enough to offer resistance to a new disease.

If infection is to be feared, children must be carefully watched, so as to avoid any chilling or any cause which would be likely to occasion disorders of digestion. If the family is in a position to remove with its children from the locality in which whooping-cough prevails, this is the surest way of protecting them. Besides, it is certain that epidemic cases are more readily infectious than sporadic ones. No catarrh of the respiratory organs is to be regarded as trivial during the prevalence of whooping-cough.

The prophylactic employment of vaccination, of belladonna, and of preparations of chlorine has proved delusive.

If infection has taken place and the first stage of whooping-cough has set in, there is no remedy known to us by which we can certainly or even probably arrest the further development of the process. An attempt may be made to take the children away from the locality of the disease and seek a healthy atmosphere. Also the results of inhalations of nitrate of silver

and of salts of quinine, and of the internal use of the latter call for further experiments. Otherwise we must confine ourselves to keeping the patients quite warm and in a uniform but pure air, since this, as well as the spasmodic stage, are rendered worse by impurity of the atmosphere, especially by carbonic acid, dust, etc. In mild weather patients may with advantage be allowed to be much in the open air, but should return home as the sun goes down. Let the patients be well fed, in anticipation of the disturbances of nutrition in the spasmodic stage, and see particularly that the digestion is kept in good order. When there is active catarrh, expectorants, such as certain kinds of teas, sulphurated antimony, ammoniacal solutions, ipecac, etc., will not be out of place. If the attacks of coughing are violent and painful, we give belladonna, opium, morphine, or hydrate of chloral, in suitable doses.

During the spasmodic stage we must, in the first place, help the children in the attack. If they lie down, set them up and support their heads. Cold compresses upon the forehead and on the chest are said to moderate the attack. I have never seen any advantage from them.

At the time when the ideas of the humoral pathologists prevailed, emetics and cathartics played the chief part. Fr. Hoffmann, Hufeland, Cullen, and others were enthusiastic for the former, and allowed them to be employed often in excess—some, every second day. Although this method was abandoned long ago, it may still do good service in cases where the air-passages, particularly the bronchioles and alveoli, are overwhelmed with secretion, in consequence of which the respiratory insufficiency is extreme, and we are apprehensive of a secondary pneumonia. Ipecac, sulphate of copper, or subcutaneous injections of apomorphine are here the most suitable. Tartar emetic would excite diarrhœa too readily. Cathartics are now thought to be required only in passive congestion of the brain and in delayed digestion.

During the attacks nothing can be done, for at this time the children cannot swallow. Niemeyer's proposition of prevailing on children to repress the attack when it is coming on only results in its breaking out the more violently. If the children

become stupefied during the attack, we shake the body, sprinkle cold water upon the face and chest, rub the body, apply sinapisms, and place the patient in a warm bath, or even, without this, employ cold affusions to the head and neck. In strong, full-blooded children, if an extreme degree of cyanosis remains, in spite of everything, or if there is a lethargic condition, while the veins of the neck are fully distended, we may determine in very rare cases to apply leeches to the head. General convulsive seizures demand the same treatment. During the attack nothing can be done for hemorrhages from the nose and mouth ; afterwards they require the ordinary remedies. Zaniboni advises especially ergot, others the solution of chloride of iron, etc.

As the mucous membrane of the air-passages is the real seat of the disease, we are desirous of acting directly upon it. It is therefore our first duty to remove the patients to the purest possible air. If the place of residence cannot be changed, which would be the most desirable thing, and in regard to which especially a stay at the sea-shore is recommended by the older and also by modern authors, then during warm weather patients must be much in the open air. If this is prevented by the state of the atmosphere, they should not stay in their bedrooms during the day. The bedrooms must be aired in the daytime, and the patients must occupy other rooms, which, if possible, must be alternately aired during the day.

Cold and stimulating drinks must be avoided, because they irritate the larynx, and may bring on attacks of coughing.

Ebenezer Watson[1] endeavored to cure the whooping-cough, during the free intervals, by touching the larynx with a solution of nitrate of silver. But the remedy was without result, because it could act on the supra-glottic region only. In spite of this, Pearce maintains, in the Lancet of April 11, 1857, that he has proved its efficacy in seventy-five cases—thirty-two boys and forty-three girls. At the same time he administered nitric acid internally, according to Gibb's method.

In the year 1866, in six children over four years old and in two adults, Rehn employed inhalations of a solution of nitrate

[1] Edinburgh Month. Journ. Dec. 1849.

of silver at the height of the second stage. At the best, after the third inhalation there occurred a remission of the symptoms, and after from eight to ten inhalations there remained only a simple bronchial catarrh. In the year 1867 I allowed a girl of two years and three months to inhale a weak solution of nitrate of silver twice daily. After nine days a decided remission occurred, with recovery a week later.

Brünniche[1] advises the employment of compressed air.

In the year 1860 Roger advised the inhalation of chloroform. The proceeding seems, however, to have had doubtful results. West advises great prudence in the use of it.

Clar recommended inhalations of camphor or oil of turpentine. I have several times employed the latter in my private practice, and believe that I have noticed good results from it.

Kjellberg of Stockholm has advised inhalations of benzine, and Fieber employed with advantage inhalations of a solution of extract of hyoscyamus. Monti expresses himself decidedly against the inhalations of gazeol, which consists principally of ammonia and benzine. [See volume IV. of this Cyclopædia, p. 420, foot-note.—*Tr.*]

In the year 1862 Hauke published the results of experiments on the inhalation of various gases which he tried in St. Ann's Hospital at Vienna. Oxygen and illuminating gas never induced attacks of cough; on the contrary, after a short time patients were glad to inhale them. Carbonic acid and ammonia made the attacks worse; they were frequently brought on by hydrogen and nitrogen. He advises increasing the oxygen of the atmosphere in which patients stay, either chemically or by setting out certain plants.

Lochner[2] sent forty-three children into the purifying chambers of a gas-works so that they could breathe the exhalations of the lime used in purifying the illuminating gas. Twenty-three boys and twenty girls were subjected to this treatment, eleven of them being less than a year old. The results are said to have been for the most part favorable.

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[1] Bibliothek for Laeger, Jan. 1867.

[2] Bayrisch. ärztl. Intelligenzblatt. I. 1865.

Commenge[1] sent eighty-eight children to the gas-works of St. Maude. Of these fifty-four were cured, twenty-four improved, while in ten no result could be determined. Each sitting lasted about two hours, and each successful case required on an average eleven or twelve sittings.

Maingault,[2] Blache, Bergeron, Barthez, Roger, and Bouchut have declared against making this use of the gas-works, partly from the want of results, and partly because they regard these inhalations as hazardous.

J. Grantham has recommended the vapor of the water of ammonia in boiling water. The spasmodic stage is said to have come to an end after three or four days.

In the years 1864 and 1865 I directed the employment of inhalations of simple warm water sometimes, or of solutions of chloride of sodium alone, or in combination with wine of opium. I gained yet more from inhalations of a solution of tannin with wine of opium. Still the improvement consisted less in the shortening of the whole disease than in an alleviation of the attacks, and more in respect to their intensity than to their number. The details can be referred to in the Journal für Kinderkrankheiten. Vol. XLVI. 1866. p. 6.

In the winter of 1868 I directed the inhalation of the infusion of ergot for a boy of a year and a half. On the fourteenth day after beginning to use this remedy, there occurred a decided amelioration of the attacks, and thirteen days later the child was cured.

In recent cases of whooping-cough, Lesser[3] has directed the inhalation of petroleum with benefit.

Letzerich,[4] acting upon his theory that whooping-cough is due to the reception and subsequent development of fungus spores, directs the inhalation of muriate of quinine in the form of a powder in combination with bicarbonate of soda and gum-arabic. According to his report of three cases treated in this way, the convulsive attacks are said to diminish after from eight to ten days, and even the third stage to come to an end soon.

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[1] Bullet. de l'académ. XXX. p. 9. Oct. 15, 1864.

[2] Gaz. des Hôpit. 121. 1864.

[3] Allgem. med. Centralzeitung. 49. 1873.

[4] Virchow's Archiv. Vol. 57.

In the year 1869 I treated a girl of three years and a half by inhalation of a solution of sulphate of quinine. Unfortunately, the parents took the child out of the hospital before any result could have been looked for.

Burchardt[1] recommended the steam from solutions of carbolic acid of from one and a half to two per cent., and Wilde,[2] inhalations of a mixture of oil of turpentine, chloroform, and ether.

Most of the articles used by inhalation have relieved the intensity and frequency of the attacks, but have not proved capable of shortening the course of the disease as a whole. The number of cases in which these methods have been used is still too small to base a positive opinion on them. At all events, the results decidedly urge us to further experiment.

Alkalies, especially carbonate of potassa and soda-water, have been very much used to loosen the secretion in the air-passages, and to the present day they are judiciously employed.

A very great variety of expectorants play an important part, from muriate of ammonia, sulphurated antimony, ipecac, and tartar emetic, to sulphur, which last has been advised by Kopp as a very efficient remedy against whooping-cough. A cure cannot be expected from any one of these medicaments, still they may give some relief. Whoever feels an interest in these things historically, must peruse the treatises on whooping-cough of Paldamus, Biermer, Barthez and Rilliet, and others.

In the majority of cases those remedies have been principally made use of which are directed to calming the nervous system and to lowering the reflex excitability, especially in the territory of the vagus nerve. Among the foremost of these, warm baths and sea baths have been recommended. Moreover, as we get a sedative effect upon the morbid irritability of the nervous system by invigorating the body, the advice is certainly sound to nourish children who are affected with whooping-cough as judiciously and heartily as possible, the more if much of the food they take is lost again through frequent vomiting. Some authorities, such as Oppolzer and others, have pronounced against the employ-

[1] Deutsche Klinik. 41. 1874.

[2] Deutsches Archiv für klin. Medicin. Vol. XIV. Part 2.

ment of the medicaments in question, as they see in whooping-cough only the progress of a catarrhal bronchitis, and dread lest the discharge of the sputa may be prevented by these remedies.

From old times up to the present, in preference to all other articles, belladonna and atropine have found warm admirers. Among more recent authors they have been very much praised; by Trousseau especially. Different ones had special preferences for different forms of this medicament and recognized in these its special efficacy. Trousseau and others limit themselves to moderate doses, while by the majority the remedy is given until slight symptoms of poisoning appear. Formerly I used the remedy in many cases and certainly with benefit, but do not regard the symptoms of poisoning as unimportant, and besides, I have sometimes seen in the course of a longer use of it, especially in young children, a depression of the strength which threatened danger, while from moderate doses I have gained no advantage. For these reasons, I have not employed the remedy for years.

In the same rank comes hydrocyanic acid with its combinations, especially the cyanide of zinc. West properly exhorts us to great caution in employing hydrocyanic acid. Opium, morphine, narcein, according to Laborde[1] and Debout, conium, chloroform, hyoscyamus, cicuta, nicotine, veratrine (Norwood of Charleston), and hydrate of chloral may be of use in diminishing the violence of the attacks of coughing. The last has been especially recommended by Murchison, Bellantyne, Richardson, and Lorey. Lorey witnessed not merely an amelioration of the attacks, but even a shortening of the whole disease from its use. Steiner, on the contrary, denies that this article possesses the action referred to. Armstrong claims to have lost but seventeen out of three hundred and fifty-seven children treated with cicuta.

With regard to veratrine, I have made the following experiment. A boy of four years was received into the children's hospital with tussis convulsiva and atelectasis in the form of strips posteriorly on both sides. During the first week chinoidine was given internally, then I directed inhalation of this remedy. During the next week circumscribed pneumonia was developed, the whooping-cough attacks became more and more moderate, and at the height of the pneumonia seemed to have disappeared

[1] Gaz. des Hôpit. 38. 1865.

entirely. The fever was treated successfully with tincture of veratrum, with a slight addition of laudanum. After about two weeks, when this had sunk to a minimum, characteristic whooping-cough attacks again showed themselves, which were not marked, however, either in violence or frequency. Unfortunately the child was taken away by his family before complete recovery.

Adams has recommended the use of the peroxide of hydrogen, Bednar for some cases the extract of pulsatilla. Gibb and Harley have been satisfied with the action of the bromide of ammonium ; others, as Fallami and Sidney Ringer, with that of the bromide of potassium. Henoch has seen no benefit from the latter.

Wachtl has introduced the use of cochineal. This inefficacious article was administered for a long time with equal zeal and confidence.

Valerianate of zinc, also sulphur, alone or with belladonna (C. S. Shelton), nitrate of silver internally (Berger), musk, castor, valerian, camphor, assafœtida, water of ammonia, etc., help to increase the list of articles that are of little service under these circumstances. For young children Bednar praises a combination of tannin and benzoic acid.

Fluegel advises inunctions of mercurial ointment with carbolic acid, at the same time with the internal employment of calomel.

The use of nitric acid was recommended by some, especially Gibb ; of muriatic acid by others.

Griepenkerl claims to have seen good results from the internal use of ergot ; Maisch and Thomas D. Davis,[1] from the extract of chestnut. Since then the action of this article has been declared by others to be wholly uncertain. Mignot has advised the use of a decoction of sweet almond shells.

Peruvian bark had already been employed by Mellin, Rosen, Cullen, Stoll, Hufeland, and others, in tussis convulsiva, but especially for the purpose of sustaining the strength in the third stage of the disease. To Binz belongs the credit of having introduced the use of quinine against this disease. He relied upon the theory that whooping-cough is dependent upon the reception and further development of some forms of fungi. Since

[1] Phila. Med. Times. 61. 1872.

quinine in experiments acts injuriously upon the spores of fungi, and besides seems to prevent the escape of the white blood-corpuscles from the vessels, he was persuaded in advance of the favorable action of this remedy. At first he made use of comparatively small doses repeatedly, subsequently, with better effect, he gave larger doses more frequently. He advises the use of the muriate of quinine as the more soluble. Still the sulphate appears to be taken up by the system just as easily. In the Jahrbuch für Kinderheilkunde N.S.I. p. 235, he reports the case of a girl of eight months who had been ill for two weeks, and that of a girl of seven years who had been attacked with whooping-cough three weeks before. The former was completely cured in forty, the latter in thirty-eight days. In the same journal, IV., p. 103, he insists that we can attain good and prompt results only when large doses of quinine are given before the disease has reached its height. Breidenbach (1869) has reported that in an extensive and severe epidemic he has seen the best results from this treatment. In the Jahrbuch für Kinderheilkunde, N.S. IV. p. 227, I have related two cases in which large doses of quinine rendered good service.

A girl of five years came under treatment at the beginning of the spasmodic stage. She received twice a day, morning and evening, nine grains of sulphate of quinine. On the eleventh day after the beginning of treatment there was not a trace of the spasmodic attacks present. Altogether ninety grains of quinine were used. On the fourth day of treatment the number of attacks had already sunk from thirty to eleven.

The second case was that of a girl of three years who was received into the hospital with otorrhœa of the left side, tussis convulsiva, and diffuse bronchitis. Cold compresses, cooling baths. As she is not willing to take quinine, it is administered by enemata, as has been advised in the case of many of the remedies proposed. She received in this way, for two days successively, daily altogether fifteen grains of sulphate of quinine. On the third day marked remission of the attacks, while, on the other hand, there was an extension of the circumscribed pneumonia of the left side. Solution of sulphate of quinine was given. On the tenth day of the pneumonia, with the abatement of this process, the whooping-cough seizures again set in more severely; fifteen grains of sulphate of quinine were given in solution daily. On the seventh day after beginning the use of this remedy, the number of seizures suddenly sank from forty-one to six, afterwards increased again to eight, and then remained stationary for several days at an average of from two to four attacks, disappearing soon after altogether. From the

time of the subsidence of the pneumonia one hundred and seventy grains were used.

I add the following also: A boy of eight years received nine grains of sulphate of quinine twice daily. Violent seizures with strabismus. From having as many as from twenty-two to twenty-nine seizures, he had on the ninth day of the treatment only two, and those but slight. Similar moderate symptoms continuing, the disease, after some nine days more, passed into the stage of decline.

A boy of five years was received with multiple caries. Subsequently tussis convulsiva was developed. A solution of sulphate of quinine yielded no prompt result. Nine grains of sulphate of quinine, given twice daily, arrested the attacks in five days.

In a girl of six years the spasmodic seizures disappeared in just short of four weeks, after seventeen days' use of sulphate of quinine (nine grains twice daily).

I could add to these examples by many, from hospital and private practice.

It follows then that large doses of quinine are of decided influence in moderating and shortening the spasmodic stage. Whether this action is to be based upon the still doubtful theory of fungus spores, I leave undecided. At all events, it follows from the experiments of Schlakow and Eulenburg [1] that quinine reduces the reflex irritability of the nerves. Besides this, quinine fortifies the strength of the body, interferes scarcely at all with the stomach, and is readily taken by most children. It may also be given in enemata.

Up to the present time the number of cases of whooping-cough, treated with quinine, is far from being large enough to found a positive opinion upon. The results already attained, however, allow us to say as much as this, that they urgently call for further experiments with this remedy, both in the first stage and in the second. Still it should be borne in mind, in order that we may go to work without bias, that there are certain cases of whooping-cough, especially complicated ones, in which no effect can be determined even from quinine.

Among external remedies, we need only speak of transient irritants, such as sinapisms, embrocations of spirit of mustard or oil of turpentine, and rubbing with warm or cold water. More violent and lasting agencies, such as the antimonial oint-

[1] Archiv von Reichert und Dubois-Reymond. 1865.

ment, vesicants, croton oil, etc., or even the employment of the hot iron, or a seton in the neck (Mercatus), are contra-indicated, because they unnecessarily reduce the strength of the patients through pain and loss of nutritive fluids. In simple whooping-cough there is no occasion for taking blood.

In the third stage we must take pains to sustain the strength of the patient, and to moderate the cough and any profuse expectoration there may be. Above all, patients must be well nursed; they should take lukewarm baths, to which, especially, malt may be added, and they must be guarded against taking cold. The infectious character of the disease belongs only to the first, particularly the spasmodic stage. In the third stage, therefore, patients may again associate with healthy persons.

As to medicaments, the preparations of iron, weak iron-water, Peruvian bark, or sulphate of quinine, may here be given with advantage.

For the cough, narcotic and expectorant remedies of various kinds may be administered. When the expectoration has been too profuse, tannin (Geigel) and alum (Bodanus) have been used with benefit.

The complications of whooping-cough, in the course of which especially it is apt to be more or less mitigated, are to be treated according to the rules laid down for them, and we then have generally to concern ourselves only secondarily about the whooping-cough itself. In regard to the pneumonias which accompany it, I will only remark that blood-letting is not allowable; on the contrary, its action is positively injurious. We must confine ourselves to the employment of cold, quinine, veratrine, digitalis; at a later stage expectorants, and, if necessary, emetics may be used.

DISEASES

OF THE

LIPS AND CAVITY OF THE MOUTH.

VOGEL.

DISEASES OF

THE LIPS AND CAVITY OF THE MOUTH.

A.—The Lips.

In a manual of special pathology, the only pathological alterations of the lips which can be considered are those which form part of the evidences of an internal malady. Most of these diseases belong to the domain of surgery, or to that of dermatology; others, again, appear only as complications of some disease of the buccal cavity; and these, to avoid repetition, will be treated of in connection with those affections. The only special diseases of the lips, then, which remain for our consideration are *herpes labialis* and *hypertrophy of the lips*.

1. Herpes Labialis.

Bateman, Praktische Darstellung der Hautkrankheiten. Leipzig. 1835. p. 271.—*Constatt*, Spec. Pathologie und Therapie. 1845. Bd. 4. p. 1072.—*Rayer*, Traité des maladies de la peau. II. edit. Vol. I. p. 339.—*Cazenave* and *Schedel*, Abrégé pratique des maladies de la peau. Paris. 1847. p. 167.—*G. Simon*, Hautkrankheiten. Berlin. 1851. p. 185.—*Hebra*, Virchow's Handbuch der spec. Pathologie u. Therap. Bd. III. p. 250.—*Biesiadecki*, Sitzungsbericht der Wien. Acad. Bd. 57. p. 436.—*Haight*, Blasenbildung. Ebd. Bd. 57. p. 631.—*Volkmann, R.*, Einige Fälle von Cheilitis glandularis. Virchow's Archiv. Bd. 50. p. 142.

Since the time of Willan, the word herpes has been used to designate groups of small, clear vesicles, which have a determinate cyclical, acute course, becoming turbid in a short time, then drying into flat scabs, and finally healing without any trace of cicatrization.

The following characteristics of herpes in general may also be mentioned: the appearance of the eruption on various, and

still tolerably constant portions of the body; similarity in the vesicles of any given group, although different groups may show different stages of development; and, finally, relapses at definite times.

These latter symptoms are especially applicable to *herpes zoster* and *herpes iris*, and hardly apply to *herpes labialis*. There is never any tendency in this exanthem to spread or creep outwards from the periphery, and therefore the name herpes (ὁ ἕρπης, creeping ulcer, from ἕρπω, I creep), is very unfortunately chosen.

Etiology.

The causes of herpes labialis can, for the most part, be demonstrated with precision, and consist either in local irritations or in general febrile affections.

There are individuals so predisposed to it, that herpes will be induced by mere contact of their lips with salt, pepper, or other spices—aye, even with apparently indifferent substances, such as the saliva of quite healthy persons. The articles of food that produce this effect are chiefly those animal substances which are preserved by salting, such as herrings, caviar, cheese, and the like.

Though the action of these substances in contact with the lips may be regarded as a species of slight cauterization, it is, indeed, difficult to explain the injurious influence of the fresh saliva of a healthy person.

Herpes is frequently contracted by mere kissing, and especially by promiscuous drinking out of the same cup; and it is by no means necessary that the giver of the kiss, or the person who hands one the draught should himself be affected with herpes. This latter fact, the risk from common drinking out of the same vessel, played a rôle in theological controversy at the time of the war of the Hussites, the question being as to whether the holy communion should be served to the people in the double form of bread and wine, or in that of bread alone.

The relation of herpes labialis to febrile processes cannot be explained by the simple rise in temperature; for its frequency is

not proportionate to the height and duration of the fever, while it is almost always absent in certain eminently febrile processes, such as abdominal and exanthematous typhus and recurrent fever. With the exception of the last-named diseases, it appears in almost all febrile affections; especially in the inflammatory ones, and with greatest regularity in pneumonia and influenza. It is also a very common attendant on severe coryza, and its presence here may be explained in two ways—both by the attendant fever, and by the continuous contact of the secretions with the upper lip, and its continual rubbing with the pocket-handkerchief. The latter cause explains the frequent appearance of herpes in the middle portion of the upper lip. The above-mentioned rare appearance of herpes labialis in typhus and recurrent fever, may, in doubtful cases, be utilized as a means of differential diagnosis between these affections and other similar fevers. Although it cannot be affirmed that herpes labialis is never observed in typhoid fever, yet it is so excessively rare (once in several hundred cases), that its occurrence might very well overthrow a doubtful diagnosis of this disease.

Pathological Anatomy.

If a fresh herpetic vesicle is superficially punctured, there escapes a clear, colorless serum, in which, under the microscope, only very isolated cells, pus-corpuscles, can be detected. As far as I know, no examinations of fresh herpetic vesicles have been made upon the dead body; in fact, it would be hardly possible, inasmuch as such trifling amounts of fluid would dry up within a few hours after death. On the other hand, Biesiadecki[1] has carefully studied the commencement of the production of vesicles, in an experimental manner, by rubbing the ears of the rabbit with croton oil; and he has arrived at the following results: The papillæ of the skin become broader and longer, and show new cell-formation. The mucous layer, or rete mucosum, is penetrated by numerous spindle-shaped cells, which are sometimes still half-imbedded in the papillæ themselves; their

[1] Sitzungsbericht der k. Academie. Wien. 1867.

remaining portion pressing apart the cells of the mucous layer, and capable of being traced as far as the cuticular layer.

The cellular infiltration of the papillæ, and the above-described alteration of the mucous layer, occasion the first visible swelling, which, after a few hours, by the accumulation of serum, enlarges into a prominent vesicle, raising the epidermis. In a few hours more its contents become turbid from the increase of cells. Dried herpetic vesicles may be examined on the dead body tolerably often, especially after pneumonic and puerperal disease. During the process of hardening and of preparation, the crusts usually fall off, and we can then, on making a vertical section, see the loss of epidermis, and a deeper encroachment of the rete mucosum into the tissue of the papillæ, which are themselves enlarged in length and breadth. The cutis, on the contrary, appears perfectly intact. The anatomical alterations of the skin are, therefore, upon the whole, quite insignificant, and of short duration.

Symptoms.

Herpes labialis almost always appears only on certain small portions of the lips; and if it breaks out at a number of places, there are always sound portions of skin between the individual groups of vesicles.

The impending eruption is to be recognized, first by a slight hardening of the skin, the epidermis upon which very soon becomes elevated in small rounded raspberry-like masses, which grow tense and translucent. The favorite seat of herpes is the border of the lips, though it also appears further away upon the lips, as well as upon the alæ of the nose, the chin, the cheeks, the eyelids, and the external ear, so much so, that Hebra proposes to call this eruption *herpes facialis*, instead of *herpes labialis*.

No one would be apt to apply the adjective *labialis* to a herpes upon the eyelids; but, as in the great majority of cases, the eruption occurs upon the lips only, the term *herpes labialis* appears to be the most distinctive appellation.

The subjective symptoms, which exist only at the very com-

mencement, consist in tension and pain in the affected parts. The pain is increased by movement; and, on this account, labial consonants are not pronounced distinctly. These little annoyances hardly last twenty-four hours, and are, in fact, complained of only by otherwise healthy subjects, with no further febrile manifestations.

By the second, or, at longest, by the third day, the formation of the scab is completed; soreness, tension, and pain are gone. The cutis beneath the scab, however, has not yet returned to its normal condition, but still secretes a few drops of serum, so that a scab is thrown off two or three times successively, until finally the last-formed scab remains, and the reproduction of the epidermis is accomplished. Herpes labialis never heals so quickly as a simple superficial wound of the skin. After the first falling of the scab, there is a moist, slightly sanguinolent surface, the secretion from which dries into a fresh scab within a few hours. After the discharge of the last scab, the epidermis is still rather thin, and the cutis still somewhat hyperæmic—conditions which give the recent cicatrix a reddish color. In the course of a few days the normal color of the skin is restored, and the seat of the disease is no longer in anywise to be detected. Occasionally the epidermis first reproduced is again thrown off in small scales.

Herpes labialis may exceptionally make its appearance in the mucous membrane of the mouth, where it naturally presents a different aspect. The epithelium of the mucous membrane macerates far too quickly to admit of the formation of a vesicle. The first thing observed in these cases are small excoriations, isolated or in groups, the yellow floors of which make them stand out in marked contrast, and are sharply defined from the rest of the red and swollen membrane. The pain on movement and on contact, especially the contact of spiced food, tobacco-smoke, etc., is tolerably intense; but it does not continue longer than for two or three days.

Herpes of the mouth is differentiated from aphthæ and ulcerative stomatitis by its short duration, its slight extent, and the moderate infiltration of the neighboring tissues. Individuals with blonde complexions are more disposed to herpes than are

brunettes, and young people more than old ones. It is less frequent, however, in nursing infants than in older children.

Treatment.

It is self-evident that special treatment is unnecessary in a complaint that always terminates in complete recovery in a few days. Its course cannot be shortened, nor the formation of epidermis be hastened by any treatment, whether with lead, zinc, or nitrate of silver. The annoying tension, and the premature contraction of the scabs, by which slightly bleeding fissures are produced, may be avoided by frequent applications of glycerine or fresh cream.

2. Hypertrophy of the Lips.

Bruns, Handbuch der Chirurgie. II. 1. p. 599. Atlas II. Taf. XI. Fig. 1–4.—*Volkmann*, Henle und Pfeufers Ztschr. N. F. VIII. 1857.—*Billroth*, Beiträge zur path. Histologie. 1858. p. 215.—*Jacobi*, Journ. f. Kinderkrkhtn. XXIV. p. 44. Jan. Feb. 1860.—*Förster*, Handb. der spec. pathol. Anatomie. II. Aufl. 1863. p. 9.

It is not within our province here to consider either benign or malignant tumors of this region, congenital or acquired alterations of the configuration of the lips and their connections, such as hare-lip, micro- and macrostoma, distortions and eversions of the lips resulting from previous destructions, etc., because they all belong to the domain of surgery. But we may not disregard the subject of simple hypertrophy of the lips, especially of the upper lip, on account of its connection with scrofula.

Definition.

We understand hereby a painless, uniform, chronic, exceedingly gradual swelling of the upper lip, especially of its vermilion border, so that in the most extreme cases complete eversion of the same takes place.

Etiology.

Chronic swelling of the upper lip occurs almost exclusively in scrofulous children.

Notwithstanding the many attacks to which the idea of scrofula has been subjected, this fact remains: that a series of chronic inflammatory affections of the skin and mucous membranes, of the organs of sight, hearing, and smell, of the lymphatic glands, bones, and articulations alternate with each other, and that they are distinguished for their obstinacy. It is furthermore worthy of notice that these local processes are very little, if at all, ameliorated by local treatment; while, even though they have been of several years' duration, they rapidly disappear upon the establishment of a similar inflammatory or ulcerative process in any other organ. Chronic hypertrophy of the upper lip belongs to the most frequent manifestations of this nature, and is produced anatomically by simple hypertrophy of those well-known mucous glands, several millimetres in breadth, which envelop the lips like an edging.

The microscopic examination of such hypertrophic glands does not exhibit any trace of alteration of tissue, but only an increase in the volume of normal tissue. These glands may either swell spontaneously, or they may be affected secondarily by inflammatory processes in their vicinity, *i. e.*, eczema of the nostrils and adjacent tissues, ozæna, or affections of the mucous membrane of the mouth.

Symptoms.

Persons thus affected show such a conspicuous disfigurement of the mouth that the condition is recognized at a glance. The lower lip is usually less swollen than the upper, because the latter is more directly subject to the irritations emanating from the nose, and, in cases of chronic catarrh, frequent blowing and wiping of the nose and of the upper lip constitute an additional source of mechanical injury.

If the lip is taken between the thumb and index finger and

compared with a healthy lip, it is easy to satisfy one's self that it is considerably thickened. This examination is altogether painless, except when there are rhagades, or ulcerated fissures in the folds of the lips, which increase the swelling of the surrounding parts, and which, as a matter of course, are very sensitive under pressure. Hypertrophy of the lips seldom exists alone, but is usually complicated with eczema of the nostrils and swelling of the alæ of the nose, with the above-mentioned rhagades, or, further, with blepharitis, phlyctenular conjunctivitis, keratitis, with otorrhœa and widespread eczema. It is a noticeable fact that other dyscrasias, such as syphilis and lymphatic leukæmia, whose favorite seat is also in the glandular apparatus, always spare the lips. In scrofula, on the contrary, the lips remain hypertrophied for years, or even for the entire lifetime, even when the neighboring diseases of the nose and mouth, which undoubtedly caused them, have long since disappeared.

The diagnosis is of special significance, because chronic swelling of the upper lip, of many years' standing, makes its appearance only in families of more or less pronounced tuberculous tendency, so that we have here a valuable indication of the deterioration of the individuals affected and their relatives.

This affection is not to be confounded with congenital double-lip—*labium duplex*. In the latter, a longitudinal ridge, hardly perceptible in the nursling, gradually develops itself under the vermilion border of the upper lip—seldom of the lower—this ridge being separated from the true lip by a furrow, and finally constituting a duplicature of the lip.

This eversion of the mucous membrane consists, according to Förster, of hypertrophied cell-tissue and enlarged lip-glands. In another case, Billroth found in this protrusion a cavernous framework, the meshes of which, however, contained lymph instead of blood, showing a structure analogous to that of macroglossia. The frænum of the lip usually withstands the eversion better than the remaining mucous membrane, so that the lip retains its normal form in the centre and the protrusion appears distinctly divided into halves. Sometimes this double lip is not at all remarked as long as the mouth remains quiet and closed ; but as

soon as it is opened and extended in laughter, the deformity appears.

Other tumefactions of the upper lip, the result of pachydermia or of acute diseases of the mouth and the incisor teeth, cannot be confounded with scrofulous hypertrophy, partly because of their different aspect, partly because their course is by no means chronic.

Treatment.

Topical treatment with preparations of iodine, bromine or mercury, is altogether ineffectual. The internal administration of these metals usually exerts a debilitating, injurious effect upon the scrofulous organism. The only treatment from which, thus far, I believe I have seen any benefit is bathing with the mother-lye of the Kreuznach or Reichenhaller springs, kept up for many months.

As a matter of course, cod-liver oil is at the same time to be given in small doses, say a teaspoonful daily, and to be omitted again as soon as it interferes with the appetite. The use of the oil, continued for a year, materially diminishes the size of a scrofulous upper lip.

Surgical interference, comprising the excision of large wedge-shaped pieces, can be thought of only after every source of local irritation has long disappeared, and the treatment just recommended has proved unsuccessful.

B.—The Tongue.

Kölliker, Handbuch der Gewebelehre.—*Frey*, Handbuch der Histologie und Histochemie. 2 Aufl. p. 516.—*Henle*, Handbuch der Anatomie. Bd. II. p. 118.—*Loven* of Stockholm, Arch. f. mikroskop. Anatomie IV. I. 1868. p. 96.—*Schwalbe*, Die Geschmacksorgane der Säugethiere u. des Menschen. Arch. f. Mikroskop. Anatomie IV. 2. 1868. pp. 154—187.—*Schiff*, Neue Untersuchungen über die Geschmacksnerven. Moleschott's Untersuchg. IX. 1867. p. 406.

1. Anatomico-Histological Observations.

When the mouth is closed, the dorsum of the tongue, with the exception of a small median, longitudinal furrow, everywhere

touches the palate. It extends downwards, however, far beyond the soft palate, narrowing the pharynx, by its proximity to the first three cervical vertebræ, to a narrow, transverse fissure, and terminating below at the hyoid bone. Where the dorsum of the tongue and the epiglottis unite, there is a median fold of mucous membrane, the frenulum epiglottidis, which is especially prominent when the tongue is protruded. The tongue increases in thickness from its tip to the isthmus of the fauces, and decreases materially from the isthmus to the hyoid bone.

The following pairs of muscles are found in and attached to the tongue below:

1. The styloglossus.
2. The hyoglossus.
3. The chondroglossus.
4. The genioglossus.
5. The lingualis.
6. The transversus linguæ.

The greater mass of the tongue is composed of the genioglossus and transversus linguæ muscles. In the centre of the tongue there is found the septum linguæ medianum, a dense, yellowish-white vertical plate, which extends lengthwise through the entire organ, and which is composed solely of connective tissue. In the compact portion of the tongue the continuations of all the above-named muscles form a thick plexus, the fibres of which cross each other at right angles.

The genioglossus muscles draw the tongue forward, effecting its protrusion; the hyoglossus muscles draw it back again. Acting together, both these sets of muscles flatten the tongue, the genioglossi drawing it forwards, the hyoglossi drawing it backwards, and the two forces maintaining their equilibrium.

As the terminal portions of the muscles of the tongue extend until close beneath the mucous membrane, and are able independently to contract upwards, downwards, or to either side, the tongue acquires the power of bending upwards, downwards, and sideways, of arching itself, and of forming a gutter.

The vessels of the tongue are disproportionately large and numerous. The arteria lingualis, the second branch of the

external carotid, arises at the level of the larger horn of the hyoid bone, and penetrates into the substance of the tongue between the hyoglossus and the constrictor pharyngis medius muscles.

Its branches are:

1. The ramus hyoideus;
2. The arteria dorsalis linguæ;
3. The arteria sublingualis; and
4. The arteria profunda linguæ, or ranine artery.

This latter is the largest of all, and penetrates the tongue close to the lingual frænum, and, from its superficial position, is liable to be wounded in operations on the floor of the mouth, or even in careless division of the frænum.

The movements of the tongue are dependent on the hypoglossal nerve; taste and sensation depend on the trigeminus and the glossopharyngeus. The ramus lingualis, from the third division of the trigeminus, together with the chorda tympani, supplies the anterior portion of the dorsum of the tongue, while the lingual branch of the glossopharyngeus supplies its posterior portion.

The mucous membrane of the tongue is of especial interest in a clinical point of view. While the mucous membrane of its under surface appears smooth and covered with simple pavement epithelium, on its tip and upon its upper surface certain peculiar, partly filiform, partly wart-like appendages, the papillæ, are distinguishable.

We distinguish:

1. The thread-like papillæ filiformes;

2. The mushroom-shaped or pin-head papillæ fungiformes or clavatæ; and,

3. The walled papillæ vallatæ.

The *papillæ filiformes* are the most numerous, and extend over the entire dorsum of the tongue in close array. They give the tongue its characteristic velvety aspect, which, however, it loses towards the root of the organ, where the filiform papillæ are shorter and less abundant.

The *papillæ fungiformes*, at the distance of from 1 to 2 millimetres (.039 to .078 of an inch) apart, protrude everywhere, as

little red buttons, between the more yellowish papillæ filiformes.

But they are more thickly distributed at the tip of the tongue, so that they sometimes entirely supplant the filiform papillæ.

The *papillæ vallatæ* form the boundary between the dorsum and the root of the tongue. They extend, to the number of from seven to ten on each side, in a line directed obliquely backwards, meet together in the foramen cæcum, and thus present the form of a broad V. They consist of prominences, varying from the size of a millet-seed to that of a hemp-seed, which, however, project very little or not at all above the level of the surrounding mucous membrane, but lie in depressions, and are consequently surrounded with a wall. With a fine probe this wall can be sounded, and the instrument can be carried entirely around the papillæ. Such a prominence also frequently appears in the foramen cæcum itself, so that in such cases it is more proper to speak of it as an elevation than as a depression. If they are somewhat more elevated, they are transformed into veritable warts, which, where syphilis is suspected, frequently leads to false conclusions, especially among syphilophobists.

Examined *microscopically*, the *papillæ filiformes* consist of a conical base, which bears, pencil-like, at its tip a number of thin, tapering papillæ. The epithelial layer lying over them is strongly developed. Very horny in texture, it presents long, thread-like extremities, sometimes bifurcated, which, scraped from a normal tongue, appear as the well-known brownish pyramidal bodies, with a dark axis and a finely-granulated cortex. The dark axis is certainly composed of very horny epithelium, which can be isolated and rendered translucent by a solution of potassa. The fine granules of the cortex respond to reagents exactly like the fungous sporules, which appear between the teeth, and especially within carious teeth, accumulated as a yellowish-white deposit. The sharp parallel contour of the surface of these masses of spores, moreover, points to the action of some agglutinating agent originating within the axis. Unless this were the case, we could not account for the smooth, even surface.

It seems the more probable that this cortex consists of fungous spores, as true thread-like fungi, the leptothrix buccalis, are often seated in great numbers upon this horny mass.

The *papillæ fungiformes* exhibit a club-shaped mucous papilla, which, in the manner of an artichoke, is surmounted with small, conical, secondary papillæ. Their epithelium has no such prolongations as that of the filiform papillæ, but retains the characteristics of the ordinary pavement epithelium of the remaining mucous membrane of the mouth.

The *papillæ vallatæ* under the microscope appear like the foregoing. Their numerous secondary terminations are enclosed in an externally smooth epithelial covering, and upon the summits of the surrounding walls are several rows of simple little conical prominences, whose surface is rendered even by an epithelial covering filling all its depressions. The circumvallate papillæ are distinguished by their great richness in nerves.

2. Glossitis Parenchymatosa.

Kemma, Dissertat. Hal. 1773.—*Schweighäuser*, Dissertat. Strassburg. 1789.—*Bode*, Dissertat. Helmstedt. 1791.—*Breidenstein*, Dissertat. Erlangen. 1791.—*Elsner*, Dissertat. Regiomont. 1789.—*Blödau*, Dissertat. Jena. 1795.—*Otto*, Dissertat. Frankfurt. 1803.—*Raggi*, Dissertat. Pavia. 1809.—*Accardi*, Dissert. Genua. 1810.—*Viollaud*, Dissert. Paris. 1815.—*Marcoul*, Dissertat. Strassburg. 1815.—*Reinisch*, Dissert. Leipzig. 1837.—*Canstatt*, Schmidt's Encyclop. Bd. VI. p. 515.—*Maisonneuve*, Des tumeurs de la langue. Paris. 1848. p. 4.—*Schneider*, Casper's Wochenschr. 1849. No. 23.—*Möller*, Klin. Bemerkungen über einige Krkhtn. der Zunge. Deutsche Klinik. 1851. No. 26.—*Emmert*, Würtb. med. Correspbl. 1851. No. 10.—*Arnold*, Memorabilien. 1856, März. No. 6.—*O. Weber*, Pitha-Billroth, Chirurgie. Krankheiten des Mundes. p. 316.—*Bamberger*, Krkhtn. des Digestionsapparates. p. 75.—*Förster*, Handbch. der spec. pathol. Anat. 2 Aufl. p. 38.—*Mittler*, Fremdkörper in der Zunge. Wien. Wochenschr. XX. 51. 1871.

Two forms of inflammation of the tongue can be distinguished: 1, a superficial inflammation, that of the mucous membrane; and, 2, a deep or parenchymatous inflammation, that of the muscular substance.

As the mucous membrane of the tongue participates in all the diseases of the cavity of the mouth, there would be several

different varieties of glossitis mucosa to discuss. In order to avoid repetition, it appears more judicious to pass this form by at present and revert to it in connection with the subject of stomatitis mucosa, so that there remains for consideration only *glossitis profunda* or *parenchymatosa.*

Definition.

We understand by this affection a dense, firm exudation in the parenchyma of the tongue, having little disposition to suppuration, and which is divided, according to its extent, into : 1st, a *circumscribed or partial glossitis*, *glossitis circumscripta partialis ;* and, 2d, a *diffuse or general glossitis*, *glossitis diffusa universalis.*

Etiology.

It is self-evident that the superficial position of the tongue renders it liable to a variety of injuries of a mechanical and chemical nature ; splinters of bone, fish-bones, needles, nails, sharp-pointed carious teeth, etc., which bore into the tongue, do not produce a general extensive, but only a circumscribed œdematous infiltration.

The sting of a wasp, bee, or hornet—insects frequently found in wind-fallen fruit, and thus taken into the mouths of careless children—may give occasion to very extensive œdematous swelling, which in some instances is said to have increased to symptoms threatening life ; but this sort of injury never results in parenchymatous glossitis with consecutive suppuration.

How little disposed the tongue is to react against foreign bodies is shown in a case narrated by Mittler, in which a man, in a fall, knocked out two upper molar teeth. The tongue bled very profusely, became somewhat swollen, and remained completely immovable, so that solid food could not be swallowed at all, and fluid food only with difficulty; and speech was very much impeded. At the end of twelve days, an ulcer two ctm. (four-fifths of an inch) in diameter was discovered in the neighborhood of the tip of the tongue on the right border, behind which a hard body, one ctm. (two-fifths of an inch) in diameter, could be felt. In the centre of the left border of the tongue a similar hardness could be felt. The probe struck upon a smooth body, hard as bone, which was extracted without trouble, and which

turned out to be the bone mouth-piece of a pipe stem, four ctm. (one and three-fifth inches) in length, and one ctm. (two-fifths of an inch) in diameter. It had penetrated through the tongue horizontally in its entire width, it being at this point only about 5–6 ctm. broad. The patient then remembered that he had had a cigar-holder in his mouth at the time of his fall. The parts healed promptly and without untoward result, despite the great irritation to which they had been subjected for twelve days.

Where anthrax poison is brought in contact with the tongue, the most intense form of glossitis is set up, with rapidly fatal termination—*glossanthrax*, or carbuncle of the tongue. Practitioners in regions where anthrax frequently prevails—*i. e.*, those practising in southern Russia—describe the course of anthrax of the tongue as exceedingly violent and malignant.

Mercurial glossitis almost always retains the character of glossitis mucosa, and will therefore come under consideration in connection with stomacace. According to Stromeyer, however, extensive œdematous swelling of the tongue, sometimes terminating fatally, has been observed as a sequence of the use of mercury.

Although it cannot be denied that here and there true parenchymatous glossitis is excited by the causes just mentioned, yet no distinct causal agency can be detected in the majority of cases of this very unfrequent affection. It appears to be more frequent in swampy regions, and has been reported as of epidemic occurrence by some authors (Arnold); in doing which, however, no little advantage has been taken of the well-known elasticity of the term "epidemic."

In recent times no epidemics have been observed, at least in Europe, and the sporadic cases of glossitis have been in nowise etiologically explained. Authors speak also of a metastatic glossitis in septicæmia, especially following typhus, small-pox, and puerperal fever. Nothing of this kind has ever been encountered by myself.

Pathological Anatomy.

Although the various forms of glossitis mucosa sometimes extend into the submucous tissues and give occasion to consider-

able swelling of the entire organ, they will be considered to more advantage in connection with the diseases of the other portions of the cavity of the mouth; and we confine ourselves, in this place, to the consideration of genuine glossitis parenchymatosa.

I have never seen a post-mortem in one of these cases. The manuals of pathological anatomy describe the swelling of the tongue, the coating with mucus, and the impression from the teeth upon it, as we will learn more in detail under the head of symptoms. Upon section the submucous tissue is found considerably infiltrated and distended by a fibrinous exudation. The bundles of muscle-fibres are discolored, friable, or studded with abscesses.

Symptoms.

The most important symptom is an exceedingly rapid swelling of the tongue, so that, within a few hours, or at most within one or two days, it may acquire the greatest possible dimensions. The patient is then no longer able to close his mouth, the tongue protrudes far out of the mouth, and presses sidewise between the back teeth, causing deep indentations with immediate disposition to the formation of ulcers. Although the alterations already mentioned are very troublesome and painful, they are by no means of as much consequence as swelling of the posterior portion of the tongue. This causes the tongue to be pressed against the posterior wall of the pharynx and the epiglottis to become pressed down, so that the act of respiration is first impeded, and then fully arrested. The patients are subject to paroxysms of asphyxia, as in croup; they become cyanotic, and are liable to speedy death by suffocation. Movements of the tongue, chewing, swallowing, and speaking are impossible. When the swelling has progressed so far that the lips can no longer be kept closed, the saliva flows in great quantity from both corners of the mouth; but after some time an exceedingly troublesome dryness of the mouth sets in.

The mucous membrane, as far as the tongue is still located within the mouth, is covered with a thick layer of tough, adhesive mucus; the tip of the organ protruding between the lips is dry,

and becomes fissured, bloody, and marked with deep, ulcerous indentations of the incisor teeth. The neighboring lymphatic and salivary glands always become swollen. The pain is described by the patients as very severe, beginning deep in the tongue, and spreading thence in every direction towards the ears and towards the throat. The somewhat puffed-up countenance evinces an expression of the greatest depression and anxiety, readily explicable by the pain and increasing dyspnœa. The general symptoms are very serious from the beginning, and consist in high fever, dyspepsia, prostration, sometimes delirium, and strong pulsation of the carotid arteries.

The course of the affection is always an acute one. Death may occur by suffocation in a few hours; or the patient may recover entirely in from one to two weeks, the fever ceasing, and the swelling of the tongue simply subsiding; or the exudation finally becoming purulent. The pus forces its way upwards, seldom downwards, breaks through the surface, and leaves a protracted fistulous ulcer in its wake. Gangrenous destruction of large portions of the tongue has been observed as a result of neglected mercurial glossitis. Sometimes circumscribed indurations remain, and often become the starting-point of recurrences of the affection.

The symptoms of partial glossitis are very different from the complex groups just described. Here there is, usually far backwards, a circumscribed painful tumor, from the size of a pea to that of a bean, which, according to its location, whether superficial or deep-seated, sooner or later raises the mucous membrane, becomes distinctly fluctuating, and finally ruptures. The symptoms are very slight, owing to the minuteness of the tumor, and the general health is not at all disturbed.

In women, hysterical subjects especially, certain of the papillæ vallatæ sometimes swell and become painful, but they subside spontaneously after a little while. This trifling malady has been described by Requin, Grisolle, and others, as glossitis papillaris, but it has nothing to do with parenchymatous glossitis, being merely a circumscribed inflammation of the mucous membrane.

The *differential diagnosis* will hardly ever present any dif-

ficulty. The sudden commencement and rapid course of the affection do not permit it to be confounded with any other benign or malignant morbid growth.

Glossitis has this in common with tumors of the region beneath the tongue, that, in both cases, the tongue is impeded in its movements, and is pressed against the palate. Simple inspection and palpation of the cavity of the mouth, however, is sufficient to establish the difference between the two processes. Circumscribed glossitis often passes very slowly into suppuration, and may then be easily mistaken for cancerous nodules. Even old practitioners, such as Wutzer, have been deceived in this way. A physician was very much worried about a painful tumor on the root of his tongue, of several weeks' standing, and about the size of a hazelnut; Wutzer recommended the extirpation of the supposed scirrhus, which, however, at the very first incision turned out to be an abscess.

Glossitis partialis is distinguished from the nodules of syphilitic gummata by its painfulness and the absence of all other symptoms of syphilis.

The *prognosis*, despite the violence of the symptoms, is usually favorable. The three cases, which I have thus far had the opportunity of observing, terminated in complete recovery, after a few days of the greatest danger. In septicæmic glossitis the disease of the tongue is only a small portion of the severe general malady, which usually terminates fatally, even without the glossitis.

The worst point is always the remaining indurations, and the disposition to relapses thus engendered.

Treatment.

Treatment of the causes can be instituted only in so far that persons who have already been attacked by glossitis should carefully avoid all chemical and mechanical irritation of the tongue, and should therefore have all sharp stumps of teeth promptly extracted.

Glossitis once developed, we endeavor to control it as far as possible by the assiduous application of ice, small pieces being

continually held in the mouth, and ice compresses being laid on the protruded tongue and the parts adjacent. If suffocative symptoms are not allayed by these means, two or three deep longitudinal incisions should be made in the dorsum of the tongue, and the bleeding be encouraged by warm water. General blood-letting, leeching, cupping, even opening the external jugular vein, as recommended by P. Frank, are all, of course, much less effectual than deep scarifications of the tongue, which, moreover, heal very promptly and without further injury, after subsidence of the enlargement of the organ. Tracheotomy remains as a resource in extreme cases.

Under all circumstances it is judicious to secure the derivative action of the bowels, and the best method for this purpose is to use purgative clysters. If the progress of the disease is less violent, and the formation of abscess is suspected, attempts should be made to detect the abscess at an early moment, by the sense of fluctuation, and to open it promptly. Disinfecting substances are recommended for gargling, such as a one per cent. solution of carbolic acid, or three or four drops of tincture of iodine to one hundred cubic centimetres (three and a quarter ounces) of water.

3. Tumors of the Tongue.

Brown, Lancet. March 30, 1833.—*Reiche*, Rust's Magazine. 1836. Bd. 46.—*Lebert*, Traité d'anatom. pathol. II. p. 149.—*Maisonneuve*, Des tumeurs de la langue. 1848.—*Hannover*, Das Epitheliom. 1852. p. 81.—*Schuh*, Pseudoplasmen. 1854. p. 208.—*Bastien*, Bullet. de la Soc. anat. de Paris. Nov. 1854.—*Förster*, Illust. med. Zeitg. 1855. p. 63; and Handbuch d. spec. pathol. Anat. II. Aufl. p. 35.—*Weisser*, De linguæ structura pathol. Berol. 1856.—*Dehler*, Oesterr. Ztschr. f. prakt. Hlkde. 1858. No. 14.—*Paget*, Med. Times and Gaz. 1858. p. 411.—*O. Weber*, Chirug. Erfahrng. 1859. p. 351; and in Billroth-Pitha's Chirurgie. Bd. III. 1. Hft. 2. p. 330.—*Thiersch*, Der Epithelialkrebs. Leipzig. 1865. p. 292.—*Bamberger*, Virchow's Handbuch der spec. pathol. Bd. VI. 1.—*Lawson*, Congenital hypertrophy of the tongue. Trans. Clin. So. V. p. 158. 1872.—*Arnott*, Makroglossia. Trans. Path. So. 1872. XXIII. p. 109.—*Clarke*, Hypertrophy of the tongue. Ibid. p. 111.

Various benign and malignant tumors occur upon the tongue. To the former category belong simple warts, tele-

angiectasiæ, varicosities, and small aneurisms, lipomata, fibromata, cysts, and syphilitic gummata; to the latter, epithelial cancer.

Etiology.

Some benign tumors are congenital. Reiche narrates a case in which the blue-black end of the tongue, constituting a tumor the size of an apple, protruded between the lips.

Simple warts of the tongue do not coincide with the multiple formation of warts on the face and hands, so often observed in children; and they are furthermore distinguished from these ephemeral papillary developments by their life-long duration.

Epithelial cancer appears much more frequently in men than in women. In a collection of the cases reported by Maisonneuve, Thiersch, and O. Weber, there were fifty cases in men, and only seven in women. No reason for this can be given with certainty; but perhaps the use of tobacco has something to do with it. Anyhow, this is more plausible than O. Weber's opinion, according to which this remarkable difference is dependent on the better care of the teeth and the greater cleanliness of the mouth in women. That sharp carious edges of teeth are often the first exciting cause, by producing a chronic ulceration of the tongue, is substantiated by every experienced surgeon.

Finally, it is to be remarked that cancer of the tongue is not so exclusively a disease of mature age as other cancerous affections. Schuh encountered it in a man but twenty-two years of age.

Pathological Anatomy.

Benign tumors of the tongue are unfrequent and of slight significance. There are simple warty vegetations over which the epithelium has become very horny; and capillary dilatations—*teleangiectasiæ*—which may enlarge as bluish-red nodules to the size of a dove's egg, and even protrude between the lips.

In old people, disposed to *varicosities*, such developments are also found upon the tongue. In some instances small aneurisms

have also appeared, which have been cured by division, and by ligation of the arteries.

Intermuscular *lipomas* and *fibromas* occur here and there, and attain the size of a walnut. They are distinguishable from carcinomas by their lack of sensitiveness and their slow development. They can be readily removed by the knife without any danger. Sometimes small *cysts* the size of peas occur in and beneath the mucous membrane, which, when punctured, discharge thick, glutinous contents. They are mostly the result of obliteration of the excretory mouth of a gland; but sometimes, when closely examined, prove themselves to be the cysticercus cellulosæ.

Syphilitic gummata are nothing uncommon in the tongue, but they will be treated of to better advantage in the section on syphilis.

Cancer of the tongue is much more frequent and important than the benign tumors. Epithelial cancer is the only variety which occurs here as a primary affection. The opinions of older surgeons, who spoke a great deal about scirrhus and fungus of the tongue, were not based upon histological investigations, which, since they have been generally practised, have detected only epithelial cancer in the tongue.

It usually commences at the anterior portion of the tongue, on the tip, or on the border to one side; sometimes, also, on the inferior surface, where extensive destruction may ensue before any degeneration has begun upon the dorsum. The usual characteristics of epithelial cancer are sometimes no more distinct than this, when masses of papillary vegetations spring forth from the interior of the organ. Otherwise, we see either a flat ulcer with undermined, necrotic edges, or the growth appears first as a nodule, formed out of globular cancer spaces, which quickly break down and disclose deep ulcers, in the vicinity of which the cancer extends rapidly in its further growth. Histological examination reveals the well-known appearances which Thiersch has best depicted. O. Weber, also, has given an excellent illustration of a vertical section of a cancer of the tongue.[1]

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[1] *Pitha* and *Billroth*, Chirurgie. Bd. III. 1. Abth. 2 Hft. p. 331.

Symptoms.

A portion of the symptoms have already been treated of in the anatomical description, and benign tumors of the tongue do not, consequently, require any further description of their symptoms.

Cancer of the tongue is one of the most terrible of diseases. The lancinating, boring, burning pains soon reach the highest grade of suffering, robbing the patient of rest at night, and not unfrequently leading him to suicide. At the commencement, as long as the cancer has not yet ulcerated, and is only evident as a hard nodule in the border or in the body of the tongue, the condition is tolerably bearable. But when destruction and ulceration have once begun, the severest pains always set in. The cancerous ulcer distinguishes itself from every other ulcer by its continuous encroachments, by its hard, lardaceous bottom, and by the viscid, milky juice which can be expressed by pressure upon its edges.

Partly on account of the pain, and partly on account of the deepening progress of the neoplasm, the movements of the tongue become more and more impeded; speech becomes indistinct, while mastication and deglutition can only be imperfectly effected. Those portions of the tongue remaining intact sometimes become œdematously swollen, so that the tongue protrudes far beyond the lips; or it undergoes such extensive destruction that it remains nothing but a misshapen mass on the floor of the mouth.

The salivary secretion is usually increased, the saliva being copiously loaded with cell-elements, and thus rendered turbid. If disinfectant mouth-washes are not continually used, the entire cavity of the mouth and the breath acquire a cadaveric odor. Sometimes large vessels, in the reach of the destructive process, are laid open before they have become obliterated, so that alarming hemorrhage may ensue, resulting in extreme anæmia.

The further course of epithelial cancer is, next, an extension to the neighboring lymphatic glands and to the lower jaw; but

every swelling of the glands of the neck and every pain in the bone must not be taken at once for cancerous disease. They are frequently produced by simple glandular irritation, as in stomacace or in diphtheria; and radiating pains occur in the diseases of various organs, without being any evidence of the extension of the pathological process into neighboring parts. When the lymphatic glands and the lower jaw really undergo cancerous degeneration, abscesses and fistulas are soon manifested externally.

The evil effects upon the general health make themselves manifest only too soon, and arise from a double source: the cancerous cachexia, in general, and the impeded chewing and swallowing, as well as the abolition of the appetite on account of the pain.

The course of the disease is usually a tolerably rapid one, unless, at least, a temporary respite is obtained by timely extirpation. In a year, or at longest in from two to three years, the patients are released from their torments by death. Sometimes the end is hastened by pyæmia, pneumonia, or hemorrhage; while secondary cancerous infection of distant organs seldom occurs in epithelial cancer. In addition, extreme emaciation, anæmia, and œdematous swelling of the ankles complete the usual picture of the cancerous cachexia.

The *differential diagnosis* presents no special difficulty in most cases. Glossitis is an acute, febrile process, which determines itself in a few days, while cancer of the tongue begins in a gradual manner. Tuberculous ulcers of the tongue, which are of rare occurrence, are smaller, more superficial, and are present only in cases of far advanced tuberculosis of the lungs and the intestines.

The distinction between syphilis and cancer of the tongue presents greater difficulties. Gummata in the tongue deport themselves in form and size almost exactly like recent cancer, but are distinguished from it by the absence of pain. Ulcerated cancer of the tongue may present a great likeness to syphilitic ulcers. Here the difference is determined by the history of the case, the searching for other syphilitic symptoms in the favorite localities of syphilis, and the properties of the ulcer

itself. Some milky juice can generally be pressed out of the borders of a cancer of the tongue, and the floor of the ulcer is much harder than in syphilis. By the removal of a small portion of the ulcer, and its microscopic examination, we can also readily detect the characteristic signs of epithelial cancer.

Treatment.

Medication naturally exerts no influence upon tumors of the tongue. Teleangiectasiæ sometimes become shrivelled through catarrhal irritation of the mucous membrane. Brown refers in this connection to a remarkable case. The extirpation of a large vascular tumor of the tongue in a young woman, eighteen years of age, had been determined upon, when she was given, in advance, a laxative of calomel. During the severe mercurial stomatitis that ensued, the tumor subsided spontaneously.

Benign tumors, warts, lipomas, cysts, etc., can be readily and permanently extirpated. The only remedy for cancer is prompt removal with knife, ecraseur, or the actual cautery. There are well-authenticated cases in which recurrence has been prevented for a number of years by timely operation ; in one, described by O. Weber, the immunity continued for twelve years. Generally, however, epithelial cancer is reproduced very rapidly; and with the greater certainty when large portions of the still healthy tongue are not removed with the diseased portions. It is important, therefore, to insist energetically upon immediate operation, and not to lose valuable time with the very unreliable use of caustics.

If the destruction has progressed to such an extent as to leave no hope of removing the entire cancer, there remains only the ordinary symptomatic treatment of cancer patients with narcotics—above all, the internal and subcutaneous use of morphia. Hemorrhage must be combated by ice or cauterization. Disinfection of the cadaveric odor of the mouth may be attempted by means of chlorine water, permanganate of potassa, or a one per cent. solution of carbolic acid ; but this treatment is usually interfered with by the increased painfulness of the ulcerated tongue which these remedies produce.

4. The Pathological Coatings of the Tongue.

Pfeufer, Henle und Pfeufer's Ztschr. f. rat. Med. VII. 2.—*Davasse*, Canstatt's Jahresber. 1849. p. 239.—*Miquel*, Prag. Vierteljahrschr. 1850. IV. —*Höfle*, Chemie u. Mikroskopie am Krankenbett. Erlangen. 1850.—*Kölliker*, Würzburger Verhdlg. 1851. 11.—*C. Wedl*, Grundzüge der pathol. Histologie. Wien. 1853.—*Bamberger*, Krkhtn. des chylopoëtisch. Systems. II. Aufl.—*Weisser*, De linguæ structur. pathol. Berlin. 1858.—*Förster*, Handbch. der spec. pathol. Anat. II. Aufl. p. 38.

Inspection of the tongue has been practised by physicians and others from the oldest periods ; but this method of examination has not afforded any developments commensurate with its venerable antiquity. The significance attached to the coatings of the tongue has also undergone the extremest changes. While, in former centuries, it was generally regarded as the true mirror of the stomach, the recent bent of pathological anatomy has been to view it as a purely local incident, and to deny it all connection with disorders of digestion.

If we inquire what deviations from the normal appearances of the tongue can be recognized, we have to consider :

1. The manner in which the tongue is protruded ;
2. The form of the tongue, its surface and outlines ;
3. The color, thickness, and extent of the coat ; and,
4. The degree of moisture of the tongue.

1. Although the manner of protrusion does not strictly belong here, but rather in the domain of nervous affections, still, for the sake of completeness, it will be judicious to give it a few words.

Even little children, from nine to twelve months of age, frequently understand how to protrude the tongue far forwards, being taught by example. This facility is often entirely lost later, from total want of practice ; and even very sensible people, when requested to show the tongue, simply open their mouths without putting the tongue out. Only after repeated persuasion does the tip of the tongue appear between the lips, and but few such people comprehend how to lay a large portion of the tongue upon the under lip. In some cases, the cause of this is really want of use of the genioglossus muscles ; in many others, for

instance, in young women of the upper ranks, it is a false bashfulness or affectation. In disturbances of consciousness, all such voluntary movements, as a matter of course, cease.

In other cases the tongue cannot be quietly protruded and held still, but it continually trembles. This occurs even in healthy people under great agitation or anxiety, as well as in fevers with a high temperature and consequent debility, especially in typhus, puerperal fever, and septicæmia generally.

In chorea, the patients cannot long retain the tongue protruded, though for the most part they are able to show it promptly enough, but it will be spasmodically retracted all at once without the volition of the patient. Finally, the oblique protrusion of the tongue with the tip drawn to one side depends either upon a unilateral defect of the lower row of teeth or upon central hemiplegia.

2. The tongues of most persons have a tolerably smooth surface, but in some it is indented with deep furrows, which usually follow a longitudinal direction, rarely an oblique or horizontal one. On protrusion of the tongue, this wrinkled mucous membrane unfolds and flattens, and discloses on the floor of the folds remarkably long and thick tufts or villi, situated somewhat further apart than on other parts of the tongue. This condition is not connected with any local or general disease. In many cases it seems as though the tongue was simply too broad for the inner space of the lower jaw; as if there had been a disproportion between the growth of the tongue and that of the jaw. For the most part, however, it is one of the manifestations of general emaciation. This is verified by the fact that it is never seen in young people, and also by another observation, often made by myself, that the wrinkled tongue disappears entirely with increasing obesity.

These lingual furrows, in cases of catarrhal or ulcerous processes—as, for example, in typhus or in syphilis—are the favorite seats of ulcers and excoriations. The border of the tongue is, in physiological conditions, uniformly rounded, but when the mucous membrane is swollen the tongue receives the impression of the teeth against which it is pressed. The tongue is then seen bordered by a sharp-cornered edge, corresponding to the line

of contact of the upper and lower teeth ; in addition to which its oval contour is lost, as the mark of each individual tooth appears imprinted upon it.

3. The normal tongue is rose-colored on the tip and the adjacent edges, dotted with red points, the fungiform papillæ ; further backwards the white tufts become so thick and large that the entire surface appears considerably lighter colored, whitish-pink, and uneven. Still further back, especially in the central portion, the tongue is altogether white or yellowish-white. It is therefore evident that the white color of the tongue depends mostly on the length and thickness of the villi ; the latter qualities, however, depend partly upon mechanical and chemical influences on the tips of the villi, and partly upon an increased epithelial development from internal causes.

The tender, normal, light, rose-colored coating of the tongue frequently becomes altered by increasing considerably in thickness ; and then the tongue appears entirely white or yellowish-white. Microscopically examined, however, its appearance is normal, save that the pavement epithelium is remarkably strongly granulated, and the entire field swarms with spores and bacteria. In patients with icterus the coating of the tongue is not proportionately as yellow as the skin ; on the other hand, many patients with disorders of digestion have a distinctly yellow coating, in which, however, no biliary coloring matter can be detected.

The brownish-red, brown, and black coatings of the tongue occur only in severe diseases in which the mouth has a disposition to become dry. It depends partly upon the articles of food and drink, and partly upon slight admixture of dried blood, which, on sudden stretching of the dried tongue, oozes from small ruptures in the mucous membrane.

The coloring matters of food and of preparations of iron leave traces only for a short time upon a normal tongue ; while in severe diseases—especially typhus—coffee, chocolate, red wine, whortleberries, etc., will leave the tongue colored black for several days.

The thickness of the coating of the tongue may increase to that of a millimetre ; and in such cases it is sometimes possible

to detach large pieces of it with the finger-nail or a tongue-scraper.

The tongues of all nurslings, even the healthiest, show a remarkably thick, white coating. The slight movement of the lips, the absence of speech, the long and frequent sleep, the admixture of milk-corpuscles with the epithelium, may be the grounds for these appearances which so frequently cause anxiety to young mothers.

The coating of the tongue, in many cases, is not uniformly thick in all places. Sometimes a tolerably broad, longitudinal stripe is seen in the centre, without any coating at all; while the borders show a thick coating. In other, less frequent cases, the reverse occurs. At the commencement of typhoid fever, a red triangular spot, disposed to dryness, is often remarked at the tip of the tongue, while all the remaining portion of the organ is coated white.

Finally, in many persons, the coating of the tongue, for lifetime or during many years at least, forms isolated circumscribed whitish patches, circular and with other more or less tortuous outlines,—the so-called *pityriasis linguæ*, which is produced merely by a stouter layer of epithelium. Although in some cases the sharp, carious corner of a tooth may be recognized as a source of local irritation, yet, in the majority of cases, neither a topical nor a systemic origin for this affection can be detected. Sometimes, especially in chronic affections of the stomach, the tongue is seen to be quite smooth, moist, and red, so that it takes on the appearances of the remaining mucous membrane of the mouth. Only a little opalescent mucus can then be scraped from it, in which, under the microscope, no little brown tops of the papillæ filiformes can be recognized, but only squamous epithelium.

In scarlatina the tongue is affected in a quite peculiar manner. The papillæ clavatæ are swollen, dark-red, and project, as little red buttons, above the white coating; or the entire coating is thrown off, and the mucous membrane in its entirety is dark-red and uneven, so that it has been designated, not inaptly, as the strawberry-tongue.

4. The normal degree of moisture may vary in two direc-

tions. The secretion is sometimes so increased and diluted, that a drop of thin mucus accumulates on the tips of the tongue only a few seconds after its protrusion ; while in other cases the tongue appears completely dry and hard. It is sufficient, in practice, to judge of the degree of moisture by feeling the tongue with the finger ; but its amount can be more exactly estimated by absorbing the moisture upon a strip of filtering paper laid upon the tongue.

Dryness of the cavity of the mouth is always pathological. Only in exceptional cases will the tongue become entirely dry in those who sleep with the mouth open ; for, usually, the sleeper closes his mouth as soon as the cavity begins to be dry. When the nostrils are obstructed, sleep is always very unquiet, and patients are frequently compelled to moisten the mouth with water.

In general increase of bodily temperature, in the heat of fever, the secretion of the mouth is always lessened—a circumstance which in part explains the constant desire of fever patients for cold drinks. In many healthy persons the use of spirits, of highly salted and spiced food, and excessive tobacco-smoking, also cause a disagreeable dryness of the tongue.

From what has been stated, the inference is evident that careful inspection of the tongue furnishes many diagnostic points, and should never be neglected. Although it cannot be denied that sometimes purely local diseases of the mouth are the causes of the coated or dried tongue, yet daily experience teaches that acute disturbances of digestion chiefly produce a white coating, which subsides again in like measure with the improvement of the stomach symptoms. It is easy to understand that thick coating of the tongue must diminish the sense of taste, and that dryness of the tongue should impede its movements.

The treatment of coating of the tongue, which is partly mechanical and partly disinfectant, will be considered to more advantage in connection with the diseases of the mucous membrane of the mouth in general.

C.—Mucous Membrane of the Mouth.

Pfeufer, Henle u. Pfeufer's Ztschr. f. rat. Medic. VII. 2.—*Hannon*, Journ. f. Kinderkrkhtn. 1850, März—April.—*Roell*, Lehrbuch der Pathol. u. Therapie der Hausthiere. 2. Aufl. p. 307.—*Andrieu, E.*, Traité complet de la symptomatologie etc. de la bouche. Paris. 1867.—*Jardin*, Sur les différentes stomatites, leur caractères differ. etc. Annal de la Soc. de Médec. de Gonel. pp. 181–334.—*Legrand, Max*, Stomatite et glossite idiopath. Union Méd. 1870. No. 65.—*Vogel, A.*, Lehrbuch der Kinderkrankhtn. 6 Aufl. p. 89.—Consult also the different recent text-books on diseases of children, and those on special pathology and therapeutics.

In the pathological processes in the mucous membrane of the mouth we distinguish :

1st, *catarrhal inflammation* (*a*, erythematous, and *b*, phlegmonous) ; and, 2d, *membranous inflammation* (*a*, croup, *b*, diphtheria) ; 3d, the various *ulcerous affections*, aphthæ, stomacace, the mercurial, scorbutic, syphilitic, variolous, leprous, lupous, and tuberculous ulcers ; 4th, the fungus formation, *thrush ;* and, 5th, gangrene of the cheek, *noma.*

Croup and diphtheria affect the pharynx by preference, and thence, only exceptionally, extend to the anterior cavity of the mouth. The complete description of these forms of disease will therefore follow more appropriately among the diseases of the pharynx.

1. Stomatitis Catarrhalis

By *stomatitis catarrhalis* we understand a swelling and redness of the mucous membrane of the mouth, by which contact is rendered painful and the secretion is increased. According to its course, we distinguish it as acute or as chronic ; according to its method of origin, as a primary or as a secondary affection.

Authors describe two forms of stomatitis, according to its intensity, the erythematous and the phlegmonous. The most important symptom of the first is redness ; in the second a considerable amount of swelling is added thereto. The latter form appears particularly on the tonsils and the palatine arches as the well-known angina tonsillaris, and will be treated of in

another place. This distinction is of no moment as regards the anterior portions of the mouth, with which we are here exclusively concerned. Here the presence or absence of ulceration forms the most important indication in differential diagnosis. Where there is no ulceration, we have to do with catarrhal stomatitis; in other cases, with ulcerous stomatitis.

Etiology.

As causes of primary stomatitis catarrhalis are to be mentioned, in the first place, mechanical and chemical irritants. The eruption of the teeth in nurslings; salivary calculi; carious teeth with sharp points and corners; too hot and too cold ingesta; excess of spices of all kinds; tobacco smoking and chewing; excessive use of organic and mineral acids; the inordinate eating of sour, hard fruit; a great number of medicines—iodine, bromine, antimony, arsenic, mercury, potassium, sodium, croton oil, daphne mezereum, and cantharides; and the inhalation of infected air loaded with injurious materials, as chlorine, phosphorus, and sulphurous acid. Long-continued hyperæmia of the mucous membrane must be added to the mechanical irritations—such, for example, as is produced by protracted sucking, speaking, crying, blowpipe blowing, and the like.

Apart from these direct injurious causes, catarrhal stomatitis is undoubtedly produced also by cold feet, wet clothing, and draughts of air, especially when the cavity of the mouth is already rendered a *locus minoris resistentiæ* by partial disease of the gums or by caries of the teeth.

While the above-mentioned causes principally produce primary stomatitis, secondary stomatitis has much more numerous causes of origin. All the ulcerative processes of the mouth, nasal passages and pharynx, whether benign or malignant, whether specific or non-specific, produce at least a catarrhal swelling and congestion in the other portions of the mucous membrane which remain free from ulceration.

All acute infectious diseases are followed by hyperæmia of the cavity of the mouth; small-pox, frequently enough, by extensive ulceration. The peculiar swelling of the papillæ clavatæ

in scarlatina, the so-called strawberry-tongue, has already been mentioned in connection with the coatings of the tongue. Measles affect more the posterior portions of the cavity of the mouth, the pharynx and larynx. In erysipelas faciei the entire mouth is strikingly congested.

Chronic catarrh of the mouth and pharynx is most constant and obstinate in tipplers. As this generally appears for the first time after the gastric symptoms of the alcoholic dyscrasia are already plainly manifest, it is to be classed among the secondary forms of the affection.

Symptoms and Pathological Anatomy.

Redness and swelling are by no means, even in general stomatitis, uniformly developed in all parts, but vary in accordance with the quality of the submucous tissue. The more lax the tissue is, the more quickly it swells and becomes congested. The closely-attached mucous membrane of the hard palate is always proportionately the least affected, while that of the cheeks and of the soft palate shows more distinct evidences of inflammation.

The uvula becomes elongated to such an extent that it continually touches and tickles the root of the tongue, causing choking and hawking. Its free extremity is usually swollen, club-shaped, and distinctly divided by a longitudinal furrow. Its mucous membrane is at the same time remarkably glossy and translucent. The impression of the teeth appears in the swollen mucous membrane of the tongue, and also in that of the cheek, upon which can be seen whitish, elevated, longitudinal stripes, with some vertical stripes, corresponding with the outlines of the teeth. The whitish color of these ledge-like wheals is due to an excessive growth of the epithelium, which here, at the points of contact of the teeth with each other, is not subjected to such strong pressure as at other portions.

The redness, when not due to an entirely circumscribed local cause, is always diffuse and capillary; in chronic catarrh the larger vessels are also dilated, and present arborescent injections.

The mucous glands swell into knots the size of a millet-seed, which is especially manifest upon the soft palate.

A dense exudation is never met with in catarrhal stomatitis. At the commencement of the process the mucous membrane is even disposed to dryness, so that this is one of the first symptoms perceived by the patient; and it may be explained as produced by a temporary compression of the excretory ducts of the glands. At a later date there is increased secretion of mucus and saliva, inasmuch as the inflammatory irritation has extended into the parenchyma of the glands. At the same time the chemical and morphological composition of the mucus undergoes an alteration, and it now appears as a thick, tenacious deposit, rich in cells and nuclei, and adherent to the tongue, the soft palate, and the gums. The exertions of the patient to get rid of this mass by hawking and spitting remain ineffectual for a long time. This slimy deposit must not be confounded with the fine, whitish deposit, which in every febrile patient can be wiped off from the gums, in the mornings especially, and which consists merely of epithelium which has undergone fatty degeneration, detritus, and masses of micrococci and bacteria. This is no doubt due to the increased temperature, by which the reproduction of epithelium and the conditions of evaporation become modified. The lack of appetite, especially the avoidance of solid, dry articles of food, by chewing which, in the healthy state, the gums would be rubbed, contributes at any rate to the production of this delicate fever deposit.

As the mucous membrane of the tongue is diseased to a like degree, or even more intensely, it follows, as a matter of course, that there is an alteration in the sense of taste. The so-called pasty or sticky taste is evidently not a sensation of taste, but one of touch. On the other hand, the assertion of the patient, that everything is flat and tasteless, depends plainly upon the thick coating of the tongue, and this explains the desire for strongly-spiced and piquant food. Besides this blunting of the sensation, other disturbances of taste appear. Tea, coffee, and wine appear to acquire a repugnant aroma; patients maintain that fresh meat is spoiled and putrid, and they drive the cook to despair with their unjust accusations.

The frequent occurrence of a peculiar bitter taste has given repeated occasion for examining the secretions of the mouth for biliary matters, but always without any positive result. The fetid flavor complained of may sometimes be genuine, and not dependent upon an hallucination of taste, inasmuch as the thick coat upon the mucous membrane may be rapidly decomposed, and thus, as is frequently the case, especially in the mornings, give occasion to an offensive breath.

The complaint of patients is first of dryness of the mouth, then of an increased viscid secretion, then of pain in chewing, and of difficulty in swallowing (tenesmus faucium). Warm drinks and gargles increase the pain, cold ones afford relief.

The general symptoms are very trifling in primary idiopathic catarrhal stomatitis of the adult; in nurslings, on the contrary, there are reflex symptoms of various kinds, which will be treated of in a special section, on difficult dentition. If the affection is secondary, or part of the symptoms of a gastric or general febrile disease, all the general symptoms that come under observation belong to the primary disorder. The same holds true when catarrh of the cavity of the mouth extends into the larynx and bronchi, or into the nasal passages and Eustachian tubes. The symptoms thus produced, hoarseness, cough, hardness of hearing, snuffling, and pain in the forehead belong simply to the complications mentioned. Swelling of the neighboring lymphatic glands of the neck does not occur in simple catarrhal stomatitis, and is always an evidence of ulceration of the mucous membrane of the mouth, though it may be but very slight.

Course and Progress.

The course of the affection varies in accordance with its causes. If these can be removed, the primary stomatitis is cured. Secondary stomatitis, in the wake of ulceration of any sort, comports itself according to the nature of the causal affection. Stomatitis following simple syphilitic vegetations of the mucous membrane, naturally justifies a more favorable prognosis than that produced by deeply penetrating destruction of tissue. Stomatitis in the wake of an erysipelas faciei is naturally of shorter duration than

that attending a severe abdominal typhus. Finally, the chronic oral and pharyngeal catarrh of drunkards is incurable, inasmuch as the etiological element, alcohol, can hardly ever be laid aside.

Treatment.

The *indicatio causalis* can here be utilized in many cases. The removal of injurious teeth, the opening of a gum-boil, the avoidance of tobacco, the suspension of irritating medicines, are therapeutic measures often easily enforced. Some patients long ardently for an emetic ; and in such cases, where there are no distinct signs of an ulcerative process present upon the mucous membrane of the cheek, it may frequently be useful, or at least uninjurious. The certainty that the stomach is free from all indigestible materials is always of importance in the treatment of an affection of the mouth.

Deuteropathic stomatitis, as it is presented in the wake of all acute, febrile diseases, usually remains untreated, inasmuch as the original disease already calls for such varied therapeutic measures, and therefore local treatment is usually confined to frequent moistening of the parched mouth.

With reference to the *indicatio morbi*, every possible local remedy has been tried here, where the seat of disease is so accessible to all the senses. The simplest and most convenient application is the rinsing out of the mouth with pure or medicated water. The term gargle, gargarisma, is in so far not altogether appropriate here, inasmuch as real gargling, performed with a loud gurgling sound due to the trembling motion of the soft palate, is usually far too painful when these parts are swollen to permit the practice of such a procedure with benefit. The simple retention in the mouth of a large quantity of fluid, combined with a slight play of the buccinator muscles, fully suffices to wash all parts of the anterior cavity of the mouth ; and this removal of the desquamated epithelium and the tenacious mucus, as well as the prolonged moistening of the mouth, is of decided benefit in all cases.

Although gargles have been employed from the oldest times, and more or less potent remedies have from the first been held in

solution in them, the question was still an open one, in how far these substances were absorbed by the mucous membrane of the mouth alone, all movement of deglutition being carefully avoided —in how far, therefore, a gargle might exert not only a local, but also a systemic influence.

This gap was filled, at my request, by one of my pupils, Dr. J. Karmel,[1] who instituted upon himself a series of experiments bearing upon this point. After Karmel had carefully rinsed his mouth with distilled water, he took into his mouth a portion of the fluid to be examined, and retained it there a certain length of time, usually for two, sometimes for three or four minutes. He then emptied the contents of his mouth into a suitable vessel, rinsed his mouth out carefully with distilled water, and spat this also into the same vessel, so that nothing should remain adhering beneath the tongue or between the teeth.

He then proceeded in the same manner with a second portion, and so on, until the entire quantity, 200 CC., of the fluid under examination had been exhausted. Movements of deglutition were carefully avoided, as a matter of course. Exact, quantitative chemical analysis before the procedure, and after it, regularly showed that a not inconsiderable quantity of the material, dissolved in the gargle, disappeared; it had evidently, therefore, been absorbed by the mucous membrane.

The experiment extended to the following-named substances: 1, alcohol (in the form of arrac); 2, carbonate of soda; 3, tartaric acid; 4, nitrate of potassa; 5, chlorate of potassa; 6, sulphate of magnesia; and 7, grape-sugar. It was shown that the facility for absorption of these materials was in accordance with the above arrangement. Of a solution containing five per cent. of alcohol, for example, twenty per cent. of the entire quantity remained uncollected; eight per cent. of a two per cent. solution of chlorate of potassa, and six and a half per cent. of a two per cent. solution of grape-sugar remained behind.

The result of this investigation may be summed up in the following propositions:

1st. The mucous membrane of the mouth absorbs to a considerable degree.

[1] Ueber die Resorption in der Mundhöhle. Dissertat. Dorpat. 1873.

2d. The absorption varies according to the nature of the material.

3d. The more concentrated the solution, the greater the absorption.

4th. The absorption does not increase in proportion to the time [of contact?].

In ordering mouth-washes these facts should always be kept in view, and it should not be forgotten, especially in administering poisonous substances, that most patients, either without remarking it, or through awkwardness, swallow a considerable portion of a gargle.

In most cases of acute catarrhal stomatitis the simple and frequently repeated rinsing the mouth with cold water might suffice; and would, at any rate, be more serviceable than the slimy decoctions of althæa and mallow, so much beloved by the older practitioners. Chlorate of potassa, especially, exerts a beneficial influence on this process; and has, in the forty years since it was brought into use by Hunt[1] and West, very nearly rendered obsolete the use of all other gargles. With a ten-grain solution of this salt more can be accomplished than with the one-tenth grain solution of corrosive sublimate recommended by Pfeufer, or the two-grain solution of nitrate of silver recommended by Henoch—more than can be accomplished with sulphate of zinc or sugar of lead. Even the permanganate of potassa and the one per cent. solution of carbolic acid have been superseded thereby.

In the chronic oral catarrh of topers, Niemeyer recommends that a piece of rhubarb should be chewed in the evening before retiring to bed; and this remedy often does render good service. Its action appears to be especially a mechanical one; at least the administration of tincture of rhubarb or rhubarb pills does not effect the same result.

That the nourishment should always be fluid or semi-fluid, is self-evident, inasmuch as the chewing of dry, hard food produces unbearable pain. All patients prefer cold drinks to warm ones; and there is no reason to refuse gratification of this

[1] *Bärensprung*, Charité. Annal—Bd. X. p. 116.

instinctive desire. Finally we have to provide for free alvine evacuation, which may here be secured best by mild drastics, but never by calomel.

Appendix.

Difficult Dentition.

The eruption of the teeth, especially the rapid succession of the milk-teeth, is always attended by a considerable degree of catarrhal stomatitis, and this may be regarded as a physiological occurrence. The manifold irradiated and reflex manifestations, however, which almost invariably accompany this process, are so important, and so often the subject of parental anxiety, that it appears appropriate to give special consideration to the subject of eruption of the milk-teeth, and the difficulties therewith connected.

In the first place, we will take a closer view of the succession of the erupting teeth. The ossification of the dental sacs of the twenty milk-teeth takes place in the fifth month of pregnancy, and the sacs for the permanent teeth are developed on the posterior wall of the sacs of the milk-teeth. After birth the milk-teeth gradually advance against the alveolar border of the jaw, now closed by cartilage, as the roots of the teeth become more developed. At the same time the cartilage of the gums is absorbed and the upper wall of the tooth-sac also, until finally the milk-tooth appears as a white ridge between the congested gums. Sometimes the cartilage atrophies some days earlier, before the young tooth has reached the level of the gums. It is not then seen, but it can be felt, and can be particularly demonstrated by the clapping sound produced by striking upon it with the handle of a metallic spoon.

The time, and the succession in which the milk-teeth break through, is by no means entirely constant; but in the great majority of healthy children the eruption takes place in a certain series of groups and at a stated time.

1*st group*.—Between the fourth and seventh months, and pretty nearly simultaneously, the two lower central incisors

appear; after which there is a pause of from three to nine weeks.

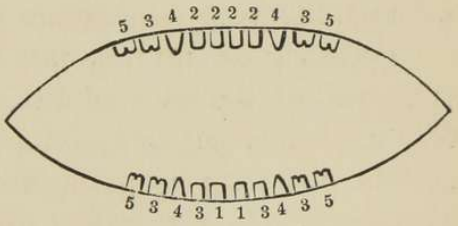
2d group.—Between the eighth and tenth months, the four upper incisors appear in quick succession within a few weeks of each other—at first the two central teeth, and then those at the side. The second pause occupies from six to twelve weeks.

3d group.—Between the twelfth and fifteenth months, six teeth appear at one time, namely, the four first molars, and the two lower lateral incisors—the molars of the upper jaw first, as a rule, then the lower incisors, and lastly the molars of the lower jaw. Then there is a pause until the eighteenth month.

4th group.—Between the eighteenth and twenty-fourth months the canine teeth break through, the upper ones being also called eye-teeth. Then there is a pause of from two to three months.

5th group.—Between the twentieth and thirtieth months, the four second molars finally appear.

We can represent these five groups in a diagram, in which the upper arch represents the upper jaw, and the lower arch the lower jaw. The numbers designate the groups to which the individual teeth belong. The incisor teeth are represented as four-cornered, the canines as pointed, and the molars as surmounted with a crown.



The first dentition is now closed with the eruption of these twenty deciduous teeth; and the second dentition, the eruption of the first permanent molars, begins in the fifth or sixth year. We can never, therefore, attribute to difficult dentition any pathological process which occurs in a child under five years of age, with twenty milk-teeth,—a blunder which occurs only too often in medical practice.

Deviations from these rules occur tolerably often; but this much can be asserted with certainty, that children, in whom the above groupings occur, as to time and succession, have the least trouble, and encounter no serious disturbances in teething.

Having studied the physiological eruption of the teeth, we

turn to the symptoms which are likely to attend upon it, to excite more or less concern, and which are designated, in general, as troublesome teething, *dentitio difficilis*.

With regard to the local symptoms, they may be aggravated in two different directions. First, the physiological catarrhal stomatitis may pass into ulcerative stomatitis; and, secondly, the secretion of mucus and saliva may increase so much that these fluids may flow for weeks from both corners of the mouth in an almost continuous stream, and soak the child's clothing.

Ulceration occurs with greatest frequency on the tip of the tongue, which is exposed in the most direct and continuous manner to friction from the new teeth. Usually a single flat, round ulcer is found, with a yellow lardaceous bottom, and with somewhat infiltrated edges, which is extremely painful to the touch, and which, therefore, greatly embarrasses all the movements of the tongue. Sometimes it heals within a few days, but in other cases it continues for weeks, and its cure may be accelerated by touching it with lunar caustic. Ulcers on other portions of the mouth are less frequent and less obstinate in teething children.

All teething children have a great desire to bite upon something hard, and for this purpose often put their fingers in their mouths. The althea-root, so much employed in the nursery to satisfy this need, is harmless as long as it is fresh; but when fermentation and decomposition has once set in, it must serve to increase the catarrhal stomatitis. It appears more desirable, therefore, to let the children bite upon a silver thimble firmly secured to the finger.

The abnormally *increased secretion of the fluids of the mouth* must be attributed, for the most part, to irritation of the mucous glands, and it has frequently this unpleasant result, that the chin and the anterior portion of the neck become reddened or even eroded if the drooling is of long continuance. Furthermore, there is a remarkably frequent concurrence of this slobbering with a pretty severe cough, so that the continuous soaking of the clothing, and the consequent chilling of the thorax, must be regarded as an etiological element of this bronchial catarrh. This is proven by the successful results of prophylaxis. If the

chest is protected by a layer of water-proof material, this cough is usually prevented or is quickly suppressed when it has already existed. These little disturbances excepted, excessive slobbering during teething can only be greeted as a good indication. Dangerous brain-symptoms are very rarely encountered in such children ; and even the usual intestinal catarrh acquires no alarming intensity, a result to be explained perhaps by the circumstance that, in these cases, the masses of mucus do not gain entrance into the stomach.

A rarer complication sometimes follows these purely local manifestations, viz., *conjunctival blennorrhœa*, which occurs most frequently during the eruption of the upper molars and canines, and which may perhaps be explained by simple extension of the irritation to the antrum of Highmore and the nasal passages. This view is a popular one, that has extended over the whole of Europe, giving to the upper canines the name of eye-teeth. Its unilateral manifestation and the absence of all contagion argues for the correctness of this etiology, so that there is not, as in other blennorrhœas of the conjunctiva, any occasion for alarm with reference to the unaffected eye. The lids swell extensively in a very short time, and it takes a good deal of trouble to expose the eyeball. The secretion is not so dark-yellow, or so thickly purulent as in real blennorrhœa, but more mucous, translucent, and stringy. The eyeball always remains intact, and the prognosis can at once be pronounced favorable with certainty, despite the serious swelling of the upper eyelid especially, and despite its painfulness. Recovery regularly follows after a few days, and no active treatment is required. Painful cauterization with nitrate of silver is here unnecessary. Cleanliness and dry warmth, best applied in the form of a dry herb-bag, answers fully.

The eruption of the teeth not only gives occasion to these topical irritations of the neighboring parts, but it also exerts an evident influence upon the entire organism, the bowels, the skin, the nervous system, and the body temperature.

Moderate diarrhœal stools during teething are evidently physiological, inasmuch as they occur in the majority of all teething children, and run their course without any injurious

results. They are explained in the simplest manner by the swallowing of large quantities of slobber, the saline constituents of which appear to act like a laxative. When moderate diarrhœa exists, other complications are hardly ever witnessed, and the dreaded brain symptoms especially are absent, so that there is no reason to treat such looseness of the bowels at once with astringents, much less with opium.

On the other hand, it cannot be denied that this simple intestinal catarrh very often affects the entire follicular apparatus of the intestinal canal, as well as the mesenteric glands, sympathetically, and thus, especially in artificially nourished children, provokes rapid atrophy and frequently death. The almost hourly recurring evacuations acquire a cadaverous odor, erode the anus and its surrounding parts, and produce a rapidly increasing emaciation. Vomiting is often added; the cavity of the mouth becomes covered with the membrane of thrush; great thirst, with entire loss of appetite, follows, and the abdomen is tympanitic. In short, the picture of follicular enteritis, with all its sad sequelæ, is produced, the further relations of which, as regards pathological anatomy and treatment, are to be found in my manual on the diseases of children.[1]

Although the diarrhœa may be in part explained by the saliva swallowed, we have no mechanical explanation for the cutaneous eruptions attending dentition. These appear usually in blonde children, with fine, smooth epidermis; and hereditary disposition thereto is not to be ignored. It is characteristic that, throughout all five teething periods, the form of eruption remains one and the same, and that the eruption either disappears entirely with the conclusion of each period of teething, or that it is at least notably improved.

The lightest form is:

a. Urticaria, an eruption of wheals, *pomphi*, exactly as by contact with the stinging-nettle, whence its name is derived. We understand by this, intensely itching, sharply-circumscribed tumefactions of the skin, from the size of a lentil to that of a bean, which show a surface but slightly prominent and flattened.

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[1] *Vogel*, Lehrbuch der Kinderkrankheiten. VI. Aufl. p. 132.

The wheals themselves are usually somewhat paler than the normal skin ; their areola is redder. The epidermis remains unaffected in all cases, except as it may be injured by continual scratching. The entire eruption fades away in a few hours without leaving any trace, not even a red point—which latter circumstance distinguishes it from flea-bites, the punctures of which remain recognizable for a longer time. Some dozen such wheals make their appearance daily, their favorite seats being the trunk and the extensor surfaces of the extremities where the epidermis is delicate.

b. Papular eruptions, *lichen*, and *prurigo* are much more obstinate and tormenting than the former variety, and the traces of scratching are so much the more defined. Their further description, pathological anatomy, symptomatology, and treatment would carry us too far. So also with the following variety.

c. Vesicular or pustular eruptions, *eczema*, *and impetigo*, as the so-called *crusta lactea* on the hairy scalp of nurslings, is one of the most frequent manifestations during teething ; they usually run an acute course—that is to say, last from six to eight weeks, and become milder, or even disappear altogether during the pauses between the individual periods of teething.

The worst and most dangerous of all the complications attending dentition are decidedly those belonging to the nervous system. We have here principally to do with convulsive phenomena. Sometimes it is general eclampsia, sometimes *slight spasms* confined to special groups of muscles. The latter are extraordinarily frequent, and are noticed to a slight degree in every child. So, for example, the sleeping with eyes half-open, evidently a slight contraction of the levator palpebræ superioris muscle, is almost constantly to be observed. The eyeballs are here directed upwards, and only the white sclerotic can be seen through the tolerably widely gaping slit between the eyelids, producing an appearance which is unnatural and alarming to the laity. In unquiet sleep slight contractions of the muscles of the face occur, so that the countenance assumes a smiling aspect ; and slight movements of the extremities occur also in most teething children. The above-described spasmodic contractions are usually of short duration, and without any bad results.

They are only an evidence that the nervous system of the children is very much disposed to reflex influences, and that this disposition is decidedly increased during dentition.

An entirely different state of things exists in *general eclampsia*, which suddenly attacks entirely healthy children, and which consists of a succession of general tetanic spasms, like multiple electric shocks. The individual eclamptic attack is in no wise to be distinguished from an epileptic fit. It usually commences with tonic spasms. The head is thrown backwards, and the back arched; the extremities are rigid, and respiration is suspended. Then the general convulsions set in; the reddened countenance becomes hideously distorted, bloody foam appears upon the lips, a general perspiration breaks out, and the resumed respiratory movements are unrhythmic and deeply sighing. Consciousness is wholly in abeyance, and the child does not respond to the most painful irritation. These convulsions often cease after a few minutes; sometimes they continue uninterrupted for several days; sometimes they pass off without leaving any traces that they have existed; but very frequently they leave behind partial, so-called essential paralyses, squint, or even idiocy, and they often terminate immediately in death.

It is self-evident that the teething represents only one of the many causes of reflex spasm; and many children are also thus affected, for example, at the commencement of the acute exanthemata and many other febrile processes, although they already have their twenty milk-teeth, and no permanent teeth are to be expected. But these nervous manifestations correspond so frequently with our periods of dentition that an intimate connection between the two is not to be denied. A more complete symptomatology and therapy cannot be given here, but it is to be found in special works upon the pathology of the nervous system.

Finally, one more was referred to among the general symptoms of dental irritation, viz., *elevation of the body temperature.* This may have its origin as well in the affection of the mouth alone, as also in its various complications; it never reaches such a height as to excite solicitude, and is seldom of long duration.

A somewhat extensive consideration and exposition of all the symptoms connected with dental irritation appeared to me the

more appropriate, because the question of their relative significance has led to extravagances in two opposite directions.

While the older physicians, and even many family physicians of the present day, who have made no special study of the diseases of children, designate as "difficult dentition" almost every pathological condition that befalls a child between two and thirty months old, there are, on the other hand, nihilists who claim that the occurrence of this set of symptoms at the same time with teething is a mere coincidence. Whoever has had much practice in the diseases of children can find in his own experience so many arguments against these last-mentioned views that only a want of professional acuteness of observation can explain the existence of such an opinion.

2. Stomatitis Ulcerosa.

The Formation of Ulcers in the Cavity of the Mouth.

For the older literature vide *Canstatt's* spec. Pathol. u. Therapie; and *Bamberger*, Virchow's Handbuch d. spec. Pathologie. Bd. VI. Abth. 1.—*Böcker*, Das chlorsaure Kali gegen Salivation. Allg. med. Centralzeitg. 1858. No. 26.—*Innhauser*, Kali chloricum. Wien. Ztschr. 1858. No. 41.—*Bergeron*, Stomatitis ulcerosa of soldiers. Union méd. 1859. No. 54.—*Kussmaul*, Untersuchungen über Mercurialismus. Würzburg. 1861.—*Herz*, Ueber einige Krankheiten Neugeborner. Wien. med. Presse. VII. 4–7. 1866.—*Bohn*, Die Mundkrankheiten der Kinder. Leipzig. 1866.—*Andrieu, E.*, Traité complet de la symptomatologie etc. de la bouche. Paris. 1867.—*Legrand, M.*, Stomatite et glossite idiopath. Union méd. 1870. No. 65.

As soon as the mucous membrane of the cavity of the mouth shows loss of substance at any one point, in addition to congestion, swelling, pain, and increased secretion, *catarrhal stomatitis* ceases and *ulcerous stomatitis* begins. There are various ulcers of the mouth, which are to be distinguished from each other in part by their anatomical constitution, color, form, and depth, in part by their more or less chronic course, and in part, finally, only by their etiology.

If, for the sake of their separate consideration, we leave out syphilitic, scorbutic, lupous, variolous, and leprous ulcers, there remain but two sets: first, the simple progress of catarrhal

stomatitis to aphthous ulceration; and, second, cancrum oris or stomacace, to which mercurial stomatitis must be added.

We may also, following the most prominent sign of distinction between the two, speak of, first, ulcers with, and second, ulcers without fetor of the breath.

Among the various ulcers of the mouth above referred to as being dependent on some dyscrasia, only those of a scorbutic character will here receive our attention, inasmuch as the others are only slight partial manifestations of dyscrasias, which do not necessarily localize themselves in the cavity of the mouth. They will also undoubtedly be fully discussed by the writers who have undertaken those portions of this work to which they naturally belong.

A.—STOMATITIS APHTHOSA.

By aphthous stomatitis we mean catarrhal stomatitis, with the addition of little, flat, yellow ulcers, usually acute in their course, favorable in their termination, and unaccompanied with fetor.

Etiology and Pathogeny.

The fact that many acute ulcers of the mucous membrane of the mouth appear in company with labial herpes, and evidently have the same etiological significance, was more particularly set forth in the first section, when treating of herpes labialis (p. 734).

Stomatitis aphthosa occurs chiefly in children; and in the form of follicular ulcers, in women during menstruation, pregnancy, or lactation, so that the older authors saw reason for accepting a specific stomatitis vesicularis materna.

The etiology of this affection is, on the whole, the same with that of catarrhal stomatitis. All said there, is true here also. If the irritation increases to such a degree that not only swelling of and increased secretion from the mucous membrane occurs, but also cellular infiltration into this membrane, and into the submucous tissue, we have a throwing off of the epithelium and the formation of superficial ulcers.

Similar ulcers of the cavity of the mouth occur also in men, from the direct transmission of the discharges from the mouth and hoof of distempered cattle. According to Roell, the use of milk from cows thus diseased is sufficient to cause ulcers of the mouth in the human subject. (Consult Bollinger, this Cyclopædia, Vol. III.)

Finally, it is not to be forgotten that scrofulous children—that is, children of tuberculous parentage—are far more frequently affected with aphthæ than those who are the subjects of no dyscrasia, and the aphthæ in these cases are to be regarded only as one of the manifestations of scrofulosis, the same thing being true with regard to phlyctenular conjunctivitis and scrofulous keratitis.

Symptoms.

Most non-professionals and many physicians speak of vesicles in the mouth ; but I have never, even upon the most careful examination, discovered a real vesicle, upon the mucous membrane of the mouth, one which, upon puncture, discharged thin fluid contents, and then collapsed. Formations are undoubtedly seen upon the mucous membrane of the tongue which bear clear resemblance to vesicles. Sometimes an isolated fungiform papilla, at the tip or anterior border of the tongue, swells very considerably and is raised up, as a little, whitish segment of a sphere, above the surrounding mucous membrane ; sometimes, also, a mucous follicle acts in the same way ; but these apparent vesicles never collapse when they are punctured. When, therefore, we read, as we so often do, that ulcers of the mouth originate from vesicles, this appears only to be a theoretical transference to the mucous membrane, of the process in the cutis, where, indeed, most ulcerations begin at first, with a watery elevation of the epidermis.

This much is positive, that a few hours suffice to change a previously healthy mucous membrane into an aphthous ulcer. These ulcers are situated chiefly on the mucous membrane of the lips and of the cheeks, especially where it is reflected on to the gums, less frequently on the gums themselves, on the palate, or

on the tongue. Their floor is whitish yellow, their edges are darkly reddened, somewhat raised, and the entire ulcer is somewhat elevated, by reason of the adjoining catarrhal stomatitis. When these ulcers proceed from a follicle (Billard's *stomatitis follicularis*) they are small, circular, and excavated ; otherwise they are oval, and may be as large as a bean.

The sensitiveness of these places is very much increased, and they thus interfere materially with speaking, chewing, and other movements of the mouth. The secretion of mucus and saliva is greatly increased ; the odor, however, as already remarked in the introduction, is but little annoying, and has in no case the nauseous intensity that exists in stomacace.

The course of the individual aphthous ulcer is mostly favorable. After a few days, the lardaceous floor reddens, and then becomes covered again with normal epithelium. The diseased spot is, it is true, still recognizable for some time by its darker redness, but there never occurs any actual cicatrization with contraction. These conditions are of course different when the causal irritation is of long continuance ; for example, the sharp corner of a tooth, or the eruption of the wisdom teeth, which is sometimes attended with very obstinate ulceration in their vicinity.

Relapses are very frequent, and many people suffer several times a year with this troublesome malady.

Besides these benign aphthæ, there appear in weakly children, especially those in lying-in and foundling establishments, very ominous ulcers upon the hard palate, which are always situated at one and the same place. Regularly at the point of transition from the hard to the soft palate, there appears first reddening, and very soon after an oval yellowish exudation within the mucous membrane. The overlying epithelium is very soon lost, and there remains only a flat ulceration, which mostly remains superficial, but which sometimes extends to the bone. These ulcers have very little tendency to heal, and usually persist until death, which is brought about by profuse diarrhœa and follicular enteritis.

Diagnosis.

With regard to the differential diagnosis, it must be distinctly understood that in these cases there is never any appearance of a diphtheritic exudation, nor of a thrush membrane, and therefore that all forms of stomatitis attended with the formation of a more lasting membrane are thrown out of consideration. An aphthous eruption is distinguished from stomacace by its seat and by the absence of fetor. Stomacace is located principally upon the border of the gums, and its ulcers are distinguished by their brownish, red, dirty floor, and by their quick decomposition. Syphilitic and scorbutic stomatitis have such decided local and general symptoms, that to mistake them for simple aphthæ is hardly possible.

Treatment.

As these simple ulcers of the mucous membrane, as a rule, heal spontaneously in a few days, when no local irritation protracts their duration, there is no occasion, in most cases, for any active treatment. The desire of the patient to be relieved as soon as possible from the annoying pains, which are much increased by speaking and chewing, can be gratified by a light application of the stick of lunar caustic, or of dilute muriatic acid, one part to three. The momentary pain produced by the application, however, is very intense; and most patients, women and children especially, in subsequent attacks, decline that method of shortening their sufferings.

Frequent rinsing the mouth with cold water, or with lukewarm water, in cases of great sensitiveness, is always judicious, and at any rate fulfils the indication to remove in good time the profusely secreted mucus and saliva. In some cases decided benefit follows from Pfeufer's method of rinsing with a weak solution of corrosive sublimate (one-tenth of a grain to the ounce). This treatment, however, it is self-evident can only be resorted to in the cases of intelligent adults, on account of the toxic effect of the sublimate in case it should be swallowed. Chlorate of potassa has here no decided action, either as a mouth-wash or

as an internal remedy; and as a mouth-wash especially it provokes too great pain to be used for any length of time.

B.—PUTRID SORE-MOUTH, STOMACACE, STOMATITIS MERCURIALIS.

By stomacace (Canstatt's Fégar) is understood a contagious, rapidly destructive ulceration of the border of the gums, which produces extensive swelling of the entire mouth, starting from the point of ulceration, and a cadaveric breath.

Etiology.

Stomacace is mainly a disease of children, and especially attacks older children, who have already completed their first dentition. In some cases contagion can be proved, and several children in the same house frequently come under treatment at the same time. Apart from children, it appears epidemically, among soldiers especially—a new evidence of which is afforded by an epidemic occurring in 1855, in the garrison of Roule, which Bergeron has described in detail. In an etiological point of view, two circumstances are of special weight: overcrowding in narrow quarters, and contagiousness. The influence of the former is uncontested; that of the latter has been doubted on many sides. Bergeron sought to furnish the evidence that ulcerous stomatitis is a contagious disease, and transmissible also by direct contact; to which end he undertook inoculation upon himself.

He dipped the point of a new lancet into the pus of one of the ulcers, and inoculated the mucous membrane of his lower lip with it. In the evening a small pustule appeared at this point, which, the next day, faded away. Six days now passed without any further recurrence, but on the seventh day, following a cold, he had a chill, nausea, and, an hour afterwards, an unpleasant heat in the mouth, especially upon the tongue, on which there was developed a complete ulcerous stomatitis, accompanied with profuse salivation. There were no other disturbances of the general system. At the end of three days the ulcers began to heal, but there still remained a sensation of heat

in the mouth, congestion of the isthmus of the pharynx, and some difficulty of deglutition sixteen days after the inoculation, and, three days after the subsidence of every symptom of disease, a slight recurrence began, with fever. Twenty-seven days after the inoculation the general health was entirely normal again, and continued to remain so. A second inoculation to which a student exposed himself remained without result.

All epidemics described in the annals of military surgery occurred in their greatest frequency between the months of April and December. Although, according to Bergeron, the disease is always endemic in the French army, the influence of the warmer portions of the year upon its extension and epidemic nature cannot be disputed. In contradistinction to this, it is worthy of remark that diphtheritic affections of the mouth are more prevalent in winter.

Among the predisposing influences are dental fistulas and the insufficient nutrition of the French soldiers, which appears to lack in variety and in alcoholic beverages. Recruits are affected most frequently, non-commissioned officers to a less degree, and officers extremely seldom.

The declaration of Hirsch must be noticed, to the effect that the disease is to be observed not only in crowded barracks, but also among troops in the field and in camp, where insufficient ventilation must be thrown out of the question.

Though we have thus far studied stomacace as a more or less epidemic disease, the very same process may occur sporadically in any individual who has taken mercury in any way. Whether the mercury comes in direct contact with the mouth, which occurs most frequently in the careless internal use of calomel, whether it is taken in the stomach, or by the skin—inunction-cure, white precipitate ointment, washings with a solution of sublimate, cauterization with Plenk's paste—whether, finally, it is inhaled into the lungs in the form of mercurial vapor or mercurial dust—by mirror-coaters, gilders, hat-makers, barometer-finishers, chemists, or miners—in all cases the earliest and most frequent symptom of intoxication is a disease of the gums, which can in no wise be distinguished from idiopathic stomacace.

Mercurial stomatitis may evidently occur in several ways, according to the manner of application of the poison. It is explained in the simplest manner by mechanical contact of the cavity of the mouth with mercury, in the careless use of calomel in powder, and gray and other mercurial ointments, and in all handicrafts in which the hands become soiled with mercury. The prolonged avoidance of such contamination is exceedingly difficult; and the most thorough washing even may not remove all traces of the poison from beneath the finger-nails and from the hairy portions of the body.

In the internal use of mercury, in the form of pills, the mercury must first pass the mesenteric glands and the liver. It is experimentally established that—1st, the mercury which reaches the liver is eliminated with the bile; and, 2d, that the glands are capable of retaining a good deal of quicksilver.

When mercury is taken up through the skin, it must first pass through the lymphatic glands before reaching the general circulation; while, when breathed into the lungs, it gains direct access into the blood, and is excreted by preference through the saliva. We have, then, in the first case, a direct contamination of the cavity of the mouth; in the second, a partial elimination and retention by the liver and the lymphatic glands; and, finally, in the third, an inevitable excretion through the salivary glands, and with it, again, secondarily, mechanical contact of the poison with the cavity of the mouth.

The salivary and mucous glands of the mouth play an active rôle in the elimination of mercury; and it can be detected, chemically, in the fluids of the mouth long after the abandonment of the remedy, as will be more particularly shown in the section on salivation.

That the medical use of mercury usually entails only severe ulcerous stomatitis and salivation, while, on the contrary, mercurialism contracted through handicrafts produces usually constitutional disturbances, such as tremors, paralysis, cachexia, etc., is explained by Kussmaul on the ground of the slight but long-continued absorption of the material in the latter cases, while, for medicinal purposes, large quantities are usually introduced within a short time.

Pathological Anatomy.

From an anatomical point of view, I must, with Bergeron,[1] deny any analogy between this affection and pseudo-membranous stomatitis. The essential nature of the process is an ulcerative destruction of the mucous membrane; and if, here and there, membranes do appear in high grades of mercurial stomatitis, they do not cover a normal, but always an ulcerated mucous membrane, and they are rather the superficial layers of the tissue itself than deposits upon it.

Microscopic examination of the ulcerated borders of the gum reveals pus-corpuscles, isolated blood-corpuscles, and granulated cells, all imbedded in an amorphous, finely granulated mass which swarms with bacteria and micrococci.

The localization and extension of stomacace is characteristic. The process ordinarily begins at the margin of the gums of the lower jaw and stretches backwards from the incisor teeth to the molars, for the most part only on one side. If there is a gap in the teeth, the ulceration creeps around the tooth into the inner surface of the jaw, and does not immediately leave the border of the gums. Very soon, however, those portions of the mucous membrane of the lips, cheeks, and tongue, directly over these parts, become affected and show the same sort of ulcers as the gum itself. The surface of the tongue, the hard and soft palate, as well as the pharynx, always remain free from stomacace. The process penetrates more deeply into the gums than into the cheeks, and utter destruction of the gums, with looseness and falling out of the teeth, results, not unfrequently, in neglected cases.

Symptoms.

The first symptom is always a marked fetor of the mouth, with great sensitiveness of the gums, which is increased by pressure, chewing, and all movements of the lips and cheeks. If the painful places are examined, the gums, around the incisor teeth of the lower jaw especially, are found reddened and swollen.

[1] Union médic. 1859. 54.

The sharp border of the gum becomes puffed out, and exhibits a grayish-yellow edge, which, on touching it with a needle, is shown to be a semi-fluid mass. In this are found, microscopically, the above-mentioned traces of rapid ulcerative destruction. Distinctly acid secretions flow from the mouth in large quantity, so that patients are hardly asleep ere they are awakened by the wet, ill-smelling places formed on their pillows.

We are not speaking here of specific mercurial salivation, which will be more thoroughly discussed when considering the diseases of the salivary glands, but of the increased secretion of the mucous glands produced by the stomatitis.

If the finger is lightly passed along the gum, it makes its border bleed at various points. Slight hemorrhages often occur spontaneously, too, and color the saliva brownish-red. Between the teeth and upon them there appears a yellowish unctuous mass, which presents the same microscopic appearances as the yellow edge of the gums already described. The somewhat swollen tongue is always thickly coated, and shows at least distinct impressions of the teeth, very often also marginal ulcers. The most adjacent lymphatic glands of the throat always become somewhat swollen.

The train of symptoms just described is completed in from two to three days, and may be entirely removed in as many more by the liberal use of chlorate of potassa. But if the case does not improve, it grows visibly worse. Distinct broad ulcers covered with a gray pulp appear in the place of the yellow margin. This pulp is sometimes membraniform, especially in mercurial stomatitis; and on this account ulcerative stomatitis is also called diphtheritis by many authors. But in the progress of the disease its difference from diphtheria becomes decidedly evident.

The marginal ulcers surround the teeth more and more completely, and finally the gum is destroyed clear to the jaw-bone. The teeth thus laid bare, totter in their sockets, and can finally be removed without pain and without hemorrhage—an evidence that the destructive process must have also penetrated into the sockets, and destroyed artery and nerve.

During this time the ulceration has extended in depth and in breadth upon the mucous membrane of the lips, cheeks, and

tongue in contact with the diseased gums. Sometimes the ulceration on the cheeks corresponds so exactly to the border of the gums that it consists of but two narrow strips running parallel with the upper and lower jaws. These ulcers are covered with a thick, grayish-white, or, when there is hemorrhage, reddish-brown, unctuous mass of exudation, which is thicker on those spots of the tongue where it lies between two corners of teeth. This exudation is cast off later, and exposes deep ulcers, with irregular, readily bleeding edges, and grayish-yellow lardaceous floors. The surrounding connective tissue is considerably infiltrated, and the cheeks in consequence are extremely swollen, and the lips strongly puffed up. The pain becomes so intense that chewing, swallowing, and even speaking, are altogether impossible; and even the slightest contact and rinsing with water increases it beyond endurance.

In the most extreme cases of mercurial stomatitis—such as should no longer occur since the introduction of the use of chlorate of potassa—Bamberger[1] states that even widespread gangrenous destruction takes place. A putrid slime of cadaveric odor forms, the denuded jaw-bone becomes necrotic, the teeth fall out, the tongue swells to an alarming degree (see Glossitis), and the patients fail visibly. Death even follows under pyæmic symptoms, metastatic inflammations, colliquative diarrhœa, and erysipelas.

The course of idiopathic stomacace is in general a very favorable one. The odor is subdued without a trace remaining after twenty-four hours' use of chlorate of potassa; the ulcers clean off, and heal usually without cicatrization. Repetitions occur, but true relapses are unfrequent. Fever is hardly ever present. Dejection of spirits and ill-humor are natural enough, and are very pronounced as long as the movements of the mouth produce severe pain. In a few cases only is there a decided disposition, from the start, to partial necrosis of the jaws. In these, while the general symptoms subside during the first few days, on the use of chlorate of potassa, the ulcers remain unaltered at some point, and after a few weeks a smaller or larger fragment

[1] L. c. p. 32.

of the margin of the jaw is thrown off. In these cases, also, complete recovery takes place; generally, however, with the permanent loss of one or of several teeth. Without the use of chlorate of potassa, simple, putrid sore mouth generally reaches a high degree, and heals only after a course of several weeks of unspeakable torment.

Diagnosis.

Mistakes are not readily possible where the symptoms are so striking. Aphthæ are distinguished from stomacace by the absence of a cadaveric odor, by not commencing at the margin of the gums, by always preserving the yellow color of the floor of the ulcer, and by spontaneous recovery in a few days, except in the case of cachectic new-born children.

If a patient with syphilitic ulcers of the mouth and vegetations acquires mercurial stomatitis, as the result of mercurial treatment, it is difficult for the inexperienced to distinguish the traces of syphilis from those due to the mercury. The occurrence of syphilitic ulcers in the pharynx and on the palate, their stability and tendency to perforation, their hard callous edges, and their slight disposition to hemorrhage, serve as guiding points. Syphilitic vegetations almost never occupy the gums, are somewhat prominent above the normal mucous membrane, and exhibit white, painless projections, which have no resemblance to stomacace.

Stomacace is distinguished from simple dental ulcer, *parulis*, by the latter remaining localized upon one individual tooth, producing a deep-seated swelling, and finally showing an abscess, after the opening of which a fistula, with granulating edges, is frequently established.

Scorbutic affections of the mouth have this in common with stomacace, that they are also confined to the gums in the beginning, and that the process terminates also with extensive ulceration and with loosening of the teeth. In scorbutus, however, the swelling and œdema of the gums is much more extensive. It takes on a dark bluish color, and frequently acquires such dimensions that it rises high above the crowns of the teeth and conceals

them. There is never anything of a yellow border to be seen.

Noma, mortification of the cheek, shows very rapid gangrenous destruction, and seizes primarily on the mucous membrane of the cheek, not the gums. In two or three days the cheek is already black and mortified, and then a mistake is no longer possible.

Treatment.

If we read the treatment of the older authors for putrid sore mouth and mercurial stomatitis, which were regarded by them as different processes, we may take it for granted from the variety of remedies employed, their heterogeneous physiological action, and the severity of the symptoms described, that the treatment was very unsatisfactory. Gargles of every sort were tried; at first those of a demulcent or mucilaginous character, both with and without the addition of opium; then the astringents with alum and tannic acid, either pure or prepared from various barks and roots; then sugar of lead, and the salts of zinc; finally, disinfectants containing chlorine, chlorinated lime, creosote, resin, oil of turpentine, and tar.

The ulcers were touched with caustics of every sort; by some practitioners purgatives were given internally; by others diaphoresis was promoted to the utmost; by a third class, the use of the so-called specifics was tried, such as iodine, sulphur, sulphate of lime, chlorine preparations, camphor, the mineral acids, and even tartar emetic. Syphilitic patients, especially, had a good deal to suffer under the manifold mercurial cures, and it required a high degree of patience and perseverance on the part of both patient and physician to carry a mercurial treatment through, in spite of such tormenting complications.

To-day, the sovereign remedy for syphilis, the inunction-cure, consisting of the daily inunction of one drachm of the ointment of mercury, has become a highly convenient and harmless procedure, which can be continued uninterruptedly for three or four weeks without any discomfort or evil results; and this revolution in practice is alone due to the use of chlorate of potassa.

When, at the same time, the patient takes every hour a tablespoonful of a ten-grain solution of chlorate of potassa, the inunction hardly produces any odor from the mouth, the gums become but slightly reddened, and never advance to ulceration unless there are too many carious teeth. The swallowing of the solution is much safer, more efficacious, and more convenient than wasting time in gargling and rinsing the mouth.

We have, then, in this remedy an eminent prophylactic against mercurial stomatitis, through which the indication for the inunction-cure has decidedly extended within the last ten years. At the same time it is also *the only positively effective remedy* for already existing stomacace, whether occurring spontaneously or from the use of mercury.

If actual stomatitis sets in during an inunction-cure, it is only necessary to suspend the inunctions for a few days, and to give meanwhile at least one drachm of chlorate of potassa daily, when the patient will be in a condition to resume the inunctions. The chlorate of potassa does not appear to have any decided power to cut short the flow of saliva.

The dose for children must be proportionately reduced, as a matter of course. A child two years of age should not be given more than fifteen grains, one of four years not more than thirty grains daily. After twelve hours' internal use of it, the fetor is completely gone, and if the stomacace has been of but slight extent and short duration, recovery begins at once. The gum does not bleed so readily any more; it becomes firmer and less painful. In all cases, even the mildest, the remedy must be continued for at least three or four days. No toxic effects are ever seen from these doses; and as the first incidental result is a somewhat increased intestinal secretion, which at once subsides under decreased doses, it is safe in all cases to push the remedy until the appearance of looseness of the bowels.

It is known from Karmel's labors (vide Stomatitis catarrhalis p. 768), that tolerably large quantities of chlorate of potassa are also absorbed when it is only used as a gargle. But as gargling is rather tiresome and fatiguing, besides being hardly practicable with young children, and as prolonged contact of the mouth-wash with the diseased mucous membrane is quite painful, I consider

that the internal administration of the remedy is safer and more convenient than its mere local use.

Before the introduction of this specific, the extraction of carious teeth, such as appear in almost every child after its fifth year, was considered absolutely necessary. At present this is by no means indicated; it rather delays recovery, inasmuch as new ulcers form at the wounded points, which resist treatment longer than the old ones.

In adults the most solicitous care of the teeth, during the employment of mercury, and frequent rinsing of the mouth with fresh cold water, are always advisable.

3. Scorbutus of the Cavity of the Mouth.

Forget, Médecine navale. Paris. 1834.—*Ozanam*, Hist. méd. des épidemies. T. IV. p. 99.—*Krebel*, Erkenntniss und Heilung des Scorbutes. Leipzig. 1838.—*Rochoux*, Diction. de Méd. Vol. XI.—*W. S. v. Himmelstiern*, Häser's Archiv. Bd. V. 488.—*G. v. Samson u. Himmelstiern*, Beobachtungen über den Scorbut. Berlin. 1835.—*Reuss*, Dissert. Scorbut. Würzburg. 1843.—*Cejka*, Prag. Vierteljhrschrft. 1844.—*A. Fauvel*, Mém. sur le scorbut, observé à Salpetrière. 1847. Arch. gén. d. méd. 1847. Juillet.—*A. Lilienfeld*, Erfahrungen über den Seescorbut. Casper's Wochenschrft. 1851. 1–3.—*Leudesdorf*, Ueber Landscorbut. Allg. med. Centralztg. 1857. 80 u. 82.—*Paul*, Scorbut in Gefängnissen. 33. Jahresber. der Schles. Gesellschaft. f. vaterl. Cultur.—*Duchek*, Scorbut. Wien. med. Jahrbücher. 1861. I.—*Senftleben*, Ueber Land- und Seescorbut von Dr. Barnes. Deutsch. Klinik. 1868. 4–6.—*Lacascade*, Quelques considérations sur le scorbut. Thèse. Montpellier. 1869.—*Hayem*, Les corbut observé à la Charité. Gaz. hebd. 1871. No. 14–18.—*Delpech*, Le scorbut pendant le siége de Paris. Annal. d'hygiène pub. 1871, avril.—*Leven*, Sur une epidémie de scorbut. Compt. rend. LXXV. No. 5. 1872.—*Da Costa*, On scurvy. Phila. Med. and Surg. Rep. 1872, Nov. 16.

By scorbutus we understand a chronic, general disturbance of nutrition, in which, with increasing debility, numerous smaller and larger hemorrhages occur in various regions of the body, being most constantly observed in the gums.

Etiology and Pathogeny.

This is not the place to recount the numerous hypotheses

concerning the production of scurvy, inasmuch as the whole process with all its localizations will be spoken of in detail in another portion of this cyclopædia.

Only this much need be here remarked, that scorbutus, so extraordinarily frequent in the Middle Ages, has become less frequent in our own times. The most recent detailed descriptions come from Paris, where, during the late siege, scurvy acquired powerful proportions. In an etiological point of view, we have always distinguished a sea-scurvy from a land-scurvy, which, however, cannot be separated from each other in any way, excepting by the difference in cause.

Sea-scorbutus appears during long sea-voyages upon sailing-vessels and as a consequence of insufficient and monotonous diet. Land-scorbutus appears in times of famine, in places undergoing siege, and in poorly-ventilated prisons, factories, and barracks. But the subject is not so simple, nor its connections so direct, as they are ordinarily supposed to be ; inasmuch as it frequently does not appear in spite of all these causes, such as the poorest nourishment, with cold, moist dwellings ; and, on the other hand, does appear in the form of little epidemics among well-nourished troops or in cleanly prisons and hospitals. That a gloomy and anxious state of mind may have an influence in determining the onset and course of scurvy has often been distinctly observed in sea-voyages and during wars. Other things being equal, tipplers are affected sooner and more strongly than temperate men. Convalescents from typhus, acute exanthemata, or surgical diseases, especially in military hospitals, frequently sicken with mild forms of scorbutus. Leudesdorf,[1] who acted as surgeon in the Turkish army during the Crimean war, reports that in the Turkish hospitals, which contained about 3,000 sick out of 25,000 troops, scorbutus became added to every possible surgical injury, to fractures, and to the wounds of venesection, cupping, and blisters. Among very old people dwelling together in masses, as in the beneficiary department of the Salpetrière in Paris, epidemics of scorbutus, according to Fauvel, often occur.

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[1] Allgem. med. Centralztg. 1857. 80 u. 82.

Pathological Anatomy.

The alterations in the cavity of the mouth are confined particularly to the border of the gums, so far as teeth are present. Sometimes these alterations are observed only on one side, sometimes in connection with but a single tooth or with several teeth. The tongue and posterior portions of the cavity of the mouth always remain free of ulcers. The mucous membrane of other parts of the mouth, especially that of the hard palate, always shows a striking pallor, upon which appear spots of ecchymosis and small vesicles the size of a pea filled with bloody serum.

The malady begins with a bluish-red border on the gums, which immediately become considerably swollen. The jagged projections, between the junctions of the teeth especially, degenerate into misshaped knobs, which can be readily bent outwards by the probe. This growth may acquire such dimensions as to cover the teeth completely, and to be raised above them in a cushion as thick as the finger. Microscopically this growth appears formed only of a bloody serous infiltration, with immense increase of dilated capillaries; besides which copious large and small hemorrhagic extravasations are found strewed about on all sides. After a few weeks these growths degenerate into a soft, pasty mass, giving way under the pressure of the finger, after the detachment of which an ulcer remains. This breaking down of tissue is inevitable in all severe cases, on account of the serous tension, the extravasation of blood, and the careless movements of chewing. If improvement begins, the swelling of the gum simply subsides, the gum recovers its former consistence and color, and resumes its position around the teeth. Exceptionally, it is said that new formations of connective tissue appear, in which case a prominent cicatrix is formed.

Symptoms.

The first indications of scorbutus are found, not in the mouth, but in the articulations of the lower extremities, which ache in such a manner as to remind one of the beginning of acute rheumatism. This pain appears with most constancy in the popli-

teal space, and on manual examination a circumscribed hardness, with a slight bluish color and somewhat elevated temperature, can early be detected. At the same time the patient complains of debility and loss of power in all his limbs, is much depressed and out of sorts, loses his appetite completely, and acquires a pale, sickly aspect.

Only after a few days does the patient remark pain on chewing ; the gums become very sensitive to every touch, and soon bleed slightly on contact. The smell from the mouth is offensive certainly, but it does not reach the intensity of the odor in stomacace. The secretions of the mouth are much increased, and the offensive saliva running out is colored brown by blood and broken-down tissue. Fever is not present. On examining the gums, the anatomical changes described in the previous section are seen.

In most patients the disease is developed equally on both sides. With the increase of the puffing and loosening of the gums, the teeth become loose also, and finally fall out, while those that remain become covered with a thick cheesy coating.

The patient's troubles increase in the most painful manner. Chewing is finally impossible, and extensive hemorrhages from the mouth ensue, even to as much as a quart in the twenty-four hours. The discharged blood coagulates only very incompletely, and is of a dark blackish-red color. At the same time the parenchymatous hemorrhages into the mucous membrane of the tongue and cheeks increase in number and in dimensions, and finally project like blisters or fungus-growths above the level of the rest of the mucous membrane. Bleeding also occurs into the papillæ of the tongue, and produces a peculiar raspberry appearance of the surface of the organ. Finally the fungus-growths of the gums disintegrate and the characteristic scorbutic ulcers are formed in their place. The edges of the ulcer are irregularly jagged, and set with fungous excrescences ; its floor is of a dirty brown tint, or is colored dark-red by fresh blood. The disintegrated mucous membrane hangs in places on the basis of the ulcer in brownish-black shreds ; and a diphtheritic layer sometimes covers the entire ulcer.

Finally, if scorbutus reaches its utmost intensity, producing

great changes in the remaining organs and a high degree of cachexia, it proceeds to veritable gangrene in the mouth. Large pieces of the mortified gums are discharged at one time, and the remainder is changed into a pasty villous mass; the necrosis seizes upon the jaw-bone itself, and the cadaverous odor poisons the entire atmosphere of the patient's apartment. Finally, after suffering from large internal hemorrhages, excessive anæmia, and general dropsy, the patient dies.

In case of recovery, which may occur at any time under favorable conditions, the hemorrhages cease, the swelling of the gum subsides, many teeth, already tottering, become firm again, and existing ulcers cicatrize. If the case had advanced to actual destruction of the mucous membrane, there will naturally remain a contracted, radiated cicatrix.

The course of the disease depends entirely upon external circumstances. Even the severest case may recover when brought under favorable conditions, while under the bad hygienic surroundings which give rise to scorbutus, only a progressive aggravation of the disease is to be expected.

Diagnosis.

The local symptoms of scorbutus are so evident and manifold as hardly to leave any chance for mistake. The commencing destructive processes in the mouth might perhaps be mistaken for simple stomacace; but even here the absence of the bluish-red tumid swelling, and especially the much smaller tumor, and the presence of the yellow border in stomacace, furnish distinct differential characteristics. In addition, the destruction in the mouth is only a small portion of the general affection in scorbutus, while stomacace is a purely local process, which is never attended by disease of other organs.

Prognosis.

The danger of scorbutus is not usually as great and imminent from the manifestations in the cavity of the mouth as from the cachexia, the dropsy, the hemorrhages, and the inflammation of

the serous sacs, among which the pericardium particularly participates in the most serious manner. Death may ensue, however, from profuse hemorrhages from the mouth, gangrene of the gums, or necrosis of the jaw-bones, and the pyæmia thereby induced.

Treatment.

There is no specific for scorbutus, and a careful prophylaxis is therefore the most important point. This question has been ventilated in all large navies for centuries, and certain procedures have been recognized effectual as preventives.

During a long sea-voyage the nutrition must not consist exclusively of salt meat. The greater the heat or the cold, the longer the calm in the tropics, so much the more abundantly must fresh meat and fresh vegetables be given, in the preserving of which we have now made such progress. The favorite prophylactic is sour-kraut; then come water-cresses, horse-radish, lettuce, sorrel, scurvy-grass, and sour fruits of all kinds. The most desirable drinks are milk, fresh or condensed, good beer, wine, and a lemonade made of citric or tartaric acid, with some brandy added to it.

The clothing is of quite as much importance as the food. Care in the use of woolen under-clothing, and in changing garments after they have become wet, great cleanliness and efficient ventilation and disinfection of sleeping apartments are the most important prophylactic measures at sea; while avoidance of excessive exertion, staying in the open air, amusement, and the utmost possible cheerfulness, are chiefly to be aimed at in prisons and hospitals.

When scorbutus has once broken out, improvement of the surroundings and the diet, as above indicated, is of much more value than all medicinal treatment. Brewers' yeast, from four and a half to six ounces daily, certainly answers the best purpose. Besides this, the mineral acids, and a multitude of aromatic and bitter remedies, such as cinchona, gentian, buck-bean, hops, fumaria, calamus, wormwood, pomegranate-rind, cinnamon, and black mustard, have been recommended. Brechet

extols charcoal-powder, from three to six drachms a day, Bergmann tar-water and creosote.

Just as with all these internal remedies, so it is with the numerous anti-scorbutic gargles. If the patients remain under the baneful conditions which gave rise to the scorbutus, no beneficial result is to be seen from any local treatment, but if these are removed, the gums heal with tolerable promptitude under the general invigoration of the organism.

The favorite additions to the gargles are, vinegar, vegetable-acids, spirit of scurvy-grass, sage, walnut-leaves, oak-bark, rhatany, chlorate of potassa, and alum. In hemorrhages, the last is used with great efficacy, sprinkled as a powder; or the parts may be pencilled with chloride of iron solution; and in the worst cases the hot-iron may become necessary. In great tumefactions of the gums, Paul employs the scissors, the bold use of which he has never seen followed by any alarming bleeding. The ulcers of the mouth are touched with dilute hydrochloric acid (one part to six), or with a solution containing chlorinated lime, myrrh, or tincture of catechu, or with a stick of lunar caustic. Others especially extol sprinkling with powdered cinchona.

4. Thrush. Fungus Formations in the Cavity of the Mouth.

Billard, Maladies des enfants nouveau-nés. (German translation, Weimar, 1829.)—*G. C. Oesterlen*, Heidelberger klin. Annalen. 1831. Hft. 1.—*Guersant*, Muguet, im Dict. méd. Vol. XVIII.—*J. Vogel*, Allg. Zeitschrft. f. Chirurg. etc. 1841. No. 24.—*Gruby*, Compt. rend. 1842. T. 14. p. 634.—*S. A. Hönerkopf*, De natura vegetabili aphtharum. Gryph. 1843.—*Valleix*, Guide du méd. prat. Tom. IV.—*Kronenberg*, Journ. f. Kinderkrkhtn. IV. 164. VIII. 2; u. IX. 1.—*Trousseau et Delpech*, Journ. de méd. 1845. Jan.-Mai.—*F. J. Berg*, Ueber Aphthen bei Kindern. Trans. v. Busch. Bremen. 1848.—*Hauner*, Journ. f. Kinderkrkhtn. XVI. 215.—*Robin*, Histoire naturelle des végétaux parasit. Paris. 1853.—*Reubold*, Lehre vom Soor. Virchow's Archiv. 1. 1854.—*Bamberger*, Virchow's Hdbch. der spec. Pathol. Bd. VI. 1. Abthlg.—*Küchenmeister*, Die Parasiten. 2. Abthlg. 1855. p. 82. Leipzig.—*A. Vogel*, Zur Lehre vom Soor. Henle u. Pfeufer's Ztschr. f. rat. Medic. N. F. VIII. Bd. 2. 1856.—*A. Förster*, Handbuch d. spec. path. Anat. II. Aufl. 1863. p. 28.—*Steiner u. Neureutter*, Pädiatr. Mittheilg. Ref. in Schmidt's Jahrb. 1866. Bd. 130.—*Mettenheimer*, Die Saughütchen von Kautschuk. Memorabil. XI. 1. 1866.—*E. Wagner*, Zur Kenntniss des Soors. Jahrb.

d. Kinderheilkde. N. F. I. 1. p. 58. 1868.—*Hallier*, Parasiten. Absch. IV. p. 86. —*J. Schiffer*, Die saccharificirenden Eigenschaften des kindl. Speichels. Arch. f. Anat. u. Phys. 1872. p. 469.—Manuals on Diseases of Children generally.

Parasitic sore mouth—Soor of the Germans, thrush, muguet of the French, *stomatitis aphthophyta*—has no connection with any of the affections thus far spoken of; neither with inflammation nor with the formation of ulcers, but depends upon the abundant development of a microscopic fungus, *oïdium albicans*, which combines with the epithelium into thick, white membranes, and covers a great portion of the surface of the mouth.

Etiology.

The formation of fungus is always favored by acid fermentation, the tendency to which, in the mouths of nurslings, who are most frequently affected by thrush, is present even under physiological conditions.

The secretion of the mouth is a mixture of the secretions of various glands—the parotid, submaxillary, and sublinguai glands, and finally of innumerable mucous glands which are distributed over the entire mucous membrane of the mouth. The secretion of the salivary glands is well known always to have an alkaline reaction, more distinct after a meal, very indistinct on an empty stomach. Pure buccal mucus, on the other hand, such as is collected in animals, after ligature of the execretory ducts of all the salivary glands, has always an acid reaction; and this acid reaction is visibly increased on contact with atmospheric air, inasmuch as acid fermentation then begins at once.

According, therefore, as the quantity of the one secretion or the other predominates, the fluids of the mouth have at one time more the peculiarities of saliva, and at another more those of mucus. Much debate has been had upon the saliva of nurslings, since it was first announced by Bidder and C. Schmidt[1] that it was incapable of converting starch into sugar, and that even the substance of the glands exerts not the least action upon starch.

[1] Die Verdauungssäfte und der Stoffwechsel. p. 22.

The recent researches of Julius Schiffer, however, furnish positive results of an opposite nature. He placed well-washed tulle bags, filled with starch-paste, in the mouths of new-born babes before any nourishment was given, and allowed them to remain there for five minutes. After this time, Trommer's test gave evidence of sugar in every instance, while the pure fluids of the mouth never gave the same reaction. Schiffer acknowledges that the saliva of new-born children changes starch into sugar much more slowly than that of adults. These facts have been explained on the ground of the general absence of the salivary secretion, just as the secretion of tears, or at least crying with shedding of tears, does not occur in very young children. At all events, there is an undue predominance of mucus over saliva in the mouths of nurslings. To this may be added the fact that in children artificially nourished, who are affected with thrush much more frequently than children at the breast, the movements of sucking are lost.

While sucking at the nipple, the nursling draws the saliva from its own salivary glands as well as the milk from the breast, while in the slight sucking from a rubber teat or in feeding from the spoon this stimulus is not brought to bear upon the salivary secretion.

As a further promoter of acid fermentation in the mouth may be reckoned the sucking at those little bags filled with bread, milk, and sugar, which, especially among the poor, are so much employed to quiet screaming children, and which undergo fermentation in a very short time.

In lying-in and foundling institutions the communication of thrush from one child to another through the medium of a nurse in common, or the promiscuous use of artificial teats, drinking-glasses, etc., has been observed, though the affection is not, therefore, to be regarded as a directly contagious disease. The germs seem to be spread everywhere, and only require favoring circumstances to overrun the mucous membrane in great quantities. After the example of Remak, who cultivated favus crusts, I made experiments in cultivation more than twenty years ago.[1]

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[1] *Henle* u. *Pfeufer's* Zeitschrift f. rat. Med. N. F. Hft. 2.

If a piece of the thrush membrane is placed upon a slice of apple, covered with a bell-glass, and kept moist and warm, there appears, after from four to five days, on various portions of the slice of apple, a fine microscopic fungus-layer, the morphological elements of which can in no wise be distinguished from the spores and thallus filaments of thrush membrane. A parallel experiment without the parasitic membrane always gives negative results in this short space of time. The *oïdium albicans* of Robin is, according to Hallier, nothing else than the usual *oïdium lactis*, the fungus of the acid fermentation of milk, and belongs to the budding spores. These are distinguished by the size and elliptical, sometimes four-cornered, configuration of their individual cells or limbs, and acquire their name because they multiply by separation of processes, or budding. They are the leaven of acid fermentation, and form in all hydro-carbons, when they contain enough carbonic acid gas for the growth of a fungus, on the one hand, and sufficient oxygen can gain access to them, on the other.

These are sufficient grounds for the explanation of thrush in nurslings. In adults it occurs only in protracted, debilitating diseases, especially in phthisical, diabetic, and cancerous cases, and can be explained here also by anomalies in the chemical composition of the fluids of the mouth, accelerated acid fermentation, increased growth of epithelium, and the absence of the movements of chewing. The condition for its productions are the most favorable in warm seasons of the year and in damp residences. In these chronic patients true croup of the mucous membrane of the mouth, a formation of grayish-yellow membrane, consisting of molecular masses, of fat and of pus-corpuscles without thallus filaments, occurs more frequently than simple thrush. A detailed description of croup will follow later among the diseases of the pharynx.

Pathological Anatomy.

Before the membrane becomes visible, the mucous membrane of the mouth is very red and somewhat sticky ; *the secretion always gives an acid reaction*, and this acid reaction is evidently

not due to retained and soured milk, for when the mouth is thoroughly cleansed with water, and nothing is allowed to be introduced for an hour after, the acid reaction still continues. At this time already the mucus of the mouth invariably contains numerous spores of fungi, egg-shaped, sharply-outlined bodies, sometimes hanging together in twos and threes, which can be seen in every preparation if the mucous membrane is only lightly scraped. These spores appear only very isolated, or not at all, on the normal mucous membrane.

After a few hours white points, of the size of a pin's head, appear on the mucous membrane of the mouth, mostly at first on the inner surface of the cheeks, which quickly increase in number, and also appear in various other places; and as early as on the second day extensive white membranes are formed, which finally cover the entire cavity of the mouth, and even the pharynx and œsophagus.

Fortunately, the fungus confines itself rigidly to the squamous epithelium, a fact which was first remarked by Reubold, and thus always spares the larynx and the trachea. Even when the entire pharynx is covered with this membrane, it never extends to the vocal cords, and the patients thus remain free from any symptoms of stenosis of the larynx. After a long continuance the white membranes acquire a yellowish color, and even a brownish color if bleeding occurs from rough contact with them.

For the first few days the membrane sits tolerably firmly on the mucous membrane, and can hardly be removed without some injury to the latter; at a later date it becomes quite loose, and can be wiped from the mouth with a damp cloth in large quantities. The histological description of parasitic vegetation of the œsophagus given by Wagner answers thoroughly for that of the remainder of the cavity of the mouth. I once had the opportunity, in the post-mortem examination of a child, to examine the mucous membrane of the mouth, covered with a recent deposit of thrush. After the mucous membrane had been well hardened, on microscopic section, after coloring with carmine, I saw on the upper surface of the membrane a thick network of thallus filaments and spores imbedded in an amor-

phous granular mass. At several places no epithelium could be detected; at others the upper layer of the epithelium could be seen; the middle layer was entirely gone, the lower layer again, in part, retained. The thallus filaments are decidedly of two sorts: 1, broader, with many transverse striations, showing the exact form of yeast fungus; and, 2, smaller, slightly granulated, with almost no transverse striation, and little sharpness of outline. The latter fibres appear more numerous than the former.

The further growth of these fibres in depth is interesting. We see a tolerably large number of spores and thallus filaments penetrate into the surface of the mucous membrane itself, and into the mucous glands; and in favorable sections we can follow the individual filaments in as far as the smooth muscular fibres. Care must be taken to make the section from within outwards, from the lower towards the upper surface, because, otherwise, the fungus might be readily pushed downwards mechanically by the direction of the cut. Wagner saw also individual filaments enter into the lumen of the vessels, where he could isolate them almost completely from the blood corpuscles by brushing them off in glycerine. In my case this did not occur, despite numerous attempts to accomplish it. Sometimes we are lucky enough to loosen large pieces of membrane at once during life, and then we can examine the upper and lower surfaces separately. We find most spores on the upper free surface, fewer filaments, and much well-preserved squamous epithelium; on the opposite, lower side, little or no epithelium, and few spores, but a thick tissue of thallus-filaments.

If such a piece of membrane is laid for a day in a solution of carbonate of potassa, the epithelium disappears first, then the molecular mass appears homogeneous and translucent, while the spores and thallus-filaments, whose matted tissue then becomes clear throughout the entire thickness of the membrane, have suffered no change.

If a piece of the membrane is burned on a plate of platinum, a strong smell of burning feathers is produced. Finally, the membrane is entirely soluble in concentrated alkalies, and by the addition of acid to the solution white precipitates fall in a little while. The presence of fat in the membrane can be determined

by its treatment with ether. After evaporating the ether upon a watch-glass, there is left a distinct layer of fat, which is seen under the microscope to consist of amorphous fat globules.

These investigations show that the thrush fungus is developed upon sound epithelium. The uppermost layer of the epithelium is always the thickest and densest, and the fungus seeks a path between these layers to a lower depth, like the roots of a tree in stony soil. The softer forms of the middle layer of epithelium admit of its rapid, luxurious growth, and the development of the epithelium is thereby impeded, in all probability, so that the molecular mass may be regarded as the material of the incomplete epithelium. There is no trace of pus formation to be found at any spot. The nearest capillaries are somewhat dilated and distended with blood corpuscles.

It has already been remarked that this parasitic growth is developed on squamous epithelium only, and never upon cylindrical and ciliated epithelium, and its appearance upon the mucous membrane of the stomach and the intestines is therefore an impossibility. On the other hand, it does sometimes appear upon the lowest portion of the rectum, and upon the female genitals. If loose thrush membrane is found, post-mortem, in the stomach or intestines, it is no evidence that it has been formed there upon the mucous membrane. In the extensive implication of the mouth and œsophagus a large portion of the membrane thrown off must always be swallowed. The nasal cavities always remain uninvaded, as can be most beautifully seen in children with congenital fissure of the palate. On the other hand, excoriations of the external skin, especially in the vicinity of the mouth, on the chin and neck, usually become covered with the growth. Furthermore, we find in the corpses of children, besides the fungus, a high degree of atrophy and follicular enteritis; in adults, tuberculosis, carcinoma, extensive suppuration, pyæmia, and other severe exhausting diseases.

Symptoms.

The most essential symptoms, relative to the form and the anatomico-histological characteristics, have already been spoken

of in the previous section. In lying-in and foundling institutions the first commencement of the disease can frequently be observed. The normal, light-red color of the cavity of the mouth becomes uniformly darker, but never in the form of patches or islets. Where the mucous membrane lies directly over the bone, as on the hard palate and on the free border of the jaw-bones, the redness cannot acquire as great an intensity as in the remaining portions; the tongue, on the other hand, as the most vascular portion of the cavity of the mouth, is always the most intensely reddened. Its turgescence is so great that the fungiform papillæ become distinctly prominent.

If, in this stage, the cavity of the mouth is examined with the finger, a decided elevation of temperature is felt, and a disposition to dryness. The congested parts are, furthermore, painful to the touch, for children do not suck at the finger-tip put into their mouths, as is always the case in health, but move the head back and forth impatiently until the finger is removed. Nor do they suck at the breast any more with the pleasure previously manifested, but they frequently drop it with cries of pain. Adult patients also complain of the pain upon movement of the mouth, or when it is touched. The acid reaction of the mouth, which appears so early and continues during the entire course of the disease, is of special significance, making it probable that the acid reaction is the occasion of the entire fungus-formation.

As long as the affection is not complicated with intestinal catarrh, as occurs in the majority of cases, its symptoms are altogether very slight, and the entire malady runs its course in a few days, if proper attention is paid to cleanliness. On the third or fourth day, the membrane can be readily removed with a moist rag. Sometimes it is reproduced once more, but does not again offer any special difficulty to cleansing, and there is no loss of substance in the mucous membrane.

When thrush attacks artificially fed children, quite a different picture is presented. An uncontrollable diarrhœa is then associated with the disease, consisting of green, acid stools, by means of which the folds of the nates, the inner surfaces of the thighs, and the heels are deeply reddened and eventually excoriated. Rapidly increasing emaciation soon supervenes; the larger fon-

tanelle sinks in, the eyeballs lie deeply in their sockets, the nose and chin become sharp, the skin loses its normal elasticity and warmth, and the children die within a few weeks of atrophy. In such cases, the thrush always reappears, even under the most careful treatment, and persists until death; in which case, on post-mortem, we find the upper portion of the œsophagus also clothed with a solid cylindrical membrane; with the evidences of follicular enteritis in the intestines and in the mesenteric glands.

One of the chief obstacles to recovery is the continuous restlessness of the children, which the attendants usually quiet by giving them a sugar-teat to suck. The sweet contents of this teat, though effecting momentary quiet, always excites acid fermentation anew. This unfortunate complication is so constant an occurrence in foundling-establishments, that physicians, whose observation is confined to such institutions, as, for example, B. Valleix, in the Parisian institution for foundlings, learn to look upon it as an integral portion of the disease—a view which, introduced by Valleix, was adopted in the manuals of diseases of children for many years.

But one who has frequent opportunity to see thrush in private practice, and in children at the breast, can readily convince himself that even an extensive formation of membrane may disappear fully within a few days without the slightest sequel.

Diagnosis.

Thrush may be confounded with the first appearance of aphthous ulcers on the hard palate. As already remarked concerning aphthous stomatitis, this begins as white fibrinous exudations, the size of pin-heads, situated beneath the epithelium, and thrown off after a few days. This eruption is bounded by a dark-red border, and under the microscope shows no thallus-fibrils nor spores.

Thrush is likewise distinguished from croup and diphtheritis by the microscopic constituents of the membrane, by its white color, and by the absence of fever, fetor, and laryngeal symptoms.

Prognosis.

The thrush of children at the breast, which runs its course without diarrhœa, is a very harmless malady, which terminates in recovery in a few days. When in feeble, artificially nourished children follicular enteritis is complicated with it, recovery is not to be expected, and the children gradually perish, with no hope of rescue, after months of continuance of the disease.

The thrush of adults is of the worst prognostic significance, not that the affection of the mouth entails any great danger, but as an evidence of far-advanced cachexia. In phthisical and cancerous patients it indicates with certainty the approaching end. In protracted puerperal fever and in typhus it vitiates the prognosis in the worst manner; yet even in these cases recovery is said to take place now and then.

Treatment.

My experiments have proved that the membrane of thrush, placed in pure water or in any solution of a non-alkaline salt, after a few days develops little tendrils of new thallus-filaments. This experiment succeeds most exquisitely with sugared water. In dilute alkaline solutions, borax and phosphate of soda, as well as in solutions of the metallic salts, nitrate of silver, corrosive sublimate, and sulphate of copper, on the contrary, no new formation of fungus takes place. In concentrated alkaline solutions, finally, the membrane becomes first soft and transparent, and finally dissolves into a light cloud, which, under the microscope, consists entirely of a mesh of thallus-fibrils.

As no remedy has yet been discovered to dissolve the membrane in the cavity of the mouth without considerable injury, we must be satisfied to restrain the further growth of fungi by dilute alkaline solutions. Solutions of carbonate of potassa or soda, or of borax (twenty-five grains to the ounce), effect this end thoroughly. A rag is moistened in the fluid, and the entire mouth washed out with it every hour. These small doses, even when swallowed, do not produce any deleterious effect

upon the appetite nor upon the action of the bowels. The linctus of borax and honey kept by the apothecaries fails in its object, inasmuch as the favorable action of the borax is annulled by the injurious effect of the sweet honey.

In thrush, complicated with enteritis, the usual practice is to employ nitrate of silver locally, touching the parts several times a day, either with the stick or with a ten-grain solution; but the results are not decided.

There is only one remedy for the thrush and follicular enteritis of artificially nourished children, and that is the breast of a healthy nurse. Children that are quite atrophied, and nearly dying, even when they have been for some months without the mother's breast, take to it greedily, and after a few efforts drink like healthy nurslings. The previously ineffectual treatment of thrush is substituted by simple cleanliness, and after a few days sound sleep returns as well as normal stools and rapid increase of fat.

The removal of the sugar teat is absolutely necessary, if improvement is to be expected. If the employment of a wet-nurse is impossible, the children are best nourished with glutinous beef broths; and they may be given for drink a decoction of salep with a few drops of red wine.

5. Gangrene of the Cheeks. Noma.

Baron, Journ. de méd. par Leroux, etc. 1816. T. 36. Bulletin de la faculté. 6 and 7.—*Isnard-Cevoule*, Journ. complem. du dict. des sc. méd. 1819. Cahier. 16.—*Siebert*, Hufeland's Journ. Bd. 33. Dec. p. 74.—*Romberg*, Rust's Magazin. Bd. 30. Hft. 2. p. 344.—*A. L. Richter*, Der Wasserkrebs der Kinder. Berlin. 1828.—*Jg. Wiegand*, Der Wasserkrebs. Erlangen. 1830.—*J. Frank*, Præcept. etc. V. III. Vol. 1. sect. I. p. 698.—*Froriep*, Patholog. anatom. Abbildgn. Weimar, 1836. Lief. 1 u. 2.—*Taupin*, Journ. des Connaiss. méd.-chir. Avril, 1839.—*Canstatt*, Schmidt's Encyclopäd. Bd. IV. p. 658.—*Hunt*, Med.-chir. transact. Vol. XXVI.—*J. Tourdes*, Du noma de la bouche chez les enfants. Strassburg, 1848.—*Albers*, Arch. f. physiol. Heilkde. Jahrg. IX. p. 515.—*Rilliet et Barthez*, Traité des maladies des enfants. 2. Edit. Paris. 1853. T. II.—*A. Keiller*, Cancrum oris. Edinb. Med. Journ. 1862, April.—*Glynn*, Cancrum oris. Brit. Med. Jour. 1869, Mch. 13.—*Kellner*, Ueber Noma. Berlin. 1870. Dissert.—*Edgar, W. S.*, On cancrum oris. St. Louis Med. and Surg. Journ. 1870. Sept.—*Lange*, Ein Fall

von Noma, geheilt durch äussere Anwendung von Ol. terebinth. Memorabil. 1871. No. 2.—*Schmid, A.*, Ueber das Verhältniss von Noma zu Gangræna oris. Bair. a. Intellgzbl. 1872. No. 39.—Various manuals and treatises on diseases of children and on special pathology and therapeutics.

SYNONYMS: *Water-cancer; cancer aquaticus; carbunculus labiorum et genarum; gangræna oris; stomatomalacia putrida; stomatosepsis; stomatonecrosis.*

By Noma (ἡ νομή, pasturage; feeding tetter) we understand a rapid gangrenous destruction of the cheek, commencing at one corner of the mouth. Even Hippocrates recognized νομαί, eating ulcers, though we cannot find in the older authors any distinct description of the process which we at present call noma. The first one who gave a characteristic description was, according to Bamberger, the Hollander Battus, in the beginning of the seventeenth century; and another Hollander, Van de Voorde, was the first to use the name of water-cancer—a very inappropriate appellation, because there is not the remotest idea of its being a cancer or a neoplasm. Finally, Richter and Tourdes have earned credit by their historic researches on the subject of noma.

Etiology.

Noma is an unfrequent disease. West has observed but seven cases among 30,000 sick children, six of which cases terminated fatally. I myself remember but five cases, of which only one recovered. In the surgical clinic of Dorpat it is proportionately frequent, one or two cases being brought there annually. Children of from two to twelve years of age are most disposed to it; it does not appear in nurslings at all, and in adults extremely seldom. Girls are more frequently affected than boys. Entirely healthy, strong children are never attacked with noma, but only those convalescent from severe diseases, such as scarlatina, measles, small-pox, typhus, whooping-cough, intermittent fever, and dysentery; and then, as a rule, only those who, before the onset of these infectious diseases, had suffered from debility, scrofulous affections of all sorts, diarrhœa, etc.

That poverty, poor nourishment, and damp residence may aid in promoting this evil, will not be denied ; but the effect of these deleterious influences cannot be very potent, or noma would be much more frequent among the impoverished masses in large cities. Edgar, an American writer, declares that the view that noma appears only in children's hospitals, or in the thickly populated districts of large cities, is incorrect, at least for America. He himself observed a series of cases in children under five years of age, and three well-developed cases in adults, during the year 1844, in a district opposite St. Louis, where, after an overflow, the remains of drowned animals and of destroyed vegetation were left to decompose during the autumn. Of my five cases two belonged to well-to-do peasantry.

The abuse of mercury—which was frequently given, and is still given, in the form of large doses of calomel, at the commencement of acute diseases—has always been complained of as a further cause of noma. In a large number of reports on noma we find it noticed that some calomel powders had been taken in the course of the previous infectious disease ; but even here all that has already been said, on the etiological relations of the impoverished condition of the lower classes, still holds true. If noma were the result of the use of mercury, it should appear much more frequently. Edgar and Keiller expressly mention that in their cases no preparations of mercury had been administered.

Its proportionately frequent occurrence in Holland, and in the coast-regions especially, where inundations are frequent, while, according to Eisenmann, it is entirely unknown in the south of Germany, is striking. Its infrequency evidently stamps it as a sporadic disease only, and nothing regarding its contagiousness has ever been reported. The few cases in adults that have thus far been described were among convalescents from typhus and those who had suffered from the abuse of mercury. In malignant epidemics of typhus, we notice an especial disposition to gangrene ; and the parotitis, ending in rapid destruction of tissue, found in typhus cases, may be regarded as an analogous process.

Symptoms.

In a convalescent from the above-enumerated infectious diseases there is formed, without any local cause, a hard place on the cheek, the size of a hazel-nut, generally quite close to the corner of the mouth. If the opportunity is presented of examining the development of the case during the first few hours, it is said that the affected portion of the mucous membrane of the cheeks will be found transformed into a flat ichorous bulla. My patients did not come under observation at so early a stage ; in those cases which I saw the earliest I found the mucous membrane of the cheek floating in loose shreds upon a black gangrenous floor. The tumor palpated from the outside feels as hard as a board, and it is extremely sensitive to the touch, especially at its periphery. As early as on the second day the cutis assumes a bluish tint, the epidermis falls off, and there is revealed a black eschar, which usually has a decided disposition to shrivel and dry up. Although by this shrivelling a line of demarcation is apparently formed (a deep groove filled with ichor, between the normal and the mortified tissue), the gangrene still progresses rapidly in every direction, towards the periphery as well as towards the deeper parts. In a short time the cheek is broken through, and a dirty, shreddy saliva flows out beside the eschar. The gangrene invades the lips, where, however, it almost always stops at the middle line ; and it also invades the corresponding alæ of the nose and the entire cheek as far as the lower eyelid and the external ear. Ordinarily it does not extend downwards below the border of the lower jaw. At the same time the jaw becomes necrotic, the loosened teeth can be removed by the fingers in a few days, and frequently carry off pieces of the jaw-bone with them. The tongue usually remains uninjured. The entire side of the face becomes swollen and œdematous, and the neighboring cervical glands become infiltrated. The nauseating gangrenous odor fills the entire apartment of the patient, and causes most serious embarrassment in caring for these unfortunates. Sometimes new, independent centres of gangrene are formed at some distance from the main one, which spread at once

and eventually come into communication with the primary circle of destruction. The salivary secretion is considerably increased, and progresses even to ptyalism. Finally, the cheek is entirely cast off in large black eschars, whereupon the necrotic jaw, denuded of its gum, can be seen lying at the bottom of ragged, gangrenous ulcers. The gangrenous border finally extends, when life is sufficiently prolonged, over the entire cheek, and the children undergo a fearful loss of substance, so much so that they can no longer be recognized.

It is very remarkable that this process should almost invariably commence at one and the same point, the vicinity of one corner of the mouth. In only one case did I see the gangrene begin beneath the lobe of the ear; from which point, however, it rapidly extended forwards and progressed in depth at the same time, so that when the case ended fatally after a few days, the destruction presented the picture of ordinary noma. It is further worthy of note, that the destruction almost always remains unilateral, as is especially evident on the nose and upper lip. This stereotyped restriction to a certain spot, as well as the constant limitation to one side of the face, points very closely to a local originating cause, though thus far our anatomical studies have not been able to give us the explanation of the matter.

The first thought that suggests itself is, that it might depend upon embolism of a large artery, from debility of the cardiac muscle or the greater coagulability of the blood; but it would remain incomprehensible why embolism should not occasionally occur in other vascular territories, such as the lower extremities, the liver, the kidneys, etc. We must, therefore, if we intend to seek an explanation, turn our attention to the nervous system, the trifacial, the facial, and the vaso-motor nerves, of late so often quoted; but we will not here enter this domain of hypothesis. Only so much will be mentioned that, on dividing the trifacial in dogs, as Magendie[1] demonstrated, the eyeball is destroyed, the half of the tongue becomes dry, brown, and fissured,

[1] *Magendie*, Leçons sur les fonctions et les maladies du système nerveux. Paris. 1839. T. II. p. 31.

the gums become spongy and hemorrhagic, and the teeth become loose. In animals tenacious of life, the batrachians, for example, *the soft portions of the face are cast off in gangrenous shreds, just as in spontaneous gangrene.* After three or four weeks only one-half of the face remains. The disproportionate liability of girls to noma points also to the more irritable nervous system of the female sex.

The constitutional disturbance is slight at the commencement, the temperature often only moderately elevated, the pulse not alarmingly hurried, the pains insignificant. On the other hand, the cachectic, collapsed, pale appearance, which is still more increased by a shining œdema of the affected cheek, sometimes also of the well side, is conspicuous. Most children are apathetic, deeply depressed, are at best able to take fluids only, and give expression to their lamentable condition merely by slight whimpering. How rapidly the strength declines, and nutrition recedes, may be known from the fact that œdema of the feet sets in within a few days from the commencement of the noma. Usually, towards the end, delirium supervenes, with profuse diarrhœa and a purulent, ichorous, and even gangrenous infiltration of the lungs, sometimes also gangrene of the female genitalia. Death follows, with few exceptions, within a few days after the commencement of the gangrene.

In the exceptional cases of recovery, the gangrenous edges become cleaned off and covered with granulations, the jaw-bone is cast off as far as it is necrotic, and at the end of months, cicatrization occurs with horrible disfigurement of the face. Permanent injury may be developed through ectropion, loss of the jaw-bone, and impeded movement of the lower jaw, in consequence of firm cicatrization.

Pathological Anatomy.

The corpse has a very penetrating gangrenous odor, and soon becomes putrid ; the general integuments are shrivelled ; the face is always turgid with œdema, the ankles frequently so. The entire body shows a striking lack of blood ; the gangrenous parts are transformed into a syrupy blackish-brown mass ; and the

bones are uncovered, brownish in color, and friable. In the adjacent portions of the cheek, not yet occupied by the gangrene, there is a dense exudation ; the undestroyed portions of the mouth, the palate, tongue, tonsils, etc., are swollen, and covered with black scales or crusts. If such an eschar is hardened, and examined in fine transverse and horizontal sections, we find much free fat and traces of muscle; the nerves are somewhat yellowish externally, but their elements can still be distinctly distinguished ; the vessels thickened in their walls are filled with thrombi. This is also the reason why hemorrhage occurs so seldom during life ; though Hueter reports a case in which fatal hemorrhage occurred. Rilliet and Barthez, in one case, found the duct of Steno permeable and uninjured in the midst of the eschar. The blood does not exhibit any characteristic peculiarities ; sometimes it is remarkably diffluent, sometimes it contains considerable normal coagula.

In the lungs there are frequently found hemorrhagic infarctions, lobular or metastatic lobar pneumonia, and even gangrene. The intestines usually show evidences of catarrh more or less distinct, which could hardly be otherwise, in view of the quantities of gangrenous ichor swallowed.

The remainder of the body will be found in a condition corresponding to the nature of the previous disease. While measles and scarlet fever will leave no traces behind them, the diagnosis of typhoid fever, of dysentery, or of malarial cachexia, can still be made with certainty.

Diagnosis.

Certain of the older authors consider this disease as carbuncle or anthrax (J. Frank); others consider it as malignant pustule (Canstatt). Carbuncle always begins in an aggregation of furuncles, which excite such swelling of the intermediate cutis that it becomes gangrenous. It passes from the cutis downwards, and is limited to the subcutaneous connective tissue. Noma extends, on the one hand, from the mucous membrane outwards, by which the cutis is at once rendered gangrenous, without any previous

appearance of furuncles; on the other hand, it extends in depth with equal rapidity, and destroys the bones of the jaw.

Noma certainly presents a great similarity in form to malignant pustule, but the infiltration of the cutis is much denser and wider-spread in the latter affection, and the general condition of the system takes part on the very first day in a much more serious manner. In addition to this, we have in noma, usually, no special etiological evidence of contagion. Malignant pustule occurs simultaneously with epizootic disease, and especially attacks those previously healthy persons whose occupation obliges them to work over the tissues of dead animals and their offal. In the noma of children there is not one of all these causes to be discovered; on the other hand, a debilitating febrile disease always precedes it.

By a few authors, Siebert, Hildenbrand, Henke, Jörg, and others, noma is considered a malignant form of scorbutus. Here we have, inversely, as with anthrax (Milzbrand), similarity of cause, but no similarity of form. Both processes occur in enfeebled individuals rendered anæmic by previous infectious disease; but in scorbutus the disease of the mouth is only one of the manifestations of the general affection, and, in its outward manifestations (proliferation and hemorrhage of the gums), has nothing to do with gangrene.

Extreme grades of stomacace and mercurial stomatitis may eventuate in destruction of the mucous membrane of the cheek, the gums, and the jaw-bones, which can hardly be distinguished from noma; but it requires a much longer time, and wanton neglect, and even then the most essential indication of noma, the rapid gangrene of the skin, does not present itself. The course of the affections, also, is quite different, for while from eighty to ninety per cent. of the cases of noma perish in a few days, a fatal termination is an infrequent result in the other process.

In gangrenous diphtheria and in malignant scarlatina the posterior portions of the cavity of the mouth become gangrenous, but not the cheeks; and distinct diphtheritic membrane is seen on the remaining non-gangrenous portions; otherwise these processes resemble it very much, especially as far as concerns the

rapid collapse and the regularly unfortunate termination. Confusion with syphilitic, lupous, and cancerous ulcers of the lips is so unlikely, that it does not appear necessary to indicate more definitely their points of difference.

Treatment.

Only so much can be said concerning prophylaxis, that, in the treatment of all the infectious diseases known as precursors of noma, all debilitating and powerful remedies, especially mercury, should be rigidly abstained from, and care be taken to secure systematic ventilation and the most efficient nutrition. If noma is once developed, the first indication is to sustain the rapidly waning strength by tonics and stimulants. The treatment universally recommended by authors, with decoctions of cinchona, solutions of quinine, wine, and coffee containing large proportions of milk, is mostly thwarted by the unwillingness with which patients, children especially, drink—an unwillingness which soon amounts to inability, when perforation of the cheek has once taken place and the fluids taken into the mouth flow back over the gangrenous tissue.

With regard to general hygienic measures, we have, above all, in patients with gangrenous destruction, to secure the freest ventilation. In warm seasons of the year the patients should lie in the open air day and night, and in winter the windows should be opened several times a day, a powerful heat being maintained, and efforts made to destroy the cadaverous odor by chloride of lime and the burning of the flowers of sulphur.

As patients soil themselves very much with the ichorous discharges, and frequently are unable to endure any covering bandage, daily complete cleansing in the warm bath and entire change of clothing is absolutely necessary. To prevent the continuous swallowing of the discharges, Marjolin advises laying the patients on the affected side, the frequent rinsing of the mouth with disinfectant gargles, and washing out the mouths of little children frequently with a syringe, especially before giving them nourishment. In all the cases thus far treated by myself, all these precautions were very little or not at all

practicable, on account of the resistance of the very unwilling little patients; and morphia, therefore, was the only quieting medium.

Under such forlorn circumstances, the aim has always been to restrain the evil by the most various topical remedies. There is hardly a caustic that has not been tried. Billard advises making a crucial incision in the infiltrated cheek, and the insertion into it of the butter of antimony. Van der Voorde recommends concentrated sulphuric acid, which should be laid upon the entire diseased surface several times a day until a line of demarcation is seen. Hydrochloric acid has come very much into use, chiefly by the advice of Siebert. It is laid upon the parts several times a day, just as is recommended with sulphuric acid, and after each application of the acid the wound is covered with charpie. Rey and Hermes obtained successful results with charpie saturated in a concentrated solution of table salt. Hunt dresses with chlorate of potassa; Deutsch with a camphor-paste. Nitric acid is recommended by Baron; pyroligneous acid by Klaatsch. Rust and Young dress several times a day with a solution of corrosive sublimate; Marjolin and Corrigan with nitrate of silver; Busch with charcoal. Keiller cauterizes, under chloroform narcosis, with concentrated nitric acid or with chloride of iron, and then syringes the mouth out diligently with a solution of chlorinated lime—under which treatment two cases recovered out of six. Lange employed oil of turpentine in a severe case, in which a part of the cheek was already destroyed by gangrene as well as nearly half of the tongue, by laying saturated charpie upon the parts and renewing the application every two hours. The result was surprising, inasmuch as the gangrene soon became limited, and cicatrization set in.

With the exception of charcoal, all the above-named materials are very powerful, and produce severe pains when coming in contact with the parts not yet gangrenous. It is also to be remembered that if the cheek is once perforated, a large portion of these caustics may get into the mouth and be swallowed. As nearly all these procedures are much too painful to be repeatedly employed without chloroform, it seems evidently more rational

to apply, under chloroform narcosis, the most radical and safest of all caustics, the hot iron.

The one patient that I have thus far seen recover from a severe noma, with loss of the entire cheek, and a portion of the upper jaw, was several times touched by me with concentrated hydrochloric acid, and the cavity of the mouth as well as the entire gangrenous surface thoroughly syringed with chlorate of potash. After a few months several plastic operations were performed on the child, which may be denominated successful in a surgical sense, but which left what the laity would consider an extensive permanent disfigurement.

6. Neuroses of the Mouth.

Canstatt, Schmidt's Encyclopäd. Bd. VI. p. 523.—*Romberg*, Lehrbuch der Nervenkrankheiten. Berlin. 1850.—*Bamberger*, Virchow's Handbuch der spec. Pathol. Bd. VI. 1. Abth.—*Hasse*, Virchow's Handbuch der spec. Pathol. Bd. IV. 1. Abth.—*Lotzbeck*, Deutsche Klinik. 1858. No. 12.—*Noël Gunéau*, Paralysis following diphtheritis. Revue de thérap. méd. chir. 1859. No. 15.—*A. Maingault*, Paralysis following diphtheritis. Arch. gén. de méd. 1859. Aout-Dec.—Bericht über Diphtheritis. Med.-chirurg. Monatshefte. 1861. Bd. I. p. 307.—*Lée*, On diphtheritic paralysis. Gaz. hebd. 1860. 43.—*Donders*, Paralytische Sympt. nach Diphtheritis. Arch. f. holländ. Beiträge z. Natur- und Heilkde. Bd. II. Heft 4. —*Trousseau*, Clin. médic. de l'Hotel Dieu. Cullman's translation into German. Würzburg. 1866. Bd. I. p. 357.—*Jaffé*, Berichte über die gesammte Diphtherie. Schmidt's Jahrbch. Bd. 140. p. 207. u. Bd. 149. p. 217 u. 321.—*Hirschberg*, Berlin klin. Wochenschrift. 1868. 48. u. 49.—*Guttman*, ibid. No. 51.—*A. Foville*, Paralysis after diphtheria. Annal. méd. psych. 5. Sér. II. p. 267. Sept. 1869.—*Lussana*, Recherches expérimentales et pathologiques sur les nerfs du gout. Arch. d. physiologie. 1869. Sér. 1. p. 20.—*Eulenburg*, Lehrbuch der functionellen Nervenkrankhtn. Berl. 1871. p. 292.—*J. Burney Yeo*, Temporary loss of taste. Trans. Clin. So. V. p. 219. 1872. Schmidt's Jahrbcher. Bd. 159. p. 128.

The diseases of the nerves will be thoroughly discussed in another place, and it is not intended, therefore, to enter here upon all questions concerning the neuroses of the cavity of the mouth. I shall content myself, therefore, for the sake of completeness and of differential diagnosis, with giving a general outline of the sensory and motor disturbances which primarily and solely affect the cavity of the mouth.

A.—SENSORY DISTURBANCES.

We distinguish here the disturbances of the sense of touch from those of the sense of taste. While by the first we distinguish temperature, consistence, volume, surface, pressure, etc., the latter recognizes the chemical peculiarities of the object of taste. Though controversies with reference to the nerves of taste are by no means at an end, it has still been established by physiological experiment and clinical observation that the perceptions of touch and taste in the tongue must depend upon different nerves. Each one can be individually diseased, whether in the excitation, the diminution, or the entire abolition of function, with complete integrity of the other.

1. *Anomalies of Tactile Sensation.*

a. *Hyperæsthesia*, sensation of pain in the mouth, is generally only one of the manifestations of neuralgia of the fifth pair, but it occurs also, exceptionally, confined to the gums (alveolar neuralgia), or to the tongue. Patients describe these neuralgias as exceedingly painful and severe. The pain appears mostly in paroxysms, remains sometimes concentrated at one point, but often shoots like lightning through an entire row of teeth, or through one-half of the tongue. In half a minute or a minute these pains suddenly cease, to return again as suddenly. In addition to such slight attacks, special paroxysms occur, which continue for many hours, even for days together. Gradually a permanent irritability and sensitiveness of one or several points supervenes, on contact with which the severest pains spread over a greater extent of surface. The second branch of the trifacial is affected the most frequently, and the pain shoots along its course in the upper row of teeth, the upper lip, the palate, and the alæ of the nose. The third branch is less frequently affected, in which case the pain shoots into the lower jaw, the lower lip, the border and tip of the tongue. The salivary secretion is usually augmented in both these varieties of neuralgia, and may increase to complete salivation.

The *differential diagnosis* is not always easy. First of all,

a careful inspection and palpation of the entire cavity of the mouth is to be made, in order to find whether a small ulceration of the tongue, caries of the teeth, or an affection of the bone is not the source of the trouble. The removal of single, painful teeth is indicated under all circumstances, even when no alteration is visible upon the crown. What further concerns the etiology and treatment must be relegated to the section on the neuralgiæ.

b. *Anæsthesia.*—Diminution of sensitiveness is produced either by central disease of the brain, in which case the opposite half of the face will be insensible, or by peripheral injury of the second or third branch of the trifacial. It usually appears, unilaterally, immediately after apoplexies and embolism of the brain, but it is only very transitory, and will hardly attract further notice on account of the general severity of the case. In peripheral paralysis of the trifacial and facial nerves, this anæsthesia offers such slight symptoms that they sometimes remain entirely concealed from the patient. The most prominent symptom always is, that, when the patient drinks out of a tumbler, he imagines that half the border of the glass is broken off. Patients likewise do not feel the bread-crumbs which remain, after a meal, between the cheeks and the gums ; and the gums on the diseased side become spongy, and frequently bleed.

According as the injury to the fifth nerve exists nearer or further from the centre, the tongue is also affected unilaterally, in which case, however, the disturbances of taste almost always occur on the anterior and middle portion of the half of the tongue. Then there appear also the other signs of paralysis of the trifacial: ulceration of the eyeball, loss of sensation over the entire half of the face, loss of smell in the nostril of the affected side, and paralysis of the muscles of mastication.

The *diagnosis* of these conditions is usually simple, and it is hardly possible to make a mistake. The *prognosis* depends altogether upon the etiological condition. In central disease, at most a stand-still, but usually an aggravation, is to be anticipated ; peripheral circumscribed disease of the trifacial may continue many years without untoward results.

The *treatment* does not include any special indication with

reference to the cavity of the mouth, but is to be pursued with reference to the general condition, concerning which the details may be consulted under the head of the diseases of the nerves

2. *Anomalies of the Sense of Taste.*

The sense of taste may be—1st, lessened; or, 2d, entirely lost; or, 3d, intensified; and, finally, 4th, special sensations of taste may present themselves spontaneously, which are usually called forth only by certain foods and spices. We can clearly distinguish but four distinct sensations of taste, viz., *sweet*, *bitter*, *sour*, and *salt;* and we test the tongue by allowing such substances, preferably in a soluble form, to come in contact with the terminal filaments of the nerves of taste. The most convenient substances for these experiments are sugar-water, tincture of quassia, dilute acetic acid, and a solution of table-salt. By various dilutions of these solutions the investigation may be varied in great measure, as has been more thoroughly set forth by Eulenburg.[1]

Disturbances of taste depend most frequently upon alterations of the lingual mucous membrane; less frequently upon diseases of the brain and the glosso-pharyngeal nerve. Conditions approaching psychological affections, such as hysteria and hypochondria, often alter the taste in various ways.

a. *Anæsthesia of the nerves of taste. Ageustia.*

This may be either complete, so that the patient cannot perceive the most intense impressions of taste; or incomplete, in which case only strong spices, very sour, bitter, or sweet substances can be distinguished. There is also a slowness in perception, and the patient cannot decide as to the taste until after some seconds. The anæsthesia is generally extended over the entire surface of the tongue; sometimes, however, it is only unilateral; and, in less frequent cases, it is limited to isolated small localities upon the tongue.

The *causes* of this condition are usually great changes of

[1] L. c. p. 294.

temperature. If ice is kept upon the tongue for a long time, or if the tongue is superficially burned by hot food or drinks, ageustia follows, though it may be but transitory. The same thing occurs with every thick coating upon the tongue, as is observed in many general affections, and especially in local diseases of the mouth. Finally, ageustia may be produced in a perfectly sound mouth, by a disturbance of the nerve conduction, a peripheral or central paralysis. It has been shown by a series of well-authenticated reports of disease and of autopsies that the source of the trouble is to be sought sometimes in the trifacial, sometimes in the glosso-pharyngeus, sometimes in the chorda tympani, and sometimes in the peripheral portion of the facial nerve. Although there is much that is vague in the combined action of these nerves with regard to the sense of taste, so much has been settled with certainty, that anæsthesia of the base of the tongue always depends upon the glosso-pharyngeus alone.

The *prognosis* is especially favorable in all local diseases of the mouth, and in rheumatic paralysis of the facial, in which taste is restored soon after the disappearance of the paralysis. The prognosis is unfavorable in most of the cerebral affections, when several of the nerves of special sense are involved, and also in hysteria.

The *treatment*, in as far as no local disease furnishes any special indication, is governed solely by the nature of the causal malady. Duchenne, in some cases of ageustia of hysterical nature, and in other cases of unknown origin, saw rapid recovery from local faradisation of the tongue.

b. *Hyperæsthesia of the Nerves of Taste. Hyperæsthesia Gustatoria. Hypergeustia and Allotriogeustia. Gustus Depravatus.*

An abnormal increased gustatorial sensitiveness of taste is not always a pathological condition, but may be a sharpening of the sense of taste, due to favorable circumstances and practice. Many specialists in wine and tea acquire an extraordinary dexterity in the recognition and testing of these articles without giving evidence of any increased irritability of their nervous systems in general.

The sense of taste shows the same variations in sensitiveness as the other senses, as may be easily proved by letting a number of healthy individuals taste any sapid substance, as wine, sugar, salt, or the like in varied dilutions.

Pathologically, hypergeustia is observed in infrequent cases in hysterical individuals and so-called clairvoyants, who, by the excessive irritability of their gustatory nerves and the unpleasant sensations, which are provoked by food of altogether pleasant flavor to other people, are compelled to adhere to a special diet. In most such instances a tormenting hyperæsthesia of the olfactory nerves, hyperosmia, is associated with it.

Allotriogeustia, perversion of taste, occurs much more frequently than the condition already described. Either the patients interchange their sensations of taste, sugar tasting bitter, and salt tasting sweet, or these gustatory sensations occur spontaneously without any active influence from a sapid substance. They are then not free from the same taste the entire day, and suffer from complete gustatory hallucinations, which are mostly of a disgusting nature, and which in insane patients so frequently lead them to imagine that they are poisoned, and to refuse nourishment.

Ordinarily, allotriogeustia is produced by diseased conditions of the mouth, and chemical change in its secretions, which then add an insipid, salt, bitter, or metallic taste to the usual symptoms. As certain substances are directly excreted in the saliva, all these perversions of taste are not due to illusion. The metallic taste in metallic poisoning, the bitter taste after santonine, or after the injection of morphia, are produced by the actual commixture of these substances with the saliva.

Icterus acts in a singular manner; for although the evidence of bile is conspicuous over the entire body, there is usually no bitter taste. In facial paralysis patients sometimes complain, concerning the affected half of the tongue, of a sharp or bitter taste instead of the much more frequent abolition of the gustatory sensation.

Finally, we may allude to the well-known peculiar desires of pregnant and hysterical women, sometimes even for very disgusting things; while the great and unconquerable desire of

children of from one to three years of age, for chalk and sand, can be explained very simply by the great need of the organism for the salts of lime. The amount of lime necessary for the rapid development of the skeleton apparently does not always exist in the food.

The *treatment* of these different anomalies of taste coincides in most cases with that of the causal affections of the mouth, or with that for the general nervous affection, and that for hysteria. For long-continuing anæsthesia of the gustatory nerves we employ irritating mouth-washes, ethereal oils, and light applications of nitrate of silver.

J. Burney Yeo narrates a case in which a man showed symptoms of concussion of the brain after a fall from a house, but recovered entirely on the following day, with the exception of complete loss of the senses of taste and smell. These sequelæ continued for fully four months, and the patient could not taste salt, sugar, acids, nor quinine, whether these substances were placed upon the lips or upon the tongue. The ordinary sensation and power of motion of the tongue were unimpaired. After six weeks' use of small doses of iodide of potassium, to which the patient was unusually susceptible, and which produced prompt iodic coryza, the taste returned completely; but smell returned only to a limited degree. A subjective olfactory sensation set in for a long time, which the patient compared to the smell of an old, foul tobacco-pipe.

B.—MOTOR DISTURBANCES IN THE MOUTH.

Spasmodic affections of the mouth, of the tongue particularly, very seldom occur alone, and we therefore pass over all convulsions of the tongue and of the muscles of the cheeks, which appear in mimetic and masticatory spasms of the face, as also those of chorea and of the commencement of certain central disturbances.

The territory of the hypoglossus is but rarely subject to spasmodic contractions. In hysterical attacks, convulsive movements set in as accompaniments; the tongue twists and arches, making a smacking noise. The same thing is sometimes observed in acute miliary tuberculosis of the meninges.

Paralyses of the tongue are also, as a general thing, only part of the evidences of central disturbances, and accompany, as uni-

lateral paralysis, especially extravasations, embolism and inflammatory deposits in the larger ganglia of the brain. In unilateral paralysis of the tongue, its tip turns toward *the paralyzed side*, when the effort is made to protrude it. The tyro is the more struck with this phenomenon, because, in central hemiplegia, the tip of the nose, the chin, and also the uvula, are drawn towards *the sound side*, on account of the more unilateral action of the symmetrical muscles. If we take into consideration the manner in which the tongue is protruded, the matter is readily explicable. It depends upon the fact that the two genio-glossi muscles, which are attached by one end to the base of the tongue, and by the other to the inner surface of the lower jaw, contract, and thus draw the root of the tongue towards the lips, and thus the anterior portion of the tongue must be protruded as a matter of course. Now, if one genio-glossus muscle is paralyzed, the sound half only will approach the lips when the attempt is made to protrude the tongue, the other half remaining behind; and this retention of one half of the tongue exerts its influence clear to the tip, which then naturally lies nearer the corner of the mouth on the paralyzed side.

The movements of the tongue are twofold: masticatory and articulatory. Mastication is the commencement of the act of deglutition, and the tongue collects the contents of the mouth into a bolus, then presses this bolus upon the palate, and drives it behind the anterior palatine arches. The articulatory movements of the tongue permit the pronunciation of letters, especially the lingual consonants D, T, N, L, R, and S. In paralysis of the tongue, both these functions are more or less impeded.

Passing over the various processes in which paralysis of the tongue and of the soft palate occurs as part of the symptoms of mere general affections, we will confine ourselves to two processes in which the paralytic symptoms commence in the cavity of the mouth, and frequently remain localized there: *bulbar paralysis*, and *diphtheritic paralysis of the soft palate.*

a. *Progressive bulbar paralysis* (Wachsmuth), or *paralysis glosso-pharyngo-labialis* (Duchenne), was first accurately defined as a characteristic form of disease by Duchenne in 1860, and then

explained by Wachsmuth as a progressive degeneration of the medulla oblongata, the *bulbus medullæ spinalis.*

We are completely in the dark as to the etiology of this affection. The first and most important symptom is a gradually increasing paralysis of the tongue, by which both mastication and articulation are in like manner impeded. If we press the finger hard upon our own tongue and attempt to speak, we get exactly that interference with speech which is produced by bulbar paralysis, inasmuch as patients thus affected are no longer able to raise the tip of the tongue and lay the organ against the palate. At the same time difficulty of swallowing sets in, and there is a very profuse secretion of mucus. The continuous spitting, however, has its cause partly in the difficulty which swallowing causes the patient. The viscid composition of the mucus of the mouth, which incommodes the patient very much, and impels him to many mechanical attempts at its removal, evinces an altered chemical composition of the fluids of the mouth, with excessive secretion of mucus. The tongue either lies quietly within the mouth, or is in continued trembling motion.

In the course of the malady the paralysis extends to the soft palate and to the orbicularis oris muscle, in consequence of which the difficulty in articulation and in deglutition increases, and the lips can no longer be pursed up into the position for whistling. As the facial muscles participate in the paralysis, the patient acquires a characteristic vacant, but at the same time lachrymose aspect, the latter peculiarity being most distinctly marked if the levator labii superioris alæque nasi muscle is not yet affected by the paralysis. Finally, paralysis of the glottis sets in, and with it its attendant difficulty in respiration. Here, usually, the paralyzed territory has its limit, and the remainder of the territory to which the cranial nerves are distributed remains intact. From his imperfect ability to swallow, the patient gradually emaciates, while the mental functions remain unaffected, in most instances, to his very death.

The disease almost always ends fatally in from one to three years, by gradual exhaustion of strength and general progressive atrophy, or by a suffocative spasm of the larynx. Further

anatomical and theoretical details can be found in the recent treatises on nervous diseases.

The *diagnosis* can hardly be made at the beginning; but the fully developed disease is readily recognized, and the above-described symptoms are correctly given by intelligent patients, without further examination. Only when, exceptionally, the trouble begins with the lips and not with the tongue, might bilateral paralysis of the facial be suspected; but here, also, the partial immunity of individual muscles of the face, and the limitation of the paralysis to the lips and the chin, furnish a safe differential guide. Bulbar paralysis is distinguished from the diphtheritic paralysis of the soft palate by its chronic course, and by the alterations in the tongue and lips, which never occur in diphtheritic paralysis.

The *treatment* of bulbar paralysis is, according to my own observations, entirely hopeless. All recent writers express themselves in a similar manner, except that Tommasi believes he cured a case by faradisation, and Benedict speaks of decided results by galvanization of the sympathetic and the mastoid processes. It is impossible to arrest the failure of strength by any remedy, either with iron or with quinine, and the patients sink away more and more. If the difficulty of swallowing has become very great, and the patients frequently swallow the wrong way, then nourishment, attempted by the pharyngeal catheter, introduced most effectually through the nose, and the administration of nourishing enemas, afford the only methods of prolonging life. Sleeplessness and dyspnœa are combated in the usual manner, by chloral hydrate and morphia, administered either by the mouth or subcutaneously.

b. *Diphtheritic paralysis of the soft palate.*

Etiology and Pathogeny.

Besides the paralysis of the soft palate in bulbar paralysis (see previous section), and the unilateral paralysis of the soft palate in general hemiplegia and paralysis of the facial nerve, there is another very extreme form of paralysis, which follows

so frequently upon diphtheria that this infectious disease must be regarded as its special cause. Isolated references to the same were made in the last century by Ghisi, in 1749; Bretonneau distinctly recognized its dependence upon diphtheria; and Trousseau, finally, by his comprehensive labors, banished the last doubt upon the subject. The extraordinary extension of diphtheria during the last decennium has produced a great number of works upon this theme, and in them all this characteristic paralysis is spoken of in very much the same way. Oertel's beautiful investigations show that the diphtheria begins as a local process, extending itself in a shorter or longer time over the infected body; that by the invasion of vast numbers of micrococci various tissues are destroyed; and, finally, that by general blood poisoning the vitality of the entire organism is extinguished. This view also explains very naturally why the paralysis appears to be confined very frequently to the soft palate, the locality from which diphtheria generally starts, and on which the first membrane is deposited.

Symptoms.

At the termination of diphtheria, or in from one to four weeks after its apparent cure, there is noticed a gradually increasing nasal quality of speech, and frequent swallowing the wrong way during eating. If the palate is inspected it will be seen that it hangs relaxed, and cannot be raised, whether by movements of inspiration, phonation, or deglutition.

Speech may become indistinct, and even incomprehensible, especially in children; and the difficulty of deglutition becomes marked, especially in drinking, during the efforts at which the fluids chiefly return through the nose. By participation of the constrictor muscles of the pharynx in the paralysis, the difficulty of swallowing becomes still more increased; but it does not often proceed to utter impossibility of swallowing food, as is seen in bulbar paralysis. If the uvula is titillated it remains immovable, nor is the soft palate arched upwards.

Faradic contractility is well maintained in the earlier days of the affection, but soon disappears, while, at the same time, a

weak galvanic current causes visible contractions in the azygos uvulæ and levator veli palatini muscles. The general symptoms are not especially striking. The children are very irritable and anæmic, but have no fever, no disturbance of the digestion, and no further pathological manifestations generally.

The malady frequently remains limited to the symptoms just described, and is then regularly recovered from in a few weeks.

In other cases there is added to it paralysis of the muscles of visual accommodation, of those of the extremities and trunk, and finally of the muscles of respiration, upon which death follows as a matter of course.

The frequency of this characteristic sequel of diphtheria varies according to the special epidemic, but is by no means governed by the severity of the epidemic. I observed paralysis of the soft palate most frequently in an extensive epidemic, in which diphtheria of the larynx and secondary albuminuria were rare; while, on the other hand, a thick membrane occupied the soft palate almost regularly.

Sée asserts that in some epidemics, thirty, or even fifty per cent. of all diphtheritic cases acquire this after-disease. Roger speaks of fifteen per cent.; and others, again, are of the opinion that it occurs in only about two per cent.

The *diagnosis* is extremely easy, and it is only possible for it to be confounded with the paralysis of the soft palate attendant upon bulbar paralysis. In the latter, however, the course is eminently chronic, and leads to death; it exhibits no etiological origin, and soon involves the orbicularis oris muscle. A mechanical paralysis of the soft palate, or one produced by a neoplasm, would easily be detected on inspection, and thus differentiated from that which ensued as a sequel of diphtheria.

The *prognosis* is, as already remarked, always favorable, when the trouble remains localized upon this little territory; and even the general paralysis following diphtheria deserves a proportionately favorable prognosis, if the muscles of respiration are not involved.

The *treatment*, accordingly, is tolerably inactive. Perhaps the malady may be shortened by the faradic current, and, apparently, even more so by the galvanic current. A result of this kind,

however, is difficult to be distinguished from rapid recovery under entirely expectant treatment.

D.—The Parotid Gland.

Anatomico-Physiological Introductory Remarks.

The parotid gland (*παρά*, near to; *τὸ οὖς*, the ear) extends, according to Henle, from the region of the external auditory meatus downwards to the angle of the lower jaw. It is flattened and triangular, with its apex directed downwards. It rests with its posterior border upon the anterior margin of the sterno-cleido-mastoid muscle, and with its anterior border overrides somewhat the masseter muscle. Its superior border follows exactly the curvature of the cartilaginous portion of the external auditory meatus. Its external surface is even, covered by the skin and the fasciæ of the subcutaneus colli and risorius muscles, and tensely attached to their under surface. Inwards, the parotid gland fills the gap between the lower jaw, the sterno-mastoid muscle, and the cartilaginous auditory meatus. Its superior border is formed into a groove that takes in the cartilaginous meatus and surrounds it closely. It is separated from the angle of the lower jaw by the trunk of the external carotid artery and the external jugular vein; and it is divided into a larger external and a smaller internal layer by the facial nerve.

The excretory duct, or *ductus parotideus sive Stenonianus*, or duct of Steno, issues from the anterior border of the gland, about at the level of the nasal openings, and passes forwards in a horizontal direction over the masseter muscle. It dips inward over the anterior border of this muscle, crosses the zygomatic muscle, and penetrates the buccinator muscle and the mucous membrane of the cheek in an oblique direction, opening into the cavity of the mouth opposite the second molar tooth of the upper jaw.

The opening of the duct is recognized on the everted cheek of the living subject as a small black point as large as a pin-hole, or an oblong dimple, into which a delicate button-headed

probe or canula can readily be introduced. In individual cases the opening is marked by a small wart-like elevation.

The noise which one hears in his own ear when the air in the respiratory tract is compressed by abdominal pressure while the nose and mouth are kept closed, is at least in part explicable by the pressure of the air-distended acini of the parotid gland upon the external auditory meatus. Although it cannot be denied that a portion of this sound depends upon the current of air forced through the Eustachian tube into the cavity of the tympanum, the distention of the parotid gland by air can be experimentally proved. For if, while this forcible expiration is being made, the duct of Steno is at the same time compressed by the finger against the upper molar tooth, this sound is not produced.

The salivary secretion (and the influence of the nerves upon it) is the best understood of all secretions. Since the path was broken by the labors of Ludwig, these investigations have been undertaken in great numbers and with various modifications, the details of which may be consulted in the more recent compendia of physiology. For our clinical purposes, the investigations of Mosler only need be cited, these having demonstrated the behavior of the parotid secretion in various pathological processes. Mosler obtained the parotid saliva by the method of Eckhard, which consists in introducing a canula through the mouth into the duct of Steno, and then exciting the salivary secretion by irritating the mucous membrane of the mouth with vinegar, sugar, and other materials. In this manner the saliva was examined in cases of diabetes mellitus, icterus, stomatitis mercurialis, and typhus. We shall have occasion, in the course of this section, to refer several times to these interesting experiments.

Parotitis. Inflammation of the Parotid Gland.

Cruveilhier, Revue méd. 1830.—*Naumann*, Hecker's Annal. 1833. Mai.—*Eisenmann*, Die Familie Rheuma. Bd. 3. p. 374.—*J. Houghton*, Cyclopæd. Vol. III.—*Leitzen*, Hufeland's Jour. f. prak. Heilkde. 1838. Bd. 86. St. 4. p. 101.—*Bérard*, Les maladies de la glande parotide. 1841.—*Clemens*, Brand der Ohrspeichel-

drüse. Deutsche Klinik. 1850. p. 479.—*Röser*, Würtemburg. Correspbl. 1855. p. 36.— *Virchow*, Annal. d. Charité. Berl. 1858. Jahrg. VIII. Hft. 3.—*Schmidtmüller*, Bair. ärztl. Intelligzbl. 1858. p. 151.—*Bruns*, Handbch. d. prakt. Chirurgie. II. 1. p. 1053. Tübingen. 1859.—*Meynet*, Parotitis, metastasis to the ovaries. Gaz. hebd. 2 Sér. III. 31. 1866.—*Mosler*, Das Parotidensecret in verschiedenen Krankheiten. Berlin. klin. Wochenschr. III. 16 u. 17. 1866.— *Wittich*, Einfluss des Sympathicus auf die Function der Parotis. Virchow's Arch. Bd. 37. 1866. p. 93.—*Martini*, Militärsanitätswesen. Schmidt's Jahrbcher. Bd. 133, p. 133. 1867.—*Eckhard*, Zur Lehre. v. d. Speichelsecretion. Henle und Pfeufer's Ztschr. XXIX. 1. p. 74. 1867.—*Combeau*, Des oreillons. Thèse. Paris. 1868.—*Sallaud*, Des oreillons. Thèse. Montpellier. 1868.—*Guéneau de Mussy*, *Noël*, Studies on parotitis. Gaz. hebd. de méd. 1868. 40-43.—*Ropas*, Sur les oreillons. Thèse. Paris. 1869.—*Debize*, De l'état typhoïde dans les oreillons. Thèse. Paris. 1869.—*Duroziez*, Five cases of parotitis. Gaz. des Hôp. 1870. No. 93.—*Blondeau*, Sur une épidémie d'oreillons. Gaz. des. Hôp. 1870. No. 79.

By parotitis we understand a considerable, painful enlargement of the parotid gland, acute in its course, in most cases subsiding without leaving any traces behind, but becoming purulent in others. Based upon these two different courses of behavior, we naturally distinguish two forms of the disease, viz. : 1, *idiopathic*, and 2, *metastatic parotitis*, which are also distinctly separated from each other etiologically. For the second form there exists only the single scientific appellation, *parotitis metastatica ;* while for the first, besides its scientific synonym, *parotitis epidemica* or *polymorpha*, there are a number of names, in part quite vulgar, by which it is known in the German provinces : *mumps*, *Ziegenpeter*, *Bauerwetzel*, *Tölpelkrankheit*, *Klirren*, *Kehlsucht*. The peculiar love of the German populace to designate this disease by comical, contemptuous appellations is in part explained by the really ludicrous appearance which the face undergoes as a consequence of the swelling of the parotid, and its œdematous surroundings.

Etiology.

Parotitis frequently appears epidemically and contagiously, and thus approaches the infectious diseases in many respects, from which, however, it is distinguished by its short duration, and the slight participation of the organism in general. This

disease, like whooping-cough, indicates the presence of a contagion, which, although the local manifestations are very considerable, excites little fever, or none at all.

In the occurrence of epidemics of mumps, some remarkable peculiarities are to be observed. First of all, they are much less frequent, for instance, than the acute exanthemata, often avoiding a locality for thirty years or more; interesting examples of which have been narrated by Behr of Bernburg, and Hinze of Waldenburg. In some localities, on the contrary, mumps is a very frequent disease, and appears almost annually, chiefly in the spring or autumn.

The participation of the population in this malady is never a uniform one, inasmuch as the male sex shows a much larger percentage of attacks than the female. Children from two to fifteen years of age are most prevalently affected; nurslings almost always possess complete immunity, and very old people likewise. Older persons are especially little disposed to it, partly to be explained by the circumstance that they may have already once gone through the disease in early life. The law of protection by one attack is almost as unexceptional for mumps as for the acute exanthemata.

Military surgeons have remarkably much to report of epidemics of mumps among their troops. Bruns cites a work of Rochard, dated 1757, which asserts that in Belle-Isle-en-mer the parotid tumor was endemic; not that it existed continuously, for there were pauses sometimes of eight or ten months' duration. It occurred at all periods of the year, but most frequently in autumn and winter, and affected those soldiers only who stood guard; while non-commissioned officers, drummers, and the like, who shared the diet and quarters of the soldiers, were never affected. In these cases, at any rate, we must give up the idea of a contagion multiplying itself in the body, else this immunity of the remainder of the men would be inexplicable. The etiology of this kind of mumps seems much more linked to that of malaria, which causes intermittents among those on guard in the ditches and outworks of some fortifications, without its further extension among the rest of the troops. The preference manifested by the disease

for the moist seasons of the year also speaks for its telluric origin.

Epidemic parotitis stands in a certain relation to the acute exanthemata, that is, it frequently appears before or during epidemics of measles, or follows upon them. A similar relationship to scarlet fever and diphtheria is also claimed by some authors, but is apparently much less frequent than the connection with measles. The frequently expressed opinion that, when scarlatina and mumps appear simultaneously at one place, being taken sick with one of the two diseases is a protection against the other, I am able to refute by many examples from my own experience.

The duration and intensity of an epidemic are very variable. While at one time it becomes extinguished in a few weeks, and spreads so little that it can hardly be termed an epidemic, at another time it will attack nearly every child and many adults, and is not exhausted until after the lapse of several months. The same holds good for the extension of individual epidemics. In many cases it is limited to one institution, or to one garrison, in others it spreads over whole cities and extensive circuits.

That mumps is sometimes decidedly contagious, is clearly evident from the way in which it spreads in many epidemics confined to houses, of which the following description of Leitzen furnishes a striking example.

In the latter half of April, 1837, while mumps was neither prevalent in the institution, namely, Franke's establishment in Halle, nor in the town, the scholars returned from vacation. One of them had sojourned at Pritzwalk, in the Mecklenburg district, where mumps prevailed, and where he had a good deal of intercourse with patients thus affected. After his return, this lad sickened with a moderately severe epidemic parotitis, from which he recovered in five days. During the latter days of April, six orphans who had had considerable intercourse with him, sickened, and then the malady spread further in this section. Of the scholars who dwelt apart from these orphans, but who were instructed in common with them, the first sickened on May 16th, and then the mumps spread among them. The first cases in the city occurred at the end of May, and only among individuals who visited the orphan asylum; and then the mumps spread so rapidly that some three hundred cases came under treatment in the polyclinic. During the Whitsuntide vacation an orphan visited Trotha, an hour distant from Halle, took the mumps in the early days of his sojourn there, and infected two children in the same house, without the

disease spreading any further in Trotha. It is to be remarked, in this connection, that the last two cases were carefully confined to the house.

Bruns[1] reports in his exhaustive description of parotitis a series of such facts, through which he undoubtedly proves the *occasional contagiousness* of the malady. The period of incubation appears to vary between four and twelve days. Undoubtedly, at other times and in other places, in spite of repeated cases being introduced, no further extension takes place, on which account its contagiousness is wholly denied by some authors. We have here a history analogous to that afforded in typhoid fever, in which the physicians of large cities can never observe any distinct contagion, while those in the country do so very frequently.

The remaining causes of idiopathic parotitis are very subordinate in character; wounds, bruises, the penetration of foreign bodies from the mouth into the duct, obstruction of the duct by salivary calculi, and swelling of the lining membrane, are here to be mentioned. The inflammations of the gland thereby provoked, as well as that ensuing from extension from neighboring organs, from the skin, the mucous membrane of the mouth, the bones, and the auditory apparatus, therefore, in fact, the secondary or deuteropathic inflammations proper, belong to the domain of surgery.

Taking cold should be mentioned as the last cause of a benign sporadic parotitis, which, if the salivary gland, by previous disease or by irritations in its neighborhood, has already become a *locus minoris resistentiæ*, may well give occasion to the disease in individual cases. It may be well to remark here that the lymphatic glands swell much more frequently than the parotid, from partial cooling off of the skin, and that by the position of the lymphatic glands of the neck a mistake in diagnosis may readily be made.

We now come to the causes of *malignant parotitis, which proceeds to suppuration*—the so-called metastatic parotitis. This accompanies a great number of severe, acute diseases, and is therefore also called symptomatic parotitis. It is most fre-

[1] Handbuch der prakt. Chirurgie. 2. p. 1056.

quently associated with typhus, in all its forms—abdominal and exanthematous typhus, recurrent typhus and the plague; it is furthermore associated with the acute exanthemata, scarlet fever, measles, and small-pox; with pyæmia, puerperal fever, dysentery, typhoid cholera, and, in the tropics, with yellow fever also. The earlier the parotitis sets in, the more dangerous it is; while that which appears towards the end of a typhus or in a puerperal fever which has lasted longer than a week, frequently terminates in recovery, so that a critical significance has been erroneously attributed to it.

In severe fevers in which the cavity of the mouth is always disposed to dryness, and sometimes grows quite hard and scaly, the ensuing metastatic parotitis might be explained on purely mechanical grounds. By the drying of the mouth of the excretory duct, a damming up and decomposition of the saliva may take place, and thus develop an irritation of the gland, which finally leads to its purulent destruction. Upon the basis of this view, Mosler even recommends catheterization of the duct of the parotid in typhus, in the hope of thereby preventing further swelling after commencing tumefaction of the parotid.

Certain objections, however, can be urged against this mechanical explanation. In the first place, it is remarkable that parotitis so seldom occurs, though dryness of the cavity of the mouth continues for weeks in almost every severe case of typhus; and, in the second place, it is never seen to occur in many processes seriously affecting the mucous membrane of the cheek, as in thrush for example, in simple and mercurial stomatitis, or in corbutus. Nothing remains, therefore, but to take refuge here, lso, in a pure theory of changes in the composition of the blood.

Pathological Anatomy.

The anatomical alterations in mumps are exceedingly little known, for the reason that the entire malady is altogether acute and devoid of danger, and therefore affords no opportunity for autopsies. I have never once seen on the dissecting-table a parotid gland swollen by benign mumps, and can only refer to the views of others, above all to Foerster. According to Foerster,

the inflammation always begins with hyperæmia, and the gland appears in consequence brightly reddened on its cut surface. Serous exudation is immediately associated with this hyperæmia, by means of which the cut surface of the then not only reddened but also swollen gland presents a uniform, flesh-like, succulent, but no longer granular aspect. Sometimes the connective tissue surrounding it is also infiltrated with serum, and the tumor thus still more enlarged. In other cases the entire swelling has consisted of this infiltration of the surrounding connective tissue, and the gland itself has remained unchanged.

Further anatomical alterations cannot be supposed to take place in benign parotitis polymorpha. Fibrinous exudations would necessitate deviations from the normal form and consistence of greater permanence, and purulent destruction would give rise to much more intense symptoms and finally to discharge externally, etc.

Hyperæmia and serous infiltration alone are susceptible of so rapid a subsidence as we regularly observe in mumps. Whether the exudation is greater in the interstitial connective tissue or in the gland and its excretory ducts, does not appear to be established. By analogy with croupous pneumonia, whose prompt favorable course most recalls mumps, I am inclined to sustain Virchow, who holds to a simple catarrh of the mucous membrane of the salivary canals as far as into the small aggregations of gland substance proper.

According to Virchow, the essential foundation of all forms of parotitis is only a more or less malignant catarrh of the ducts of the gland. He divides them into three groups :

1. Primary simple catarrh, usually appearing epidemically (*angina parotidea*, mumps), without disposition to suppuration and ulceration.

2. Secondary, purulent catarrh, readily productive of abscess (*blennorrhœa parotidea*), usually in connection with previous catarrh of the cavity of the mouth, and not unfrequently with affections of the middle ear.

3. Specific catarrh, almost always leading to ichorous degeneration, usually associated with ichorrhæmic or embolic metastasis in other places.

The second form, *metastatic parotitis*, has been much more thoroughly investigated, anatomically, than the other form, for the reason that it is frequently found in the corpses of persons dead from severe infectious diseases. Some authors distinguish two different forms, in one of which the inflammation proceeds from the connective tissue of the gland, and in the other from the mucous membrane of the ducts and from the acini of the gland. I could never make out a division so distinct in all the numerous autopsies that have come under my notice. The condition of things is naturally different, according to the duration of the parotitis previous to death. If the patient dies when the swelling of the gland has existed but one or two days, the glandular tubes and acini are found swollen and reddened, while the connective tissue of the gland is yellowish-red and infiltrated with serum. A viscid, ropy, gray-white secretion accumulates in the ducts, which soon takes on a purulent character. By pressure on the gland, and by stroking the duct towards its orifice in the mouth, this fluid can be forced from the orifice, according to Bruns, and we can also, according to him, distinguish the purulent contents of the ducts (the main one and its branches), upon section through them, from their thickened walls.

If the parotitis has existed a few days longer, purulent softening of the acini of the gland sets in, and this always begins in the centre and spreads towards the periphery, so that the acini finally become changed into small collections of pus. Finally, the ulceration also seizes upon the interacinous connective tissue, and the originally multiple little abscesses unite into one or a few great cavities of pus. This pus now seeks an outlet. It either breaks directly towards the outer surface, which always requires a considerable time, on account of the toughness of the parotido-masseteric fascia, or it invades the neighboring organs. Among these, the first place to mention is the external auditory meatus, and the rupture generally takes place on the line between the cartilaginous and osseous portions of the auditory meatus, or else further forward.

The remaining ways in which the abscess may open into the mouth, the pharynx, or the œsophagus, or, still further, by working its way down into the anterior mediastinum, along the

sheath of the sterno-cleido-mastoid muscle, are represented in literature only by a few isolated reports.

Besides this travelling of the pus, other inflammations also occur, from the very start, in the neighborhood of a metastatic parotitis, namely, in the masseter, pterygoid and temporal muscles, from whence the pus forces its way inwards and upwards towards the base of the skull into the temporal or the zygomatic fossa. The purulent process may involve the periosteum of the adjacent bones, the temporal portion of the sphenoid bone and the lower jaw, and even the bones themselves; and it may finally spread through the cranial bones to the cerebral membranes and the brain. Not at all unfrequently the labyrinth and middle ear participate, in which case the pus probably passes directly along the vessels and nerves that go from the parotid gland to the ear. The ossicles of the ear thus become destroyed, and, at the best, life-long deafness ensues.

The lymphatic vessels, veins, and nerves that traverse the parotid gland are naturally drawn into the disease on suppuration of the gland. As a result of the irritation to the lymphatic vessels, the neighboring lymphatic glands are always found swollen, reddened, or even likewise in course of suppuration. Thrombosis frequently takes place in the jugular vein and its branches, and the breaking-down of the clot leads to septicæmia and ichorization of the sinuses of the dura mater. The nerves traversing the parotid, the facial nerve, and some twigs from the second and third branches of the fifth, are also destroyed in a complete breaking-down, especially in gangrenous destruction of the parotid gland; but in case of the simple formation of an abscess, they appear to remain intact a long time; otherwise paralysis of these nerves would be more frequently observed as a result of the disease. Apart from this, the facial nerve seems especially calculated to conduct the inflammation into the auditory apparatus, while the twigs of the trifacial favor its transportation into the brain, as would be inferred from the observations of Virchow, who has several times seen the environs of the Gasserian ganglion infiltrated with pus.

The parotid sometimes rapidly undergoes gangrenous degeneration from causes unknown. From the purulent cavities gangre-

nous cavities form, filled with ichor and gas, out of which, after they are opened, black shreds of connective tissue and of gangrenous acini can be drawn. The gangrene may destroy the gland in part or in toto, in which case, all the tissues, even the facial nerve, are destroyed ; and, after recovery, a deep chasm remains beneath the ear.

Symptoms.

Idiopathic parotitis is so different from the metastatic form, not only in its causes, but also in its course and termination, that a separate consideration of both varieties appears necessary.

a. *Idiopathic Parotitis.*

Its precursors are in no wise characteristic ; frequently, even, they fail entirely. The first symptom, then, is the local pain on opening the mouth. In many epidemics, again, the precursors are more severe, and continue several days. They consist in feelings of depression and vague pains in the limbs, headache, loss of appetite, slight chills, and even distinct febrile movements towards evening. Sometimes even alarming symptoms are developed, such as frequent vomiting, diarrhœa, great anxiety, a disposition to syncope, and sometimes convulsions in nervous and irritable children, so that we have before us the entire array of symptoms which indicate the onset of the acute exanthemata. Trousseau reckons the period of incubation at from ten to fourteen days ; though there are various older and more recent authorities, according to which they continue but from six to eight days.

The commencement of the glandular swelling is first indicated by fleeting stitches of pain in the region of the parotid gland. These may spread upon all sides, especially towards the ear, and are increased by movements of the lower jaw, by speaking and by chewing, as well also as upon pressure from without.

As the pains increase, a distinct, visible tumor becomes developed, almost always on one side only, beneath the lobe of the ear, which very soon appears somewhat raised from the middle line, so that, on looking in the face of the patient, the distances of

the ear-lobes from the middle line become dissimilar. In simple mumps the tumor never acquires that degree of hardness which is shown in suppurative parotitis; it always feels somewhat doughy, and is not immoderately painful to the touch. The greater alterations of form depend rather more upon the œdematous swelling of the neighboring connective tissue than upon that of the parotid gland itself; and hence the tumor is never sharply circumscribed. It loses itself superiorly towards the cheek and the eyelids, anteriorly towards the corner of the mouth, and inferiorly towards the clavicle. The inferior maxillary and sublingual regions, especially, undergo a remarkable expansion, which even extends beyond the middle line, and finally gives the chin the appearance of resting against a padded wall. It is even claimed that the circumference of the neck has sometimes exceeded that of the head, so that the shoulders, neck, and head together have formed a truncated pyramid. With such a swelling all movements of expression of the face are naturally suspended; and the staring aspect of the visage, combined with the thickness of the neck, gives the patient an idiotic appearance, which has occasioned the many vulgar appellations by which the disease is popularly designated.

The consecutive œdema not only extends towards the periphery, but also in depth, so that the tonsils, the pharynx, and even the larynx may be invaded by it. The symptoms resulting from this extension are, a further increase of the difficulties in chewing and swallowing, an altered, nasal intonation, and sometimes, though but seldom, a sense of suffocation, which may advance to stenosis of the larynx.

The skin over the tumor is not essentially altered, either in respect to color or to temperature. With a moderate-sized tumor it is somewhat reddened; but when tightly stretched, on the contrary, the filling of the capillaries is impeded, from the effect of which a pale, waxy, glistening appearance results. The skin cannot be raised into a fold anywhere over the tumor, on account of the œdema of the subcutaneous connective tissue. The pain remains tolerably moderate during the entire duration of the swelling. The three points of pain indicated by Rilliet and Barthez, namely, at the articulation of the lower law, under

the mastoid process, and over the submaxillary gland, are not always distinctly pronounced, though readily to be explained by the compression of the periosteum, and the submaxillary gland. The œdematous parts at a greater distance from the parotid are almost always altogether painless.

The patient holds his head stiffly towards the diseased side in unilateral parotitis, and immovably straight in bilateral parotitis. Opening the mouth is for some days entirely impossible. Hardly the point of a teaspoon can be forced between the rows of teeth; all movements of chewing and swallowing require a great effort; and hunger and thirst are endured a long time before the patient makes up his mind to allow anything to pass his lips. As local disturbances yet to be mentioned, are: hardness of hearing, shooting pains and continuous ringing in the ears, increase of the salivary secretion to salivation, or its decrease with a sensation of dryness in the mouth, loss of appetite, vomiting, constipation, and, finally, symptoms of cerebral hyperæmia, the result of pressure upon the veins in the neck.

The inflammation is often limited to one parotid, while the gland of the opposite side remains entirely free or shows only slight evidences of inflammatory swelling, and this tendency to unilateral disease may distinguish whole epidemics. On the other hand, there are also epidemics in which both parotids are affected in like degree in most cases, though never simultaneously, the gland last diseased always reaching its culminating point two or three days after the first has done so, the course of the process being thus lengthened a few days.

Elevations of temperature and increased rapidity of pulse are of slight moment and of short duration in most epidemics—no trace of fever remaining, usually, by the fourth or fifth day; sometimes, however, a pronounced typhous condition is said to be developed. Debize, of Paris, relates some cases in which dryness of the tongue and lips, great prostration, apathy, sometimes even delirium or sopor, picking at the bedclothes, and a brown coating of the lips and tongue, were observed, while, in one case, the evening temperature remained at 104° Fahr. for several days.

The course of regular mumps is favorable, as a rule. After

the local and general symptoms have culminated in from three to six days, they gradually subside in about the same length of time, so that recovery is complete in from one to two weeks. It is naturally understood that the inflammation of the gland, in such instances, does not go on to suppuration, as often occurred in former epidemics, but that it subsides. The absorption of the exudation takes place with remarkable rapidity, so that in a few days everything has disappeared, leaving no trace. In scrofulous children this sometimes occupies a somewhat longer time, and may be protracted during several weeks; though here, also, resolution is always finally accomplished. First the œdema disappears, and then the swelling of the gland, during which the epidermis is cast off in fine scales. Critical excretion by means of the perspiration and the urine frequently does not occur.

Swelling of the testicle, usually unilateral, is a very characteristic complication of this epidemic parotitis, and was known to Hippocrates. It hardly ever occurs in childhood or in old age, but is usually seen only in pubescent youths and virile men. In most instances the testicle itself becomes swollen, constituting orchitis; therefore, less frequently, there is an epididymitis, and with it, usually, an acute hydrocele and œdema of the subcutaneous connective tissue of the scrotum. The symptoms are not very annoying; the pain is dull, in spite of considerable swelling, even to two or three times the normal volume, and the spermatic cord is not sympathetically affected. In very severe cases there is also a gonorrhœa-like discharge from the urethra, and burning pain in micturition. The fever, which, as a partial symptom, had already subsided, now reappears for a few days, but usually disappears promptly after the administration of an emetic—a favorite method of treatment at all times for this complication.

The course of the inflammation of the testicle is similar in duration to that of the mumps, increasing for from three to six days, and then as rapidly subsiding, with complete recovery in from one to two weeks. The rapid and safe resorption of the exudation shows that, as in mumps, it can only be serous in nature. This characteristic complication begins about six or eight days after

the appearance of the mumps, and the testicle remains swollen for several days after every sign of disease has disappeared from the parotid gland. Usually but one testicle becomes affected, even in bilateral parotitis, and most commonly that of the right side, while the left parotid is most frequently affected. When both testicles are involved, it is, according to Gravis and Stevenart,[1] as unfrequently simultaneously as when both parotids are affected, one testicle being always affected first, and then the other a few days later.

An observation of Blondeau is also interesting, viz., that mumps patients suffering under gonorrhœa are not at all disposed to swelling of the testicle, and the mumps generally pursues its course free from all complications. Cases, too, in which a repeated alternation of the swelling between the parotid gland and the testicle takes place are very rare in the literature of the subject.

The testicle usually resumes its normal condition after the subsidence of the tumor, but in some epidemics it is said to undergo complete atrophy, a remarkable example of which is cited by Bruns.[1] Dogny[2] reports the same concerning an epidemic which raged in the garrison of Mont Louis (Pyrenées orientales), in January, 1828. Of eight hundred men, eighty-seven were affected—all of them between twenty-two and twenty-eight years of age, except one who was thirty-two years old. The course of the disease was favorable; its duration was from eight to twelve days; metastasis to the testicle was very frequent. Of sixty-nine bilateral and eighteen unilateral cases of parotitis, metastasis to both testicles occurred in four cases, and to one testicle only in twenty-three cases, *all of which resulted in atrophy of the affected testicles*—of which the reporter convinced himself several months afterwards. This report, unfortunately, is silent as to the condition of the sexual functions—whether seminal emissions and the power of reproduction were lost in those patients who had been affected with bilateral atrophy.

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[1] *Bruns*, Hdbch. p. 1091. [2] Ibid. p. 1089.

[3] Journ. de méd. et chirurgie. Paris. 1832. T. III. p. 107.

In the female sex, who are much less frequently attacked by mumps, there occurs, though in much more isolated instances, a secondary swelling of the external genitals, of the ovaries, or of the inguinal glands. Reports of this character are so scarce that it is doubtful whether they actually represent a metastasis, or only the spontaneous occurrence of both affections in one individual. A case of M. Peter,[1] of recent date, is worthy of mention, by which it apppears that *not only may there be a metastasis from the parotid glands to the genital organs, but that, vice versa, metastasis may take place from the latter to the former.*

In a woman twenty-three years of age the previously regular menstruation ceased in the year 1848. Since that time she had been subject to a series of inflammations of the parotid, always on the left side, and always readily subdued by leeches and cataplasms. They occurred at the periods of the failing menses. At times a sanguineous tumor of the left larger or smaller labium of the left side occurred, at the same period, instead of the swelling of the parotid.

Finally, Combeau adduces observations which show that the conjunctiva and the mucous membranes of the throat, urethra, and vulva may become inflamed during the course of the disease. On this account he is decidedly in favor of regarding parotitis as a general affection with manifold localizations, and opposes the theory of metastasis.

b. *Deuteropathic or Metastatic Parotitis.*

The local differences between idiopathic and deuteropathic parotitis are slight during the first few days. In the latter variety the gland is harder, denser, and somewhat better definable, on account of the lesser amount of œdema in the surrounding parts. The temperature of the skin rises over the inflamed gland, so that the difference is readily appreciable by the hand; and the tensely stretched skin soon reddens. The painfulness is much greater, also, when purulent destruction begins, and first decreases after distinct fluctuation is perceptible. But as this metastatic parotitis frequently appears in the earliest

[1] Gaz. des Hôpitaux. 1868. No. 37.

days of severe infectious diseases, typhus and scarlatina, the pain is not often loudly complained of on account of the clouded condition of the sensorium. The same is true with reference to the remaining subjective troubles in the ear, in the throat, in the movement of the lower jaw, and of the neck, etc. In metastatic parotitis absorption hardly ever occurs, but it goes on regularly to suppuration or even to mortification, provided that the life of the patient is prolonged long enough. A few days, usually, are sufficient to conduct the process to maturity. First, we observe a doubtful, then a gradually more distinct and more extended fluctuation beneath the blue-red skin, and then the skin becomes thinned and elevated at the point of fluctuation, which is always a sign that the parotid fascia has been penetrated.

In infrequent cases the course is protracted and extends over several weeks, until, finally, the abscess is ruptured spontaneously, or is evacuated by incision. Cicatrization by granulation follows in most instances, often protracted by the formation of abscesses in other portions of the gland, and discharge of the pus through the skin at several points. The abscess, however, may also become transformed into an ichorous cavity, and finally becomes gangrenescent, in which case there may be further infiltration and gangrene of the adjacent parts, as has been already described in detail under the head of pathological anatomy.

The mortification of the gland is recognized by the cadaverous odor, the blackening of the skin, the cavity formed, and the discharge of gangrenous shreds. Gangrene is not frequent, however, in metastatic parotitis, but is more often observed in sporadic parotitis from unknown causes, of which the reports of cases cited from Bruns, and two cases treated by myself, give evidence.

The course and termination of metastatic parotitis depend much more upon the nature of the disease which it attends than upon the amount of local disturbance ; and here it may be set down as a rule that the parotitis is the more unfavorable and dangerous the earlier it appears in connection with a typhus, a scarlatina, etc., while its course is mostly favorable when it occurs during convalescence from these infectious diseases.

Diagnosis.

The superficial position, and the characteristic form of the inflamed parotid, prevent all doubt as to the seat of the disease. The elevation and distortion outwards of the lobe of the ear excludes its confusion with simple tumefaction of the lymphatic glands, which frequently occurs in scrofulous subjects. The acute commencement and course of the disease readily show that the process depends upon an inflammation, and is not due to a morbid growth or to the very infrequent chronic hypertrophy of the gland.[1]

Idiopathic parotitis, by its usually epidemic appearance, its mild course, and the extreme rarity with which it ends in suppuration, is sufficiently distinguished from the deuteropathic form, which is, furthermore, always complicated with other severe processes. Whether the suppuration, in the metastatic form, originated in the salivary ducts and glandular acini, or in the interstitial connective tissue, is said to have been determined, in some cases, on the living subject by the fact that, in the former case, external pressure on the gland caused the escape of a drop of pus from the orifice of the duct into the mouth, while in the latter case it did not. I have repeated this experiment a number of times, always without any result. But I do not, on that account, adhere to the doctrine of primary interstitial suppuration, inasmuch as the salivary duct may become so swelled as no longer to give passage to the pus.

Prognosis.

The prognosis in mumps, as already appears evident from the consideration of its symptomatology, is exceedingly favorable, so much so that in many cases professional assistance is not solicited, especially towards the end of an epidemic. A fatal

[1] We omit in this manual of special pathology the discussion of hypertrophy and benign and malignant tumors of the parotid; likewise of salivary tumors, salivary fistules, and salivary calculi, all of which have been exhaustively considered in Bruns' *Handbuch der prakt. Chirurgie.* II. Abth.

termination appears never to occur in individuals previously healthy.

In metastatic parotitis the prognosis varies: 1st, according to the nature of the inducing disease; and, 2d, according to the period at which the parotitis shows itself. At the commencement, or during the height of a typhus or scarlatina, this complication is regarded as very dangerous, and almost always leads to a fatal termination. After a disposition towards improvement has set in, its course is mostly favorable, and on this account it was included by our predecessors among the critical secretions. We can, however, observe no remarkable improvement, even at this stage, on the additional appearance of parotitis, and must acknowledge, at most, that convalescence is not evidently interrupted nor delayed thereby. It is to be regarded at any time as an undesirable complication. The unfavorable results of purulent or gangrenous destruction are sufficiently apparent after study of the pathological anatomy of this affection. Apart from the conspicuous defect which is manifested by the tolerably deep depression beneath the ear, there ensues, on account of the destruction of the facial nerve, a marked and incurable paralysis of this nerve; and by the extension of the destructive process upon the neighboring bones, protracted suppuration results with partial exfoliation of the bones; and by the escape of the pus into the inner ear, life-long deafness and manifold brain symptoms are produced.

Treatment.

The only effectual prophylaxis against an epidemic disease—removal from the infected locality—would be made very little use of for mumps, inasmuch as the danger from the disease is too slight in proportion to the inconvenience of a change of residence. Other prophylactic measures have been recommended by officious practitioners; but they are all without any rational foundation, and afford no efficient protection.

Epidemic parotitis does not ordinarily require any medicinal treatment. Avoidance of injurious influences, rest, anti-febrile diet, and equable warmth suffice, as a rule, for the restitution of

normal health. For constipation slight laxatives may be given, but not calomel; and acid drinks may be allowed for the fever. Emetics are highly recommended by some physicians, by military surgeons especially, and do appear to afford relief in very severe bilateral parotitis with great subjective troubles. I have never yet found any pressing indication for this treatment, however, in practice among children.

As to the requirements for topical treatment, an inunction with grease and oil is much simpler and more appropriate than the application of heating herb-bags or cataplasms. Very great local distress may be somewhat alleviated by a few leeches and by ice-compresses. The use of cold is never dangerous, but is found very unpleasant by many patients, and, in most instances, is soon suspended. Delayed absorption in scrofulous children is hastened by ointments of mercury and iodine or painting with tincture of iodine.

For the inflammation of the testicle, which occurs now and then, in adults only, a somewhat elevated position of the scrotum and warm applications to it usually suffice; at most the attempt may be made to produce a slight diversion to the mucous membrane of the intestines by the administration of mild laxatives. In cases of threatening brain symptoms, cutting off the hair, local and general venesection, drastics, and irritants to the skin are indicated.

The treatment of deuteropathic parotitis is principally directed towards facilitating suppuration and favoring its escape externally, which is best effected by active poulticing. According to Bruns[1] we should not neglect to search for the salivary duct in the mouth, and then endeavor, by pressure upon the parotid from the outside, or by the introduction of a probe into the orifice of the duct, to remove any obstacle that may exist in the duct of Steno, and to press out any stagnant saliva or pus. In this way, perhaps, resolution may be promoted, or suppuration be prevented. In typhus, Mosler has employed the introduction of a canula into the salivary duct, with success, as a remedy for parotitis. He has succeeded in this way in effecting

[1] L. c. p 1106.

the subsidence of commencing tumefaction, and in preventing further inflammation; so that he strongly recommends this method as prophylactic treatment in the parotitis of typhus.

If suppuration has once taken place, the fluctuating point should be punctured as soon as possible, and so deeply as to penetrate the fibrous parotid fascia. In this way the pain is most rapidly subdued, and more deeply extending suppuration is averted.

Artificial openings, as well as spontaneous ones, have a great disposition to close too soon; and it is therefore often necessary to dilate them, in doing which a direction downwards should be given in order to facilitate the flow of the pus.

The general treatment depends much more upon the nature of the infectious disease which has given rise to it, than upon the inflammation of the gland itself, and for the most part must be of a stimulating character.

2. Ptyalism. Salivatio.

The older literature has been very exhaustively brought together in *Canstatt*, Bd. IV. Stomatitis mercurialis u. Ptyalismus.—*Frerichs*, R. Wagner's Handwörterbuch d. Physiol. Artikel Verdauung. Bd. III.—*Jacubowitsch*, De saliva. Inaugural Dissertation. Dorpat. 1848.—*Bidder und Schmidt*, Die Verdauungssäfte und der Stoffwechsel. Leipzig u. Mitau. 1852.—*Christison*, A treatise on poisons. 1845. p. 408.—*Duclos*, Canstatt's Jahresber. 1846. Bd. V. p. 180.—*Behrend's Arch.* Bd. I. 1846. p. 197.—*Lehmann*, Lehrbuch der physiol. Chemie. 2 Aufl. Leipzig. 1853. Bd. II. p. 9.—*Bamberger*, Virchow's Handbch. Bd. VI. Abth. I. p. 66.—*Hebra*, Allg. Wien. med. Zeitung. 1861. No. 20.—*Kussmaul*, Untersuchungen über den constitut. Mercurialismus. Würzburg. 1861.—*C. L. Sigmund*, Die Einreibungskur mit grauer Salbe. Wien. 1866. 3 Aufl.—*O. Eckhard*, Zur Lehre von der Speichelsecretion. Henle u. Pfeufer's Zeitschr. 3. Reihe XXIX. 1. 1867.—*C. W. Knight*, Spontaneous ptyalism. Philadelphia Medical and Surgical Reporter. 1870. May 21.

By *salivation*, *ptyalism* (πτυαλίζω, to spit saliva), *sialorrhœa* (τὸ σίαλον, saliva), we understand such an increase of the secretion of saliva that the patient is no longer able to swallow it, but is compelled to spit it out frequently and in large quantities. The physiological limits of normal secretion of saliva are very

broad, according to Herman[1] from 1 lb. and 4 oz. to 5 lbs. and 4 oz. in the twenty-four hours, and therefore the limits between physiological and pathological secretion cannot be strictly drawn. Although salivation cannot be considered as an independent specific disease, but only as a symptom of various local and general disturbances, it will nevertheless be treated here in detail, on account of its practical diagnostic importance.

Etiology and Pathogenesis.

a. Excessive secretion of saliva is, above all else, an attendant symptom upon almost all diseases of the cavity of the mouth, and is even temporarily produced by irritant and piquant articles of diet. It sets in also in all wounds and surgical diseases of the mouth and the tongue, in caries of the teeth, in necrosis of the jaw, during the eruption of the milk-teeth, and in acute and chronic irritations of the parotid and of the submaxillary glands.

The eruption of small-pox in the mouth, sometimes the commencement of typhus also, give rise to a very severe salivation, and, finally, the same is true of both the external and internal use of mercury—which latter subject has been considered in detail in treating of mercurial stomatitis.

Preparations of iodine, gold, copper, and lead, and also some irritating vegetable remedies, as jalap, digitalis, and balsam of copaiba, produce ptyalism in some individuals.

The influence of the nervous system—and particularly of the trifacial, the facial, and the glossopharyngeal nerves—on the secretion of saliva, has been established by the well-known experiments of Ludwig. Irritation of these nerves increases the secretion of saliva, and this increase also appears in a remarkable manner when the irritation is applied to the central extremity of the divided gustatory and glossopharyngeal nerves. Here, evidently, a reflex action must take place, and, if so, then all the local irritants just cited must act through the termination of these nerves in a reflex manner.

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[1] Grundriss der Physiologie. 4 Aufl. 1872. p. 102.

Mercury appears, also, to excite an inflammation of the mucous membrane of the mouth, in the first instance, and salivation afterwards, in the manner above indicated; but here the possibility must not be excluded that the metal, by its passage through the salivary glands, may provoke a direct irritation upon their parenchyma. The susceptibility of the salivary secretion to mercury, and the results that follow it, are very diverse, and, according to Kussmaul, depend upon the following conditions:

The topical action of mercury upon the cavity of the mouth and the parotids produces a more regular and prompter flow of saliva than its contact with the skin causes. Anointing with the ointment of the gray oxide of mercury and the white precipitate ointment, washing with a solution of corrosive sublimate, even the incautious administration of calomel in powder, cause salivation in almost all cases.

The presence of teeth disposes to ptyalism. It is, therefore, very seldom encountered in toothless children, or, according to Ricord, even in toothless old persons. Caries of the teeth, development of the wisdom teeth, and diseased gums especially, favor the onset of salivation.

Catching cold during a mercurial treatment hastens the outbreak of the salivary flow; so also pregnancy and constipation. Diarrhœa, on the contrary, retards it. Persons who have already been strongly salivated, are very readily salivated again by much smaller doses of mercury. Finally, it is still to be mentioned, that long-continued taking into the body of small doses of mercury, as occurs in various trades, seldom produces severe stomatitis and salivation, but much more surely the constitutional effects of the metal, and that, on the other hand, the administration during a short time of large doses of our ordinary medicinal mercurial remedies produces the opposite state of affairs.

b. In many cases salivation occurs in mouths entirely sound, and is produced by diseases of the stomach, bowels, or the female genitalia. The paths which convey the impression, in these instances, are the pneumogastric and sympathetic nerves. Various irritations of the mucous membrane of the stomach

increase the secretion of saliva at once, as was first demonstrated by Frerichs, in dogs with gastric fistulas. Salivation is often induced by indigestion, acute and chronic gastric catarrh, distention of the stomach with gas more particularly, penetrating ulcer and cancer of the stomach, and, finally, also from the irritation of worms. The morning water-brash of gastric patients is caused only by the large amount of saliva swallowed during the night, as is clearly demonstrable by chemical examination of the vomit. Here, also, belongs the salivation in diseases of the pancreas and the spleen, in intermittents, during pregnancy, in disturbances of menstruation, in the climacteric years, and in many hysterical conditions.

c. Finally, psychical influences alone may produce an increase of saliva, though only of a temporary nature. It is a proverbial saying, that the sight of favorite food, nay, even the mere speaking of it, causes the mouth to water; so also does the impression of disgust, the inspection of a foul corpse, the minute examination of fæces, vomit, or bronchiectatic sputa; while among all nations the sign by which disgust is exhibited is spitting. This last category of psychical influences never causes a long-continued ptyalism, and therefore does not belong particularly to the subject, being merely introduced to show in what manifold manners innervation works upon the salivary secretion.

Symptoms.

As all the inflammations of the cavity of the mouth have already been treated of—among them mercurial stomatitis—we need only mention here the symptoms produced by the salivary flow itself. This is never painful, though extremely annoying. The continuous collection of saliva impedes distinct speech; the patients complain of a bad, stale, often metallic taste in the mouth, and are no longer able to retain the saliva in the mouth nor to collect it in mass. It flows continually from them, and they are, therefore, compelled to have a spit-cup or a cloth in hand the entire day. Rest at night is naturally very much interfered with when the pillow becomes saturated in a short time.

There are cases reported in which five quarts of saliva have been collected within twenty-four hours. From one to two quarts is the daily average in mercurial stomatitis.

The chemical alteration of the saliva produced during salivation has been examined by many chemists. Gmelin, Thomson, Simon, Ure, Bird, Garrod, Lehmann, and Wright have worked at this subject. At the commencement of the flow, the specific gravity is usually very much increased, being as high as 1059, while it hardly reaches 1010 in normal saliva. This increase depends upon a greater admixture of mucus—the sympathetic saliva of the physiologists,—some albumen, and an increase in the salts. In the course of the salivation, however, the specific gravity falls almost down to that of distilled water. Wright found it 1001.5 in one instance. With the subsidence of salivation, the saliva usually acquires its normal characteristics again; but exceptionally it remains thin and clear for a long time (trigeminus saliva of the physiologists). Its reaction is either normal, slightly alkaline, or it is neutral or even slightly acid, due to increase of the readily souring mucus. According to Wright, a strong alkalescence may be produced by treating the ptyalin with ammonia. The amount of sulpho-cyanide of potassium present is much diminished, and it is hardly possible to produce a red color with chloride of iron, even by evaporating the fluid to one-fourth or one-sixth of its volume. Wright alone has found an increase of this salt in the majority of his cases. In mercurial salivation, traces of mercury may regularly be detected, even a long time after the discontinuance of the remedy. According to Bamberger, this succeeds best by the galvanic test. The saliva to be examined is slightly acidulated with hydrochloric acid, and then connected with a simple copper and zinc element, in such a manner that the metallic connecting-wires dip into the saliva. At the free immersed end of the zinc-pole a small bright leaflet of copper is fastened, upon which, after twenty-four hours, the mercury is deposited as a whitish layer, which disappears on being heated over a spirit lamp. It is of special importance, finally, according to Wright's observations, that the catalytic power of mercurial saliva is much enfeebled or entirely lost.

It is self-evident that with so great a loss of water the remaining secretions and excretions must be somewhat impaired. The amount of urine is always decreased, and constipation sets in. In many cases the rapidly progressive emaciation is conspicuous, and it is not very readily explicable, in view of the slight specific gravity of the saliva, and the extremely small loss of nitrogen. It is due much more to the long continuing loss of appetite, produced by the continuous dilution of the gastric juice with saliva, by the distention of the stomach with gas, and by the above-mentioned much diminished action of the saliva of salivation upon the starches.

Course.—Terminations.—Prognosis.

Mercurial salivation continues for a period of from one to three weeks, and is essentially dependent upon the cure of the ulcers in the mouth. It soon lessens upon the subsidence of the swelling of the mucous membrane, and disappears, leaving no traces behind, even without treatment. The salivation produced by remote causes is influenced in like manner, principally in accordance with the curability of the fundamental disease. In chronic diseases of the stomach a high degree of salivary secretion is rare; but usually there is frequent spitting, and water-brash may be produced by the saliva that is swallowed. In chronic gastric catarrh, and in round ulcers of the stomach, these symptoms may disappear after having persisted for years.

Salivation in uterine, hysterical, and pregnant patients is temporary for the most part, though it continues sometimes during an entire pregnancy, and sometimes assumes an intermittent character; examples of which are recounted by Rayer and Wright. Spontaneous ptyalism, of which Tanquerel des Planches has collected twenty-nine cases, is for the most part chronic, and continues for months and even years. In eight cases recovery followed spontaneously, twelve were cured by various remedies, and nine remained uncured.

The prognosis, as far as life is concerned, is always favorable; for, although patients with long-continued salivation now and then die, there is always found on dissection some other cause of

death in the stomach, intestines, or female genitals. With reference to the duration of the affection, no definite termination can be predicted, except in the case of mercurial salivation, inasmuch as the irradiated ptyalism may continue days, weeks, or years. The emaciation and anæmia hereby produced soon cease if the fundamental cause can be removed.

Treatment.

Prophylactic treatment can be spoken of only with reference to mercurial salivation, and the salivation produced, in rare instances, by the use of iodine. The remaining forms, which are complicated with diseases of the stomach, intestines, and uterus, fall into the same category with the very incomplete prophylaxis of these processes. The avoidance of mercurial ptyalism is best accomplished by the sparing administration of mercury. Calomel should be entirely done away with as a simple laxative; and if its use cannot, in some cases, be avoided, it should at least be administered in an appropriate envelope, either in the form of a pill or contained in a well-closed wafer.

The treatment of constitutional syphilis with mercury, and the after-results that ensue from it, have given rise to a great deal of writing and of controversy. This question will not here be further ventilated, but only the precautions will be given under which an inunction-cure of from three to four weeks' duration may be effected without injury, according to my own experience, verified in a hundred instances. These consist simply in the maintenance of an equable temperature, in the bed-chamber, of not less than 66° F., in good ventilation, open bowels, careful cleansing of the teeth, and the daily use of one drachm of chlorate of potassa dissolved in from six to nine ounces of water. With these precautionary measures, the treatment hardly produces even slight fœtor from the mouth, and almost never results in profuse secretion of saliva.

If the salivary flow is once established, it is not possible to stop it suddenly. Mercurial ptyalism ceases in from one to three weeks; the other forms pursue very various courses. Hysterical ptyalism, as well as that of pregnancy, may continue a few hours

and not return at all, and, on the other hand, may recur frequently, or may last for several months without being controllable by any remedy. If salivation has occurred through the influence of mercury, the skin should be carefully cleansed at once with soap, in a warm bath; and, where possible, the patient should be taken into another room, in order that no more vapor of mercury may come in contact with him. The administration of mercurial preparations should be suspended, as a matter of course. If mercurial stomatitis exists, the treatment recommended on p. 790 will be applicable. Chlorate of potassa is powerless against the flow of saliva itself. Some effect is observed from mild laxatives, tamarinds, or, at most senna, which here excite an antagonistic intestinal catarrh. The internal use of iodide of potassium is recommended by many authors, acetate of lead by others, and opium by most of them. As we have learned, through Ludwig's exact experiments, the influence of the nerves upon the salivary secretion, causing its increase on irritation of the nerves, we shall combine theory and practice in the happiest manner in these cases, if we administer full doses of opium.

As a troublesome dryness, and, finally, a complete suspension of the saliva, belong prominently to the earliest symptoms of atropine-poisoning, the use of this remedy has been quite rationally tried. It does not refuse its service here, but the additional effects of atropine, dilatation of the pupils, rapidly appearing delirium, and so on, are so disagreeable, that the remedy will never find an extensive use.

The topical treatment of the cavity of the mouth with gargles has no distinct influence upon the course of the salivation, but is agreeable to most patients, by allaying the mawkish taste. Astringents are used here for the most part—decoctions and infusions of sage, oak-bark, cinchona-bark, krameria, tormentilla, etc., or solutions of alum, tannin, acetate of lead, sulphate of zinc, and carbolic acid, to which the experienced practitioner always adds some tincture of opium. Knight cured a severe spontaneous ptyalism with gargles of table-salt.

The diet, as long as stomatitis is present, naturally consists only of fluids, or at most of broths; but, inasmuch as the patients

lose strength rapidly, it should at all times be the most nutritious possible, consisting of such food as milk, eggs, finely-chopped meat, beer, and wine.

E.—Deep Inflammation of the Connective Tissue of the Throat.

Angina Ludovici. (Ludwig's Angina.)

Craigie et Aquié, Journ. de Montpellier. Vol. II. Livr. 10.—*Ludwig*, *Theurer*, *Bosch*, *Heim*, *Höring*, *Schmetzer*, *Leube*, collectively in Schmidt's Jahrbcher. Bd. XV. pp. 27–29.—*Heyfelder*, Studien im Geb. der Heilwissenschaft. Stuttgart. 1838. Bd. I. p. 236.—*Camerer*, Würt. Correspbl. Bd. VII. No. 10.—*Rösch*, Würt. Correspbl. VIII. Nos. 41 u. 42.—*Martini*, Rust's Magaz. Bd. 27. p. 431.—*Bermann*, Casper's Wochenschr. 1840. No. 46. u. 1841. p. 76.—*Canstatt*, Schmidt's Encyclop. Bd. V. p. 323.—*Tempe*, Casper's Wochenschr. 1841. No. 18.—*Zellner*, Zellgewebebrand am Halse. Oesterr. med. Wochenschr. 1845.—*Velpeau*, Gaz. des Hôp. 1849. No. 12.—*Stanelli*, Deutsche Klinik. 1850. No. 5.—*Sprengler*, Deutsche Klinik. 1851. No. 4. etc.—*Bamberger*, Virchow's Hdbch. Bd. VI. Abth. 1. p. 59.—*Emmert*, Lehrb. der spec. Chirurgie. 2. Aufl. Bd. I. p. 834.—*Wernher*, Hdbch. d. Chirurgie. 2. Aufl. Bd. I. p. 636.—*Binswanger*, Arch. f. physiol. Heilkde. VII. p. 578.—*Förster*, Hdbch. d. spec. path. Anat. 1863. 2. Aufl. p. 50.—*Grohe*, Berlin. klin. Wochenschr. 1869. No. 31.—*Niemeyer*, Lehrbuch d. spec. Pathol. 7. Aufl. Bd. I. p. 519.

By *angina Ludovici* (Ludwig's angina), *cynanche cellularis maligna* (κυνάγχη, dog-choke, dog-collar), *pseudoerysipelas subtendinosum colli*, we understand, as the above names indicate, a very acute inflammation and suppuration of the cellular tissue beneath the chin, in the environs of the submaxillary glands, which has been named after its earliest describer "Ludwig," and which has appeared epidemically at various times.

Etiology.

Canstatt considers it a metastatic deposit following anomalous typhus, or acute exanthems; and in such cases the process would be considered analogous to that of metastatic parotitis, the submaxillary glands being the seat of suppuration, instead of the parotid. In many reports of the disease, however, there is no mention of previous acute infectious diseases, and the malady is

described as an idiopathic, primary affection, which by no means always originates in the submaxillary glands. The cause adduced by Niemeyer, viz., periostitis of the lower jaw, has naturally an entirely different significance, and is never accompanied by such violent symptoms as angina Ludovici.

Emmert attributes it to the following causes: topical irritations, catching cold, especially during times of prevalent rheumatism and erysipelas; and exanthematic, typhous, and puerperal conditions. Zellner, finally, saw it appear, in Steiermark, simultaneously with scorbutus and typhus. In recent times the disease has almost disappeared, and I can find only a few short accounts of it in the various journals of the last few years. During the third decade of the present century, however, it seems to have made much sensation, especially in Württemberg, of which the numerous, simultaneous reports of the physicians of Württemberg furnish distinct evidence. Isolated cases, moreover, are also found in the older literature, collected by Craigie and Alquié, and Bamberger even cites Hippocrates as the earliest author on this subject.

Pathological Anatomy.

The inflammation generally begins in the supra-hyoid region, and usually on one or the other side of this region; rarely in the middle, over the hyoid bone. The extensive exudation thus set up is sero-plastic, and has a great disposition to undergo purulent, and, still more so, to undergo ichorous degeneration. In cases terminating fatally, the connective tissue and the muscles of the entire sub-mental region are found, according to Bamberger, transformed into a semi-fluid, brownish mass, mixed with necrotic shreds of connective tissue; the submaxillary glands and the parotid are also destroyed by gangrene, and the neighboring connective tissue is densely infiltrated, for a distance extending even to the larynx and the pharynx. Sometimes even the periosteum of the lower jaw is loosened.

In addition to this, we usually find considerable hypostatic trouble in the lung, and signs of embolism or septicæmia in various organs.

Symptoms.

The above-described swelling of the hyoid region sets in with lighter or severer, or even with complete typhous symptoms. The tumor is moderately painful, becomes harder and harder, extends in various directions within a few days, and finally takes in the entire anterior half of the throat as far as the sternum. A great deformity is thus produced, the chin and sternum being on the same level. The skin of the throat can no longer be moved nor raised, but is tightly stretched and reddened. The lower jaw naturally becomes immovable, and deglutition is impossible. With compression and œdema of the larynx and trachea, great dyspnœa, even orthopnœa, sets in, and this soon influences the circulation, so that the patient, whose breathing is loud and rattling, finally becomes cyanotic, with a small, sinking pulse. The floor of the mouth is elevated by the tumor, in such a manner that the tongue is pressed against the hard palate and rendered immovable, in consequence of which speech is also very much interfered with. The mucous membrane of the cavity of the mouth, as far as it can be seen, is swollen and reddened.

Finally, hyperæmia of the brain results from compression of the internal jugular vein, giving occasion to headache, vertigo, obnubilation, and delirium. In the metastatic form such cerebral symptoms may be explained by the infection alone, without any local cause, as in these cases, especially, the disease takes on a typhous character.

Course and Terminations.

The disease always maintains an acute course. After a week a standstill occurs, after which, in the idiopathic variety especially, the tumor sometimes entirely subsides, without passing into suppuration. This resorption never takes place rapidly, but leaves a hard swelling after it, that persists for weeks. In most cases suppuration sets in, the exudation giving way at several points. The skin covering it becomes raised and reddened in places, and, after incision, a brown, shreddy pus escapes, or, in severe cases, a gangrenous ichor. Some collections of pus

rupture towards the cavity of the mouth or the pharynx, and, with the breaking up of the exudation, the enclosed connective tissue and the adjacent skin also become gangrenous. According to Wernher, the disease terminates fatally in almost half of the cases, especially when attacking typhus convalescents, and death may occur either after a few days or not until after from three to four weeks. The less acute the course of the affection, the more favorable the prognosis. Death takes place through exhaustion, suffocation, or septicæmia. Bad after-effects and subsequent exacerbations have been observed as a result of the abscess.

The ordinary termination is purulent, gangrenous destruction, on which account the designation, *cynanche gangrœnosa*, appears the most appropriate. Further injurious results are fistulous ulcerations, strongly contracting cicatrices, which produce torticollis and impeded mobility of the neck, and, finally, caries and necrosis of the jaw-bones.

Treatment.

It is claimed that in some cases suppuration may be prevented and prompt resolution be effected by powerful antiphlogistic treatment, consisting in local and general venesection, and also by the administration of emetics and drastics. In individuals of debilitated constitution, however, such weakening treatment cannot be thought of, and here the attempt is made to favor softening by dry or moist warmth. In all cases, but at any rate in cases associated with danger of suffocation, early and deep incisions are requisite, with the insertion of blunt hooks and thorough separation of the fascia, by means of which, as a rule, a deeply-seated collection of pus is reached. By the discharge of this, as well as by the copious flow of blood and of serous exudation, the alarming symptoms are often overcome in a short time. The after-treatment is regulated in accordance with ordinary surgical rules.

If the dyspnœa continues after the discharge of the abscess, its cause must be sought for in an œdematous swelling of the larynx ; in which case death by suffocation can only be avoided by the prompt performance of tracheotomy.

DISEASES

OF THE

SOFT PALATE.

WAGNER.

THE SOFT PALATE.

THE diseases of the soft palate are usually treated of simultaneously with those of the cavities of the mouth and pharynx. Their separate consideration, as undertaken here (chiefly for outside reasons), is nevertheless fully justifiable. A great number of affections of the palate either occur alone, or else the participation of the palate is of special importance. In the following exposition, these diseases are especially considered; the others are treated of more briefly.

The diseases of the soft palate are of importance:

1st. Locally, by impeding deglutition, respiration, articulation, etc.

2d. By their extension into the mouth, throat, nose, ear, larynx, etc.

3d. By the simultaneous existence of general diseases (acute exanthemata, diphtheritis, syphilis, etc.), or of nervous affections, etc.

Introductory Anatomical Remarks.

Concerning the gross anatomical relations, consult the well-known manuals of anatomy; also, *Tourtual*, Neue Unters. üb. d. Bau des menschl. Schlundkopfes. 1846.—*C. L. Merkel*, Die Funct. d. menschl. Schlund- u. Kehlk. 1862.—*Luschka*, Der Schlundk. des Menschen. 1868.—Concerning the histological relations, consult the manuals of histology; in addition, the monographs of *von Szontágh*, *Klein*, *Huxley*, *Sappey*, *His*, *Henle*.—Concerning the tonsils, consult especially *Billroth* (Beitr. z. path. Histol. 1858), and *Th. Fr. Schmidt* (Z. f. wiss. Zool. 1863. XIII. p. 221).

The following account of the anatomy and histology of the soft palate will be justified by every one who is conscious of the gaps in his own anatomical knowledge. Without this minute knowledge many diseases are incomprehensible.

We distinguish, in the first place, an anterior and a posterior surface of the soft palate; then a central portion, including the uvula; then the two anterior and two posterior palatine arches; and finally the tonsils. The soft palate merges into the hard palate without any distinct line of demarcation. In different places, and in different individuals, it is of varying thickness (from three-tenths to five-tenths of an inch), and of varying length. The free space lying between the arches of the palate and the root of the tongue is the so-called pharyngeal opening, or pharyngeal entrance, *isthmus seu vestibulum pharyngis*, *interstitium arcuarium*. The entrance of the pharynx is of variable size—partly due to individual peculiarity, partly to the size of the surrounding parts.

The anterior surface of the soft palate is, normally, moderately and uniformly reddened, generally paler than the anterior surface of the uvula and the anterior palatine arches. It is but slightly movable and wrinkleable. It exhibits, especially in its central portions, the mouths of numerous mucous glands, at a uniform distance (about $\frac{1}{2\frac{1}{3}}$ of an inch) from each other.

Microscopically examined, the anterior surface of the soft palate consists most superficially of a many-layered (30–50) stratum of pavement epithelium about $\frac{1}{5\,0}$ of an inch in thickness, which rests upon long papillæ containing loops of capillaries. At the outer surface lie from five to ten layers of flattened cells, which, like the epithelium of the mouth, are continually thrown off; the remaining epithelium is likewise similar to that of the cavity of the mouth. The moderately vascular stroma, formed of connective tissue, shows no sharp division between *mucosa* and *submucosa*. It consists of a thin tense uppermost layer; then follows a thicker, looser stratum as submucosa; still deeper there lies a thick tense stratum of connective tissue (continuation of the aponeurosis of the hard palate), which, towards its inferior portion, encloses numerous, mostly flattened, little leaflets of fat. The numerous mucous glands which form a stratum as thick as $\frac{3}{1\,0}$ of an inch above the tonsil, decreasing in numbers towards the uvula, have the usual characteristics of such glands. Solitary follicles are altogether wanting on the anterior surface or are very incon-

stant. Below all lies the striated muscular structure, which sends isolated bundles between the mucous glands towards the above-mentioned third fibrous layer.

The *uvula* is from $\frac{4}{10}$ to $\frac{6}{10}$ of an inch in length. The greater portion of its substance consists of mucous glands, which are larger and more numerous in the anterior half than in the posterior half. Between these lie some twigs of connective tissue, and especially some twigs of muscular tissue, the latter forming plexuses not only between the glands *in toto*, but also between the lobes of the glands. The submucosa is more extensive anteriorly than at the sides, and here again, more extensive than at the posterior surface. Follicles exist, but are not constant. The epithelium is like that of the soft palate, but is less thick and thinnest on the posterior surface. In the latter position, it is, during the first months of existence, ciliated almost to the very tip.

The lateral portions of the soft palate pass, on either side, into the palatine arches: the anterior, *arcus glosso-palatini*, which bound the *isthmus pharyngo-oralis*, and the posterior, *arcus pharyngo-palatini*, which bound the *isthmus pharyngo-nasalis*.

The *anterior palatine arches* are shorter, lie about $\frac{4}{10}$ of an inch higher, and reach further outwards than the posterior arches. Towards the middle, each anterior palatine arch terminates flatly upon the base of the uvula, and laterally it passes over to the border of the tongue. The anterior palatine arches resemble the soft palate, being richly supplied with mucous glands. The solitary follicles are inconstant, but are present in most instances, especially at the lower end.

The *posterior palatine arches*, towards the median line, pass into the lateral borders of the uvula, and externally they pass into the lateral walls of the pharynx. At their inferior portion is found, upon either side, the *plica or arcus pharyngo-epiglotticus*. Solitary follicles are frequently found on the posterior surface, seldom on the anterior; on the latter portions they are sometimes gathered in groups, several lying close to each other, like an additional tonsil, which may lie above, but is more likely to lie below the tonsil.

The relations of breadth between the anterior and posterior palatine arches vary; the smaller the former, the broader the latter appear.

The *tonsils* (tonsillæ palatinæ) lie between the two palatine arches, and generally project distinctly beyond them, though to a variable degree. Their form is that of an oval disc, sometimes of a flattened globe. Their size varies so much that no positive volume can be determined on. Their length amounts to from half an inch to an inch; their transverse diameter, in those more round than oval, amounts to almost as much; their thickness varies from $\frac{4}{10}$ to $\frac{6}{10}$ of an inch.

The free superior surface of the tonsils is moderately red, and presents variously numerous round or linear openings (from ten to sixteen in number). These are mostly so narrow that they are just visible; less frequently they are wider and more like fissures. They lead into longer or shorter fissures (lacunæ, sinuses), running perpendicularly or obliquely in various directions, and sometimes giving off branches. In their normal condition these sinuses do not stand open, but they gap distinctly on being cut across. They are surrounded by a grayish-red, homogeneous mass, which, in thin sections, presents little follicle-like divisions, for the most part visible even to the unaided eye. Externally from these, therefore between them and the follicular coverings of the adjacent lacunæ, there lies a narrow strip of connective tissue.

The lacunæ are lined with a thin but uniform layer of epithelium, similar to that covering the external surface. The epithelium sits on shorter papillæ (also containing capillary loops), which are extensions of the thin capsule of the surface penetrating into the lacunæ. The tissue surrounding the lacunæ consists of follicles and interfollicular tissue. The follicles are composed of reticular tissue, with few capillaries, and of small round cells imbedded in it. The reticulum itself, the size of its spaces, the number, size, etc., of the cells contained in it, are subject to manifold physiological variations. The follicles are not capsular, but yet they are sharply defined. The inter-follicular tissue has an essentially similar character, only its reticulum is more scanty, thicker, poorer in cells, and

not only richer in capillaries, but it also contains arteries of the smallest calibre, and tolerably large veins, which surround the periphery of the follicle. The larger vessels lie in the firm, fibrous connective tissue, which forms a framework through the entire tonsil, continuous on the one side with the thick capsule on the attached portions of the tonsil, and on the other with the scanty connective tissue under its free upper surface. It is poor in capillaries, contains hardly any vessels except those for the substance of the follicles and the papillæ of the lacunæ.

The human tonsil does not contain any mucous glands. Only exceptionally one of those lying outside of the tonsil opens into the depth of a sinus.

The free surface of the tonsil exhibits as numerous differences as its size and form do in healthy persons, and in those who cannot remember ever to have had any disease in them; so that it is more rare to see a normal tonsil than to see manifold deviations from the normal condition.

Only the tonsils of new-born babes and of small children are, for the most part, normal. They are small relatively to the size of the body, and composed of three or four lobes, between which lie the shallow lacunæ. Cytogenetic tissue is found about the latter, indistinct in new-born babes, distinct in older children, but not yet arranged into follicles in the first period of life. The lacunæ gradually become deeper, and new ones appear; the latter by a characteristic chamber-formation of the epithelium. The capsule of the tonsil also first becomes more distinct after birth. The same is true of the connective tissue forming the partition walls of the lacunæ.

The alterations which the tonsils undergo, besides their normal growth, are still but little known. It is probable that the follicles continually perish, and that new ones appear. Equally probable is the formation of new, and probably always shorter and narrower, permanent lacunæ.

The tonsil, where its surface is not free, is smooth, moderately arched, and surrounded in front and behind with loose connective tissue. On the outer side there is a layer of pretty tense connective tissue. The former is bordered by bundles of the palato-pharyngeus and palato-glossus muscles. Into the latter are inserted bundles of the superior constrictor muscle of the pharynx, which draw the tonsil inwards, as well as

bundles of the stylopharyngeus muscle, which draw it outwards. Still further externally there lies the bucco-pharyngeal fascia with adipose tissue; and on this borders the internal pterygoid muscle.

The space in which the tonsil lies is the anterior portion of the so-called interstitium pharyngo-maxillare. In its posterior portion lie the external and internal carotids, the first removed six-tenths, the latter eight-tenths of an inch from the outer surface of the tonsil. Still further externally, we find the internal jugular and posterior facial veins. The glosso-pharyngeal nerve lies much nearer the tonsil. At the anterior portion of the external surface of the tonsil is found the pterygo-mandibular or pterygo-maxillary ligament.

The posterior surface of the soft palate presents several differences from the anterior surface. Its mucous membrane is thinner, and of a pale red. The epithelium is squamous in the grown subject, ciliated in the fœtus and in the new-born babe. The mucous membrane contains scanty and small papillæ, scanty mucous glands (above the uvula they are altogether wanting); and the solitary follicles are very variable in their number. The remaining layers are similar to those on the anterior surface, but are thinner, so that the muscular substance lies nearer the surface.

The *muscles* of the soft palate and of the palatine arches are partially recognizable by their action in an examination of the palate. They almost all exist in pairs, and always act simultaneously under normal conditions.

The *levator veli palatini muscle* (*musculus petro-salpingo-staphylinus*) raises the soft palate and narrows the Eustachian tube.

The *tensor veli palatini* muscle (*musculus spheno-salpingo-staphylinus; musc. circumflexus palati mollis*) stretches the so-called aponeurosis of the palate, that is to say, the fibrous membrane springing from the posterior border of the hard palate and losing itself in the soft palate. It further effects a dilatation of the Eustachian tube in each act of deglutition, and so ventilates the middle ear.

The *levator uvulæ*, or *azygos uvulæ* muscle shortens the

uvula vertically, so that its mucous membrane becomes laid in transverse folds ; at times it also bends it backwards.

The *palato-glossus* muscles, lying in the anterior palatine arches, can close the *isthmus pharyngo-oralis* by approaching the middle line, and by elevating the back of the tongue. In this action, for the most part, the *stylo-glossi* and *palato-glossi* muscles participate.

The *palato-pharyngeus* muscles lying in the posterior palatine arches transform the *isthmus pharyngo-nasalis* into a narrow elongated fissure running from above and before, downwards and backwards. If the levator muscles act at the same time, the soft palate becomes horizontal.

The blood-vessels of the soft palate are closely connected with those of the posterior oral and nasal cavities, and of the anterior portion of the pharynx.

The arteries are given off from the external carotid, and especially from the facial and internal maxillary arteries. From the internal maxillary arises the posterior or descending palatine artery (or pterygo-palatine artery), for the soft palate (—hard palate, etc.). From the facial artery (or from the descending pharyngeal artery, sometimes from the occipital artery—or as independent trunks) arise : the inferior or ascending palatine (or pharyngo-palatine) artery, for the mucous membrane, muscles and glands of the soft palate (—the mouth of the Eustachian tube and its surroundings) ; the tonsillar artery, for the tonsils (—lateral wall of the pharynx and root of the tongue).

The not unfrequent variations of the arteries are almost exclusively of surgical interest.

The veins form two plexuses : the posterior venous plexus, which is continuous with the veins of the nasal mucous membrane, and empties into the plexus of the temporal fossa ; the anterior venous plexus, which is connected with the root of the tongue, and empties through the pharyngeal vein into the internal jugular.

The lymphatic vessels of the palate, including those of the tonsils, are very numerous. The finest lie as a net-work in the interfollicular tissue of the tonsils. Larger but still microscopic

vessels are found in the entire mucous membrane of the palate, especially that of the uvula; they lie partly in the neighborhood of the capillaries, and in part they form special strands, parallel to the surface, coursing near the blood-vessels. Among the larger trunks we can discover an anterior and a posterior lymphatic plexus. The latter is in connection with the lymphatic vessels of the floor of the nasal cavity. The former, much more strongly developed, forms two groups; the anterior group follows the anterior palatine arches, and is in connection with the absorbents of the root of the tongue; the exterior group descends from the tonsils outwards, and takes up these absorbents.

The lymphatic glands belonging to them lie at the angle of the jaw, at the bifurcation of the carotid, beneath and near the upper portion of the sterno-cleido-mastoid muscle, and at the side of the hyoid bone and the larynx. The lymphatic glands lying at the angle of the jaw, from two to four in number, receive their lymphatic vessels from the tonsil, and participate so constantly in certain affections of the tonsils, that they are sometimes described as the tonsillar glands.

The nerves of the soft palate, as regards their distribution, are not yet sufficiently well understood. The physiological views concerning them present as numerous contradictions as the conclusions drawn from some pathological conditions.

With reference to the *motor nerves*, the motor portion of the third branch of the trifacial, especially the internal pterygoid branch, supplies the tensor palati muscle; according to others, it is the lesser superficial petrosal nerve (branch from the otic ganglion). The remaining palatine muscles are supplied by the facial nerve: the levator palati and azygos uvulæ by the superior palatine branch, the so-called larger superficial petrosal nerve, and the palato-pharyngeus and palato-glossus muscles by the inferior palatine branch.

The sensory nerves preside over the sense of touch, taste, pain, reflex muscular contraction, and secretion. The anterior face of the soft palate, down to the borders of the anterior palatine arches, is supplied from the trifacial, and especially from the descending or palatine branches of the spheno-palatine ganglion belonging to the second branch of the fifth nerve. The

opposing sides of the palatine arches, the lateral wall of the entrance into the pharynx, with the tonsils and the posterior side of the soft palate, are supplied by branches from the glosso-pharyngeus, pneumogastric, and spinal accessory nerves. The glosso-pharyngeus, that is, the tonsillar nerve of the lingual branch, is the nerve of taste for the lateral portions of the soft palate and the anterior palatine arches. According to others, it also permits the sense of touch, as well as reflex movements (gagging, etc.), and excites the secretion of the parotid gland. The pneumogastric and accessory nerves send sensory and motor filaments especially to the pharynx.

The secretory nerves lie in the chorda tympani. Their excitation produces a secretion of the submaxillary and sub-lingual glands, and the mucous glands of the mouth and the palate.

The centre of the co-ordinate movements of deglutition probably resides in the medulla oblongata.

A number of individual variations occur in the soft palate, which are congenital, but do not constitute deformities exactly, nor yet diseases. They have received but little attention as yet, although they have a practical significance under some circumstances. Among those worth mentioning are :

Great breadth of the entrance into the pharynx, with normal volume of the surrounding soft parts ;

Great narrowness of the entrance into the pharynx, under similar conditions ;

Higher or lower insertion of all four palatine arches, or only of those of one side, or only of the anterior or the posterior, or only of one of these ;

Varying size of the palatine arches, the uvula and the tonsils, with normal structure of these parts ;

Variations of height in the position of the tonsils ;

Marked inclination of the tonsils towards the middle line ;

Variations in the size and form of the tonsils, in the number of their lacunæ, and so on.

The physiological irritability of the soft palate varies very much in different persons ; in many, all contact with the parts at once excites powerful retching movements ; while others bear

this, and even greater irritation, much better. The reflex secretion of mucus and saliva is variable in like manner.

General Symptomatology.

The estimation of the diseases of the soft palate is formed from inspection, in comparatively rare cases also by palpation of the palate itself and of the parts connected with it (bones, blood-vessels, lymphatic glands, etc.), as well as from the impediment to function.

A. *Inspection* direct, or by means of a mirror for the posterior surface of the soft palate, readily permits us to recognize most of the diseases, on attentive consideration—many of them better than on the dissecting-table. With the mouth widely open, the anterior surface of the soft palate and uvula, together with the tonsils, is readily inspected, either without any assistance, especially in persons accustomed to it, vocalists and the like, or with the assistance of a tongue depressor. The use of the depressor, for which the handle of a spoon, a paper-cutter, the finger of the physician, etc., can be substituted, is so managed in adults, whether standing, sitting, or lying down, that it is not placed too far back upon the tongue, and the tongue should be gradually depressed only as far as is necessary. The gradual depression of the tongue, and the avoidance of placing the depressor too far back, are important, because, in so doing, retching occurs much less frequently. It is advisable, at the same time, for many reasons, to cause the patient to inspire and expire deeply, or to make him say, "ah," or "eh." In little children, who resist the ordinary examination, the procedure is more difficult. In most instances two assistants are requisite—one to fix the child's head, the other to hold the arms. If the mouth is not opened voluntarily, the first assistant holds the child's nose at the same time, and the examiner places the depressor rather deeply in the mouth. If milk or some more solid food has been taken just before, of which light particles remain hanging on the palate, some water should be drunk, or the mouth rinsed out before the inspection is made.

Consult the article on Rhinoscopy, concerning the inspection of the posterior surface of the soft palate.

The inspection of the palate furnishes us, without any further research, with conclusions concerning the size and form of the parts, the various disturbances of circulation, the presence of inflammation, gangrene, morbid growths, and the mobility of the parts. The diagnosis of the diseases of the soft palate is especially pathologico-anatomical, and moderately easy, if the examiner is familiar with the appearances of the different morbid changes and possesses the necessary technical skill. For beginners, and under certain circumstances for practical men also, the recognition of the so-called deposits presents the greatest difficulty (by deposits we have reference to those membranous spots, circumscribed or occupying the entire surface of the palate, at first whitish, and later grayer, which are elevated above the free mucous membrane adjoining them). A similar coloration is shown by exfoliating epithelium, epithelial suppuration, epithelial thickening and infiltrations, especially of syphilitic and tuberculous nature, which render the mucous membrane anæmic *in toto* or in its uppermost layers. The projecting plugs of epithelium and pus from the lacunæ of the tonsils, and the spots of mucous membrane affected by thrush, are both alike whitish in color and elevated.

B. *Palpation* is employed to form conclusions as to the consistency of individual portions of the soft palate in inflamed or hypertrophic conditions, or as to the size, consistence, and so on, of morbid growths.

Inspection, and especially palpation, are much more frequently employed in estimating the size and other peculiarities of the lymphatic glands connected with the palate, especially those at the angle of the jaw. Slight enlargements of these glands are without diagnostic interest, especially because, for the most part, it cannot be determined whether the enlargement had not previously existed. Extensive enlargement leads to the conclusion that not only the surface of the soft palate, but its parenchyma also, especially that of the tonsils, is quite actively diseased—sometimes in the form of inflammation and gangrene,

sometimes in that of morbid growths (syphilis, tuberculosis, cancer).

The frequent assertion of authors that the swollen tonsil can be felt from the exterior at the angle of the lower jaw, is incorrect for the ordinary forms of inflammatory swelling, and for hypertrophy itself. Minute examination upon the living, section of the head through the tonsil from before backwards (Braune, Luschka: illustrations—original sections), the free exposure of the normal and swollen tonsil in the corpse, have convinced me of this fact. What is really felt in the cases mentioned is either infiltrated connective tissue of the pharyngo-maxillary space and the infra-maxillary region, or swollen lymphatic glands, or both of these conditions together.

C. *The local symptoms* of diseases of the palate, besides those already mentioned, are disturbances of motion and sensation.

1. *Motor Disturbances.*

Consult *A. Brücke*, Nachschrift zu *Kudelka's* Abhandlung. u. s. w. nebst einigen Beobachtungen über die Sprache bei Mangel des Gaumensegels. 1858.—*Schuh*, Wien. med. Wochenschr. 1858. No. 3.—*H. Smith*, Dubl. Jour. 1862. XXXIV. p. 19. On certain movements of the throat and chest in the acts of respiration (singing, speaking, yawning, snoring).—*Passavant*, Ueber die Verschliessung des Schlundes beim Sprechen. 1860.

In looking at the soft palate, either no movement is seen, or there is a simple contraction, an elevation of the palate, especially a shortening of the uvula or an alternate contraction and relaxation. The last regularly occurs when the patient says "ah" or "eh." In diseased conditions these movements are either entirely absent, or they are labored or incomplete, or they are possible on one side only.

The most important motor disturbances are the following:

a. *Difficulty in swallowing* in the most various grades up to complete impossibility, either on account of the great pain (see below), or on account of impeded muscular action.

The movements of deglutition, which here come under consideration, comprise merely the first two acts of the process: namely, 1st, the conveyance of the bolus behind the anterior arches of the palate, principally by the action of the palato-glossus mus-

cles; 2d, the onward movement into the middle of the pharynx, implying the elevation of the root of the tongue, the action first of the palato-glossus muscles and then of the palato-pharyngeus muscles, and the closure of the upper pharynx and the posterior nares—depending on the action of the levator and tensor palati muscles, the superior constrictor muscle of the pharynx as well as of the larynx; finally, the elevation of the larynx, descent of the epiglottis, and so on.

These difficulties occur in all superficial diseases of the palate with pain occurring spontaneously or on movement, in case of diminished lubrication of the bolus (closure, and so on, of the mucous glands), in disturbed muscular movements from various causes (pain—collateral œdema and inflammation which extend to the muscles—diseases of the muscles and of the nerves).

Swallowing is either impeded or impossible, or the first act of deglutition is performed normally, while, as a result of an incomplete or a complete paralysis of the soft palate, the food, especially in fluid form, escapes through the mouth or nose again, either wholly or in part.

The swallowing is either continuously abolished, or it is only impossible at times. After a long rest, especially, the first attempts at deglutition are often unsuccessful, sometimes from pain, sometimes from other causes, while subsequent attempts succeed. Small amounts are in many cases more difficult and painful to swallow than larger ones, because a greater muscular contraction is necessary in the former case than in the latter.

The ordinary expression for most of the diseases of the palate, "angina" (ἀγχω, to strangle, to narrow, to bind tightly), shows that the impeded deglutition was the most prominent symptom from the earliest times. The earlier expression "Cynanche" (the tongue hanging from the mouth, as in an overheated dog), is no longer in use. The expression "Bräune" (from the browning of the countenance) is only used in [German] popular parlance. Our Angina corresponds with the French word *esquinancie* (*esca* and *ango*) and the English term *sore throat.*

b. *Difficulty in the specific or modified movements of respiration:* gargling and hawking. *Hawking*, a frequent symptom of affections of the palate, consists in a prompt and powerful current of air driven from the lungs into the pharynx and posterior portion of the mouth. In its performance there is a trem-

bling of the walls of these parts, especially of the soft palate, by means of which the well-known sound is produced, and mucus, etc., removed from the surface, and propelled outward.

c. *Difficulty in speech.* The speech is either altogether difficult or uttered in a low tone, on account of the pain it causes, or the articulation of the palatal letters (*k*, *g*, *ch*, *j*, guttural *r*) is more difficult. The labials also (*b* and *p*) are articulated with difficulty, or are altogether indistinguishable, because, as a result of paralysis of the palate and the inefficient closure of the posterior nares, a portion of the expired air escapes through the nose, and the remaining portion is too small to move the lips in the proper manner. By compressing the nostrils, the articulation of the labials becomes normal, or the speech contains a peculiar additional *timbre* or clang—sometimes as if a foreign body was in the posterior portion of the mouth, which prevented resonance in the naso-pharyngeal space (anginose speech, *voix amygdalienne* of the French); sometimes the speech is nasal (rhinophonia), that is to say, there is a consonance of the voice in the nasal cavities. The latter has its origin in the impeded action of the levator and tensor muscles of the palate, and the consequent imperfect closure of the nasal outlets, the complete closure of which is essential for the clear enunciation of almost all the consonants and vowels. (A second variety of nasal speech, which occurs in articulating the consonants *m* and *n*, may also occur by narrowing or closure of the nasal canal.)

d. *Altered* (diminished or increased) *reflex movements.* Their cause lies partly in disturbances of sensibility, and partly in lessened mobility of the palate.

2. *Sensory Disturbances.*

a. *Disturbances of objective sensitiveness to pressure and temperature.* The first rarely comes under consideration. The latter is either increased, as is especially observed in the use of hot drinks, and even weak alcoholic beverages (light wines and beer), or it is diminished (anæsthesia) in many inflammations, morbid growths, and nervous diseases.

The proposed local anæsthetising of the palate, by painting it with concentrated solutions of morphia, bromide of potassium, and so on, are of almost purely surgical interest.

b. *The disturbances of the sensation of taste* in the soft palate, are only unfrequently utilized clinically.

According to the investigations of Klaatsch and Stich (Virch. Arch. 1858. XIV. p. 225), of Stich (ibid. 1859. XVII. p. 80), and Schirmer (Deutsche Klin. 1859. No. 13. et seq.), the greatest perception of taste is found upon the anterior palatine arches and in the anterior surface of the soft palate; the uvula, the tonsils, and so on, exhibit no such perceptions. Besides the glosso-pharyngeus, the lingual branch of the trifacial is also probably a nerve of taste. (See several pathological cases given by Schirmer.)

c. *The disturbances of ordinary sensation* are various.

The sensation of stickiness and that of dryness are mostly not to be disconnected. They have various causes, frequently appearing together: lessened, because impeded introduction of fluids,—diminished or altered secretion of mucus in the mucous membrane of the mouth and palate; sometimes, also, perhaps diminished formation of saliva; keeping the mouth open on account of diminution of the space in the posterior nares; fever

The sensation of a foreign body (dust, hair, and so on) is especially found in many chronic superficial affections. This produces either the so-called empty swallowing, with which the swallowing of a little fluid of the mouth, and so on, is no doubt generally connected, or frequent hawking.

The sensation of nausea, such as attends touching or irritating the palate, appears under similar conditions. This, however, is a feeling which is gradually blunted by habit. This sensation is absent in some healthy people, not unfrequently also in the insane, in hydrocephalic children, etc.

The sensation of fatigue in swallowing, and especially in speaking, has, mostly, the same origin.

d. *Pain* occurs prominently in acute superficial and parenchymatous affections, generally also in cases of intense hyperæmia, attended or not attended by erosions and ulcerations; while in other affections, croupous and diphtheritic inflammations, for example, in most of the chronic affections, especially in hyper-

trophy of the tonsils and in syphilitic angina, pain is slight or altogether absent.

The pains are spontaneous, or occur on contact (mechanical causes—very cold or hot food and drink—some medicines), and on movement (swallowing, speaking). The pains dependent on swallowing occur both in swallowing fluid, and particularly solid, ingesta, and in the swallowing of the mucus in the mouth. They are recognizable by the aversion which patients exhibit to drinking, etc., as well as by that which they manifest to making certain movements: the patient distorts his face, and brings the chin near the breast during the second act of deglutition (as if to shorten the road for the bolus), and not unfrequently spits the half-swallowed mass out again. The pains vary in intensity, but are, in general, moderate during rest. They are frequently referred to the external auditory canal. The pains are most frequently those of pressure; on touch or movement they are frequently stinging, seldom pulsating; or they have the character of the above-mentioned disturbances of ordinary sensation. In numerous cases pain also exists behind the angle of the jaw, either spontaneously or on pressure, associated with either perceptibly swollen glands or merely with swollen connective tissue.

The substances removed in diseases of the palate by hawking, coughing, etc., are more frequently mucus, pus, and blood, in varying quantity and in varying proportions. They do not present anything characteristic. Furthermore, there are croupous diphtheritic masses, dead, inflamed, and gangrenous portions of tissue, morbid growths, which sometimes, by their form and structure, permit the distinct recognition of the locality which they occupied. Still further, the masses existing in the lacunæ of the tonsils are worth mentioning: epithelial and purulent plugs in various stages of metamorphosis (so-called Spinnehusten, in German), and calculi.

A foul odor from the mouth occurs in many acute diseases, as a result of the stagnation and decomposition of food, mucus, etc.; further, in gangrene; and, finally, in chronic affections, in which decomposition of substances of different kinds occurs in the various cavities.

The symptoms of spreading of affections of the palate are readily explicable by the anatomical relations of the parts. They occur in some disturbances of circulation, in most inflammations, and in some few morbid growths. The disturbances caused thereby are sometimes the sole, or else the most important, subjective symptoms. Extension may take place:

Towards the cavity of the mouth, including the tongue, even as far as the lips, and the skin; from the former, also, sometimes to the excretory ducts of the salivary glands;

To the cavity of the pharynx, and thence, on the one hand, towards the nasal cavities (even to their adjacent cavities, the lachrymal canal, the conjunctiva); on the other hand towards the Eustachian tube and the middle ear (from which frequently proceed pains in the ear, or even hardness of hearing); thirdly, towards the larynx (epiglottic ligament, the epiglottis itself, the cavity of the larynx);

Towards the articulation of the jaw and its surroundings; producing in this manner, or by transplantation to the muscles controlling this motion, or to the pterygo-maxillary ligament, difficulty of opening the mouth, or impossibility of so doing;

Towards the corresponding lymphatic glands.

The increase in the production of the mucus of the mouth and palate, and of the saliva, a frequent symptom of many affections of the palate, has not yet been minutely explained.

Cough occurs in part directly from elongation of the uvula till it reaches the epiglottis, or even below it; partly from the entrance of mucus, etc., into the interior of the larynx, from want of action in the epiglottis; partly from extension of the inflammation to the laryngeal mucous membrane. It occurs as the chief symptom in many cases of chronic disease, especially catarrh, which affects the palate and the throat at the same time. The cough is short, for the most part, dry, or else accompanied by expectoration similar to the so-called laryngeal cough; or it approaches hawking in various degrees.

Vomiting occurs at the commencement and during the course of many acute affections of the palate. It is sometimes without significance, and is sometimes an important symptom.

The general symptoms of diseases of the palate exhibit very

little that is remarkable. Temperature, pulse, and respiration are, in no case, characteristic. The same holds true with regard to certain symptoms dependent partly on fever, partly on inanition. The headache that exists in many acute inflammations, and the albuminuria occurring often in some of them, are due to causes thus far not thoroughly understood. A few peculiarities attendant upon special affections will be spoken of in their appropriate places.

General Treatment.

The treatment of affections of the palate is either expectant, or local, or—and that only in a few affections (diphtheria, syphilis, and some others)—general.

The ordinary rule, avoidance of movements, etc., of the diseased parts, is unnecessary in most cases of acute disease, because the patient himself avoids every movement as much as possible on account of the pain it produces; and also, because, on the other hand, thirst and hunger compel movements of deglutition. Speaking, also, is avoided by the patient, for the most part without professional prohibition. In a number of chronic affections, especially those that are painless, the avoidance of the cause (screaming, smoking, etc.) is a self-evident rule.

The treatment of most of the acute affections is expectant. Nevertheless, professional views on this point are very diversified, as will be seen below.

The *local treatment* of the diseases of the palate is partly an external one (venesection, cataplasms, ointments to the region of the angle of the jaw), and partly an internal one (scarification, cauterization, pencillings, inhalations, gargles or mouth-washes, etc.).

Mouth-washes and gargles are employed when the disease is of considerable extent, and especially involves the anterior surface of the palate; when gargling is not impossible from pain, disease of the muscles, etc., and when the patients have the skill to do it properly (they are not used, therefore, in

children, in those dull of comprehension, etc.). For gargles, either pure water, or diluted milk, or decoctions of sage, mallow, or althæa are employed—sometimes with the addition of tincture of opium, or astringent substances (solutions of alum, tannin, decoction of oak-bark, etc.), or chlorate of potassa, lime-water, or permanganate of potassa in dilute solution. All these substances are employed cold or luke-warm, or, in some cases, warm.

Inhalations are ordered in a similar manner with gargles. In affections of the posterior surface of the palate, *the nasal douche* recommends itself.

All the topical remedies thus far mentioned have especially the advantage of keeping the diseased surfaces cleansed. The contemplated detachment of some exudations, or more rapid absorption of infiltrations, or astringent influences in hyperæmias, etc., are, on account of the short period during which gargles can be applied, but little more than pious hopes.

Pencillings, used frequently with borax in honey of roses, or in water, alcohol, or glycerine, or the similar use of chlorate of potassa, iodide of potassium, corrosive sublimate, and so on, are at present less frequently employed, and mostly only for diseases of a circumscribed nature.

The *stronger astringents* (alum, tannin, etc., in substance) and the *caustics* (nitrate of silver, in substance or in solutions of various strengths, less frequently caustic potassa, concentrated acids, etc.), of late years, especially, the modern carbolic acid, are employed sometimes in circumscribed affections (ulcers) ; sometimes, also, in widely extended affections, acute (diphtheria), as well as chronic (hypertrophy of the tonsils, chronic angina).

General treatment, which was formerly much employed in the form of emetic and cathartic remedies, venesections, specific remedies (belladonna, aconite, chlorate of potassa, etc.), is at present used almost only in diphtheritis and syphilis. In the first-named disease it is now especially tonic and supporting ; in the latter, specific.

None of the abortive measures employed against certain acute diseases are reliable. That which is still most frequently employed is the vapor bath at the commencement of an acute angina. Venesection in the usual manner, or from the ranine

vein, scarification of the diseased parts, painting them with nitrate of silver, and so on, are measures but very rarely employed.

I. Disturbances of Circulation in the Soft Palate.

The disturbances of circulation in the soft palate, with the exception of hemorrhage, possess no great practical interest in themselves, for they affect the palate alone in but few cases, usually involving adjacent parts (mucous membrane of mouth, pharynx and nose, the larynx—the brain) simultaneously. On the other hand, these disturbances are not seldom of diagnostic importance, inasmuch as they, like the external skin and the red of the lips, afford reliable indications of general blood diseases and disturbances of circulation (anæmia, plethora, passive hyperæmia, scorbutus, icterus, and so on). (In icterus the soft palate is distinctly colored yellow, in remarkable contrast to the hard palate.) The disturbances of circulation in the palate are still further important, in that many of them—for example, collateral and venous œdema—indicate the existence of disturbances of the surrounding parts (posterior portion of the floor of the mouth, root of the tongue, submaxillary salivary glands, base of the skull, vertebræ of the neck, bones of the nose.

The importance of anæmia, generally coincident with atrophy of the soft palate, in severe chronic diseases, especially in phthisis, has attracted but little attention in Germany. Consult, on the other hand, *E. Smith*, Consumption; its Early and Remediable Stages. 1862.—*H. Green*, A Practical Treatise on Pulmonary Tuberculosis. 1864.

1. *Anœmia of the Soft Palate.*

Anæmia of the soft palate is characterized by the usual features. In high grades of anæmia the palate is always diminished in size, and the isthmus, in consequence, is widened. The anæmia is either general, or it affects only isolated spots, which then always exhibit other deeper disturbances (cicatrices, pressure from adjacent abscesses, tumors, etc.).

General anæmia is distinctly evident in the palate in every

instance in which the amount of blood in the body is diminished from acute or chronic causes (after blood-letting, secretory losses, inanition; in chlorosis, hydræmia, advanced phthisis). In these cases the existence of anæmia is readily and more reliably confirmed in the soft palate than in the external skin, or even in the lips, because it can be seen there without pressure, turning the parts over, etc. The well-known causes of local anæmia (pressure, closure of arteries, spasmodic conditions) very seldom affect the palate.

2. *Congestive Hyperæmia of the Soft Palate.*

Congestive hyperæmia occurs under different conditions. In the first place, it occurs in many individuals during every ordinary inspection of the throat, especially when this is of long duration, or is undertaken in an unskilful manner. It is then sometimes seen to occur suddenly, like a blush upon the face. Then it is found in general plethora, and sometimes in congestive conditions in the direction of the cranial organs. In the latter case, however, the mucous membrane of the soft palate is often normal, even when there is intense congestion of the face. Hyperæmia frequently occurs, further, in habitual smokers and tipplers. Finally, it is observed in some cases of excentric hypertrophy of the left ventricle of the heart.

Congestive hyperæmia passes over, without any sharp line of demarcation, into the commencing stages of most acute and many chronic inflammations. It is sometimes found for hours together, sometimes for one or two days, at the commencement of other acute diseases (abdominal typhus, basilar meningitis), and may divert the physician's attention for a time from the latter affections.

The clear reddening of the soft palate, while the mucous membrane of the hard palate, and the remaining structures in the mouth, retain their normal color, or vary very little from it, is characteristic of congestive hyperæmia. In case of long continuance, the clear red frequently passes into a bluish color, and more or less numerous large veins become prominent.

3. *Passive Hyperæmia of the Soft Palate.*

Excess of venous blood takes place in long-continued or frequently-repeated congestive hyperæmia, as in tipplers; also as a result of the different stases of blood in diseases of the heart and of the lungs (weak heart, etc., emphysema, chronic phthisis with stasis, etc.).

The mucous membrane of the soft palate (and of the pharynx) shows the same cyanotic coloration as the lips, for the most part to a degree even greater. If, however, general anæmia and emaciation already exist, the bluish color is only slight, or is wanting altogether. Frequently, moreover, especially in catarrh from drink, numerous superficial veins are uniformly, or not uniformly, largely dilated. Sometimes the uvula is slightly œdematous at its tip. Hemorrhages occur not unfrequently.

4. *Hemorrhage from the Soft Palate.*

Hemorrhages occur from very different causes. In the first place, it is to be remarked that the blood present on the palate (and in the pharynx) may have arrived there from the cavities of the mouth or the nose, or from the air-passages and the lungs, or from the stomach. In all these cases the proof of the source of the blood in the organs mentioned is more important than the want of proof of the source of the blood on the palate. The posterior portion of the palate and the superior and lateral portions of the pharynx are hard to explore, especially in case of an exhausted condition of the patient.

The hemorrhage may occur from wounds of various kinds, and also from wounds occurring in the act of eating (bits of bone, fish-bones, etc., too hot food), or they have their origin in diseases of the vessels in the neighborhood of the palate. The first-named forms of hemorrhage belong in part to surgery, and in part will be subsequently discussed. Among the last-named causes of hemorrhage, aneurism of the internal carotid is of great significance.

Hemorrhages from the palate occur, furthermore, as a result

of congestive, as well as passive, hyperæmia, in the so-called hemorrhagic diathesis, in inflammations, in ulcers of various kinds, and in ulcerated morbid growths.

I have seen excessive hemorrhage of the palatine arches and the glosso-epiglottic ligament, after the application of the hot iron to an ulcerated carcinoma of the tongue. I once saw slight hemorrhages from the palate, with copious flow of blood from the gums (fatal hemorrhage from the latter), in a case of abdominal typhus; and, at another time, in a man fifty-eight years of age, who died from hemorrhagic diathesis (besides hemorrhage from the kidneys, and, at last, copious capillary hemorrhage in the brain, with hemiplegia).

Regular hemorrhages from the palate, after suppressed menstruation, occur unfrequently. (See case reported by Dunlap, New York Jour., May, 1850.)

The hemorrhages that occur in consequence of congestive or passive hyperæmia are mostly small in quantity, and of the character of ecchymoses. They are present in but small numbers; or, if more abundant, are still not very numerous. Sometimes they are interstitial, sometimes free. The interstitial hemorrhages lie either in the epithelium or in the mucous membrane; in the latter cases gray or blackish gray discolorations of the palate remain for a long time, in which case granular points of red or black pigment occur in the mucosa and in the capsule of the tonsils. The variations of seat just mentioned are not always easy to discover in life, and are practically without significance. The mucous and sub-mucous hemorrhages flow together sometimes to a clump the size of a hazel-nut, especially towards the tip of the uvula. These hemorrhages have, for the most part, no significance; but they may be of diagnostic importance after epileptic attacks, as well as after other forms of spasm with disordered respiration. Sometimes such hemorrhages occur in very severe efforts of hawking and coughing (whooping-cough, etc.,—chronic pharyngitis). When they are free, they produce a bloody color of the sputa, and are then sometimes subjects of medical inquiry.

In consequence of the hemorrhagic diathesis, and rarely of scorbutus, sometimes ecchymoses only, and sometimes larger bleedings, veritable hæmatomata occur, especially on the uvula.

The hemorrhages in inflammations of various kinds are of different, but mostly slight intensity (see further on).

The hemorrhages from ulcers and morbid growths may attain various proportions. Severe hemorrhages are infrequent in the former. In the latter they may induce or precipitate a fatal termination.

Hemorrhages of the soft palate and the uvula, so-called *staphylo-hæmatomata*, are readily recognizable. The blood is either reabsorbed, or it remains for a long time in the form of pigment.

Treatment.—Slight hemorrhages from the free surface subside of themselves. In severe hemorrhages the source of bleeding is to be sought for and is to be treated in the usual manner (chloride of iron, etc.).

Consult *Lasègue*, Traité des angines. 1868. p. 19.—*Lasègue* and *Legroux*, Arch. gén. July-Dec. 1871.

5. *Œdema of the Soft Palate.*

Acute and chronic local œdema very often occurs in the soft palate, especially in the uvula, and sometimes reaches a very high grade. Œdema of these parts seldom occurs in connection with general œdema.

Local œdema of the soft palate and the portions of the mouth and pharynx adjoining, has its origin most frequently in inflammation of the soft palate itself and the adjacent parts (bone, teeth, gums, external skin, and subcutaneous connective tissue, salivary glands, angina Ludwigii, etc.); it is a collateral œdema. This may be slight or very extensive, according to the seat and extent of the cause. When extensive, the uvula may be transformed into a flabby, reddish-white, translucent tumor of the size of the thumb; the palatine arches may become equally thick. If the œdema of the uvula is less marked, it is, in correspondence with the varying condition of the submucous tissue, most at the anterior surface, less so at the sides, and least on the posterior surface. The œdema sometimes occupies the uvula only; sometimes one or both palatine arches; sometimes the entire soft palate; and sometimes, finally, the adjacent glosso-epiglottic and aryteno-epiglottic ligaments also.

The diagnosis of œdema is easy. The results are very different, according to the amount and extension of the œdema. The treatment, if any is necessary, consists in that of its causes; or, locally, in scarification.

Local congestive œdema very seldom occurs in the soft palate.[1]

Œdema of the soft palate, occurring in general œdema from various causes, comports itself, in regard to its local conditions, just like local œdema.

II. The Inflammations of the Soft Palate. Anginas.

The inflammations of the soft palate are so different, according to their causes, nature, extension on the surface and in depth, their course, local and general effects, etc., that we must divide them into numerous varieties, most of them readily to be distinguished, but in part merging into each other. All these, as well as some inflammations of the pharynx, have this in common, that they cause difficulty in swallowing, *angina;* hence, the old names, also still employed, *angina catarrhalis, crouposa, tonsillaris, etc.*

Anginas have been known since the remotest times; not only in general, but also in several special forms. Their history is in part connected with that of medicine in general, and, therefore, cannot be given here in detail. Only during the present century have their pathologico-anatomical conditions been closely observed, chiefly by physicians especially devoted to diseases of children, and by the French in particular. Bretonneau's work (1826), as well as the extensive spread of pharyngeal diphtheritis, awakened especial interest in all the diseases that occur in the palate. The best works since that time are still those of the French. (Consult the various well-known encyclopædias and dictionaries; among the recent ones especially the article Angines, of Desnos, Nouv. dict. de méd. et de chir. prat. par Jaccoud, 1865. II. pp. 111 and 448; and an article on the same subject by *Peter*, Dict. encyclop. des. sc. méd. par Dechambre. 1866. IV. p. 689). Unfortunately, the French authors have fallen into the error of too extensive a specialization of anginas, in contradistinction to German authors, who separate the different forms of angina too little.

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[1] Concerning one especially interesting case of the kind which I observed for months together, see *Cuntz*, Arch. d. Heilk. 1874. XV. p. 63.

The most worthy articles, apart from the well-known works on special pathology, and the better works on diseases of children, are those of *Vidal*, Du diagnostic différ. des diverses espèces d'angines. Thèse. 1832.—*Chomel*, Art. Angines, in Dict. de méd. en XXX. Vol. III. 1833.—*Rilliet et Barthez*, Tr. des. mal. des enfants. 1853. I. pp. 197 and 223.—*Trousseau*, Clin. méd. 1861. 1. p. 431.

Inflammations of the soft palate are either *primary*, idiopathic, or they accompany any other, for the most part inflammatory, affections of the mouth, pharynx, nares, and larynx ; or a similar affection of the skin (acute exanthems, erysipelas, herpes, etc.) ; in which case they are called *symptomatic* or accompanying anginas. They may represent the most important localization of a *general disease* (diphtheritis, syphilis), or they may be *secondary* (in typhus, etc.). According to the nature of the inflammation, we distinguish catarrhal, phlegmonous, croupous, diphtheritic, and abscess-forming inflammations, inflammations with prevalent morbid formations, etc. For the most part, these forms are clearly distinct ; less frequently they pass one into the other. According to the extent of the process we distinguish inflammations of the soft palate, or of one of the palatine arches ; of one, or, more frequently, of both tonsils ; of the uvula, as well as inflammation of the pharynx, which often coexists with the others. Most of the inflammations coming under the notice of the physician are acute. Some of these recur frequently. Chronic inflammations are, on the whole, less frequent ; still, chronic non-inflammatory disturbances (hypertrophy, etc.) not unfrequently remain after acute inflammations. The anginas either heal, or additional new-formation processes and atrophy take place, or else gangrene sets in.

1. ACUTE CATARRHAL INFLAMMATION OF THE SOFT PALATE. ANGINA CATARRHALIS, SUPERFICIALIS, ERYTHEMATOSA.

Catarrhs of the palate occur very frequently, especially during youth. They are infrequent in the new-born, more frequent in little children, nurslings included, most frequent in boyhood and youth, much less frequent in full-grown manhood and old age, except in those who have already often been affected with

them. They are manifested in greatest number in spring and autumn especially, not unfrequently in quite an epidemic manner. In some families there exists a special predisposition to angina, gradually subsiding after puberty.

The causes of primary, acute catarrhal angina are, most frequently, taking cold in the throat and the feet, to such an extent that, for a period of years, certain predisposed subjects become anginous after each cold, even the slightest;—different ingesta which are injurious, either mechanically or chemically; hot food and drinks; hot vapors; less frequently so-called irritating food of all kinds, or tobacco; poisons and medicines. These act either directly (the different acids and alkalies, taken intentionally or by accident; further, nitrate of silver, some mercurial salts, zinc salts—the two latter sometimes among workmen employed in handling them—tartar emetic, phosphorus, iodine, and chlorine), or they act after being taken up in the organism (mercury, iodine, sometimes belladonna and other substances).

Angina occurs as an extension from catarrhal or phlegmonous (diffuse or suppurative) stomatitis, from various causes; especially also during dentition, and after operations upon the teeth;[1] in glossitis, pharyngitis, catarrh of the nasal mucous membrane, or of the larynx, and in almost all inflammatory affections of the tonsils.

Anginas occur symptomatically in all acute exanthems, especially scarlatina and small-pox; the congestion seems, here, frequently to skip the mucous membrane of the mouth, but is only, for the most part, not visible beneath the thick epithelium. Concerning the relation of angina to erysipelas, herpes, and pemphigus, see below.

Anginas occur secondarily here and there in acute diseases of the most various kinds (pneumonia, typhus recurrens), and in almost all chronic diseases, and in all stages of these affections, mostly without discoverable cause, sometimes from the causes already mentioned, sometimes only in conditions of acute and

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[1] Concerning the diseases of the teeth, which are results of anginas, and concerning Angina Dentaria, see *Magitot*, Tr. de la carie dentaire. 1867.—*Hohl*, Schmidt's Jahrb. 1870. cxlvii. p. 222.

chronic cachexia. In some cases they have nothing peculiar about them (thus catarrhal, croupous, and aphthous anginas occur, for example, in typhus and tuberculosis); in others they present special forms.

In all the anginas named, the mucous membrane of the mouth is normal; or it exhibits, at the same time, the same and frequently more severe alterations (mercurial and scorbutic anginas).

Pathological Anatomy.

Acute catarrhal angina most frequently affects the entire mucous membrane of the soft palate, especially, also, the superficial surface of the tonsils, and the posterior surface of the velum. Less frequently it affects only one portion of these structures: the anterior or posterior surface without the uvula and arches: or the uvula only, and then sometimes in its entire surface; and sometimes only in its anterior or only in its posterior surface; or only one or more palatine arches; or only the superficial surface of the (normal-sized, enlarged, or atrophied) tonsils (so-called *angina tonsillaris*, *catarrhalis* or *superficialis*).

The affected parts are reddened in various degrees, clear or dark red, uniformly or streaked, or in patches, or in all three forms together. They are moderately swollen, as is especially evident in the uvula and palatine arches. The swelling is sometimes distinctly œdematous and sometimes not. Sometimes the parts are dry, especially when darkly congested, and at the commencement of the affection; but they are usually very moist, and abundantly covered with mucus, which may be clear or slightly turbid from the presence of pus corpuscles, or less frequently is slightly reddened from the presence of blood. If the tonsils are affected only on the surface, and not, at the same time, in their parenchyma, they frequently appear smaller than they really are, on account of the swelling of the palatine arches.

The histological conditions are such that a return to the normal state usually and readily occurs. The hyperæmia of the blood-vessels disappears, likewise the surcharge of the lymphatic vessels; the serous infiltration becomes reabsorbed, partial

alterations of epithelium either recede or become adjusted by the subsequent growth of epithelium. Only unfrequently small, shallow losses of substance may be discovered, almost of the size of a lentil, involving the epithelium alone (so-called erosions), and most frequently situated in the environs of the normal or enlarged lacunæ of the tonsils. Still less frequently, such losses of substance occur in the superficial corium (catarrhal ulcers).

The submaxillary lymphatic glands are not at all, or only slightly swollen.

Various modifications of simple catarrh not unfrequently occur.

Sometimes the surface of the palate, somewhat oftener the surface of the tonsils, presents spots, or somewhat more extensive areas of a whitish or yellowish color, slightly elevated above the remaining surface, and resembling the deposit of diphtheria; but it is much thinner, and does not become thicker during the course of observation. This coloration is due to a suppuration of the epithelium. Microscopically, the uppermost epithelial layers are absent, for the most part; the next uppermost layers—rarely all of the layers—show the epithelium bloated, as though dropsical, or the epithelial cells contain from two to ten pus corpuscles, occasionally a few red blood corpuscles; or the surface exhibits a peculiar uneven, raw, and grayish appearance. In two such cases I found the upper epithelial cells very irregularly distributed, as if in the process of desquamating; between them were numerous large drops of fat (apparently from milk that had been taken into the mouth); the epithelium of the middle layers contained remarkably large or double nuclei. The lymphatic vessels of the mucosa were very broad, with copious lymphatic corpuscles within them and in their environs. (See below, *Angine Pultacée.*)

In individual cases the *œdematous swelling of the submucosa is especially extensive.*[1]

The *mucous glands*, ordinarily imperceptible or indistinctly perceptible, sometimes project so upon the surface that we must

[1] Consult *Wertheimber's* Angina pharyngea œdematosa. Journ. f. Kderkrkh. 1859. XXXII. p. 12.

suppose a constriction of their secretory ducts, through swelling of the epithelium of these ducts or of the tissues surrounding them, or of the stroma of the mucous membrane, and a retention of the secretion in the smallest glandular ducts and acini—so-called *acute glandular angina.* In other cases the principal excretory duct, at least its mouth, appears remarkably broad. In both classes of cases, especially in the first, shallow ulcers sometimes occur, whose centres are the deepened, round, excretory ducts, sometimes filled with muco-purulent contents.

The *solitary follicles* of the palatine arches and of the uvula, even normally inconstant as to existence and number, are, for the most part, normal; occasionally they are swollen to a moderate degree. In very infrequent instances they exhibit more extensive, small-celled, new formations which rupture, and then form small, crater-like ulcers. These *follicular ulcers* continue several days longer than the superficial catarrh.

Sometimes the surface of the mucous membrane exhibits variously numerous, irregularly distributed *vesicles*, the size of millet-seeds, filled with clear fluid. In one series of cases these occupy the epithelium, and are without relation to the excretory ducts of the mucous glands. They collapse after an existence of from one to two days, and leave shallow losses of substance of the character of erosions, which heal up in a few days and seldom pass into ulcerations. This is the true *angina catarrhalis vesiculosa* or *herpetica*, analogous to vesicular stomatitis or the herpes of the external skin. (See below.) The vesicles, which continue days and weeks, even after subsidence of the remaining inflammatory evidences, are either accumulations of mucus in the upper excretory ducts of the mucous glands (analogous, therefore, to sudamina, *i. e.*, comedones of the sweat glands or the sebaceous glands of the skin), or accumulations of mucus in the acini (analogous, therefore, to miliaria of the external skin).

A further vesicular form shows itself as uniformly distributed (in the manner of the mucous glands) sparser vesicles similar to those first-named, which rupture just as promptly, and leave a deep, roundish loss of substance as large as a lentil, of varying form from the running together of several of them, for the most

part with very hyperæmic areola, and with a yellowish deposit. After several days the vesicles heal without visible traces, while frequently new ones appear. These vesicles, etc., are *suppurations of the epithelium of the uppermost excretory ducts*, and their environs.

According to the statement above, the vesicles of catarrhal angina are very various. I have seen all the forms mentioned on the dead body, and have examined them microscopically, but do not find myself in entire accord with the clinical descriptions of most authors. The names *aphthæ* or *aphthous angina* I have designedly avoided in the above description, because they have several significations anatomically as well as clinically. Consult in this connection *Bohn*, Die Mundkrankh. d. Kinder. 1866, p. 3.

Symptoms.

The local troubles are different in the different acute catarrhs of the soft palate. They are not only dependent upon the extent and intensity of the processes, but also upon the rapidity of the onset of the disease, upon the individuality of the patient and the frequency of the attacks—the latter in so much that the troubles are slighter generally, the more frequently the patient has already had anginas. The affections of the uvula and the anterior palatine arches produce, in general, more inconveniences than those in other localities.

The local manifestations are: a feeling of pressure even during rest, of severe pressure or stinging, burning, etc., in speaking, but especially in swallowing. Deglutition is performed with difficulty. At the commencement the diseased parts are generally dry, the secretion of mucus being diminished; later in the disease the latter is increased. The excessive secretion of the fluids of the mouth and saliva, frequently present, is rendered still more annoying by the difficulty of swallowing. Little children, nurslings, refuse nourishment on this account. The mouth, also, can sometimes only be opened with difficulty. The submaxillary region is painful spontaneously, and especially on pressure, without any perceptible glandular swelling. Speech is also impeded, and in extensive swelling acquires a nasal tone—so-called anginose speech. In slight cases the impediment in speech is trifling,

and after a moderate amount of speaking these impediments often become less noticeable. Sometimes there are rheumatic pains, or slight contractions, or tonic spasms in the muscles of the neck.

Severer manifestations on the part of the respiratory organs (hoarseness, dyspnœa) seldom occur in pure catarrhal angina, or only with simultaneous intense influenza of new-born, and very young children, as well as in case of participation of the glosso-epiglottic and ary-epiglottic ligaments.

The general disturbances are very various. In adults fever is sometimes present to a slight or moderate degree, and sometimes it is absent. In children, especially very young ones, there is usually severe fever, and with it the entire series of its attendant general symptoms. Nausea, loss of appetite, bad taste, and increased thirst are frequent; sometimes there is vomiting. Headache, most frequently in the forehead, is not uncommon; dizziness is rare; delirium occurs often in small children, seldom in adults. Sleep is usually disturbed. The question of confining the patient to bed depends partly upon the fever, and partly upon the remaining general disturbances. Many patients without fever, or with slight fever only, do not consult the physician at all, or do so only incidentally, and pursue their accustomed avocations in the usual manner. Others, on the contrary, especially children and effeminate subjects, present the appearance of being rather severely ill.

The anatomical modifications of catarrhal angina, already detailed, do not materially alter the general picture of the disease. They cannot always be distinguished during life, in case of difficulty in inspecting the palate. Diphtheritis is frequently diagnosed, especially in cases where the surface exhibits a discoloration like that of a deposit.

The commencement of acute catarrhal angina is sometimes sudden. Chills at first are infrequent. Most frequently the patient notices the local or general ailment, or both together, early on rising, chiefly after a restless night.

The course of the disease is usually uniform, without fluctuations. After increase of intensity in the symptoms for one or two days, they remain unchanged equally long, or even as long as a week, and then usually subside gradually in from one to

three days. Sometimes the objective and subjective symptoms exist upon one side alone, or are especially severe on one side, and then either pass over upon the other side, while they disappear from the side first affected, or they affect both sides together for a day or for several days.

The disease does not terminate suddenly, but gradually. Most frequently the symptoms of the slighter anginas disappear during the night. Nevertheless relapses take place not unfrequently during the disappearance of the local annoyances, as well as during actual convalescence.

Gubler has seen paralysis of the soft palate, and once even general paralysis, after catarrhal angina. Similar cases have been reported by Barascut, Lucé, Bergeron, and others.

Modifications of Acute Catarrhal Angina.

1. *Rheumatic Angina.*

This is characterized by a disappearance of the throat symptoms of a severe catarrhal angina which has lasted for one or two days, and with this the appearance of a rheumatic affection of a muscle, a group of muscles, or an articulation.

The cause of this angina is catching cold, usually a severe cold. Its onset is sudden. Locally there is shown deep reddening, like that of scarlatina, and moderate swelling of the entire soft palate, and, in most instances, of the pharynx also. The pains are very lively, and the difficulty of swallowing considerable. The movements of the neck are much impeded and painful, partly on account of the symptoms mentioned, and partly in consequence of acute torticollis which frequently exists simultaneously. The fever is usually very severe and out of proportion to the local symptoms. The symptoms connected with the palate almost always cease suddenly, on the appearance of the rheumatic affection of the muscles or joints. The prognosis is good.

As already stated, the angina usually precedes the rheumatism; occasionally it attends it, or comes on after the rheumatism has passed off. Vesicles appear upon the mucous membrane of the palate in infrequent cases (Monneret and others).

Rheumatic angina was known to Stoll, Bouillaud, and others, but was first brought again into prominent notice by Trousseau and his followers. It is but little observed in Germany, but more frequently in England (*Ogle*, Med. Times and Gaz., Feb. 11, 1865). If, however, we include in this category those cases in which, in addition to the pain in the palate, severe pain occurs in the nape of the neck and at the side of the throat, especially on movement, but also during rest, it is also frequent with us (in Germany).

Gouty angina behaves itself in essentially the same manner.

2. *Intermittent Angina.*

This occurs with ordinary or masked intermittent, and is characterized by its periodicity.

It is little known in Germany. Since Jos. Frank's time it has been most frequently described by French authors (Barbette, Sédillot, and others).

3. *Cachectic Angina* (*Angine Pultacée of the French*).

Cachectic angina occurs only with acute or chronic cachexias (typhus, scarlatina, old age, chronic phthisis, etc.). Its anatomical characteristics (see p. 891) are those of distention and shedding of the epithelium. This shows itself in the form of a spotted or uniform whitish discoloration and rawness of the entire soft palate, with but slight congestion of the surrounding parts. The patches may be wiped off without exciting bleeding and without loss of substance. Local annoyances do not exist, or, if present, are those of a slight angina. The general disturbances recede before those of the previously existing general affection. The duration is only a few days.

I have seen pultaceous angina only upon the dead body, and only in three instances, which are described on page 892. French authors regard the nature of the disease to be an epithelial hypergenesis (?). The disease has nothing in common with aphthous angina, and does not even appear to predispose to it.

The *treatment* of acute catarrhal angina consists in rest of the parts, and, when attended by severe fever, in the usual procedures. Locally nothing at all is required for the most part; or we may advise the frequent administration of lukewarm drinks

(ice and cold water are not desired by most patients), and, in addition, mucilaginous or slightly astringent mouth-washes or gargles.

Opinions are much divided concerning the value of early cauterizations with nitrate of silver, in substance or in strong solution. I never observed any benefit from it in my own person or that of others. To guard against the frequent recurrence of these anginas, we may recommend keeping the neck bare, washing the neck with cold water several times a day, cold frictions to the entire body every day, and cold baths.

2. CHRONIC CATARRHAL INFLAMMATION OF THE SOFT PALATE. ANGINA CATARRHALIS CHRONICA.

Consult the literature of chronic pharyngitis.—In addition, *Chomel*, Gaz. méd. 1846. p. 310. Gaz. des Hôp. 1848. p. 251. 1851. p. 141.—*H. Green*, Treatise on diseases of the air-passages, etc. New York. 1847. 4th edition. 1858.—*Yearsley*, A treatise on enlarged tonsils and elongated uvula. 3d ed. 1848.—*Guéneau de Mussy*, Tr. de l'angine glanduleuse, etc. 1857.—*Gibb*, On diseases of the throat, epiglottis, and windpipe. 1860.—*James*, Sore throat, its nature, etc. 1861.—*Valentiner*, N. Ztg. f. Med. u. Med.-Ref. 1850. No. 24.—*Lacase*, Gaz. d. Hôp. 1851. No. 36.—*Tufnell*, Dublin Journ. 1862. XXXIV. p. 439.—*Dürr*, Ueb. d. Verbindung von Ophthalmia u. Angina granulosa. 1867.

Chronic catarrh of the palate is likewise frequent, and often occurs in middle life. It is more seldom a separate affection, being, for the most part, present at the same time with the various forms of chronic pharyngitis, of coryza, and, less frequently, of laryngitis ; but it is always much milder than these affections.

The causes of chronic angina are the same as those of congestive and venous hyperæmia (see pp. 883 and 884). Hence the names *angina clericorum*, *cantatorum*, *potatorum*. The cause of injury lies partly in straining the palatine structures in speaking, screaming, etc., partly on the inspiration of cold or dusty air, etc., through nose and mouth, etc., during these acts. In all these instances chronic angina becomes developed gradually, or it is a consequence of frequently recurring acute catarrhs. Less frequently the causes are unknown.

Chronic angina generally affects the entire soft palate, less frequently individual portions of it alone or in greatest part, most frequently the uvula. The mucous membrane is moderately, uniformly, or irregularly (in patches) reddened with almost always isolated broad and tortuous veins, some of them of the character of true varices. The mucous membrane is usually thicker than normal, seldom thinner—thicker usually as a result of œdematous infiltration of the submucous tissues, but sometimes in the form of an hypertrophy. The œdema is most conspicuous on the uvula, which may be doubled in length (in very rare cases reaching as far forward as the lips), and may become as thick as the thumb. In these instances the mucous membrane and submucosa may reach much further downwards sometimes than the muscular substance, so that only incomplete shortening of the entire organ is possible. The thickening of the palatine arches is less distinct. The mucous glands are distinctly swollen in many cases (hence the name *angine glanduleuse*), as well with thickening of the mucous membrane as in thinning of it, and not unfrequently discharge mucus during the movements of the palate. Occasionally the solitary follicles of the palatine arches are enlarged to double or quadruple their normal volume, most frequently those at the lower end of the anterior palatine arches—hence, no doubt, the name *angine granuleuse.* The lymphatic glands at the angle of the jaw are chiefly normal, sometimes moderately swollen, and then most frequently in scrofulous subjects, as well also as in those previously syphilitic.

Local disturbances are frequently entirely absent, or the patients become accustomed to them. They are not proportionate usually to the visible alterations of the parts. Most frequently there exists an annoying sensation of dryness, seldom one of burning. Impediments in speech or alterations of voice are rare, and difficulty of deglutition is still less frequent. All the symptoms are especially present soon after rising from bed, or after more severe use of the voice (by ecclesiastics on Sunday). Many patients have the habit of hawking very frequently (the *hem* of the English) ; others have only a very frequent but usually short cough, dry or attended with expectoration.

Chronic inflammation of the uvula, and its *elongation* especially, is sometimes unattended with any local symptom. More frequently there is a tickling in the throat, or the sensation of the presence of a foreign body, especially on bending the head backwards and on lying down. This, as well as the hawking, is probably chiefly dependent upon occasional adherence of the lower end of the uvula to the posterior wall of the pharynx. Sleep is frequently disturbed from the same cause. In some cases nausea, retching, and disposition to vomit, or even regular vomiting, are the most prominent symptoms—so much so, sometimes, that the diagnosis of chronic gastric catarrh is made.

The uvula, in this condition, sometimes retains its normal mobility, and sometimes its contractions are inactive and incomplete. In the latter case speech is sometimes altered, the voice is not clear, and in singers it sometimes loses in pitch. Some observers have seen nightly nightmare in consequence of this affection.

General disturbances occur mostly only when there is simultaneous chronic coryza and pharyngitis. They consist not unfrequently in hypochondriac and melancholic disposition, dread of pulmonary tuberculosis, etc.

The disease *terminates* in complete recovery only on removal of the cause. In other cases improvement only is possible, interrupted, however, by frequent relapses. Thus the disease *continues* not unfrequently for years and for decades, but usually disappears in old age.

The *treatment* is especially local, inasmuch as the causes cannot be removed for the most part, or can be only insufficiently removed. Excessive smoking is to be forbidden. In slight cases residence in an atmosphere free from dust, especially a forest atmosphere, the inhalation several times a day of warm steam, the use of the mineral waters of Salzbrunn, Ems, or Kissingen, alone or with milk or whey, are to be recommended. In severe cases, in addition, daily cauterization for a week, with solutions of nitrate of silver (1 part to 20 or to 10), or with solutions of sulphate of zinc (1 part to 30 or to 20), or sprinkling with powdered alum or with calomel (1 part to 12 of sugar), is useful.

Simple gargling with these substances is mostly useless. Even in obstinate cases resort to the springs named, among which Ems has the greatest renown, as well as resort to sulphur springs, will frequently secure immunity from the trouble for months together. If the uvula is abnormally long, its lower end should be excised.

3. THE SYMPTOMATIC ACUTE CATARRHAL ANGINAS.

Acute catarrhal anginas are more or less regular attendants upon several infectious diseases, especially the acute exanthemata, but not unfrequently also upon erysipelas, herpes, and pemphigus, especially when the disease affects the skin of the face alone or in part. Catarrh of the palate occurs much less frequently without a simultaneous affection of the skin. In these cases, however, it has the same causes, the same course, the same sequelæ, as when the exanthem of the skin is present. In all cases there is a catarrhal stomatitis at the same time, and, less frequently, pharyngitis also.

The symptomatic anginas are only unfrequently, however, so characteristic that the diagnosis is possible from inspection of the palate, the local disturbances, etc., without the aid of etiological and other clinical relations.

1. The *angina of measles*, *angina morbillosa*, is not absent in any case of measles. Its severity, usually, is in direct relation with the severity of the eruption on the skin, and does not depend upon the character of the epidemic. It is present usually from twelve to twenty-four hours earlier than the eruption on the skin, in rarer instances even forty-eight hours earlier. It affects the mouth and the palate at the same time, especially the posterior arches and the contiguous portions of the pharynx. An ordinary, uniform reddening of the mucous membrane is seldom observed. There are usually present, alone, or in addition to the redness, numerous red circumscribed or diffuse patches, from the size of a millet-seed to that of a lentil—and, at a later period, papules also. Not unfrequently local hemorrhages occur. At the end of from twenty-four to thirty-six hours the patches and papules have disappeared—the latter,

sometimes, after they have become transformed into little vesicles. The local disturbances are mostly slight; somewhat greater when considerable swelling of the palate and the tonsils occurs at the same time. The general disturbances are dependent upon the coetaneous affection of the skin.

Consult *Thomas*, Measles (this Cyclopædia, Vol. II.).—*Rehn*, Journ. f. Kinderkrkh. 1868. 1. p. 93.—*Monti*, Jahrb. f. Kinderh. 1872. VI. p. 20.

2. The *angina of rubeola* (Rötheln; German measles) is, on the whole, similar to that of the morbillous variety.[1]

3. *Scarlatinous catarrhal angina* varies according to the character of the epidemic, etc., and bears no relation to the eruption upon the skin. It remains either catarrhal, or changes in the shortest time into a croupous angina, etc. (see below). It is hardly ever absent in scarlatina. In exceptional cases it occurs without subsequent or simultaneous exanthem of the skin. It precedes the cutaneous eruption usually from twelve to twenty-four hours, occasionally as much as forty-eight hours and even longer. Seldom does it first appear at the same time with the eruption on the skin. In general it is distinguished by an intense uniform or spotted congestion over the entire surface of the palate, followed, after from twelve to twenty-four hours, by moderate tumefaction, as well as sometimes by small papular elevations. The lymphatic glands of the neck are, as a rule, moderately swollen. The subjective disturbances are frequently entirely absent at the commencement, and in general reach only a moderate grade. The appearances, on the part of the surface of the tongue and the general symptoms, are those of scarlatina. The commencement is usually sudden. The duration, in favorable cases, is from four to six days.

The scarlatinous angina without simultaneous or subsequent exanthem of the skin, so-called *scarlatina anginosa* (*sine exanthemate*), is, after the ordinary exanthematous, the most frequent form of the scarlatinal disease. It exhibits the same alterations of the mucous membrane of the mouth and the palate as the ordinary exanthematous form. Desquamation of the

[1] See *Thomas*, Jahrb. f. Kinderh. 1872. V.; and this Cyclopædia, Vol. II.

skin seldom occurs after it; albuminuria occurs now and then.[1]

4. *Variolous angina* is a regular attendant upon *variola vera*, an inconstant one upon *variola hemorrhagica* and upon *varioloid*. It is characterized by the same conditions as in the eruption upon the skin—a high grade of hyperæmia at first, and then suppuration of the epithelium, only that the throwing off of the epithelial layer is more rapid than the analogous process in the skin, and the transformation of the pustules of the mucous membrane into ulcers is more rapid. When the pustules or ulcers are rather numerous, there is a uniform, dense, purulent infiltration of the sub-epithelial connective tissue, sometimes of the interacinous tissue, and even of the acini themselves. The lymphatic vessels are large and frequently filled with fibrin. The variolous eruption on the mucous membrane of the palate appears at the same time as the affection of the skin, mostly somewhat earlier, never later. Thus it acquires a certain diagnostic interest, especially when the number of efflorescences upon the skin is small, or when they are too little developed, or when they have become indistinct through scratching, etc. The local disturbances are sometimes slight and sometimes very severe, according to the number of pustules and the general condition; and they are especially severe when the pustules are confluent, so that the surface of the palate has a diphtheritic appearance. Distinct cicatrices do not remain. The general disturbances depend upon the affection of the skin, the simultaneous presence of pustules in the larynx, trachea, bronchi, etc.

5. *Erysipelatous angina; erysipelas of the palate.* There are several different relations between erysipelas of the skin and catarrhal angina. Most frequently an erysipelas of the face passes over the lips upon the mucous membrane of the mouth and palate, and produces the symptoms of angina, on the palate especially;—less frequently an erysipelas of the face passes through the nose and the pharynx to the soft palate; erysipelatous angina seldom exists in connection with erysipelas in the

[1] Consult the manuals on Diseases of Children. Further, *Thomas*, Jahrb. f. Kinderh. 1869. II. p 373.—*Monti*, ib. 1873. VI. p. 227.

most frequently affected portions of the skin ; it often appears after the sudden or gradual disappearance of the latter, sometimes with the ordinary symptoms, sometimes with more extensive and much more speedy swelling of the submucosa—the so-called *erysipelas vagum* or *volaticum*, in contradistinction to the other form, *erysipelas migrans*. The least frequent complication is for an erysipelatous angina to pass over to the skin of the face, either directly or in the form of a metastasis.

Erysipelatous angina is characterized clinically by an intense purplish congestion, sometimes by a marked lustre, usually without any conspicuous tumefaction. The sensation of burning and of dryness is tolerably severe. Large vesicular elevations of the epithelium, which collapse after a duration of a few hours, exist but very rarely. The lymphatic glands of the jaw are but little swollen. The general symptoms are analogous to those attending erysipelas of the skin. The fever is more severe than in ordinary angina ; the gastric symptoms, nausea, pain at the epigastrium, and constipation, are sometimes very persistent. The disease lasts only a few days—a week at the most. The termination is usually favorable if the erysipelas travels from within outwards. If the opposite is true, death frequently occurs, chiefly through œdema of the glottis, less frequently through capillary bronchitis, etc., and sometimes without any discernible cause.

The diagnosis of erysipelas of the palate is only to be made with safety in connection with the previous, simultaneous, or subsequent erysipelas of the skin, especially of the face.

Erysipelas of the palate seems to have been known to Hippocrates. It has certainly been described by Sydenham, van Swieten, and Frank. For our minute knowledge of it we are indebted to the French authors, especially to *Cornil*, Arch. gén. 1862. XIX. pp. 257 and 443.—Consult, also, *Heusinger*, Arch. f. klin. Med. 1866. II. p. 523.—*Mettenheimer*, ibid. 1868. IV. p. 203.—*Bohn*, l. c., p. 34.

6. *Herpetic angina* (*Herpes pharyngis* of Bretonneau and Trousseau ; *herpes gutturalis* or *angina herpetiformis* of Gubler ; *angina aphthosa*, *aphthous sore throat* of the English ; *angine couenneuse commune*, etc.).

Herpetic angina is characterized by a vesicular affection of the soft palate analogous to herpes of the skin, but which continues

for a short time only, and, for the most part, undergoes transformation into peculiar shallow ulcers with a fibrino-purulent deposit. It is also characterized by the almost simultaneous presence of groups of herpes vesicles upon the skin of the face, especially of the lips, and upon the mucous membrane of the mouth. The mucous membrane of the soft palate, if the disease is observed at an early enough period, shows a number of vesicles, from the size of a millet-seed to that of a lentil, filled with contents more or less turbid, and with somewhat swollen surroundings strongly injected and hemorrhagic in places—the vesicles, sometimes sparse and sometimes numerous, being uniformly distributed, or else arranged in groups. The vesicles occupy the uvula and the velum more frequently than other localities; sometimes they are confined to the uvula; occasionally they exist only upon the tonsils. After a day and a half or two days, or three days at the furthest, the vesicles, in many cases, will have disappeared without leaving a trace, especially when they are few in number, and when their inflammatory areola is small. More frequently they rupture and become transformed into shallow ulcers, which heal up in from one to three days. Sometimes, however, especially when present in great numbers, with severe inflammation and tumefaction of the surrounding parts, they become covered with a whitish or yellowish pellicle (so-called *angine couenneuse*), which sometimes spreads also upon the surrounding parts, so that the greater portion of the soft palate may become covered by it. The pellicle can be removed with a hair-pencil; and, upon such removal, the shallow ulceration is seen beneath it, which heals in a few days—or, if the affection has already existed a few days, the mucous membrane underneath may be normal. The lymphatic glands of the jaw are little, if at all, swollen. In the majority of cases similar groups of vesicles are seen at the same time, or one or a few days later, upon one or more spots in the cavity of the mouth (tongue, cheeks, hard palate). There is almost always a coexistent herpetic eruption of the lips, less frequently of the skin of the face, and very rarely of parts at a distance from the face.

The *local disturbances* are those of an ordinary catarrhal angina, more frequently unilateral than bilateral. Sometimes the

pain and difficulty of swallowing are very great. Frequently an acute pharyngeal catarrh exists at the same time. The *general disturbances* always precede the local affection, sometimes by from one to three days. They consist in tolerably severe fever, often preceded by a chill, in gastric symptoms, etc.

The *duration* of the entire disease is from three or four days to a week. Sometimes the affection repeats itself once, rarely a number of times. The *termination* is always favorable.

The *causes* of herpetic angina are unknown. It occurs in adults more frequently than in children, more frequently in females with disturbances of menstruation. It appears at all periods of the year, and is sometimes especially frequent, at times when herpes in general and pharyngeal diphtheritis are prevalent.

Herpetic angina is little known in Germany, but does not appear to be any more rare than in France. I have repeatedly seen it in polyclinic practice. In most instances several cases appeared in rapid succession; after which, again, pauses of years would intervene. The vesicles themselves I have seen but a few times only, and always upon the uvula. In most cases there was, besides herpes of the lips, a group of shallow ulcerations present, some as large as lentils. I have never seen the fibrinous layer (the so-called *angine couenneuse*). In my cases the erosions healed in a few days.

Herpes of the palate has long been known. Boerhave and van Swieten, Stoll, and others observed it. The French authors, however, have described the disease with the greatest precision. *Gubler*, Bull. de la Soc. de Méd. des Hôp. 1857. III. p. 86. Arch gén. Mai, 1857.—*Feron*, De l'angine herpét. Thèse de Paris, 1858, et al. —*Bertholle*, l'Union méd. 1866. Nos. 65–70.—*Wittmeyer*, Corr. Bl. d. ärztl. Ver. v. Thüringen. 1873. II. No. 3.

Neumann (Lehrbuch d. Hautkrankh. 1873. 3d Aufl. p. 194) describes a disease perhaps belonging here. After two or three days of fever vesicles appear on the face, on the extensor surface of the extremities, and on the trunk; at the same time swelling and congestion of the soft palate and of the tonsils set in. After four or five days the vesicles desiccate. Relapses are common.

7. *Pemphigus of the soft palate* is found, according to Bärensprung (Char.-Ann. 1862. X. p. 55), in the form of an acute œdematous swelling with vesicular detachment of the epithelium, and is almost constantly associated with pemphigus of the external skin (*febris vesiculosa et bullosa*—water-blebs).[1]

[1] Consult also the case of *Rollett*, Wien. med. Ztschr. 1862. No. 19; also *Hebra*, in Virchow's Hdb. d. spec. Path. u. Ther. 1865.

The *treatment* of all the anginas just described is like that of ordinary catarrhal angina.

4. PHLEGMONOUS ANGINA.

Phlegmonous angina occurs in several grades of severity which have no sharply marked lines of demarcation. Further, the slighter grades pass over in every gradation into the higher grades of catarrhal angina, and the higher grades pass imperceptibly into gangrene of the soft palate.

The slighter and middle grades of phlegmonous angina occur both primarily and secondarily from the same causes as those which produce catarrhal angina, and occur to the same extent. Not unfrequently they affect the same individual several times. They are less frequent in childhood, more frequent at puberty and in manhood, infrequent in old age. The higher and highest grades seldom occur from the same causes: sometimes they occur in scarlatina, and, finally, under a few special conditions, which, when of slight intensity, may also excite slight phlegmonous angina. These are, in the first place, high temperatures; the inhalation of very hot air or of flame by persons whose clothes are burning, or who are in immediate proximity to a conflagration; the inhalation of hot steam or the swallowing of very hot water, most frequently observed in England in the persons of small children who steal drinks from the long-nozzled tea kettle; the application of the hot iron in surgical operations deep in the mouth, etc. Furthermore, phlegmonous angina occurs in voluntary or accidental poisoning with concentrated acids (most frequently sulphuric acid, less frequently nitric, muriatic, and other acids) or alkalies, most frequently with caustic lye, which is kept for washing and other domestic purposes, seldom with phosphorus.

[1] Recently Fraentzel observed (Berl. klin. Wschr. 1874. No. 9) phlegmon of the palate, pharynx, etc., after the internal use of *liq. ferri sesquichlor.* in typhus.

Phlegmonous angina, in its lower grades, is characterized, anatomically, by a moderate, and, in the higher grades, by a

much more intense hyperæmia, connected with more or less frequent hemorrhages, but chiefly by a more severe and uniform swelling of the tissues, not confined to the submucosa alone. In most instances the soft palate is affected by it in its entire extent; less frequently the affection is unilateral. The swelling is the greatest at those points provided with abundant submucous tissue. The uvula may become as thick as the thumb, and the arches of the palate may become so much thickened that the anterior arches may retain an impression from the last molar tooth.

The microscopic appearances are various. The blood-vessels are always contorted, dilated, and swollen with blood. In milder cases we find that, beneath the normally adherent or less firmly adherent epithelium, the mucosa is infiltrated with moderately or excessively numerous pus corpuscles, sometimes only in its papillary portion, sometimes in its entire thickness, especially in the neighborhood of the blood-vessels. They are also found in the submucosa, but for the most part in much smaller quantity. The swelling of this tissue is chiefly due to serous infiltration and to dilatation of the lymphatic vessels, especially at the excretory ducts of the mucous glands. The mucous glands themselves are normal, or else considerable quantities of pus corpuscles lie along their excretory ducts and between the acini, while the outer surface of the acini and their cavities are for the most part normal.

Hemorrhages and collateral œdema prevail in these cases of phlegmonous angina resulting from external heat. In cases of poisoning the same conditions sometimes prevail, and sometimes there are more or less characteristic alterations of the epithelium, the blood-vessels, the mucous glands, etc.[1]

Simple ulcers on different points of the soft palate, occurring under various circumstances, for the most part without symptoms during life, exhibit as regards their edges and their floors the usual characters of phlegmonous inflammation.

The *local manifestations* in the milder cases are like those of

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[1] Concerning these last relations, consult the well-known exposition of *Rokitansky*; further, *Nager*, Arch. d. Heilk. 1872. XIII. p. 213.

a severe catarrhal angina. In severer cases they are much more serious. There is intense spontaneous pain, increasing on pressure and on movement, except when gangrene has already taken place, or unconsciousness exists. Deglutition and speech may be in complete abeyance, especially when there is simultaneous phlegmonous stomatitis and pharyngitis. The tongue is thickly coated for the most part, and strongly retracted in severe cases, as if shortened and thickened. Sometimes there is copious bloody mucus in the mouth. The lymphatic glands of the neck are little, if at all, swollen.

The *general disturbances* consist in fever of moderate intensity, with evening exacerbations, and but slightly accelerated pulse, for the most part. The respiration is little hurried in uncomplicated cases, but is increased in various degrees when there is œdema or inflammation of the larynx, etc. Headache is sometimes present and sometimes absent. Vertigo is rarely present. Vomiting occurs frequently, especially in cases from poisoning.

The disease usually *commences* suddenly, even in cases of so-called spontaneous origin. Frequently there is first pain in the throat and a sense of pressure, which rapidly and continuously increase, and, according to the cause, at once, or in the course of one or two days, impede deglutition to a very great degree. Less frequently the disease begins with chills or rigors—the local manifestations first appearing some hours later.

The *duration* is about one week in ordinary cases of medium severity. In the severest cases death usually occurs before the end of this period; or, if recovery takes place, improvement begins at a much later date.

The *termination*, in moderate cases, is in return to the normal condition, sometimes with a transition into chronic angina. In severe cases this termination is less frequent. More frequently death occurs from the poisoning, or from the simultaneous or subsequent affection of the larynx (especially œdema of the glottis), or from suppuration in the subcutaneous and inter-muscular connective tissue of the neck, or of the mediastinum; or from inflammation of the lungs (catarrhal

pneumonia, or pneumonia from a foreign body), or by termination of the inflammation of the palate in gangrene (poisoning, scarlatina).

The *diagnosis* of phlegmonous angina is easy in itself. But the further question in point is, whether a primary phlegmonous angina is present, that is, one occurring spontaneously or through some poison, or whether it is connected with scarlatina, or whether it is secondary. In the latter instance the question arises whether the angina is secondary to suppuration in the surroundings of the tissues of the palate, or whether it occurs in consequence of abscess situated in the palate itself or more especially in the tonsils.

The existence of an angina produced by heat or cauterizing agents is established either by the history of the case, or, in the absence of this, by the burning of the external skin, present at the same time, the cauterization of the mucous membrane of the lips and the mouth, the discovery of the remnants of the poison, or the sudden appearance of a violent grade of inflammation. Scarlatinal phlegmonous angina is almost always associated with considerable tumefaction of the tonsils, in most cases with phlegmonous pharyngitis, frequently also with infiltration of the parotid gland, the lymphatic glands of the jaw, and the surrounding connective tissue. The absence of any external cause for the sudden and intense inflammation, the severe general manifestations (high temperature, rapid, and usually small pulse, severe cerebral symptoms), the eruption on the skin (either just commencing or already distinctly developed), are furthermore important in the diagnosis of the phlegmonous angina of scarlatina.

The discrimination of phlegmonous angina from collateral inflammatory œdema is frequently possible from the aspect of the parts (pale color, extensive œdematous swelling), but chiefly by its limitation. In *angina Ludwigii* and in anthrax, which appears in the upper part of the neck, only one-half of the palate is affected; in abscess of the gum of the back teeth only the anterior palatine arch of the affected side is swollen in most cases. In scarlatina it is very unusual to see one side alone affected, or one side more prominently than the other.

In small children phlegmonous angina may proceed like a severe pharyngeal diphtheritis, with laryngeal croup, so that tracheotomy may be necessary. I saw this occur in a child one year old, in whom death took place after thirty-six hours' sickness, in spite of this operation. On dissection, considerable congestion and swelling of the soft palate was found, with an insignificant amount of swelling of the ventricular bands (upper vocal cords) of the larynx. The mucosa and submucosa exhibited the conditions above described. The swollen epithelium was found to consist of numerous large cells, which contained from ten to twenty pus corpuscles, either uniformly distributed or collected in a large vacuole.

The *treatment* of phlegmonous angina is, in mild cases, like that of catarrhal angina. In severe cases it consists in the administration of draughts of cold water, ice-pills, and cold compresses about the throat. If there seems to be danger of an extension to the larynx, doses of calomel at short intervals are advisable. In the severe pains of larger children or adults narcotics are to be administered.

The angina accompanying glanders, and that accompanying anthrax, may be regarded as further, infrequent *modifications of phlegmonous angina.*

In the *angina of glanders* the local manifestations are those of an intense phlegmonous angina, sometimes with the production of pustules and ulceration, or with transition into mortification, and with great swelling of the lymphatic glands of the jaw, sometimes with considerable œdema of the face and neck. The local disturbances are marked, the general manifestations severe.

In the *angina of anthrax* similar conditions exist; sometimes with gangrenous emphysema of the skin of the neck.

In both classes of cases the etiological conditions, the occupation of the patient and the previous local manifestations, are of considerable importance in diagnosis. The prognosis is almost absolutely unfavorable. (See Gangrene.)

Psoriatic angina, psoriasis of the soft palate, I have seen occasionally in general non-syphilitic psoriasis of the cutaneous surface. It consists in several distinctly circumscribed patches on the soft palate, but little elevated, uniformly reddened, of the size of one or two lentils, and usually not very numerous, the epithelium upon which does not exhibit any essential altera-

tion to the unaided eye. The patches remain for weeks and months, and disappear with the eruption on the skin, or before it.

5. INFLAMMATION OF THE TONSILS; AMYGDALITIS; TONSILLITIS; ANGINA TONSILLARIS.

The different inflammations of the tonsils require special consideration, partly because they are frequently present alone, and partly because their appearance in conjunction with catarrhal and other inflammations of the palate materially modifies these affections.

Inflammations of the tonsils are acute or chronic. They affect either both tonsils or only one of them, in either case affecting the organ in its entire extent or only in part. They may either attack the epithelial structures only, or the glandular structures only, or chiefly the connective-tissue stroma, with the capsule or without it, or several of these tissues together. They seldom occur in normal tonsils, but often in partially or wholly hypertrophied or atrophied tonsils.

Etiologically, inflammation of the tonsil is like the ordinary anginas. The same form or different forms of tonsillitis frequently affect the same individual a number of times, even as many as from twenty to fifty times during his life—less frequently, as a rule, as he grows older—sometimes at longer, and sometimes at shorter intervals, sometimes at intervals of only days or weeks. This frequent recurrence of amygdalitis often has its cause in alterations of the tonsils (retention of secretion in the lacunæ, etc.), and often its causes are unknown.

The following forms of *acute angina tonsillaris* are to be distinguished *pathologico-anatomically.*

a. *Simple or superficial catarrhal tonsillar angina; superficial catarrh of the tonsils;* pure catarrhal inflammation of the mucous membrane of the tonsils. The lacunæ and the parenchyma of the tonsils are entirely unaffected in uncomplicated cases; but one of these structures will often show a similar slight catarrh, or a retention of the secretion from swelling, etc. of the superficial epithelium, which is slightly infiltrated with serum.

b. *Lacunal* (according to others *follicular*) *catarrh of the tonsils*, usually appears in connection with superficial catarrh; but it is generally of longer duration, and is therefore often thought to be the only change that has occurred. Many cases of so-called *angina tonsillaris* of mild grade belong here. Individual lacunæ of the tonsil, or all of them, those of normal as well as those of hypertrophied or atrophied tonsils, become filled, under conditions of hyperæmia, with a whitish or whitish-yellow, thin or thick curdy substance, flowing out when thin, but, when thick, removable only with difficulty, or even not at all. This substance consists of epithelium and pus in varying proportions. If these contents do not escape from the lacunæ, they frequently undergo desiccation, and sometimes bacteria are developed in various amounts; fat also is formed, and needles of fatty acid; sometimes cholesterin, and even masses of chalk in molecular form or in the shape of stones as large as peas. (See below). The yellowish or whitish masses seen on the superficial surface of the tonsils correspond in number, size, form, and distribution with the orifices of the lacunæ which themselves exhibit numerous variations: they vary much in number; their form is round or oval, three- or four-cornered, etc. When the surface is not kept very clean, the purulent points are not unfrequently covered with mucoid pus, and thus concealed. Sometimes, upon removal of this substance, shallow, circular erosions of the epithelium become apparent around the yellow points. The conditions are almost always easily recognized on the corpse. In the living subject, especially at the commencement of the disease, it is frequently confounded with herpetic angina, with mild diphtheritis, and with superficial abscess of the tonsil. The parenchyma of the tonsil is affected in different ways. In acute cases it is always swollen in a moderate or severe degree, which is the more important if the disease occurs in an hypertrophied tonsil. The swelling is produced by hyperæmia and serous infiltration, but in greatest part by the simultaneous proliferation of cells. The tonsils then project beyond the arches of the palate to a variable degree. In chronic cases the tonsil is only moderately enlarged, or not at all enlarged. In addition to the follicular tonsillitis, acute catarrh of the palatine arches is always found, frequently

also of the uvula and the soft palate, less frequently of the mucous membrane of the pharynx, and hardly ever of that of the mouth.

Tonsillitis of the lacunæ heals in many cases by the spontaneous evacuation of the epithelial and purulent contents of the lacunæ, or by their expulsion in efforts of hawking; or the contents may desiccate, and then become foul, or become calcareous; or, as a result of the suppuration of the epithelium, there is a loss of epithelium at points in the lacunæ and an adhesion of their opposite surfaces. In this way a diminution of the size of the lacunæ results, sometimes amounting almost to obliteration, and then the remaining portion may undergo cystic distention.

c. *Parenchymatous Amygdalitis* seldom occurs by itself, but, as a rule, in connection with superficial inflammation or with inflammation of the lacunæ, or it is preparatory to the formation of an abscess. It is either primary or produced by extension of the inflammation from the lacunæ or from the adjacent structures. It is frequently found in scarlatinous, variolous, croupous, and diphtheritic anginas.

Parenchymatous amygdalitis is characterized, in the first place, by congestive hyperæmia of a high grade and serous infiltration. The tonsils may become swollen to double their normal bulk, and may even exceed this. If the affection involves both tonsils, the entrance into the pharynx is decidedly narrowed, and the uvula is dragged forwards or backwards to allow the two tonsils to approach each other, which they may do till they almost touch. If the affection is unilateral, the uvula is drawn over to the opposite side. The enlarged tonsil projects out of its bed, because it cannot project externally. In this stage there results either return to the normal form or an infiltration of small cells, both in the interior of the follicles and between them, and sometimes also in the inter-lacunal connective tissue and in the capsule of the tonsil. These conditions, likewise, may undergo retrogression, or a new formation of reticulated substance with permanent hypertrophy of the tonsils may result. In other instances abscesses occur in varying number.

I have examined parenchymatous tonsillitis several times—twice in its transition

to the formation of abscess, where the hypertrophied tonsil had been extirpated during the first days of the inflammation. Both times the larger portion of the tonsil exhibited the characters of hypertrophy without inflammation. On isolated places of the capsule, beneath the surface of the tonsil, I found a considerable infiltration with small cells. This also occurred in several places in the epithelial lining of the lacunæ, either there alone or in the adjacent cytogenetic tissue at the same time—in the latter case with escape of the pus into the cavities of the lacunæ.

d. *Amygdalitis with abscess; tonsillar abscess.* Tonsillitis proceeding to abscess rarely occurs by itself, but usually at the same time with one of the previously-mentioned inflammations. Sometimes it affects both tonsils, sometimes only one, or one in a greater degree than the other. It seldom occurs in young children, but often in larger children and in youthful adults.

Amygdalitis with abscess consists in the formation, usually, of several abscesses which do not lie in the lacunæ, but in the parenchyma of the tonsil, and seldom remain isolated, but usually become confluent. If the abscesses are near the surface of the tonsil or a lacuna, they perforate the former or penetrate into the latter. The previously existing hyperæmia, and the serous or cellular infiltration of the remaining tonsillar tissue then subside, and the tonsil returns to its normal form. If the abscesses are at a distance from the surface, a desiccation of the pus usually occurs, sometimes with final resorption and formation of cicatrices, and, at a still later stage, atrophy of the affected places. The tonsils acquire varied proportions according to the number and size of the abscesses and according to the condition of the remaining tonsillar tissue. The palate exhibits evidences of catarrhal or phlegmonous inflammation, sometimes only in the arches, sometimes in its entire extent.

e. *Peritonsillar, or retrotonsillar abscess*—that is, the formation of abscess in the connective tissue surrounding the tonsils, most frequently between the tonsil and the affected palatine arch, usually the anterior arch. The affection ordinarily involves but one tonsil, sometimes the other also several days afterwards. The abscess may acquire the size of a walnut, so that the arch of the palate at the affected spot is bent forwards as much as half an inch, and the tonsil is drawn towards the middle line,

sometimes also drawn downwards. The usual termination of the abscess is perforation, and then rapid return to the normal condition. If the affection occupies the anterior surface of the tonsil, we observe a round projection of dark-red color, which may be as large as a walnut, and which, at a later period, appears gray at its most prominent part, and then sometimes yellow, the tonsil itself being frequently invisible as a consequence of the great œdema of the palatine arch. The uvula, usually markedly œdematous, is sometimes drawn forwards, or backwards, or to the side. If the abscess occupies the outer or the posterior surface of the tonsil, the projection is slight, or there may be none at all; on the other hand, the palatine arches are very œdematous, the tonsil sometimes being crowded out of its bed between the arches of the palate.

Combinations of the inflammations of the tonsils just described are frequent. An acute or chronic inflammation of one or more lacunæ most commonly causes a parenchymatous inflammation or a suppuration of the contiguous tissue, or of the surrounding connective tissue, if the lacunæ are very deep. The inflammation of the lacunæ may irritate the surrounding structures mechanically, by the formation of stones, or, chemically, by the decomposition of their contents. Processes occur here analogous to those in the external skin, where the accumulation of secretion in the hair and the sebaceous follicles (the so-called comedones, miliaria, etc.) excites inflammation of the surrounding tissues, producing the various forms of acne.

Clinical Considerations.

Superficial tonsillar catarrh gives rise principally to local symptoms. The general symptoms are similar to those of catarrhal angina. Tonsillitis affecting the lacunæ, parenchymatous tonsillitis, and tonsillitis with abscess, are frequently not to be distinguished from each other at the commencement of the disease, but are usually readily distinguishable after they have existed for a few days. Sometimes they give rise to slight and sometimes to very severe local symptoms, and to mild or severe general symptoms. Both series of disturbances are, as a rule,

slightest in inflammations of the lacunæ and in mild inflammations of the parenchyma, and most severe in those with abscess.

The pathologico-anatomical division of tonsillitis into five different forms, as already described, cannot be clinically carried out in every individual case: 1st, because, not unfrequently, several forms are combined; 2d, because a minute inspection during life is sometimes utterly impossible (in the case of very young children), and sometimes only possible during the first days of the disease, the difficulty of opening the mouth during the later days, the concealment of the diseased tissues by coatings of mucus, etc., interfering with a careful examination; and, 3d, because the deeper inflammations of the lacunæ and the deeper abscesses are not recognizable before section of the tonsil, whether in the living subject or on the corpse.

The *local symptoms* are found either on one side only or on both sides; in the latter case they may be equal or unequal in degree. They consist in a feeling of pressure or pain, usually present even during repose, increasing upon external pressure, movements of swallowing, speaking, hawking, etc., and upon movements of the neck and head. In general, they are greater the more intense the hyperæmia, the greater the swelling and the more rapidly it has formed; and also, for the most part, the less frequently the affection has previously existed. The pains are variously described according to their nature (pressure, burning, stinging, scraping, etc.), and according to their seat (in the palate, angle of the jaw and vicinity, ear), sometimes without any explanation for these differences from the visible extension of the inflammation. The movements of deglutition are painful and impeded, at first with solid substances and afterwards with fluids also, and, in cases of tonsillitis with abscess, may be almost impossible or quite so for some days. Everything taken flows out of the mouth again, and so also with the copiously secreted mucus and saliva already in the mouth. Speech is painful, incomprehensible, and exhibits the characters of anginose speech in the highest degree; in unfrequent cases it is lisping. There is difficulty in opening the mouth—so much so, sometimes, that the front teeth cannot be separated from each

other. Usually there is also some impediment in the movement of the cervical vertebræ in turning the head.

With almost all tonsillar anginas there is hyperæmia or collateral œdema of the contiguous portions of the pharynx at the same time. If this extends to the Eustachian tube, there will be noises in the ears and continuous or flying pains in the ears, described by some patients as a very distressing symptom; if it extends to the entrance of the larynx, there will be dyspnœa, especially on lying down, or paroxysms of suffocation may occur. The encroachment upon the naso-pharyngeal space causes the patient to keep the mouth half open. Hoarseness occurs in some cases. Some patients have a short and dry cough, especially in the recumbent posture, probably from the flowing of mucus into the upper part of the larynx. In tonsillitis with abscess there sometimes exists, from the second or third day on, ordinary or painful œdema of the submaxillary region, and also, less frequently, of the upper portion of the neck and of the face, on one side.

The lymphatic glands of the jaw are, in all forms of tonsillitis, rarely swollen. Extensive swelling of these glands occurs (if the glands had not been previously enlarged) almost only in cases of frequent recurrence and in subacute cases with abscess.

The *general symptoms* are slight, or absent, in superficial tonsillitis, and not unfrequently also in lacunal amygdalitis, at least in adults and in frequently recurring cases; while they are usually very distinctly pronounced in all other varieties. They consist in fever of different grades, in gastric disturbances, and frequently in head symptoms.

Fever is not present in most cases of superficial tonsillitis, in many cases of tonsillitis of the lacunæ, and in some cases of parenchymatous tonsillitis. In the remaining forms, especially that with abscess, there is an increase of temperature of from 1° to 1.5° C. (1.8° to 2.7° Fahr.), or even as much as 2° or 2½° C. (3.6° to 4.5° Fahr.), and moderate acceleration in the frequency of pulse and respiration. The temperature usually reaches its maximum in the first few days, especially on the third day: 39°–40° C. (102°–104° F.) in cases with abscess, below 39° C. (102 F.) in the remaining cases. The fever, as a rule, soon shows moderate

remissions with evening exacerbations, the latter sometimes occurring at noon or early in the night; both are more marked in case of the formation of an abscess. Sometimes a considerable increase of fever is suddenly manifested after several days of slight febrile phenomena. Defervescence is usually critical, the crises continuing ordinarily from twelve to twenty-four hours. The crisis begins most frequently from the third to the fifth day; but, in the variety with abscess, mostly from the sixth to the seventh day, or even later.

Gastric disturbances occur regularly in the variety with abscess, irregularly in all other forms. The tongue becomes coated from the second day of the disease, especially at its posterior portion. The appetite fails altogether in most cases, or its gratification is rendered difficult by the local conditions of the parts. Thirst is moderate in most instances; it is greatest, usually, in the earlier days of the disease. Constant nausea is not infrequent, but vomiting is. Intestinal disturbances are rare. In cases of deficient cleanliness a bad breath is frequent.

Cerebral symptoms are slight in adults, but in children they are frequently very pronounced. They consist in pains of varying intensity over the head generally, or limited to the forehead, in disturbed sleep with frequent dreams, while during the daytime a more apathetic condition prevails for the most part. Delirium is frequently present, especially at night, in children; less frequently in adults. Convulsions also sometimes occur at the commencement of the affection in children.

The *general sensation of illness*, the feeling of languor, etc., is most marked in the forms with abscess, and, for the most part, it is pretty well marked in the other varieties only when the disease is present for the first time. It has its origin in the local disturbances, in the fever, the inanition and the sleeplessness—the latter being sometimes produced only by the greater secretion of saliva and the consequent necessity for swallowing.

The *commencement* of the disease is seldom sudden in the milder forms, but more frequently gradual; in the severer forms it is usually sudden. The latter class of cases generally announce themselves as a general sense of being unwell, early in the morning, before rising, after a somewhat restless night.

Upon rising, or during any time of the day, a severe chill may occur, or sensations of chilliness of several hours' duration, followed by heat, thirst, loss of appetite, sometimes vomiting, headache, and occasionally epistaxis. At the same time, often only after some hours, occasionally only after one or two days, the local disturbances in the throat become manifested, slight at first, but usually increasing rapidly in intensity. If the tonsils are examined at this period, they usually distinctly exhibit, sometimes without the previous existence of pain and dysphagia, the evidences of superficial or lacunal tonsillitis—the remaining forms being chiefly characterized by a moderate enlargement of the tonsil. When the affection commences gradually, the first evidences of disease are sometimes the general, sometimes the local disturbances, and sometimes both together.

The *course* of the disease presents no essential stages.

The *duration* is different in the different forms of amygdalitis, varying between two and fourteen and even twenty days and more. The superficial and lacunal forms of tonsillitis continue usually from three to four, at most for eight days. In the latter form some isolated yellowish points frequently remain after the congestion and tumefaction, and with them the local disturbances have disappeared. Parenchymatous tonsillitis and tonsillitis with abscess continue at least a week, frequently a week and a half, and even as long as two or three weeks.

The *termination* is always favorable, except in the smallest children, and in certain very infrequent individual cases in adults. Most frequently complete recovery takes place within one or a few days, or in a few hours, or all at once. The latter occurs only in case of abscess in the tonsil or in its surroundings, and upon its rupture and discharge ;—patients who, a few hours or a few minutes before, were complaining of severe symptoms, local and general, becoming relieved almost immediately. The point of rupture of the abscess is only occasionally to be seen on inspection ; the patient has expectorated or swallowed odorless or fetid pus, as the case may be, or knows nothing about it. If this takes place during sleep, temporary suffocation may be produced by its penetration into the larynx. In a few days the tonsil diminishes in size, prominence, and vascularity ; the collateral hyper-

æmia of the surrounding structures, and the increased temperature disappear after a few hours, and the pulse not unfrequently sinks below its normal frequence. At once, or after a quiet night, even without having as yet appeased his hunger, the patient, who previously presented a mournful aspect, feels as if new-born. It is true that often, although complete recovery takes place, a great disposition remains to the renewal of similar disease, which follows, sometimes within a few days, sometimes only after months and years.—Parenchymatous tonsillitis frequently passes into hypertrophy, and tonsillitis with abscess into partial atrophy of the tonsil.

Cases of *fatal termination* are exceedingly infrequent. They occur almost only in connection with other severe diseases, and in small children—in both classes of instances as a result of several days' inanition and the febrile movement. The few remaining fatal cases have their cause in extension of the disease to the larynx (œdema of the glottis, etc.), or upon the lateral walls of the pharynx (erosion of large vessels with fatal hemorrhage), or in the descent of pus into the thorax, or in severe cerebral symptoms the causes of which are not determinable, even by post-mortem examination.[1]

Chronic amygdalitis, chronic tonsillar angina, presents itself almost exclusively in the form of hypertrophy of the tonsils; less frequently, in that of atrophy of the tonsils, to which one of the previously mentioned five anatomical precedents (superficial catarrh, catarrh of the lacunæ, etc.), becomes superadded, sometimes in the acute, sometimes in the subacute, sometimes in the chronic form. (See Hypertrophy and Atrophy of the Tonsils.)

Chronic ulcers on the surface of the tonsils are seldom of catarrhal or inflammatory nature. They are most frequently syphilitic in origin.

The *treatment of tonsillitis* in superficial catarrh and in catarrh of the lacunæ is similar to that of acute catarrhal angina. Most practitioners treat parenchymatous amygdalitis in the same manner. Others recommend the application of cold to the sides of the upper part of the neck, the frequent dissolving of bits of ice

[1] Consult *Chassaignac*, Gaz. des Hôp. 1854. No. 65.—*Hauff*, Württ. Corr.-Bl. 1863. No. 43.

in the mouth, and even leeches behind the angles of the jaws. Only in very severe swelling, that undergoes no alteration for days, is it recommended to make several scarifications into the tonsils, and to repeat the operation after a day or two.

Abscess in and about the tonsil is treated at first in the same manner—especially so, as its diagnosis is not possible for the first few days. In case of the diagnosis being made, warm mouth-washes and gargles are recommended, and warm compresses externally, if they do not produce headache, or increase headache already existing. The much-recommended timely opening of tonsillar abscesses is of little use, because it is very seldom successful, and, even when it succeeds, hardly ever relieves the patient. It is somewhat different in anterior peritonsillar abscess; but even then the relief to the patient is seldom as great as after spontaneous discharge. Deep incisions are not advisable, owing to the contiguity of the carotid artery.

Tonsillar abscess is not to be aborted by any treatment (ice, scarification, etc.); venesection is at most to be performed in cases of severe cerebral symptoms in larger children and in adults; tracheotomy may become necessary in case of extension of the inflammation to the upper portion of the larynx. (Case of *Puech*, Gaz. hebd. 1857. IV. No. 34.)

The treatment of *chronic tonsillitis* is essentially the same.

6. ANGINAS WHICH REGULARLY APPEAR WITH STOMATITIS.

A precise discrimination cannot be made between those inflammations which affect the soft palate especially, and during which inflammatory conditions of the mucous membrane of the mouth recede, and those in which the reverse conditions exist. Nevertheless several of the affections belonging to the latter class deserve a brief consideration in this place also. (For more details consult the articles under Diseases of the Mouth.)

a. The *so-called toxic anginas*, which are produced by tartarized antimony, by the internal use of mercury, iodine, etc., are always associated with a tolerably intense stomatitis.[1]

[1] *Lollier* (Union, 1873. No. 4) describes an intense stomato-pharyngitis, which the patient had intentionally excited by means of cantharides.

b. *Aphthous angina* always occurs only upon isolated portions of the soft palate, while aphthæ are found in much greater number in the most different portions of the mucous membrane of the mouth. The greatest differences of opinion still exist concerning the pathological anatomy, etc., of the disease.[1]

c. *Ulcerous angina* always occurs with an *ulcerous stomatitis* (thrush), usually of severe grade. The latter occurs much more frequently without simultaneous ulcerous angina. The characteristic ulcers and general catarrhal inflammation occur upon the mucous membrane of the mouth, especially upon the edges of the gums and the inner surface of the cheeks, frequently upon one side only. (See above.) The soft palate, especially its central portion, as well as the uvula in its upper half, occasionally the palatine arches and the tonsils, exhibit in the earliest days small yellowish spots, or spots as large as an inch square and larger, distinctly elevated (suppuration of epithelium), and usually interrupted by small, regularly distributed red points (excretory orifices of the ducts of the mucous glands). After from three to six days, grayish-red depressions are found in place of the yellowish patches (losses of epithelium), and after a few days more the normal conditions are resumed. The local manifestations are those of a severe catarrhal angina, combined with those of stomatitis. Salivation is very profuse, and the breath usually foul. The lymphatic glands of the jaw are little or not at all swollen. There is little or no fever. The duration of the affection is from two to three weeks. The termination is always favorable.

Treatment.—Assiduous cleansing with chlorate of potassa (one part to thirty).

Ulcerous stomatitis and ulcerous angina, so-called thrush, have excited the greatest interest for several decennia, partly on account of their relation to scurvy and partly on account of their being often confounded with diphtheritis. I have discussed the subject briefly in this place because I have observed the above-described marked participation of the soft palate in many cases treated in the polyclinic. I have not observed it epidemically or endemically. It also appears to be absent in our barracks.

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[1] Consult the manuals of Diseases of Children and the work of *Billard*. Further, *Bohn*, l. c. p. 67.—*Gerhardt*, Lehrbuch der Kinderkrankheiten. 1871. 2 Aufl. p. 368.

Consult the well-known works of *Guersent*, Dict. de méd. 1827.—*Taupin*, Journ. des conn. méd.-chir. April, 1839.—*Rilliet-Barthez*, l. c. I. p. 197.—Especially *Bergeron*, De la stomatite ulcéreuse des soldats et de son identité avec la stomatite des enfans, dite couenneuse, diphthéritique, ulcero-membraneuse. 1859.—*Roger*, l'Union. 1859. No. 54.—*Bohn*, l. c. p. 97.—*Hirsch*, Hdb. d. hist.-geograph. Path. 1862. II.

d. *Angina mycotica, thrush of the palate.* Thrush (aphthæ) of the cavity of the mouth, in severe grades, almost regularly attacks the anterior portion of the soft palate, occasionally passing over the greater part of it, the uvula, the contiguous portions of the palatine arches, and the pharynx. Sometimes it forms elevated patches from the size of a millet-seed to that of a lentil, sometimes a tolerably uniform, whitish, milky-looking adherent layer, which is interrupted for the most part by red points (mouths of mucous glands). In the masses, which are readily removable, even without hemorrhage when care is exercised, we find the cylindrical and dendritic fibres, individual spores and aggregations of spores of the *oïdium albicans*, lying in greater part upon the epithelium, but in part distributed through it. The remaining mucous membrane of the palate exhibits the characters of a mild catarrhal angina. The local symptoms are also of the same character. For all further particulars, see Thrush of the Mouth.

In a case of true dysentery I saw an example of thrush affecting the palate especially, and in its entire extent, the mucous membrane of the mouth being involved in a much slighter degree. The patient recovered from the thrush, despite the severity of the intestinal affection, in the course of twelve days.

Concerning the histological relations in reference to the epithelium and the mucous membrane, see *Wagner*, Jahrb. f. Kinderk. N. F. 1868. I. p. 58.

Nothing positive is known concerning *other mycotic anginas*. The micrococci, etc., occurring in healthy persons, on different portions of the mucous membrane of the mouth, the leptothrix, do not appear on the palate of healthy persons; but they occur frequently in pharyngeal affections of every kind, especially when the superficial surface of the epithelium is not engaged in the process of constant renewal, and when the secretion of the mucous glands is diminished.

Fraenkel (Sitz. d. Berl. med. Ges. vom 29. Jan. 1873) describes a benign mycosa of the pharynx in a student of medicine. White, discrete elevations, as much as 1 mm. in height and as large as the underlying glands, were seen upon the tonsils and upon the glands at the base of the tongue. They did not simulate firm membrane, but were more like mould. When removed, they promptly recurred. They consisted of epithelium and many lively, moving micrococci, and of numerous partly moving rods of various lengths.

7. CROUPOUS AND DIPHTHERITIC INFLAMMATIONS OF THE SOFT PALATE. ANGINA CROUPOSA ET DIPHTHERITICA. ANGINA MALIGNA. PHARYNGEAL DIPHTHERITIS. DIPHTHERITIS.

The literature of pharyngeal diphtheritis is unusually extensive. I give below only the most important works on the disease in general, and especially its clinical relations. Treatises on the other aspects of the disease will be referred to later. Numerous unimportant works, such as ordinary descriptions of epidemics and endemics, recommendations of remedies, etc., I leave out altogether. In like manner, I refer to their appropriate chapters the relations of the accompanying affections of other organs, such as the larynx, kidneys, and nervous system.

The older literature, especially the Italian, Spanish, French, and English of the seventeenth and eighteenth centuries, is detailed in the Dict. encycl. des sc. méd. by *Dechambre*, 1866. V. p. 1. 45. The most important are: *Ghisi*, Istoria delle angine epidemiche dell' anni. 1747 and 1748. Cremona. 1749.—*Samuel Bard*, Researches on the nature, etc., of sore throat. New York. 1771.

Among recent works the most important is that of *Brétonneau*, Des inflammations spéciales du tissu muquex, et en particulier de la diphthérite, ou inflammation pelliculaire. 1826.—Further, in Arch gén. de méd. 1855. E. V. p. 1. VI. p. 257.—*Guersant*, Art. Angine couenneuse, in Dict. en XXI. Vol. 1821. II. and in Dict. en XXX. Vol. 1833. III.—*Becquerel*, Gaz. méd. 1843.—*Empis*, Arch. gén. Feb. and March, 1850.—*Isambert*, Arch. gén. E. 1857. IX. pp. 325 and 432.—*Trousseau*, Clin. Méd. 1861. I.

Good clinical German works which treat of pharyngeal diphtheritis in general do not exist (except the short descriptions in works on special pathology and on diseases of children). In special respects recommendable are *Bartels*, Arch. f. klin. Med. 1867. II. p. 367.—*Wertheimber*, Die Schlunddiphtherie. 1870.—Consult also the compilations of *Jaffé* in Schmidt's Jahrbb. since 1862: CXIII. p. 97; CXIX. p. 236; CXLIX. pp. 217 and 321.—Further, *Roser*, Arch. d. Heilk. 1869. X. pp. 103, 201, 303, and 366.

Croupous and diphtheritic angina are characterized by the appearance, always in an acute manner, under mild or severe local and general symptoms, of peculiar whitish elevated patches on the surface of the soft palate—so-called deposits, of varying

extent, which disappear after a duration of several days, but sometimes reappear upon the same spot. If the tissue beneath the deposit is only hyperæmic and infiltrated with serum, and the lymphatic glands of the jaw are not at all or only a little swollen, the angina is of a *croupous* nature. If, on the contrary, the tissue beneath the (slight, or, for the most part, dense) deposit is very hyperæmic, or at the same time hemorrhagic and infiltrated with sero-purulent matter, and if the lymphatic glands of the jaw are considerably swollen, then the angina is *diphtheritic*. Even in the latter class of cases, the tissues may return to their normal condition after two or more weeks' continuance of the disease. Or they may become gangrenous, involving the palate, especially the tonsils, as well as the lymphatic glands—so-called *gangrenous diphtheritis*. The three forms mentioned run into one another in various ways, in their local manifestations as well as in the participation of the system in general.

I have wavered for a long time as to whether I should take up pharyngeal diphtheritis in the description of the diseases of the pharynx, inasmuch as it has already had a detailed consideration in the first volume of this cyclopædia. I have finally decided to take it up, however, chiefly for the sake of completeness. I could not bring myself to give up writing about that very disease of the pharynx, which has attracted the most general professional interest for the last decade. Furthermore, I take up the subject of diphtheritis because I have followed it with interest since its first appearance in Leipsic, and have closely studied it, at least from one point of view. Finally, I have included it because I differ essentially from Oertel (Vol. I.) in several theoretical points.

My description, which is to follow, will be more a picture of diphtheritis at Leipsic, than a general delineation of that disease. On this account I have failed to give an extended review of the literature of the subject as well as an exhaustive consideration of many etiological and general pathological questions. My experience is based upon numerous mild cases, and upon about one hundred fatal cases, for the most part severe, and principally fatal by croup of the air-passages. These were either seen by myself or by Prof. Wunderlich, who has very kindly placed his clinical notes at my disposal. Of these I have made a complete dissection in forty-three instances, and a dissection limited to throat and thorax upon about as many more. All these fatal cases were cases of pure primary pharyngeal diphtheritis and not of scarlatinous diphtheritis. I saw the first fatal case in September, 1856; the second in October, 1861. Cases of the disease and of death from it have become more frequent only since the autumn of 1862, and since this period only has the

disease been known here as pharyngeal diphtheritis. Of the three physicians engaged in the hospital service during the years from 1860 to 1870, each one has to mourn the loss of one, and one of them of two of their own children from diphtheritis. Fatal croup of the air-passages without an affection of the pharynx has occurred much less frequently since that time. I have seen, and in fact dissected, only three cases; while, of the above-mentioned forty-three cases, thirty-nine died essentially from the croup of the air-passages; it was absent in four cases only. The condition of things is similar in Dresden, and also, according to West, in London.

The *deposit* furnishes the most important diagnostic feature in croupous-diphtheritic angina. It shows itself in patches of various sizes, dead-white at the beginning (of a dull silvery lustre, parchment-like, lardaceous), roundish, closely adherent to the subjacent tissue in the early days of the disease, and detachable, for the most part, only at the expense of a hemorrhage. These patches increase in thickness and in area in a few hours, or a few days, and distinctly project beyond the adjacent surface. The latter, as well as the underlying mucous membrane, is dark-red in color. The patches are either sharply circumscribed, or they gradually merge, throughout their circumference or at certain points, into the contiguous tissues; in the former instance they do not enlarge in area any more, while in the latter instance they continue to increase.

The *recognition of the deposit* is easy in the great majority of cases, if not at the first examination of the patient, at least upon the second, undertaken after some twelve or twenty-four hours. Its recognition is difficult in cases where the deposit lies upon the posterior surface of the palate, or when the surface is covered with mucus, or fluid food (especially milk), and cannot be effectually cleansed, and where cauterization of the surface has already been made with acids, but more especially with nitrate of silver. The deposit is confounded most frequently with the whitish plugs which project out of the normal or enlarged lacunæ of the tonsils, as well as with the whitish or yellowish, somewhat raised patches which occupy the interior of the lacunæ without projecting through their (narrowed or closed) outlets. In both classes of cases the questionable patches occupy only the surface of the tonsils, not the uvula, soft palate, etc.; their size and distance apart correspond with the lacunæ; the first-named

plugs project too much, for the most part, with but a slight extent of surface, and those last mentioned come distinctly nearer the surface after half a day or a day. Less frequently a croupous deposit is confounded with suppuration of the epithelium, as in catarrhal angina, ulcerous angina, etc. (see p. 891), and in variola of the soft palate (see p. 902); or with shallow syphilitic ulcerations, beneath which the mucous membrane is anæmic from cellular infiltration; or with thrush of the soft palate (see p. 923); or with gangrenous processes (which see).

Croupous-diphtheritic angina occurs, with many similar characteristics, anatomically and clinically, under four different conditions.

1. As a *primary disease*, the so-called true pharyngeal diphtheritis in healthy persons, less frequently in persons otherwise diseased.

2. As a *complication of scarlatina*, the so-called scarlatinous pharyngeal diphtheritis.

3. As a *secondary disease* in the other acute exanthemata (measles and small-pox), in the acute infectious diseases (typhus, cholera, pyæmia, puerperal fever), in various chronic diseases (tuberculosis).

4. As a so-called *non-specific angina*, with croupous exudation.

Pathological Anatomy.

Croupous angina is characterized especially by the deposits described. These form smaller or larger patches, or occupy a larger surface. In the latter instance the entire surface of the soft palate, including the uvula and the tonsils, may become covered with the deposit. Not unfrequently it is simultaneously found at the root of the tongue, upon the contiguous portions of the mouth, and upon the pharynx, nose, and larynx. (These do not come under consideration just now.) The deposits, in a recent condition, are white; they have a dull lustre, and are of varying thickness up to $\frac{8}{100}$ of an inch, sharply bordered, firmly adherent to the subjacent tissue at first, and elastic. After a continuance of several days they become gray, even blackish,

turbid, less adherent to the subjacent tissue, and also somewhat less elastic.

Microscopically, the fresh deposits consist of a clear, homogeneous, glistening network of varying thickness, the interspaces of which contain serum, or blood or pus corpuscles. The interspaces are for the most part very small; only here and there are they larger, even microscopic. Sometimes the latter is the case in the entire deeper half of the deposit. In the beginning the uppermost, flattened epithelium is still present upon the deposit; but after a few days' continuance it will have disappeared. The lowermost epithelial layers are always still present in the beginning, and sometimes remain throughout the entire disease.

The croupous network occupies the place of the epithelium, and never extends into the mucosa; but it extends to various depths into the lacunæ of the tonsils, though very seldom into the subepithelial portion of the excretory ducts of the mucous glands; even the portions of the excretory ducts lying in the epithelium seldom participate in the formation of the network. The mucous membrane beneath the croup membrane is in uncomplicated cases at first markedly hyperæmic, less so at a later date—sometimes dotted with variously numerous hemorrhages, for the most part considerably infiltrated with serum, sometimes with sero-pus. Its lymphatic vessels are mostly considerably dilated and filled with serum, or—and more frequently—with fibrine and pus corpuscles.

Many different views are held concerning the nature of the deposit—the croupous or diphtheritic membrane. Most authorities regard it as a fibrinous exudation, which, according to many, is located upon the epithelium, according to others beneath it, and according to others, again, replaces it. Buhl (Verh. d. Bayr. Akad. 1863. II. p. 59. Zeitschr. f. Biol. 1867. III. p. 341) and myself (Arch. d. Heilk. 1866. VII. p. 481) regard it as a product of the epithelium. According to my views, the epithelial cells swell; their protoplasm contains interspaces in numerous different places, which become filled with serum or pus corpuscles, etc., while the remaining portions become particularly clear, glistening, resistant, etc. This metamorphosis does not take place in the uppermost flattened epithelial cells,

and ordinarily the lowermost ones do not take part in it either. It begins in the upper half of the middle layers of epithelium, and appears to extend thence more rapidly upwards than downwards. In consequence of the metamorphosis mentioned, and of the swelling of the underlying mucous membranes, the affected places project comparatively far, and sometimes, fungus-like, above the surrounding tissue.

Not unfrequently globular bodies, which consist of finely punctated masses of fungus, are found in one or more layers, upon the upper surface of the croup membrane, or, when the uppermost flattened layer of epithelium is still preserved, above this, and later in spots also between the latter and the croupous reticulum. The deep boundary of the croup membrane sometimes consists of one or more layers of not materially altered epithelium, and sometimes of the uppermost layer of the mucosa. (The contrary view that the croup membrane reaches into the tissue of the mucosa itself, I have never found confirmed.)

The detachment of the croup membrane occurs after several days' continuance. Either the upper portion alone is thrown off, the membrane splitting in thickness, so to speak, or it is thrown off in its entire thickness. The first method occurs by the appearance in the thickness of the membrane of large spaces filled with serum, by means of which the network is drawn out into fine threads and finally torn. The latter method occurs by arrest of the metamorphosis at the lowermost layers of epithelium, and in the mildest cases by the continual growth of new epithelium. In cases less mild, a penetration of serum takes place between the membrane and the still remaining epithelium or the upper surface of the mucosa. Furthermore, the croup membrane is loosened from its connection with the tissue beneath it, occasionally by the swelling of the latter and the enlargement of its superficial surface, but generally by reduction of its swollen condition and diminution of its surface. In both instances rents occur not unfrequently, forming spaces in the croup membrane, which frequently contain at the same time the above-mentioned fungus globules.

The mucous glands do not have any essential relation to the

croup membrane. The latter usually comports itself in essentially the same manner in places where these glands are very numerous (the uvula, etc.), and where they are entirely absent (the tonsils). Either the epithelial portion of the excretory duct of the mucous glands (and unfrequently their subepithelial portion also) is at the same time croupous, and thus more difficult to recognize, or it is not altered, or it is even somewhat dilated. Sometimes the croup membrane (less frequently in the palate, often in the larynx and trachea) then exhibits regularly distributed round holes, empty or filled with mucus. These are the excretory orifices lying in the epithelium. Those portions lying in the mucosa are frequently dilated and filled with mucus. The follicles of the glands are normal.

Croupous-diphtheritic, or so-called *purely diphtheritic angina* is characterized by the same croup membrane, sometimes remarkably thick, and often extended over a large surface; while the mucosa, in severe cases the submucosa also, the interacinous tissue of the mucous glands, and the intermuscular connective tissue, exhibit a considerable sero-purulent or purely purulent, or thick cellular infiltration, in addition to the hyperæmia and the frequent and extensive hemorrhages ordinarily present. It is worthy of remark that this cellular deposit reaches much further on the surface, in most instances, than the croup membrane, so that sometimes the most posterior portion of the root of the tongue is densely infiltrated with cells, while that region exhibits but little alteration in its epithelium, or even none at all.

In the above description I have, like most authors, designated as diphtheritis the deposition of cellular elements in the tissue of the mucous membrane, etc., although the word diphtheria signifies what we now ordinarily mean by croup membrane. I follow in this only the present habitual use of the word diphtheritis, in which its true meaning (skin, membranous) has gradually been lost.

Fungus-globules, such as are found in croup, and ordinary micrococci occur much less frequently in the tissue of the mucous membrane and the deeper structures than in croup membrane. I have never been able, in spite of frequent investigations in that direction, to confirm the opposite views on this subject announced by some authors, from Tommasi and Hueter to Eberth. (For further particulars, see below.)

The *lymphatic glands of the jaw*, in special cases the lym-

phatic glands of the entire side of the neck, especially along the external jugular vein, behind and beneath the upper half of the sterno-cleido-mastoid muscle, as well as the glands at the back part of the neck, are always enlarged, sometimes to from five to ten times their normal volume. They are soft, deeply congested, homogeneous upon section, at first grayish or dark-red, later grayish-yellow in places. They exhibit the character of a high grade of hyperæmia, hemorrhages in places, and considerable proliferation of cells; small gangrenous foci are sometimes found in their central portions. The *spleen* is usually normal; occasionally it is acutely swollen.—The *solitary follicles* of the small and large intestines are often moderately swollen (Rud. Maier, Arch d. Heilk. VI. p. 171).—The *liver* is for the most part normal to the unaided eye, but sometimes contains the smallest lymphatic new formations.

Consult the remarkable case of numerous lymphomata of various organs, described by Roth (Virchow's Arch. LIV. p. 254).

Gangrenous diphtheritic angina exhibits the same croupous deposit upon the surface, while the underlying portions undergo the already described hemorrhagic, or purulent, or dense cellular infiltration in a high degree, and show gangrenous degeneration in places, most frequently starting from the tonsils. The same alterations occur in isolated lymphatic glands.

For the remaining pathologico-anatomical relations, see further on.

Clinical Considerations.

A. *Primary pharyngeal diphtheritis* occurs in several forms, whose extremes can be well defined, while there are many intermediate forms. The deposits described are common to all forms, while the affection of the lymphatic glands, the extension to the pharynx, nose, larynx, trachea, and bronchi, the participation of the kidneys, the nervous system, the remaining organs, and the general system, as well as the sequelæ, are present in various degrees of intensity, or fail altogether. Our justification in considering these various forms as different varieties of one main

affection lies not only in their essentially similar pathologico-anatomical and histological relations, not only in many clinical peculiarities, but more particularly also in their etiology. Every practising physician knows of examples where several individuals of the same family have become affected with croupous angina at the same time, or within a short period, say within a few days of each other—one or more in a severe form, the rest of them in milder forms. The former may die, while the majority of the latter recover promptly and permanently, with the exception, perhaps, of one, in whom severe sequelæ became established. Instances are still more frequent in which the mother or nurse of a diphtheritic patient (both in primary and in scarlatinous diphtheritis) becomes attacked a few days afterwards with a croupous angina, usually of a mild character.

Cases of so-called diphtheria, without a diphtheritic membrane, are tolerably infrequent. In families where several members are affected with severe or mild croupous angina, one member sometimes shows only an intense hyperæmia of the palate, without any discernible deposit, but, in spite of this, may exhibit later the sequelæ of diphtheritis.

a. *The Milder Forms of Pharyngeal Diphtheritis; Croupous Angina; Pharyngeal Croup.*

The surface of one tonsil, less frequently of both, from the commencement shows several deposits as large as lentils, which increase in thickness and size from one twelve hours to the next, and usually run together in such a manner that the entire tonsil becomes covered. Only unfrequently does one tonsil alone remain affected; usually the other becomes covered also on the second day, or, less frequently, on the fourth or fifth day. In the great majority of cases the deposit remains limited to the surface of the tonsil only. In another series of cases it extends from the tonsil upon the contiguous portions of the palatine arches—in some upon the lateral portions of the uvula also. In all the last-named localities the deposit ordinarily remains small in extent, and is usually circumscribed from the commencement.

Beneath and beside the deposit the mucous membrane is usually strongly injected, dark-red, and moderately swollen. The arches of the palate, as well as the uvula, are sometimes highly œdematous, even without the presence of the deposit. The tonsils swell beneath the deposit usually to double or quadruple their normal volume (parenchymatous tonsillitis: see p. 913), sometimes to such an extent that they flatten the uvula upon both sides. Acute catarrh exists in the posterior portion of the mouth and in the pharynx. The lymphatic glands of the jaw are somewhat swollen in almost every instance, but not to a great degree.

The deposit is usually distinctly visible on the first day of the disease. Where this is not the case, it either occupies a position unfavorable to vision (behind the palate, or in the depth of a tonsillar furrow), or it is not yet thick enough to admit of its recognition upon the hyperæmic substratum. It frequently spreads for several days from the localities first affected, or else new deposits take place independently of the old ones. All this occurs more promptly, on the whole, in younger individuals. After from three to four days, frequently not till after six or eight, and sometimes not till after from ten to fifteen days, the deposits become loosened and are swallowed with the drinks or expectorated; those of the posterior surface of the palate may even be sneezed out. The portions of mucous membrane upon which they sat exhibit no macroscopic alteration in the great majority of cases, or they may appear somewhat thinner, and, very seldom, superficially eroded.

The *local disturbances* of the patient are either present from the beginning, or they appear for the first time after the existence of general symptoms of several hours or even of one or two days' duration. They are not distinguishable from those of a moderate grade of catarrhal angina, consisting in difficulty of swallowing, somewhat nasal speech, slight spontaneous pains, and moderately severe pains on pressure and movement, salivation, sometimes impeded nasal respiration, sometimes severe aural pains. In some cases local disturbances are altogether absent.

The *general symptoms* are very various. They are so slight sometimes that the patient continues his avocation during the

entire duration of the disease, or during the first few days of its course; children even continue to frequent their schools, and are with difficulty confined to bed. On the other hand, there is sometimes a severe sense of illness from the commencement.

Among the special manifestations, the *fever* is neither typical nor does it bear any proportion to the extent, etc., of the deposit nor to the hyperæmia, etc., of the mucous membrane. On the first day of the disease there is usually a moderately high temperature, or it may be quite high from the beginning, from 38.5° to 40° C. (101.3° to 104° F.). This temperature either increases during the following days, say to 41° C. (105.8° F.), or it remains without rising any further, or it decreases by from 1° to 2° C., principally in the mornings. It presents only moderate evening exacerbations. From the fourth to the sixth day, sometimes earlier, the temperature usually declines, sometimes from 41° C. to 38° C. (105.8° to 100.4° Fahr.), within one or two days, and the patient is usually free from fever a few days before the disappearance of the deposit. Only very seldom is there a total absence of fever. The *pulse* exhibits moderate acceleration, proportionate in general to the temperature. In some cases it is unusually high in the first few days, from 120 to 140 in adults, and after a few days it sinks to its normal frequence or even below it. The *respiration* sometimes corresponds to the frequence of the pulse, and sometimes it is somewhat in excess.

The remaining general manifestations depend partly upon the local affection and partly upon the fever. In most instances there is more or less, sometimes excessive, *languor* and debility, indisposition to work or to play, either from the very commencement or after a few days' sickness. *Headache*, or discomfort in the head, usually of a moderate degree, general or confined to the brow, is frequently complained of by adults. This exists from the beginning or appears only after a few hours or a few days. With it, the countenance is seldom flushed, but rather pallid, the latter sometimes despite strong pulsation of the carotids. The *alimentary organs* are usually markedly affected: the tongue is coated; the appetite is usually much diminished; there is slight thirst; seldom vomiting; constipation is more

frequent than diarrhœa. The urine is almost always normal. Herpes of the lip or the nose is sometimes found to exist with the commencement of the disease, occasionally in children, more frequently in adults.

The *onset of the disease* exhibits remarkable differences in small children on the one hand, and in larger children and adults on the other. In the latter class of patients, in the majority of cases, there is moderate pain in the throat and difficulty in swallowing, bilateral more frequently than unilateral, coming on most frequently early in the morning, after awakening, or upon getting up—less frequently in the evening, and least frequently during the course of the day or of the night. Sometimes there is only pain at the angle of the lower jaw. Chilly sensations are frequent, downright chills rare. Unfrequently the last two symptoms are present first, the difficulty of swallowing appearing several hours later. Sometimes there is only a general feeling of discomfort, languor, etc., or only headache, or gastric disturbances may be most prominent—loss of appetite, nausea, even vomiting. The same initial symptoms sometimes occur in young children, as far as they can be recognized; or there is moderate or severe aural pain, usually unilateral; or the disease may be recognizable only by its local manifestations.

The *course of the disease* usually exhibits no material fluctuations. The difficulty of swallowing and the remaining subjective disturbances increase in the following days, so that not unfrequently professional assistance is not solicited until the third day. With the subsidence of the hyperæmia and swelling of the mucous membrane the local disturbances diminish, although small deposits may still be present.

The disease *terminates*, in ordinary favorable cases, in from five to fourteen days—most frequently at about the end of a week. Convalescence is usually rapid, but it is sometimes slow in children.

The *complications* which appear during the course of the disease are not very frequent, but they are of importance, because the danger in so-called mild diphtheritis is principally due to them. They are: extension of the deposit into the larynx, etc., diseases of the kidneys, very rarely pneumonia, etc. As a

consecutive disease, paralysis of the palate, etc., occurs, but is likewise infrequent.

b. *The Severe Forms of Pharyngeal Diphtheritis; Diphtheritic Angina; Diphtheritic Pharyngeal Croup.*

The surface of one tonsil, seldom of both tonsils in the beginning, becomes covered, in from twelve to twenty-four hours, with a uniform deposit; or deposits, small and scattered at the beginning, coalesce within a similar period. The deposit, however, does not remain limited to the tonsils, but extends, in one or a few days, upon the arches of the palate, (the anterior arches more frequently), upon the anterior and posterior surfaces of the soft palate, and upon the uvula—sometimes in the form of small isolated patches the size of a lentil and larger, sometimes as a uniform covering over the parts mentioned in their entire extent. The thickness of the deposit, and the marked infiltration of the parts can be determined, in the living subject, by the size of the tonsils and the uvula, and the thickness of the border of the palatine arches. The mucous membrane beneath the deposit, and alongside of it, is intensely reddened, and frequently marked with slight hemorrhages. The infiltration of this membrane is of a sero-purulent nature, or composed of small cells compactly arranged; that of the tonsils consists in a cellular hypertrophy. The swelling of these parts materially narrows the passage between the arches of the palate; this narrowing may be symmetrical, or one-sided, if there is unequal swelling of the tonsils. Copious amounts of turbid mucus, frequently commingled with blood, are found on all these parts, unless the patient has recently swallowed, or rinsed out his mouth.

If the opportunity is presented of examining the palate before the appearance of the deposit, the mucous membrane is seen to be darkly reddened, moderately swollen, and dry. After a number of hours, its surface appears peculiarly gray, in places or in its entire extent. The deposit is still very thin, and the hyperæmic mucous membrane beneath it masks its color. A few hours later it has acquired its whitish aspect, and is distinctly raised above the contiguous surface.

The deposit sometimes extends from the parts named a certain distance on to the mucous membrane of the back of the mouth, more frequently to the root of the tongue, and, furthermore, upon the lateral, upper and posterior walls of the pharynx, and upon the inferior and posterior portions of the nasal passages. At all these localities it is sometimes in patches, sometimes uniformly distributed.

Considerable *swelling of the lymphatic glands of the jaw* is present in all cases, usually from the second day of the disease. This is at first unilateral, or greater on one side than upon the other, but it seldom continues so during the entire disease. The swelling of the lymphatic glands is a tolerably safe measure of the extent of the process in the tissue of the mucous membrane and of the tonsils, especially when a quiet inspection of the palate is impossible, as in the case of small children, or when it is not known whether the swelling of the tonsils appeared in connection with the present disease or existed previously. Here, too, as in most diseases of the lymphatic glands, many individual differences are observed. The swelling of the lymphatic glands appears earlier, and is more pronounced on the side on which the affection is most severe than on the other. The lymphatic glands may become as large as cherries or larger; they are distinctly circumscribable at the beginning, but their contour sometimes becomes indistinct at a later period. In the most severe cases the skin on the upper portion of the side of the neck is œdematous, and frequently slightly hyperæmic.

Sometimes all the lymphatic glands of the upper and middle portion of the neck are swollen, especially in pharyngeal diphtheria, and when the attack is very severe the skin of the corresponding region is œdematous and hyperæmic.

The *deposits undergo alterations* during the next few days. Besides the increase in surface already mentioned, and which is mostly over after three or four days, there is also an increase in thickness, which is especially distinguishable at the edges of the deposit, and in the increased thickness of the deposit upon the uvula. Furthermore, it becomes dirty white, yellowish or brownish, from imbibition of food and medicaments, soot, etc. Unfrequently it is dirty red from the beginning, from

hemorrhage into the membrane and into the underlying mucous membrane. The elasticity present at first is lost, and the deposit becomes brittle. From this cause, and from the diminution in the swelling of the mucous membrane, its lustre is lost. Isolated portions of the membrane become detached from the fourth to the sixth day—sometimes only superficially, in instances in which an increase occurs in the thickness of the deposit; sometimes in its entire thickness.

The deposit exhibits certain characteristics according to its locality—whether observed *in situ* on the living subject, or only after its detachment. The uvula is sometimes enveloped by a pseudo-membrane in its entire extent, like a sort of glove-finger or thimble—the latter form being then still easily recognized in the expectoration. The deposit upon the uvula, the soft palate, and the anterior palatine arches is sometimes perforated with regular holes, which are the unaltered but dilated mouths of the excretory ducts of the mucous glands—those on the posterior surface of the palate being sometimes ribbed longitudinally. The deposits on the tonsils, which are usually expectorated in fragments, not *in toto*, sometimes exhibit prominences of varying thickness on one surface—the under surface; they are the adherent deposits of the lacunæ. (Similar appearances exist on the pseudo-membranes from the turbinated bones, the pharynx, larynx, trachea, and bronchi.)

The hyperæmia and swelling of the mucous membrane usually become distinctly diminished previous to the removal of the deposit. After this, the affected portions of the mucous membrane appear less hyperæmic and swollen, without loss of substance, though occasionally with shallow erosions. The mucous membrane either acquires its normal condition in a few days, or it remains hyperæmic and infiltrated a longer time. In the latter case new deposits often form after a day or two, usually thinner than the previous ones, and more promptly detached. This may recur several times on the same spot, or on different spots, usually small in extent, and in infrequent isolated cases may be repeated for from four to six weeks.

Shortly before the detachment of the deposit, and soon after it, the tonsils, in particular, not unfrequently appear to be prac-

tically destroyed, either superficially or more deeply. On the following day it is seen that this was not the case; either the deposit was located upon a spot previously atrophied, or the sur rounding tissues were still considerably swollen.

The *local disturbances* are the same as those of a moderate or severe grade of catarrhal angina. They are slight in some cases; severer in some; very severe in others. They depend upon the thickness of the deposit and the amount of hyperæmia, as well as upon the swelling and mobility of the palate, and upon the size of the tonsils. They usually increase continuously during the first few days, and are most severe from the third to the fourth day. Difficulty of swallowing is the most prominent symptom. This is especially troublesome, inasmuch as the copiously formed mucus in the mouth and pharynx, and the profusely secreted saliva, necessitate in part frequent hawking, and in part frequent deglutition. Sometimes the chief or the only difficulty experienced is connected with the swelling of the lymphatic glands. Speech exhibits the ordinary changes characteristic of anginous affections. The breath of the patient may be maintained odorless by constant cleansing of the mouth and pharynx. In other cases a foul, not unfrequently stinking, gangrenous odor is present, which, however, is promptly subdued by the assiduous employment of disinfecting remedies. The examination of the diseased parts is unsatisfactory, on account of the difficulty in opening the mouth.

The *general symptoms* are present much more regularly than in the lighter forms of diphtheritis. The *temperature* is very seldom normal; usually it is moderately increased, not unfrequently much increased, to 40° or 41° C. (from 104° to 105.8° Fahr). This affords, however, no estimate of the severity of the case, except when it continues at 104° and more for several days. In the latter instance, and also when morning remissions fail altogether for several days, the prognosis is bad. In some severe cases the coldness of face, hands, etc., is in contrast with the high temperature. The *pulse* is sometimes moderately, sometimes considerably accelerated; in unfrequent cases it is abnormally slow; frequently it is small and hard from the beginning. Its acceleration usually continues longer than the elevation of

temperature. *Respiration* is always moderately accelerated. Patients frequently assume a *half-sitting posture*, or they are frequently obliged to raise themselves, in order to hawk and to cough with more force. In lying down, especially if there is considerable swelling of the tonsils, or swelling of the nasal mucous membrane, etc., at the same time, the breathing is frequently impeded, noisy, and carried on through the open mouth. The masses of mucus moved in the pharynx by the respiratory current produce a sound audible over the thorax; so that auscultation of the chest does not furnish any safe conclusion as to the contents of the bronchi.

The *general strength* is but little diminished the first day or two; but from the third day on it usually becomes greatly diminished. Sitting up is found to be laborious. Sometimes the patients are remarkably *pallid* from the very first, or after a few days' sickness. The *cerebral functions* are normal; delirium is infrequent; ill-humor is frequent. Moderate headache as well as vigilance exists in most cases.

The *alimentary organs* almost always evince considerable disturbance of function. The tongue is usually thickly coated. Appetite frequently fails entirely; thirst is moderate. Sometimes there is vomiting; constipation is more frequent than diarrhœa. The *skin* is usually very dry. Sometimes there is *herpes* of the lips. The *urine* is scanty, dark-colored, and rich in salts; only the chlorides are diminished in most instances. In about one-third of the cases there is mild albuminuria; unfrequently, and mostly in severe cases, it is intense.

Thorner (Berl. klin. Wschr. 1869. No. 43) found, in several cases of severe diphtheria, an extraordinary diminution in the chlorides, the same as in pneumonia, without the previous imbibition of large quantities of water.

The *onset of the disease* is very varied, in part arbitrarily so, and in part according to the age of the patient. In some cases it is exactly like that in the milder forms—first, difficulty in swallowing, seldom severe from the beginning, etc. In other cases there are the indications of slight or severe illness for several hours or for a day, the difficulty of swallowing not setting in until from six to thirty-six hours later. Repeated rigors or

a chill may also be present. Or there may be vomiting, headache, and backache, vertigo, indistinctness of vision, and general languor. All these may exist to but a moderate degree, and are then usually attributed to some real or fancied error in diet, or they may be more severe, in both cases being associated with moderate or high fever. Or there may be disturbed sleep, sleeplessness and delirium. Or all the symptoms mentioned may occur almost simultaneously, sometimes as with one sudden blow. In little children the commencing symptoms are sometimes equally characteristic (convulsions); sometimes they are very unpronounced, similar in general to some febrile affection, or a severe coryza; or the child is peevish, has no desire to play, etc. In the sudden onset of the disease, the similarity with the onset of scarlatina is quite marked, inasmuch as an erythema is sometimes present in the beginning upon various portions of the body. Herpes of the lips, existing from the beginning, is tolerably rare; and still more infrequent are vesicular eruptions on other portions of the body, or hemorrhages of the skin.

The local disturbances usually follow the above-described general symptoms on the very first day of the disease. Inspection of the palate usually discloses this on the first day, occasionally not until the second. In addition to the high grade of hyperæmia and swelling, the deposit may be seen sometimes covering a considerable extent. The cases are very infrequent in which no evidence of the deposit is to be seen on the first or even on the second day. Either the deposit in those cases is really still wanting, or it is located upon some portion of the structure not visible without artificial aid (the posterior surfaces of the palate, palatine arches, and uvula), or the affection begins in the depths of the tonsillar lacunæ.

The *course of the disease* is tolerably uniform when the affection remains limited to the palate. In general the deposit extends more rapidly than in the mild form of the disease. The local symptoms and disturbances and the general affection increase day by day until towards the end of the first week, only the fever usually abates earlier. The symptoms mentioned then gradually subside, while the debility and anæmia usually continue somewhat longer.

Recovery takes place in the course of the second or third week. The redness and tumefaction of the palate become less, the deposit loosens and becomes detached, the swelling of the lymphatic glands likewise subsides. The fever decreases, the high temperature declining especially, or greater morning remissions set in, while the pulse sometimes still remains frequent. The skin becomes moist, the secretion of urine more copious. The swelling of the face decreases and its pallor is lost, the appetite returns, sleep sets in, the general languor gradually disappears. In occasional cases only the disease may continue a couple of months and more, in consequence of local disturbances; new deposits recur, usually diminishing in surface and thickness, ordinarily with progressively slighter local and general manifestations.

The *complications* arising during the course of the disease occur with varying frequency in different epidemics. They occur much oftener in severe than in mild pharyngeal diphtheritis. They consist sometimes in an extension of the croupous or croupous-diphtheritic processes upon the contiguous parts, sometimes in diseases of other organs, especially the kidneys, the nervous system, and the skin. They constitute almost the only danger of non-gangrenous diphtheritis.

The *extension* of the croupous, or the croupous-diphtheritic processes upon the contiguous structures, is very frequent.

Extension to the gums, the mucous membrane of the mouth generally, and the lips, and particularly to the root of the tongue (*stomatitis*, etc., *diphtheritica*), sometimes produces no local symptoms and sometimes only slight symptoms.

Extension into the *pharynx*, usually in a slight degree upon the lateral and superior walls, and in a severe degree upon the posterior walls, is very frequent; it increases the difficulty of swallowing and the general disturbance, without materially influencing the other symptoms. Extension to the *Eustachian tubes* begets pain in the ears. Extension into the *œsophagus*, and further upon the cardia and the stomach, occurs very seldom. I have only seen in two instances an extension of the deposit upon the uppermost portion of the œsophagus. West found the œsophagus free, although the stomach was covered with the pseudo-membrane.

In case of extension into the *nose*, there occasionally occurs a true croupous deposit, but more frequently a combination of croup and suppuration of the epithelium. The participation of the nose is usually painless. It is characterized less by impeded passage to the air than by a peculiar, usually thinly purulent, greenish discharge, excoriating the contiguous parts of the external nose, and on the first day not perceptibly different from an ordinary coryza. In some cases there is at the same time a moderate œdema of the upper part of the face, especially of the eyelids. Besides the nasal diphtheritis, there usually exists an intense palatal and pharyngeal diphtheritis. It is quite unusual to find the latter slight while the nasal affection is severe. It may, however, not be at all apparent, and then perhaps exists on the posterior surface of the palate. Occasionally pharyngeal diphtheritis commences upon the posterior surface of the palate and on the nose at the same time, and first extends upon the lateral and anterior surface of the palate on the second or third day, remaining slight at the latter location or extending considerably. From the nose the croupous inflammation may extend into one or both *lachrymal ducts*, and, still further, even upon the *conjunctiva*, in which case there will be overflow of tears, etc.[1]

The extension of croupous and diphtheritic angina upon the *larynx*, *trachea*, and *bronchi* forms the chief danger in the first-named affection. The disease of the air-passages is usually purely of a croupous nature, or there may be at the same time extensive infiltration of the mucous membrane of the larynx and upper portion of the trachea—the remaining portions showing evidences of ordinary croup. The extension of croup to the air-passages occurs oftener in children than in adults, and much more frequently in severe than in mild cases of pharyngeal diphtheritis. The relation as to time between the affection of the palate and that of the larynx is a varying one. In one series of cases, probably the majority of them, both affections appear at the same time, or the laryngeal affection follows hardly a day later.

[1] Consult *Hirschberg* (Berl. klin. Woch. 1869. No. 3) upon the etiology of conjunctival diphtheritis, and its connective relations with the diphtheritic affections of the pharynx and larynx.

These are the cases in which distinct evidences of laryngeal croup (long-drawn respiration, frequent dry coughs, toneless, hoarse voice, great disquiet, and pains in the throat) also occur from the second to the fourth day, and symptoms of stenosis become manifest from the fourth to the sixth day—the latter first appearing, in most instances, on the partial detachment of the croup membrane. In a further series of cases, the symptoms of laryngeal croup first make their appearance at the end of the first week, occasionally still later, even towards the middle of the third week.

The period at which croup of the air-passages becomes added to pharyngeal diphtheritis is generally misjudged. This is due to the circumstances that the patients are already suffering from dyspnœa, due to the angina itself, especially in case of simultaneous collateral œdema of the ary-epiglottic ligaments—that movements of the body do not take place to account for the dyspnœa, and that usually, also, the speech is feeble from the pain, etc. Therefore the laryngeal affection is most frequently not recognized, until, upon more or less forcible examination of the child's pharynx, it holds its breath for a long time and then screams. In adults and in older children the croupous affection of the air-passages is attended by much slighter symptoms than in young children. In the case of one of my assistants, Dr. O. B., who died on the fourth day of the disease, pharyngeal diphtheritis of a severe grade existed; the croup of the air-passages extended to the third bronchial division, and, in places, to the fourth division, without any decisive symptoms of the condition having existed during life. In general, if the deposit on the palate is considerable, the croup of the larynx is considerable also; but in many slight cases of the latter condition a diagnosis cannot be made with certainty. But the reverse condition occurs also; slight affection of the pharynx and intense affection of the larynx being combined. The opinion of some, that primary laryngeal croup never occurs, is erroneous, according to the observations made here. Since the domestication of pharyngeal croup, the number of such cases has at any rate diminished. (See p. 925.)

Pneumonia, as well in the form of lobular (catarrhal) as in that of lobar (croupous) inflammation, occurs usually only in connection with croup of the air-passages. I have seen two fatal cases, verified by the post-mortem examination, as well as several cases which were not fatal, in all of which pneumonia existed in the lower lobes, without simultaneous evidence of croup of the air-passages.

The *lymphatic glands* of the neck, and especially those of

the jaw, diminish in size with the subsidence of the pharyngeal affection, and usually become normal a few days after; not unfrequently, however, they remain enlarged to a slight extent for several weeks. Suppuration occurs only occasionally in spots, and gangrene still less frequently.

Albuminuria seldom occurs in slight cases, but occurs in about half the number of severe cases. It is not, however, proportionate to the severity of the disease. It is seldom encountered from the commencement—usually not until after several days, sometimes not until towards the termination of the disease. It is usually slight, continuing but a few days, or irregularly intermittent, so that, in the same case, with a duration of two weeks' sickness, the urine may contain from one-fortieth to one-fourth of its volume of albumen at one time, and may then be free from albumen for a day or for several days. Frequently the urine contains a few pale tube casts. In cases where the albuminuria has existed, and also where it has not existed, hyperæmia, enlargement, and diminished consistence of the kidneys will be found on post-mortem examination, and microscopically a strongly albuminous or partially adipose turbidity of the epithelium of the kidneys.[1]

Consecutive dropsy occurs very unfrequently: Demme (Jahrb. f. Kinderh. N. F. I. 1868. p. 21) saw seven cases, four of them fatal, among forty-two cases of primary non-scarlatinous diphtheria. I have never observed it here.

c. *The Gangrenous or Septic Form of Croupous-Diphtheritic Angina; Angina Gangrænosa.*

We find here *locally* the same deposits as in the form already described, the same or still more decided hyperæmia and infiltration of the mucous membrane, etc., and the same rapid enlargement of the lymphatic glands. The process usually grows more quickly in thickness and surface, on the tonsils, and rapidly extends upon the pharynx and into the nasal passages, sometimes also upon the posterior portions of the mouth and upon the gums. On the first and second day, the local appear-

[1] Consult *Bartels*, Mitth. d. Ver. schlesw.-holst. Aerzte. 1873.

ance does not differ essentially from that of the forms previously described. Several differences, however, make their appearance after the third or the fourth day. The diseased surfaces of the palate and nose bleed either spontaneously or upon very slight provocation—at first moderately, later sometimes profusely; or there appears an apparent, much more unfrequently an actual, superficial, or deep gangrene of the affected places, especially of the tonsils. At the same time the swelling of the lymphatic glands becomes constantly greater, and the overlying skin, as well as the adjoining skin to a considerable distance, is frequently hyperæmic, and, to a variable degree, œdematous.

I have not myself seen actual destruction of the tissues of the palate in primary diphtheritis, but only in scarlatinous diphtheritis. From the descriptions of most authors it appears that they have not observed actual gangrene upon the corpse, but have taken this for granted from the presence of gray shreds similar to dead tissue, and from the odor. (See also West.)

According to Millard (Sur la trachéotomie, 1858) and Peter (l. c.) the blood, in gangrenous diphtheritis, is brown, similar to prune-juice, or liquorice-juice, and leaves marks on the fingers like ink. It is turbid, viscid, and readily absorbed; the coagula are not firm.

The secretion flowing from mouth and nose is grayish-yellow, frequently mixed with blood, at first still purulent, later ichorous. It first reddens and then corrodes the skin of the lips and nostrils. At the same time there is frequently a gangrenous or cadaverous odor in the entire atmosphere surrounding the patient. This, however, is no indication of gangrenous destruction, for the proper cleanliness of the diseased parts may be impossible for various reasons.

The *local disturbances* are either the same at first as in the previous form, or they are remarkably slight from the beginning. In the former case they diminish with the appearance of the gangrenous manifestations.

The *general symptoms* are particularly characteristic. The *temperature* is usually high, amounting to 40° and 41° C. (104°–105.8°). At the same time the head, hands, and feet are frequently *cold*. The *pulse* is always much accelerated, small, empty, not unfrequently irregular. The *respiration* is usually much hurried, even without the larynx or the lungs being affect-

ed (in consequence partly of the difficulty in expectorating the mucus that accumulates in the upper part of the larynx, and partly from the swelling of the upper vocal cords—ventricular bands). The patient is remarkably *pallid;* the face is sunken, especially the eyes—this condition contrasting strongly with the swelling of the neck. The entire carriage of the patient is *apathetic* and indolent, so that even those usually ill-natured make no special complaint, readily permit an examination, etc. There exists either a great amount of *disquiet*, or, more frequently, slight *somnolence.* Consciousness is usually unclouded, as appears on one's addressing the patient. Adults have a distinct cognizance of the severity of their disease.

The *appetite* fails completely. The tongue is dirty-red or brown, smooth or thickly coated. *Thirst*, even, is sometimes slight, while the drinks administered are usually quickly and readily swallowed. Sometimes vomiting occurs regularly after drinking, but it seldom sets in spontaneously. The bowels are constipated, or there may be copious, sometimes involuntary *diarrhœa.* The *urine* is usually diminished in quantity, is frequently albuminous, and sometimes dark, brownish like beer (containing blood).

Hemorrhages take place tolerably often in gangrenous angina. They are important sometimes on account of their copiousness, sometimes on account of their prognostic signification (thinning of the blood). They sometimes take place from the portions of mucous membrane affected with the croupous-diphtheritic deposit; less frequently from the palate and pharynx; more frequently from the nose. They sometimes occur in the air-passages and in the lungs, even without croup of these structures; sometimes they take place from the kidneys and urinary passages (hæmaturia), sometimes from the genitals, sometimes from the bowels. Finally, numerous smaller or larger hemorrhages of the skin take place, interstitial or free (from the mouth and from blistered surfaces). If the hemorrhages are copious, the temperature sinks from 1½ to 3½ degrees Fahr., but again reaches its former elevation after from twelve to thirty-six hours.

In addition to the extension of the diphtheritis by continuity

of surface, *diphtheritic deposits take place at a distance from the palate*, especially on the *skin*, in the gangrenous form, and less frequently in non-gangrenous diphtheritis.

Diphtheria of the skin, which was very unfrequently observed in our cases, never appears upon the normal skin, but only upon such portions as have been denuded of epidermis, on accidentally wounded surfaces (intertrigo of small children and fat adults), on sore nipples, on blistered surfaces and incised wounds. The affected portions of the skin present a grayish deposit of varying thickness, finally amounting to several millimetres, and similar to that upon the palate. The parts are painful, a clear or slightly turbid fluid, sometimes offensive in odor, oozes from them. The borders of the skin are bluish-red, and strongly prominent—sometimes erythematous for a considerable distance. Upon these places then appear minute vesicles which run together, burst, and at once become covered with the croupous deposit. In this way the malady may extend over half the length of the body, usually from above downwards. Diphtheritis of the skin is usually secondary to diphtheritis of the pharynx ; the reverse condition of things occurs very rarely.

Diphtheritis of the genitals affects the glans and prepuce in boys, the vulva and vagina in girls and women, seldom the uterus ; it also affects the neighborhood of the anus in both sexes.[1]

The *onset* of gangrenous angina is in general similar to that of the other forms. Usually, however, the general symptoms are severer from the beginning.

The *course of the disease* exhibits two different varieties. In one series of cases the above-described severe diphtheritis continues for a day or for several days, and the gangrenous form becomes superadded. In another, less frequent series, the angina begins to be gangrenous from the start. After an illness of a few hours the palate exhibits the above-described deposit, which, on the second or third day, extends over the pharynx and nasal passages, followed by equally rapid marked swelling of the

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[1] See, also, *Ebstein*, Diphtheritis, eine Gefahr der rituellen Beschneidung (Arch. d. Heilk. 1869. X. p. 393).

lymphatic glands and the adjacent connective tissue. With this there is high fever and delirium, and in most cases death occurs after three or four days.

The almost constant *termination* of gangrenous angina is in death. It generally occurs rapidly, in the course of a few quarters of an hour, or even minutes. The previous apathy usually continues to the last; less frequently death is preceded by great agitation, anxious tossing about, etc. The pulse and respiration are frequently irregular; the former is generally rapid and small, rarely abnormally slow. The temperature is occasionally high, as much as 107¼° Fahr., but usually it is diminished. Sometimes the skin perspires freely. Not unfrequently death is preceded for a few minutes by convulsions, general or of isolated parts; or it instantly follows the sudden raising of the patient, whether this act was performed by himself or through the assistance of others. Sometimes the evidences of collapse continue for several days, until finally death takes place despite energetic treatment. Less frequently the patient dies unexpectedly during convalescence, with or without previous convulsions.

The *causes of death* are sought in various conditions corresponding to the principal disturbances present during life (high fever, irrepressible vomiting, hemorrhages, albuminuria, etc.), or to the symptoms manifested shortly before death (failure of pulse, general collapse, etc.); but for the most part they remain obscure, even on post-mortem examination. In a series of cases post-mortem examination revealed intense bronchitis, mostly with catarrhal pneumonia; furthermore, dilatation of the heart from fatty metamorphosis, and severe affections of the kidneys. In one case I found numerous capillary hemorrhages in the brain and its membranes, in the heart, and in the serous membranes; in a second, œdema of the glottis; in a third, anæmia of a high grade; and, finally, in a fourth, gangrene of the lungs.

Consult *Gerlier*, Mort par concrétions cardiaques dans la dipth. 1866.—*Duchenne*, Bull. de thér. 1870. LXXVIII. p. 173.—*Löwenhardt*, Virch. Arch. 1867, XL. p. 296.—*Bouchut*, Gaz. des Hôp. 1872. No. 117.—*Mosler*, Arch. d. Heilk. 1873. XIV. p. 61.—In regard to the infrequent terminations, consult *Heubner*, Gesichtserysipel mit Hirnaffection (Jahrb. f. Kinderheilk. 1872. VI. p. 105); *Bayer*, the same with fatal hemorrhage from the bowel (Arch. d. Heilk. 1870. XI. p. 398.)—*Scholz* (Wien. med.

Presse. 1865. No. 25) saw diphtheritic angina and retropharyngeal abscess, thrombosis of the left internal jugular vein and transverse sinus, embolism of the left internal carotid artery.—*Güterbock* (Virch. Arch. LIII. p. 523) saw emphysema of the skin in a case of diphtheria.

The *diphtheritic paralyses* [1] occur more frequently in mild cases of pharyngeal diphtheritis—sometimes even in those receiving no medical treatment, than in the severe forms of the disease. They appear most commonly two weeks, sometimes one week, occasionally three and four weeks, after the healing of the local processes, and in some cases not until after convalescence. They most frequently affect the soft palate, and consist in paralysis of both motion and of sensation (*anæsthesia* and *analgesia*); less often they affect the extremities in like manner, with or without the paralysis of the palate; sometimes they are all affected at the same time, or one after the other; preferably the lower extremities only; sometimes the sphincters also. Paralysis of the muscles of the eyes and of the larynx is more rare. Not unfrequently there are analogous conditions of the higher organs: either alone or at the same time with the paralysis mentioned, most frequently of the organs of vision (presbyopia, myopia, even total blindness), more rarely of the organs of hearing, smell, and taste. Sometimes there is impotence. After a continuance of the paralysis for weeks or for months, complete recovery usually takes place—death but seldom. The latter follows much more frequently from pneumonia due to the presence of a foreign body, with or without consecutive gangrene—less frequently from larger bodies becoming detained in the glottis and in the bronchi, from paralysis of the nerves of respiration, etc.

B. *Scarlatinous diphtheritis* occurs but rarely in some epidemics of scarlatina, and in others almost as frequently as the catarrhal angina (see p. 901). It sometimes sets in during the prodromal stage, sometimes during the height of the exanthem, less frequently after its disappearance or even not till the end of the second week of the disease. It exhibits the same three forms as are observed in primary pharyngeal diphtheritis.

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[1] For special information concerning diphtheritic paralysis, see the chapters on Diseases of the Nerves. (Vol. XI.)

Pathologico-anatomically, or histologically, there is no recognizable difference between primary and scarlatinous pharyngeal diphtheritis. I have examined the purely croupous forms by detaching the membrane in the living subject, and the diphtheritic and gangrenous forms upon the corpse.

As already related (p. 946), all the cases of gangrenous pharyngeal diphtheritis observed by me were, with one exception, of scarlatinous origin. Gangrene probably begins in the tonsils, in most instances, especially in the depths of the lacunæ, and usually extends only over the tonsillar tissue—less frequently over the arches of the palate, etc. I have dissected fifteen cases of scarlatinous angina—three in adults, twelve in children. The ordinary cases of diphtheritis had died during the first week—those of gangrenous diphtheritis, between the second and fifth weeks. Among the latter were two in which gangrenous, partially cleansed ulcers on the posterior surface of the soft palate were the only thing found. In one case the purulent infiltration extended through the entire muscular tissue, as well as through all the tissues surrounding the lymphatic glands ; the latter themselves were little altered. In three cases the lymphatic glands of the neck upon one side were gangrenous, and in one case upon both sides. In five cases there was laryngo-tracheal croup at the same time, bronchial croup being also present in three of them, so that tracheotomy had become necessary twice. In a sixth case there was such a severe non-croupous laryngo-tracheo-bronchitis, that tracheotomy had likewise been performed. Among the five cases there was croup of the upper portion of the œsophagus in one instance, and of the cardia in another. In most of the later cases of death, there were numerous lobular pneumonias, and also punctated or uniform fatty degeneration of the heart. In almost all the cases the affection of the kidneys was much more intense than I have ever seen it in primary pharyngeal diphtheritis. In one there were numerous abscesses of both kidneys. In the cases that died during the first week, there was lymphoma of the liver—in one case, also in the suspensory ligament of the liver. In one instance there was a cheesy accumulation in the lower lobe of the left lung and in the bronchial glands, with acute miliary tubercles in lungs, liver, and spleen.

Clinically, the presence or prompt appearance of the cutaneous eruption, the nature of the temperature, the rapidity of the pulse, the frequently peculiar condition of the tongue, are important in reference to the diagnosis of scarlatina. The scarlatinous eruption of the skin, and that occurring in many cases of diphtheritis, mostly a very fleeting erythema, would not be often mistaken for each other. Concerning the remaining diagnostic indications, see under the head of Scarlatina. From the pharyngeal affection the differential diagnosis can only seldom be made between primary and scarlatinous diphtheritis. It can be most easily made in those instances where the soft palate is very markedly reddened in its entire extent, and is only covered in places with the croupous deposit.

It is further worthy of remark that, other things being equal, the diphtheritic anginas appearing in the earlier stages of scarlatina are less dangerous than the primary forms; that extension to the air-passages occurs less frequently; that the anginas appearing in the later stages are more dangerous than those occurring in the earlier stages; that the swelling of the lymphatic glands furnishes here also a correct measurement of the severity of the pharyngeal affection; that the affection of the kidneys in scarlatina almost always makes its first appearance in the second week; and that paralysis and cutaneous diphtheritis are not observed in cases of scarlatinous diphtheritis.

The *pharyngeal diphtheritis of measles* is regarded by many as an affection analogous to that which accompanies scarlatina, while others regard it as a secondary disease (see below). It appears in some localities and in some epidemics. It is not seen at all in Leipzig, or, at least, has not been observed for some decades. On the other hand, secondary ordinary anginas, resembling pharyngeal diphtheritis, occur here in severe measles, as in other severe diseases, always simultaneously with intense bronchitis and multiple lobular pneumonia.

Morbillous pharyngeal diphtheritis is manifested anatomically as a croupous or diphtheritic stomatitis, pharyngitis, and, for the most part, laryngitis also. Intense bronchitis, with lobular pneumonia, is almost always present. The diphtheritis sets in either a few days after the breaking out of the cutaneous

eruption or in the stage of its desquamation; it is least frequently developed at the beginning of the disease. Its local and general symptoms are sometimes very definite and sometimes insignificant. The local disturbances are very slight in many cases, and present the usual degree of gravity in others. The course of the affection is sometimes rapid, sometimes slow. The prognosis is mostly that of a fatal termination.

Consult West, l. c.

C. *Secondary diphtheritis* is characterized by its appearance in the course of other acute or chronic affections, those mostly of a severe type and presenting distinct symptoms. It generally manifests but slight local and general symptoms, and is almost always fatal. The acute diseases concerned are typhoid fever, most frequently, pyæmia, puerperal fever, erysipelas, whooping-cough, measles; the chronic diseases are tuberculosis of the lungs particularly, extensive pleural exudations, chronic diseases of the kidneys (Bright's disease so called, second and third stages), chronic suppurative inflammations of the joints, chronic diseases of the liver, especially in topers.

Secondary diphtheritis occurs at all ages. It is mostly of a croupous nature only. The mucous membrane beneath the deposit is hyperæmic, either not infiltrated at all or slightly infiltrated with pus. The croup membrane seldom occupies the palate or the pharynx to any great extent, but, as a rule, only isolated portions, most frequently the tonsil on one or on both sides, with or without implication of the uvula; or the uvula alone; seldom the pharynx also, but in the latter case usually the larynx likewise. The deposits are sometimes of the ordinary croupous character, containing usually, particularly in their upper portions, very copious punctiform masses of fungi; sometimes there is suppuration of the epithelium at the same time. (Sometimes the microscope reveals the latter condition only, though to the unaided eye a thin croup membrane appears to be present.)

The *symptoms* of secondary diphtheritis vary according to the more or less severe general condition of the patient, and—self-evidently—according to the practicability of a minute local

examination. In the majority of cases the local symptoms are insignificant, or fail entirely. In other cases the ordinary local disturbances of a slighter or higher grade are present. In others, still, the disease of the palate is first made evident by the signs of laryngeal croup. In most instances there is an aggravation of the general condition, especially increase of fever.

The discrimination between primary and secondary pharyngeal diphtheritis is easy in most cases, but difficult or impossible in others, even on post-mortem inspection. In the first place, it is worthy of mention that any patient with acute or chronic disease may become attacked with primary diphtheritis at any time, and that then the latter affection may progress in the manner above described. Oulmont (see below) saw eight patients in one ward attacked by pharyngeal diphtheritis within a few days; six of them had typhus, and five of these died. Furthermore, cases not rarely appear where individuals, apparently healthy, become attacked with pharyngeal diphtheritis and die, and in whom the previous disease is first recognized only upon post-mortem examination. Among the forty-three post-mortems after primary pharyngeal diphtheritis previously referred to, I saw ten such examples; in four instances I found tuberculosis of the lungs and bronchial glands, in one instance an old pulmonary abscess (apparently remaining after small-pox), in two instances severe pulmonary atelectasis, in two instances extensive follicular ulceration of the large intestine, and in one instance a chronic catarrhal ulcer of the cæcum, with a lardaceous spleen. The conditions were somewhat similar in the remaining cases, which were not thoroughly examined.

Oulmont, Bull. de la Soc. de Méd. des Hôp. Sept. 1865.—*Peter*, Gaz. hebd. 1866. Nos. 26, 27, 30.—*E. Wagner*, Arch. d. Heilk. 1866. VII. p. 516.—*Roser*, Ibid. 1869. X. p. 374, describes a real septic or cachectic pharyngeal diphtheritis.—*Bahrdt*, Ib. f. Kinderh. 1870. IV. p. 96: secondary pharyngeal diphtheritis and laryngeal croup after diphtheria of a wound in the skin.—*Billroth*, Wien. med. Woch. 1870. Nos. 7, 8, 20.

D. *Non-specific pharyngeal diphtheritis* (angine couenneuse simple, of Bretonneau ; Angina with plastic exudation, of Trousseau ; Angina crouposa communis, of Isambert.)

The deposits are the most striking and, in many cases, the most important indication of pharyngeal diphtheritis, to be seen either in the living patient or in the dead subject. They are the most important feature in so-called mild diphtheritis, while infiltration of the tissue of the mucous membrane is the most important in severe and in gangrenous diphtheritis. Croupous deposits, however, have been observed in various

localities—at times also situated on the palate—which have nothing in common with true pharyngeal diphtheritis. These deposits are exactly like those of diphtheritis, macroscopically and microscopically. They occur but seldom, on the whole, and are in the form of small patches, from the size of millet-seeds to that of lentils, in the catarrhal anginas, especially in lacunal amygdalitis, in which affection they form rings around the orifices of the lacunæ. They also appear in the form of large patches upon abscesses in the palatine arches; they occur in the same manner in some cases of phlegmonous angina, according to some authors in the toxic forms particularly (after mercury—nitrate of silver (?)—ammonia (?); they are also sometimes seen in syphilis, in the form of larger or smaller whitish deposits; they are rarely seen in extensive burns of the cutaneous surface, in scurvy, hospital gangrene, etc., and after sausage-poisoning (?).

In most of these cases the diagnosis between specific and non-specific diphtheritic deposits cannot be made from mere inspection of the palate. In non-specific cases the affection is oftener unilateral, and the hyperæmia and swelling are frequently slight. Sometimes specific causes (poison) or simultaneous cutaneous affections, etc. (syphilis) are recognizable. The deposits sometimes disappear with remarkable rapidity; sometimes they remain an unusually long time. They sometimes recur several times in the same patient within a short time, other members of the family remaining free from them, etc.[1]

Etiology.

The causes of primary pharyngeal diphtheritis—those of the other forms we leave out of consideration—have at all times been the subjects of most assiduous investigation, and especially so during the last ten years, when it has been thought possible to produce the disease artificially, and its ultimate origin has been thought to be discovered in the presence of a low fungus.

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[1] *Gubler*, Arch. gén. Mai. 1857.—*Bartels*, l. c.

For reasons already mentioned (see p. 925), the author avoids, in this place also, a detailed reproduction of innumerable isolated observations, but brings forward only what he has himself observed in Leipzic, and briefly defines his position with regard to the prevalent fungus theory.

Primary pharyngeal diphtheritis has occurred here in patients of all ages, most frequently in those from two to six years of age, less frequently in those under two, very seldom in nurslings, even when all their brothers and sisters were affected, never in the new-born. Up to the tenth year of age it is still tolerably frequent; from this age on it is less so, up to twenty-five and thirty years. Beyond this age it occurs very seldom, or at least almost only in the mildest forms, and it has been observed but a few times in old persons. No difference with regard to sex was observable. On the whole, the diphtheria occurred oftener in strong, even fat and well-nourished children, than in debilitated children.

Marshall saw diphtheritis in a new-born child (Schmidt's Jb. CIL. p. 324).

So far as the season of the year is concerned, it occur somewhat more frequently in the autumn, but isolated ca were not wanting at any season. During the epidemic of chol (July to October, 1866), and still more during the epidemic small-pox (February to September, 1871), it was less freque It occurred here with about equal frequency in all parts of t city. It seemed to be more frequent in narrow streets and courts in the vicinity of the water, in subterranean dwellings, and in new-built houses; but even in the best portions of the city, and in the finest dwellings, there was no immunity from the disease. When it appeared in a large, densely populated house, it almost always remained confined to the house, or even to one floor, or one part of a floor.

The predisposition of isolated families to attacks of the disease has also been observed here. For the most part, no cause for this was detected, but not unfrequently the affected members of the families had been suffering with chronic angina or with hypertrophy of the tonsils. The most severe, so-called gangrenous form of diphtheria occurred here very seldom as a general thing. If the patient belonged to a family who were natives of

this place, sometimes there would be only the one case, sometimes the remaining members of the family would be affected in a milder manner. In a few instances only, in this city and in the neighborhood, from three to even as many as six children of the same family were attacked with the severer form, and all died. The parents, nurses, servants, etc., were often likewise attacked in such instances, but almost always in a mild form, seldom fatally.

Pharyngeal diphtheritis cannot be compared with any of the known contagious and miasmatic diseases. It is without doubt, in a certain sense, "catching," *contagious;* and in other respects it is *miasmatic.* Its nature is rendered the more undeterminable in that it exhibits different characteristics, at different times, and in different places. Its contagiousness has been very much dreaded at certain times, and by some authors ;[1] and at other times and to other authors it has shown itself little, if at all contagious. A great number of trustworthy observations testify to the *great contagiousness* of the disease, by its spreading from house to house, from one member of a family to another, and from bed to bed in hospitals. Cases of this character have evidently become much less frequent during the last decennium ; in Leipzic they have appeared with this intensity in isolated instances only. The following grounds, also, are ordinarily referred to in proof of its contagiousness, although they are for the most part otherwise explicable: namely, the epidemic and endemic appearance of the disease at times ; the remarkable susceptibility of children ; the fact that several members of a family are often attacked; the fact that nurses and physicians in attendance are more often attacked than other people; and perhaps, also, the more frequent occurrence of the disease in the families of such professional attendants. With regard to the simultaneous *miasmatic* nature of diphtheritis, the principal evidence is, its attaching itself to certain localities, as has been observed from the time of Bretonneau to the present, and by others also, especially Bartels. Only a few such instances have occurred

[1] Read the celebrated descriptions of Bretonneau, Trousseau ; and, further, of Boudet, Arch. gén. Feb. 1842.—Becquerel, Gaz. Méd. 1843. p. 687.

here in Leipzic. The best evidence on this subject is an observation, substantiated from several sources, to the effect that when healthy children were brought back to the bed-chambers previously occupied by their brothers or sisters when sick with diphtheritis, especially when the cases had been fatal, or even when they were only brought back into the affected house, they sometimes became attacked by the same disease, either at once during the first few days, or not until after thirty or forty days. There appears, therefore, to be a tenacity to the contagion similar to that of exanthematous typhus, etc.

The *specific poison* of diphtheritis is still entirely unknown. It was first hoped that it would be discovered by attempts at the artificial production of diphtheritis. The possibility of the so-called production of diphtheritis by tincture of cantharides and olive-oil (Bretonneau), by acids, alcohol, nitrate of silver, ammonia, chlorine, corrosive sublimate, etc. (Albers, Duval, Valentin, Delafond, and others), no longer requires to be refuted. The impossibility of producing it by ammonia, recently again maintained by Reitz,[1] and by Oertel (l. c.), has been demonstrated by Mayer.[2] Whether the inoculation of croupous and diphtheritic membranes in the artificially opened trachea of rabbits, etc.,[3] has produced true croup or true diphtheritis, I am unable to say from my own observations. It is well known that Trousseau, as long ago as in 1828, and at a later date Peter[4] inoculated themselves with diphtheritis several times on the arm, tonsil, and palate, by means of the lancet, but without result.

The *parasitic nature* of diphtheritis rests upon still weaker evidence. According to the variously conflicting experiments of Tommasi-Hueter (Buhl), Oertel, Trendelenburg, Nasiloff, Klebs, and Eberth, and according to Letzerich and others, bacteria are the essential cause of diphtheria. The botanic characteristics of this fungus are still entirely unknown; the fungus is frequently but not regularly found in cases of diphtheritis in the human subject, as it readily adheres to the surface of the de-

[1] Sitzgs-Ber. d. Wien. Acad. 1867. LV. 2. Abth.

[2] Arch. d. Heilk. 1873. XIV. p. 512.

[3] *Trendelenburg*, Arch. f. klin. Chir. 1869. X. p. 720.—*Oertel*, l. c.

[4] Étude sur la diphthérie et la croupe. 1859.

posit, and cannot easily be removed by movements, etc.; the gross anatomical and histological characteristics of human and artificially excited diphtheritis vary in manifold and in part in the most essential points ;—on all these data consult the author's Manual of General Pathology.

The incubation period of diphtheritis, according to a number of tolerably well-attested observations, occupies from two to four days (Newman, Coulon, Bartels, Mueller, Wertheimer, Thomas, the author) ; and this is the same after inhalation of the infectious material as after its introduction through the lips, the nose, mouth, eyes, or skin. Cases have not been observed here like those repeatedly observed in France, where diphtheritic masses brought in contact with the surfaces of wounds produced diphtheritis, which either remained local, or excited pharyngeal diphtheritis. On the other hand, diphtheritis of the skin of the external nose, of the mouth, of the external genital organs in both sexes, of excoriations on other parts of the skin (blistered surfaces, leech-bites) in diphtheritic patients, have also been repeatedly observed here. Diphtheritis has been observed likewise at the corners of the mouths of nurslings where mothers were affected with pharyngeal diphtheritis. The use of the clothing and bedding of diphtheritic patients has also been observed here to produce diphtheritis in the foreskin, the vulva, the arms, and the navel of new-born babes, alone, or associated with the affection in the pharynx. I lost my own son at the age of five weeks in this way, from gangrene of the navel with diphtheritic deposit. The croup-like or diphtheritis-like affection of the wound made in the operation of tracheotomy in diphtheritic patients is very seldom true diphtheritis, according to my observations.

The *relations of diphtheritis to other diseases* will be spoken of here only in so far as it has any practical signification. With reference to the fact that croup and diphtheritis belong together, no doubt can prevail, according to the exposition already given. The relations of pharyngeal diphtheritis and scarlatina are in many ways of great interest in practice ; both frequently appear in juxtaposition, etc. In spite of this, no correlation between the two appears to exist ; for the grounds

of this assertion see p. 925. That scarlatina, with ordinarily mild or with severe pharyngeal diphtheritis, may produce in others pharyngeal diphtheritis without cutaneous eruption—in the attendants, for example—has been repeatedly observed here also.[1] It is also of practical importance that having been through the one does not protect against the other.[2]

Finally, cases have been observed in which patients with gangrenous erysipelas, with gangrenous wounds of the skin, with puerperal gangrene, excited diphtheritis of the pharynx, etc., in other patients in the same wards, or in the attendants. This did not occur here.

Pharyngeal diphtheritis, and the diphtheritis of wounds in hospital gangrene, etc., are not identical: Roser in opposition to Robert, Virchow, and others. Lasègue (Traité des angines, 1867) describes a diphtheroid angina.

Treatment.

Few acute diseases have been submitted to as numerous therapeutic propositions and measures as pharyngeal diphtheritis. There is hardly another affection concerning which the views of authors differ so widely. This has its cause in the actual or supposed accessibility of the diseased structures, and in the various views entertained as to the nature of the disease (whether it is a local or general disease from the beginning, a fungus disease, etc.), and in the so-called therapeutic results. Almost every physician of my acquaintance has his own method, his favorite remedy. One method is barbarous to the patient, another is milder, a third is expectant. The views of most observers coincide in this, that *the majority of so-called mild cases recover spontaneously;* that *gangrenous diphtheritis is intractable to every form of treatment;* that with us *the chief danger of severe angina is its extension into the larynx*, and that this extension cannot be prevented by any remedial agency.

Where the condition of things permits it, the patient is to be at once rigidly isolated, and, where possible, in such a manner

[1] See also *J. Braxton Hicks*, Guy's Hosp. Rep. 1871. XVI. p. 165.

[2] See also *Mettenheimer*, Memorab. 1869. XIV. No. 8.

that the physician and attendants do not come in any contact with the remaining members of the family, the physician visiting and examining the healthy members of the family before entering the chamber of the sick. Although isolation, at the time it is usually instituted, is frequently and perhaps constantly useless, it is nevertheless advisable, both out of regard to the sick and the well, and is in accordance with the rules of medical policy. The temperature in the bed-chamber should be regulated in accordance with the degree of fever, and the room should at all events be well ventilated.

Local treatment is practised in different ways by different physicians; cauterization in every possible degree, the use of so-called solvent substances, the use of disinfectant remedies, and simple cleansing of the palate and pharynx, being the four methods employed.

Cauterization is practised with nitrate of silver in substance or in solutions of various strengths, with concentrated or diluted acids, especially with fuming muriatic acid, also with chromic acid, with caustic potash, pure or variously diluted with water or glycerine, with iodine (Lecointe), with an iron warmed in boiling water (Danvin). This must be done daily, at least once, and where possible twice a day. Cauterization, with solid or liquid substances, frequently acts mechanically more than chemically. In children, it not unfrequently results in injury to healthy parts, hyperæmia of these parts, and also collateral œdema of the contiguous parts. The caustic reaches only a portion of the diseased surface. Children are much excited by it physically and psychically ; the pains in the palate are sometimes first excited by it or still more increased, and the administration of nourishment is then accompanied with more resistance. In spite of these drawbacks, cauterization is still much in vogue. Of late years, however, the physicians most experienced in the treatment of the diseases of children, Bartels and others, have expressed themselves in opposition to it.

Substances which dissolve the deposit and so remove it, are prescribed by many in the form of gargles, and especially of inhalations. Gargles usually cannot be employed on account of want of skill in using them, pain, etc. Those who

put faith in inhalations recommend that they be used tolerably often, every hour or two, a few minutes at each time. Some practitioners waken patients from sleep to administer them. The substances employed have been chosen less from the results of practical observation than from experiment upon membranes removed from the living subject or from the corpse. Lime water, diluted in from two to four parts of water, and lactic acid in from fifteen to thirty, are especially employed. When these remedies are diluted and used frequently, they have at least the merit of cleansing the mouth, palate, and pharynx, and of provoking movements of swallowing. When their employment is painful, or when the patient is very weak, they are injurious.

Küchenmeister (Oestr. Z. f. pr. Heilk. 1863, No. 13 u. 15, 1867. No. 38; Berl. kl. Wschr. 1869, No. 49 u. 50) first recommended lime water, since so much employed; and *A. Weber* (Med. Ctrbl. 1869, No. 22) first recommended lactic acid. Consult also *Bricheteau* and *Adrian* (Bull. de thér. 1868. LXXIV. p. 72). *Förster* (Arch. d. Heilk. VI. p. 521) recommends the carbonate of lithia; *Fraenkel* recommends a solution of pepsin.

Substances are in use, equally often, to which a *specific action* upon the diphtheritic mucous membrane is attributed, either disinfectant or parasiticidal. Neither object has ever been realized; the disinfectant action is, in most instances, chiefly a deodorizing one, though a cleansing one also; the parasiticidal action is based upon the undemonstrated parasitic nature of the disease. As a systematic employment of these remedies is impossible, we will mention those best known, in the order they happen to occur to us. They are employed in the dry or in the fluid form, by insufflation or by inhalation—in the latter instance several times a day. Those most frequently employed, and by some authors praised as unfailing remedies, are alum, tannin, and sublimed sulphur by insufflation, or chlorate of potash (from five to ten grains to the ounce), and carbolic acid (one part to three hundred) by inhalation, or they are painted upon the parts, or used as gargles, etc.

Alum as well as tannin is at present less employed. Sublimed sulphur, much recommended by Lagauterie, Barbosa, Jodin, Roger, and others, is regarded by certain authors as a sure remedy.

Chlorate of potash, prominently introduced by Isambert (Étude chim., phys., et clin. sur l'émploi thérap. de chlor. de potasse special. dans les affect. diphthér. 1856), is more frequently employed than any other remedy. Of late years it has been somewhat superseded by carbolic acid, first recommended by Calvert (Lancet, Sept. 1863.), and then by Turner and many enthusiastic German physicians. Sulphurous acid, also, is recommended by Dewar (Med. Times and Gaz. May, 1867), and various sulpho-carbolates by Sanson (Med.-Chir. Trans. 1869. LII. p. 139).

In this category also belong the employment of iodine (Warring-Curran; iodine and iodide of potassium āā. gr. iv., alcohol f. ʒ iv., water, f. ℥ iv., by inhalation); of bromine (Goldsmith-Fuckel; bromine gr. vj., bromide of potassium, gr. xxiv., water f. ʒ j., applied by the brush); of permanganate of potassa (Lessing and others); of cinnabar (Abeille); of peroxide of hydrogen (Stöhr); of ether (Black); of glycerine (Stehberger); of highly rectified spirits of wine (Lövenson), etc., etc. Schwanda recommends the galvanic current to kill the fungus!

The so-called *mechanical treatment* of diphtheritis—that is to say, the removal of the deposit by means of the forceps, proposed by some authors—is not practicable in an efficient manner.

The *general treatment* is of importance in addition to assiduous cleansing of the palate, etc., by frequent drinks, rinsing of the mouth, and inhalations of water or chlorate of potassa in weak solution. The patient should be kept in bed, even in the mildest form of diphtheritis; and only adults should be allowed to sit up, still remaining confined to the room. The diet should not be low, even if there be high fever; and, as is self-evident, fluid substances only should be allowed. According to the cravings of the patient, we give milk, warm beer, both of them with eggs, meat, broths, and nourishing soups from the beginning; or, if anæmia be present, good beer, wine, and brandy. If an adynamic condition exist, the latter drinks are to be given often, and in considerable strength. In addition to this we administer iron and quinine, the former most frequently in the form of the ethereal tincture of acetate of iron (from five to fifteen drops every three hours), and in the form of the solution of the chloride of iron (from one to five drops in sweetened water, every two, three, or four hours).

The following articles have been much recommended for internal administration, with the idea of their specific action: chlorate of potassa and bicarbonate of soda, alone or with nitrate of soda. Their merit is very doubtful. The same may be said

of the internal administration of balsam of copaiba with cubeb (Trideau), as well as that of cubeb alone (Vaslin).

The cold water treatment is justifiable only in case of the continuance of high fever for several days and the maintenance of considerable strength.

In severe cases of general debility the transfusion of blood may be tried (Demme). Swelling of the lymphatic glands is treated in the ordinary manner.

The extension of the pharyngeal disease into the larynx cannot be surely prevented by any remedy. It is recommended to keep a sufficiently large ice-bag continuously upon the upper portion of the throat, from the commencement of pharyngeal diphtheritis. It is usually well borne, and may perhaps prevent the development of laryngeal croup.

Antiphlogistics in any form, derivation by sinapisms, blisters, etc., are no longer in use—indeed, they are deemed injurious by almost all practitioners.

III. Gangrene of the Soft Palate. Gangrenous Angina. Angina Gangrænosa.

Gangrenous angina is either primary or secondary.

Secondary gangrenous angina occurs very seldom in ordinary anginas; somewhat more frequently in many forms of phlegmonous, scarlatinous, and diphtheritic anginas; unfrequently in the inflammations of the palate, complicating measles and typhus. *Primary gangrenous angina* is still more rare. It corresponds probably to noma of the external skin, of the mucous membrane of the mouth, etc. It can usually be diagnosed with certainty only when the patient has been closely observed from the beginning, and when the secondary gangrenous anginas above-mentioned are thrown out of the question, on etiological and clinical grounds. The *causes* of gangrenous angina are entirely unknown. It occurs more frequently in young persons, poor children especially, than in adults; weak and cachectic subjects, especially those who have recently recovered from severe diseases (typhus, dysentery, etc.), are those most commonly attacked.

The *local manifestations* are alike in both forms of the dis-

ease at their height. In secondary gangrenous angina there is usually at first an intense phlegmonous inflammation of the entire soft palate, upon both sides alike, or upon one half especially. Usually as early as the second or third day there are grayish or black circumscribed patches, which become transformed, after a few days, into sharp-bordered, similarly-colored ulcers. The remaining portion of the palate still remains dark-red and swollen. The ulcers maintain their circumscribed contour, or in a few days involve the entire soft palate, not unfrequently the mucous membrane of the cheeks also, as far as the lips, or the pharynx and upper portion of the larynx. They then diffuse a cadaverous or fæcal odor, if they are not too small in extent. The disease is very painful. Swallowing and articulation are very much impeded, and may even be prevented. The submaxillary glands are usually considerably infiltrated, and frequently also the tissues adjoining them.

The *general symptoms* are severe in both forms of gangrenous angina. The fever varies; the temperature may be normal, or slightly, or considerably, elevated; the pulse is sometimes much retarded, at other times much accelerated. There is complete loss of appetite, frequently vomiting, etc. Consciousness may be maintained, or there may be a comatose condition.

The *course* of the disease is acute. Limitation of the gangrene, etc., occurs occasionally, and only in inflammatory gangrene. In all other cases death takes place, usually in from four to ten days, in consequence of septicæmia, or hemorrhage from the diseased or other portions, or œdema of the glottis, or inflammation of the lungs, or, finally, from diseases which are connected with the primary disease, and not with the gangrene of the palate (inflammation of the kidneys in scarlatina, etc.).

Gangrenous angina occurs most frequently *in scarlatina.* Its commencement presents nothing characteristic. There is usually, at first, an intense dark, somewhat livid reddening, frequently accompanied by hemorrhages and marked swelling of the soft palate and contiguous parts, as well as of the lymphatic glands of the jaw. The local manifestations are very prominent when they are not concealed by the severe general affection. Towards the middle or end of the first week, less frequently during

the second week of the disease, a dirty gray spot appears upon one tonsil or upon both. This usually extends rapidly, so that the entire soft palate becomes gangrenous in a few days. The termination is almost always fatal, usually under septic manifestations.

I have carefully examined two cases of this gangrenous angina pathologico-anatomically. One was fatal from the gangrene, the other from nephritis. On portions of the tonsils, which were intensely diphtheritic at the commencement of the disease, there lay deep, gaping furrows, the immediate surroundings of which were still gangrenous, while further outwards a dense, purulent infiltration existed in many places, a line or so in width, and reaching as far as into the muscular structure. The contiguous portions of the palatine arches exhibited a slighter purulent infiltration.

Pitha (Prag. Vtljschr. 1851. II. p. 27) and others describe an *angina nosocomialis phagedænica*. It occurred during an epidemic of hospital gangrene, and affected patients with phagedenic ulceration, as well as individuals entirely healthy or in complete convalescence. It occurred suddenly, without assignable cause, for the most part over night. It involved the mucous membrane of the mouth, soft palate, and posterior walls of the nose. Grayish-white thick deposits occurred, underneath which the dark-red and acutely swollen mucous membrane underwent rapid destruction in from twelve to twenty-four hours, so that large lardaceous ulcers were formed with highly-reddened, jagged, and undermined edges.

Consult also R. K. Browne (American Medical Times, Nov. 1862), concerning a characteristic *gangræna faucium*, a new form of hospital gangrene.

It is probable that certain unclassified cases belong in this category, for example, Günsburg (his own Zeitschrift. 1850. I. 2. H.): so-called pharyngo-typhus in a girl sixteen years of age; further, Guthrie (Edinb. Med. Jour. 1862. VIII. p. 297). Jobert (Ann. de thér. Mai, 1846) saw a case of perforation of the soft palate following a wound from a crust of bread. Williams (Brit. Med. Jour. July, 1862) describes peculiar, perforating, non-syphilitic ulcers of the palate.

Gangrenous angina has long been known. Its independent existence was denied in France by Bretonneau, who recognized it as a regular sequel of diphtheritic and scarlatinous angina; on the other hand, its independence was demonstrated later by Delaberge and Monneret (Compend. I. p. 134. 1836); Rilliet and Barthez (Arch. gén. 1841. XII. p. 446); Guersent (Dict. en XXX. 1833. III. p. 134); Trousseau (l. c.); and especially by Gubler (Arch. gén. Mai, 1857.)

IV. Atrophy of the Palate, and especially of the Tonsils.

Atrophy of the soft palate and atrophy of the tonsils sometimes occur together, and sometimes the latter occurs alone.

Both affections have been practically but little investigated. The latter will almost exclusively occupy our consideration here.

The *causes* of these atrophies are various.

Congenital atrophy, deficient development of the tonsils, occurs not unfrequently. It consists in the tonsils and sometimes the arches of the palate also remaining at a standstill, in the condition of early age. The tonsils are remarkably small, the number of the lacunæ is usually small; they are shorter, and their surroundings exhibit smaller follicles, or the follicular arrangement of the cytogenetic tissue is wanting altogether.

Atrophy from disease and from senile marasmus simultaneously affects the tonsils and the soft palate, including the uvula. This atrophy occurs regularly in old age from well-known causes. The diseases in which it occurs are convalescence from severe acute diseases (typhus, etc.)—furthermore, chlorosis, tuberculosis, diabetes mellitus, etc. In the diseases last mentioned the atrophy of the parts named is as regularly present as that of the entire body, and thus the soft palate becomes a true mirror of the general nutrition. The palate and the uvula are thinner, slender, and mostly very pale. The thinning affects the mucous membrane and the muscular layer; the mucous glands are sometimes diminished in size at the same time, and sometimes they project to a remarkable degree. The tonsils are small, the outlets of their lacunæ are indistinct, their surface is level or slightly arched. Upon section they show especially a thinner, whiter, and firmer perilacunar tissue than natural, and poorer in cellular elements.

Atrophy in consequence of operation, especially that of incomplete extirpation of the tonsils, is not always distinguishable from the foregoing forms.

Inflammatory atrophy of the tonsils occurs in a great number of persons. It occurs most frequently in both tonsils, in equal or unequal degree, less frequently in one tonsil only. It affects the tonsil either in its entire mass, or only in the upper or lower half, or only in its middle portion, or only in its length, breadth or thickness, or only the anterior or posterior half in its entire length, or only partially, or in still more limited extent. In this manner there occur a multitude of variations, most of

which are not fully recognizable during life. A special description of these variations is unnecessary.

At the point of atrophy the tonsil is diminished to almost complete disappearance, in all its diameters, but especially in thickness. The cytogenetic tissue still remaining shows isolated follicles sometimes, but in most instances there is no longer any distinct follicular arrangement. Sometimes the reticular tissue is remarkably broad in its fibres, the interspaces being correspondingly small. (In individual cases large giant-cells with many nuclei were found in the atrophic cytogenetic tissue.) Sometimes the lymphatic vessels are remarkably distinct. The lacunæ are mostly narrower, or they are obliterated in various places, and to a varying extent, most frequently on the free surface. Sometimes they are dilated in places, and filled with concentric layers of epithelium. The intermediate connective tissue is usually hypertrophied. The capsule of the tonsil is normal, or thickened in places or in its entire extent, most frequently on the surface. In the latter case fibrous processes usually pass from it into the interior of the tonsil. Sometimes the tonsil is composed in greatest part of connective tissue, which may be poor or rich in vessels, and is not unfrequently bestudded with minute masses of hæmatoidine. (In one case I found all the smallest arterial and venous vessels, as well as the capillaries, narrowed in calibre, their walls being ten times their normal thickness, and composed of a homogeneous, non-nucleated, amyloid-like substance.) Or it contains nuclei here and there, equally distributed between the fibres, or in process of gradual transformation into non-follicularly arranged cytogenetic tissue. This connective tissue, in many cases, contains fat cells in varying proportion, mostly irregularly distributed, seldom in lobular form. Near the base of the tonsil, more rarely at a distance of from a half to two millimetres ($\frac{2}{100}$ to $\frac{8}{100}$ of an inch) from the capsule, several elongated dense masses, either very small or barely visible, and composed of *hyaline cartilage* or of *true bone tissue*, are not unfrequently found lying in the connective tissue. The muscular fibres on its external surface are sometimes to a great extent firmly adherent to the connective tissue.[1]

[1] Concerning syphilitic atrophy, see further on.

Atrophy is frequently associated with *dilatation of the lacunæ*, and they are filled with desquamated pavement epithelium, or at the same time with pus, or with the products of destruction of both, so that atheromatous, cholesteatomatous or abscess-like cavities are produced. The lacunæ open either on the surface in the ordinary manner, or they are contracted in places—mostly near the surface—or they are entirely closed.

When the dilated lacunæ open freely on the surface, they often reveal within them, and projecting beyond them to the size of a millet-seed and larger, grayish-yellow, dry masses which, after being removed spontaneously (by coughing, hawking, etc.), or artificially, are not unfrequently reproduced many times. This affection simulates most nearly the comedones of the external skin. This condition occurs sometimes in one only, sometimes in several, or nearly all the lacunæ of one or both atrophied tonsils, and then excites one form of chronic angina.

When the lacunæ are closed at their orifices the condition of things is different. The number and size of the affected lacunæ vary very much ; sometimes they are affected singly, sometimes in groups of from two to four, or even as many as ten. Their size varies from that of half a pea to that of two peas, and occasionally they may reach even that of a cherry. Their form is regularly round or elongated ; less frequently irregularly jagged ; in the latter instance the irregular shape is due either to the fact that lacunæ of normal or slightly dilated calibre open into them, or to the fact that two or more neighboring lacunæ have run together. If these are few and small, the size of the tonsil is not materially altered ; in other cases, in spite of the atrophy, it may be of normal size or even larger. Some tonsils are almost entirely composed of such lacunæ, and of the intermediate atrophied cytogenetic, or almost pure fibrous tissue. The dilated lacunæ sometimes lie near the surface of the tonsil, and sometimes deeper in its interior. In the former case, gray or yellowish, soft or tense, prominences form, covered with thin mucous membrane ; these prominences are distinguished from abscesses chiefly by their long duration (they sometimes continue for months), and the absence of any inflammation in the

vicinity. In the latter case they are discovered only in the dead subject upon section.

The contents of the lacunæ are gray or grayish-yellow, or pure yellow. The consistence varies from that of thick gruel to that of milk; it is uniform, or contains a variable number of roundish lumps.

The gray semi-fluid contents are composed principally or entirely of epithelium. This is arranged in layers, usually flat, less frequently in more globular form. The epithelium is often simply degenerated, less frequently in a state of fatty degeneration, sometimes with isolated larger, or numerous small vacuoles. The nuclei are single, sometimes multiple, normal or distended. Sometimes epithelial cells are found completely filled with pus corpuscles. The white, semi-fluid contents consist of pavement epithelium and crystals of cholesterine. The yellowish contents consist principally of pus corpuscles, which frequently exhibit different stages of fatty metamorphosis. The grayish-yellow, mortar-like contents likewise reveal epithelium filled with fine molecular masses of chalk, as well as larger but still microscopic globules of chalk, which sometimes adhere tightly to the inner surface of the lacunæ. In all cases, bacteria appear in these contents, either mobile or immobile, punctiform, red-shaped, or thread-like in form, or we find shreds of shagreen-like masses of zooglöa. Many exceedingly small, brownish-yellow, curdy lumps, or even those as large as a millet-seed, consist almost entirely of these bodies. They lie free, or in the interior of the pavement epithelium. In two cases I saw mould-fungus in the lacunæ, and several times fragments of food (vegetable-cell tissue, and striped muscular fibre).

The lining of the lacunæ is composed sometimes of only a few layers of pavement epithelium, sometimes of many (from ten to twenty) and is generally seated upon small, seldom upon hypertrophied papillæ. The parts surrounding such lacunæ are sometimes normal, sometimes compressed in various degrees, and atrophied; in the latter instance, there is frequently no follicular arrangement. The reticulated tissue sometimes contains red or black pigment granules.

Some lacunæ, contracted or entirely closed superiorly, contain, alone or in connection with the contents described, one, and less frequently several *concretions*. These vary generally in size from that of a millet-seed to that of a pea; occasionally they are as large as cherries or even larger, of a brownish-gray color, round and smooth, or often irregularly indented (so-called mulberry calculi). They are principally composed of phosphate and car-

bonate of lime, and of a variable amount of organic material. The stones are either borne without further local disturbance, or they finally make their way to the surface, and are then usually coughed out. This takes place sometimes without severe inflammatory and other symptoms, sometimes through the medium of a tonsillar or peritonsillar suppuration.

Wurzer (Buchner's Rep. f. d. Pharm. XXIII. 2. H.) found in a tonsillar calculus removed during life 63.8 parts of phosphorus, 15.7 of carbonate of lime, 1 per cent. of iron, 7.1 of hydrochloric acid, soda, some potassa, and 13.3 of an animal matter. Robin found 50.0 of phosphorus, 12.5 of carbonate of lime, 25.0 of water, and 12.5 of mucus.

Clinically, atrophy of the tonsils has received but little attention. It does no special damage in itself. In fact, many observations go to prove that persons with congenital or acquired atrophy of the tonsils are less subject to almost all the diseases of the tonsils, especially the ordinary inflammations, diphtheritis in its various forms, and syphilis. The dilatations of the lacunæ (like comedones) have already been mentioned as a cause of chronic tonsillar angina. The cyst-like dilatations of the lacunæ have a clinical importance in the fact that their contents act irritatingly on the contiguous parts, either mechanically or chemically. In this way are produced—and usually repeatedly—the various kinds of tonsillitis, and especially intra-tonsillar and peri-tonsillar abscesses.

The latter condition is seldom demonstrable in the living subject. When, on the contrary, many tonsils are examined from the dead subject, and especially when, after previous hardening, several parallel sections are made through them, various far-reaching cicatrices, etc., are frequently found in the neighborhood of such cysts.

Simultaneous atrophy of the soft palate and of the tonsils occurs not unfrequently in connection with chronic pharyngitis, especially with pharyngitis sicca. In what relation they stand to each other is for the most part unknown. Probably, however, the former in some cases is the predisposing cause of the latter.

V. Hypertrophy of the Tonsils.

Concerning the anatomical and histological relations, see *Billroth*, l. c.—*Virchow*, Geschwülste. II. p. 609.—*O. Weber*, in Pitha-Billroth, Hdb. d. Chir. l. c. p. 360.—Concerning the clinical relations, see *Dupuytren*, Repert. d'anat. et de phys. 1828. V.—*Graves*, Dubl. Journ. Jan. 1839.—*Shaw*, Med. Gaz. Oct. 1841. Bull de thér. May and July, 1843.—*Robert*, ibid.—*Pitha*, Prag. Vjschr. 1845. II.—*Yearsley*, On the enlarged tonsil, etc. 1848.—*Roger*, Séméiot. des mal. de l'enfance. 1864.—*Desnos*, in Nouv. dict. de méd. et de chir. prat. 1865. II. p. 138.—The copious literature upon extirpation of the tonsils belongs to the domain of surgery.

Hypertrophy of the tonsils is a true hypertrophy. All the constituents of the tonsil increase proportionately, the follicles especially becoming not only large, but also more numerous. It occurs in very different grades, less frequently in one tonsil only, oftener in both, and in the latter case to an equal or unequal degree. The size of the hypertrophied tonsil varies from a slight increase of volume to one the size of a walnut, and occasionally it reaches that of a small hen-egg. The tonsil retains its original form—only the prominences and depressions on its surface are more distinctly pronounced, occasionally to such a degree that the superficial surface becomes lobulated. Generally the entrances of the lacunæ are larger and wider, corresponding to the increase in size of the tonsil, and their already normally-arched contour is rendered still more distinct. The orifices may be empty, or filled with various substances. In most instances the position of the tonsil also becomes altered, as it becomes enlarged. Only occasionally does it remain lying chiefly between the palatine arches, so as to make them bulge forward at the corresponding point. In most instances it protrudes beyond its niche between the palatine folds towards the median line. At the same time it is usually somewhat lowered, rarely so much so as to appear pedunculated. In bilateral hypertrophy the pharyngeal vestibule is remarkably encroached upon, even so much so that only a small horizontal space remains between the root of the tongue and the soft palate. The uvula is pressed together from both sides, or it is often pushed backwards, or, less frequently, forwards. In

extensive unilateral hypertrophy the uvula is pushed over to the opposite side; it thus remains crooked, or is irregularly bent in various ways. Finally the middle pharyngeal space itself is narrowed by the corresponding projection of the hypertrophied tonsils into this space.

On *section* the hypertrophied tonsils are grayish-red, poor in blood, soft, but brittle, and homogeneous, with the exception of the fissures and their contents. The follicular arrangement on section of fresh tonsils is sometimes distinct and sometimes wanting. In the latter case it may be brought to view in the hardened tonsil, in thin sections, especially under a magnifying lens. The mouths of the lacunæ will be found in a more or less gaping condition; they are also wider and elongated in proportion to the increased thickness of the tonsil. They run vertically or obliquely into the tissue of the tonsil, and usually exhibit numerous prolongations. Around the lacunæ are the follicles, increased in number even to the number of twelve on each side of the lacuna. They vary in size from $\frac{1}{2}$ to 2 mm. ($\frac{1}{50}$ to $\frac{1}{12}$ of an inch), and are mostly round; but many of them are oval. They are usually distinctly separated from each other by interfollicular tissue; but not unfrequently two and even three of them lie so closely together that they appear as if formed by division, or some of them may be actually biscuit-shaped. Two follicles rarely lie the one upon the other. The larger follicles usually project somewhat into the interior of the lacuna. Microscopically, the follicles and the reticulated tissue between them are essentially arranged as in the normal tonsil. The septa of connective tissue and the capsule are usually somewhat thickened. The superficial epithelium is of normal thickness or thicker, and the papillæ beneath are usually increased in number, and sometimes somewhat shorter. The epithelium, etc., of the lacunæ present nothing characteristic.

Hypertrophied tonsils so frequently exhibit all of the affections already described, especially hyperæmia and the various inflammations, as well as partial atrophy, that these conditions, especially the inflammations, must be reckoned in the symptomatology of tonsillar hypertrophy. The lymphatic glands of the jaw are often moderately enlarged, probably less as a result of

the hypertrophy than of the subacute and chronic inflammation of the lacunæ, etc. Sometimes most of the glands of the throat are larger and harder.[1]

In hypertrophy of the tonsils the soft palate is either normal, or it exhibits chronic catarrh, and, with especial frequency, œdema of the uvula; or there is, at the same time, a slight hypertrophy of the mucous membrane, sometimes of the follicles also, and especially of the so-called auxiliary tonsils in the anterior palatine arches.

Hypertrophy of the tonsils *occurs frequently*. It is encountered mostly in the earlier years of life, and is then frequently inherited. In some families one of the parents and most of the children are thus affected. More rarely it occurs, for the first time, in the later years of childhood, and still more rarely after this period. It attacks both sexes with equal frequency.

The *special causes* of hypertrophy of the tonsils are unknown. In one series of cases it is developed without attracting any notice and without any previous angina, and in these cases comes to the knowledge of the physician accidentally. According to some observers the influence of the first dentition (from the sixth to the twenty-fourth month of life) is important. In a more frequent series of cases, the affected individual suffers repeatedly from catarrhal or parenchymatous tonsillar angina; the swelling of the tonsil connected therewith subsides only incompletely, and after each attack the tonsils remain somewhat larger than before. The croupous, diphtheritic, and scarlatinous forms of angina, also, sometimes leave an enlargement of the tonsil after them.

The *symptoms of hypertrophy of the tonsils* are local and general. They of course differ according to the grade of hypertrophy, according to whether one or both tonsils are involved, etc.

The *local symptoms* are in greater part readily explained on inspection of the entrance to the pharynx. Most of the pathologico-anatomical characters already described are easily recognizable. Sometimes the palatine arches are pushed for-

[1] Consult *Griesinger*, Arch. f. phys. Heilk. IV. p. 515.

ward to an abnormal degree. Only occasionally are they so broad that the hypertrophied tonsil cannot be seen by direct vision; in most instances the tonsils project far beyond them. The most striking appearance is the narrowing of the passage into the pharynx. The *difficulty of swallowing* thus produced is sometimes slight and sometimes severe, and in either case is not always proportional to the degree of enlargement of the tonsil. It is never wanting in a high grade of the affection, but it is sometimes not perceptible to the patient, though distinct enough, usually, to objective observation. *Breathing* is much more affected; when very large tonsils perceptibly narrow the upper pharyngeal space, breathing is principally, or only, possible through the mouth. The mouth is therefore always maintained half-open, giving the patient a somewhat stupid look. The difficulty of breathing is slight in the erect posture, but on lying down, especially at night, it causes unquiet sleep, frequent outcries, sometimes symptoms similar to those of night-mare, or actual paroxysms of suffocation. Frequently there is loud snoring. Not infrequently the patient is often awakened at night by thirst, which is a result of keeping the mouth open. The *speech*, in high grades of hypertrophy, is less distinct, even nasal. The enunciation of *l* and *r* is especially indistinct (so-called *paralalia literalis*). Little children acquire speech with more difficulty. The range of the voice is usually circumscribed. The pitch of tone is, as a whole, elevated. Hearing is frequently affected, in consequence of pressure of the tonsil upon the pharyngeal extremity of the Eustachian tube, etc., but more so probably from extension of the catarrh of the mucous membrane (Harvey).

The affection itself is altogether painless.[1]

A further series of local symptoms is only present at times. They are not consequent on the enlargement of the tonsil in itself, but upon the frequently coexisting catarrh, etc. Slight hemorrhages from the surface occur not unfrequently, especially at night; the patients expectorate buccal mucus mixed with blood.

[1] Consult Bennati, Die physiol. u. path. Verh. d. menschl. Stimme. Uebers. 1833. [Recherches sur le méchanisme de la voix humaine; and Recherches sur les maladies qui affectent les organes de la voix humaine. Paris, 1832. ?—*Tr.*]

The contents accumulating in the dilated lacunæ, and decomposing there, sometimes produce a foul odor of the breath. This also often provokes the frequently recurring superficial or lacunal catarrh—the former sometimes resulting in an actual hypertrophy of the mucous membrane, the latter in purulent accumulations, etc. Extension of the hyperæmia into the nasal mucous membrane produces chronic coryza and frequent epistaxis in some individuals. Sometimes the catarrh extends regularly to the larynx, and there is hoarseness or obstinate cough.

The disposition to croupous or diphtheritic angina is probably increased. In all diseases which are associated with enlargement of the tonsils, the local disturbances become greater.

Severe grades of hypertrophy of the tonsils, when they occur in childhood, and when they are of long duration, sometimes exert an influence on the *conformation of the countenance and the thorax.* The *face* does not grow in proportion to the remainder of the body, nor does the external nose; the nostrils are frequently sunken in. The roof of the mouth also remains small, and is more strongly arched; the upper alveolar process of the upper jaw especially is very small, and in this way the space for the teeth in the upper jaw becomes narrowed, so that the anterior teeth especially assume an irregular position, and encroach in part upon one another. According to some, this deformity depends upon the diminished patulousness of the posterior nasal openings; these and the contiguous structures remaining backward in development in consequence of lessened function (Robert). The deformity of the *thorax* varies. Most frequently it is compressed laterally in its inferior portion, the sternum is somewhat bulged forwards, and the vertebral column is bent—similar to the *pectus carinatum* (Dupuytren). Less frequently the lateral walls of the thorax are more prominent, the breast-bone is depressed, and the vertebræ are little or not at all bent (Coulson, Warren). In both classes of cases the muscles of the chest are sometimes remarkably weak.

Deformity of the thorax takes place after several years' continuance of the hypertrophy of the tonsils. It may disappear with the reduction in size or the extirpation of the tonsils, even when it has existed to a great degree (Shaw, Pitha, and others). Its cause is supposed to be the diminished entrance of air into the

lungs, so that the external pressure of the atmosphere produces a compression of the thorax (Dupuytren, Shaw, and others). Robert compares the condition of the parts to a syringe whose walls offer but little resistance, and therefore, when the opening is small, sink in if the piston is suddenly withdrawn.

In occasional cases there is almost continuous headache, in the forehead.

The *general system* is almost always impaired in severe forms, and after the continuance for years of hypertrophy of the tonsils. The patients are usually pale and thin ; less frequently the face is somewhat puffy and slightly bluish. These manifestations, also, disappear quickly after the cure of the enlargement of the tonsils.

Smith (Med. Times and Gaz. 1865. No. 786) refers to the danger of administering chloroform in cases of hypertrophied tonsils.

The *course* of tonsillar hypertrophy is always *very chronic*. The increasing hypertrophy may be arrested at any stage. This is the most frequent course. Or hypertrophied tonsils may gradually decrease in size, usually without special treatment—sometimes by the actual influence of gradual atrophy, as frequently occurs during puberty, or before it, or at a later age, sometimes under the influence of inflammatory atrophy. Finally, the tonsils may become so large, and the local and concomitant disturbances may become so important, that extirpation then becomes necessary.

Treatment.

No prophylactic measures are known which will prevent hypertrophy of the tonsils. Cold ablutions to the throat or the entire body, the former several times a day, and assiduous gargling with cold water, at least lessens the frequent recurrence of tonsillar catarrh, and thereby perhaps the increase in the hypertrophy also. For the latter condition itself all the various mouth-washes and gargles, the local applications of the various astringents by pencillings, etc., are probably without effect. The value of strong astringents and of superficial caustic remedies is also very questionable (nitrate of silver in solutions from

fifty grains to two drachms to the ounce, concentrated acids, caustic lime, tincture of iodine, etc.). I have seen at most superficial ulceration, but never an actual diminution of the tonsil, in cases of great hypertrophy, even after applications continued with regularity for months. The value of mud and sulphur baths, the latter much recommended by the French, is equally doubtful.

Extirpation of the tonsils is to be undertaken in all cases where swallowing, breathing, or speaking is seriously impeded, where the hearing is affected, where deformity of the thorax is present, where the general condition is disturbed without any further apparent cause, and, finally, where frequent angina occurs, especially with a tendency to extension into the larynx. In many cases the extirpation of the inner half or a large portion of the tonsil suffices; the remainder then performs its functions like a normal tonsil. Extirpation is also recommended by some observers during the existence of an acute tonsillar angina.

Voltolini pierces the hypertrophic tonsil with the galvano-cautery.

VI. Syphilis of the Soft Palate.[1]

The soft palate is, next to the skin, the most frequent seat of syphilitic disease. This is very seldom primary, usually secondary, less frequently tertiary. In reference to its method of appearance, etc., everything is true which is true of syphilitic affections in general. The following forms of syphilis of the palate are in many cases easy to recognize, and to differentiate from each other; more rarely they merge into each other. Not unfrequently the physician is brought into contact only with the severer forms (and with these not until they have existed for a long time), especially among the poor, the previous conditions not having been attended to on account of the slight amount or total want of pain, difficulty of swallowing, etc.

Syphilis of the palate usually affects both halves of the palate

[1] For history and literature of syphilis of the palate, see Vol. III. of this cyclopædia.

in equal or nearly equal degree, more rarely one side alone or to a greater degree. Most frequently it affects the surface of the tonsils, either alone or with the contiguous arches of the palate; next in frequency comes the uvula, then the remainder of the soft palate with the palatine arches.

1. *Syphilitic Erythema. Angina Syphilitica Erythematosa seu Catarrhalis.*

Catarrh frequently constitutes the first manifestation of secondary syphilitic disease of the mucous membrane, and also of the hereditary affection, but it usually produces such slight symptoms that it is not noticed by the patient. The catarrh usually involves the entire soft palate. It sometimes presents itself as an ordinary catarrh, sometimes with slight œdema, sometimes with the character of slight phlegmonous inflammation. It has no special characteristics from which the syphilitic nature of the affection can be recognized. Only its gradual commencement, its slight subjective disturbances, and its long duration, may excite suspicion. Sometimes fever exists at the same time. Frequently syphilis of the skin is present at the same time, in the form of roseola or papules. Syphilitic catarrh ceases of itself or after special medication, or it passes into the form next to be considered.

Chronic syphilitic angina seldom results from the previous form, but oftener remains after the following form, especially in those who have been subject to anginas before contracting syphilis. It likewise presents nothing characteristic.

2. *Syphilitic Alteration of the Epithelium.*

The under portion of the mucous membrane is generally diffusely, less frequently highly reddened, and probably always slightly infiltrated with cells; the surface is colored whitish, over a varying extent and contour; it is slightly elevated, smooth, or slightly rough. This condition is found most frequently on the surface of the tonsils, and in their entire extent. The whitish coloration, according to preparations which I made

in specimens taken from the living subject, is mostly due to a suppuration of the epithelium, less frequently to a true croupous metamorphosis of the epithelium. In the latter case the deposit is usually thicker, similar to that of non-specific pharyngeal diphtheritis (see p. 954). In all other respects this form is similar to that previously described.

3. *Syphilitic Papules and Patches. Angina Syphilitica Papulosa.*

Upon the mucous membrane, which is as a rule diffusely and moderately reddened, though sometimes entirely pale, are various prominences, frequently of the size of lentils, round or roundish, isolated or more frequently confluent, which depend upon a uniform small cellular infiltration of the mucous membrane, not unfrequently of the submucosa also, an œdematous condition of the latter being often also present at the same time. These elevations are always pale, mostly milk-white in color, and first catch the eye in consequence of this color. This appearance is produced by the cellular infiltration and anæmia of the mucous membrane. The surface of the elevations is sometimes smooth, sometimes rough, in the manner mentioned in section 2 (p. 979), and from the same causes there specified. This form of syphilitic angina comes most frequently under medical treatment, and may also be hereditary. The subjective disturbances are mostly moderate and similar to those of ordinary catarrhal angina. They vary according to the number and location of the diseased spots, and still more according to the severity of the catarrh of the non-papulous portions of the palate. Syphilitic patches disappear by absorption, after a variable length of time, either spontaneously or under the use of medicine ; or they ulcerate, superficially or to a variable depth. At the same time the same affection is frequently found at various portions of the mucous membrane of the mouth, on the inner surface of the lips, and at the corner of the mouth ; and papulæ, psoriasis, etc., may coexist upon the skin.

4. *Syphilitic Nodes; Syphilitic Infiltration. Angina Syphilitica Gummosa seu Parenchymatosa.*

Syphilitic patches pass in manifold ways into syphilitic nodes, or the latter exist as such from the beginning. There is then formed a roundish infiltration, mostly single, which may be as large as a cherry, and which undergoes destruction and ulceration with tolerable rapidity. The contiguous parts are only moderately catarrhal. The disturbances are usually proportionately slight. These nodes, according to their seat, either form a deep ulcer, or produce perforation of the palate, less frequently of one arch of the palate. Of especial importance and of tolerable frequency are the nodes developed on the posterior surface of the velum, mostly over the base of the uvula; they often occur without being noticed, and are only to be suspected when ordinary examination of the palate shows collateral œdema of the parts. They speedily perforate the soft palate, not unfrequently with partial or complete detachment of the uvula.

5. *Syphilitic Ulcers.*

These may proceed from any one of the three last-mentioned forms. Those proceeding from the second form are erosions or shallow ulcers; those from the third form are deeper, and likewise the most frequent; those of the fourth form are the deepest, or they perforate the palate or the palatine arches, sometimes the tonsil also. These ulcers occur on any portion of the palate. Their size, number, configuration, etc., vary very much. Their floors exhibit the known peculiarities according to their age and stage of existence. These are not characteristics in themselves. An accurate diagnosis is only possible from the condition of the parts surrounding the ulcer, from the frequently simultaneous existence of syphilitic alterations of epithelium or patches on the remaining portion of the palate, or in the cavity of the mouth, including the lips, and from the history of the case. Where these participations of the surrounding tissue, etc., are wanting; where, also, catarrhal, phlegmonous, tuberculous, and other ulcers are apparently to be excluded; where, finally, local

treatment is unsuccessful, the diagnosis can only be made by the healing of the ulcers under the influence of anti-syphilitic treatment. The sufferings of the patient are those of an ordinary chronic catarrhal angina; they are comparatively slight. Not unfrequently lack of cleanliness produces a foul breath.

I have twice seen small, serpentine yellowish stripes, some of them as much as an inch in length, in the vicinity of rather deep syphilitic ulcers of the uvula and the contiguous palatine arches—apparently lymphatic vessels, filled with pus, or an affection of the parts surrounding the lymphatics. They disappeared at the same time that the ulcers healed.

Syphilitic ulcers heal, when superficial, without leaving a cicatrix. The deeper ulcers heal under the formation of cicatrices (see section 6).

6. *Syphilitic Cicatrices.*

Syphilitic cicatrices of the palate occur almost always after previous deep ulcers. They are found but very seldom without the simultaneous formation of ulcers. They possess no special characteristics. But as other sorts of deep cicatrices are very infrequent in the palate, we have the right to regard every large undoubted cicatrix in the palate, especially in its central portion, as of syphilitic origin, if there has been no operative process, or lupous affection, and if previous severe angina, especially that of scarlatina, has not existed.

Syphilitic cicatrices occur in different forms, as follows:

a. As thin milky spots, of varying size, most frequently on the surface of the tonsil; sometimes, also, on that of the contiguous palatine arches;

b. As deeper, whitish or yellowish-white spots, of varying size, which occupy either the largest portion of the palate, or only one portion of the middle third of the palate, or of the palatine arches, or the tonsils, or the uvula. The position of the adjoining portion of the palate is normal, or may be distorted in various ways;

c. As perforations of the central portion of the palate, or of the palatine arches and tonsils;

d. As irregularly striped cicatrices of the palate, or as regular

or irregular lobulation of the tonsil. The first form corresponds to the deep cicatrices of gangrene of the external skin; the latter are analogous to the well-known lobulation of the surface of the liver and the infrequent lobulation of the tongue. Both of them, also, may be produced by syphilitic processes in the interior alone, without simultaneous syphilitic ulceration of the surface;

e. As distortions of the uvula from deep, but usually, at the same time, from superficial destructions, which leave weals behind them, analogous to those of the sterno-cleido-mastoid muscle, etc.; the uvula is bent forwards or backwards or to the side;

f. As syphilitic defects of the entire soft palate, or of only one-half, or of one palatine arch, and of the uvula in almost its entire length, or as syphilitic tonsillar atrophy;

g. As syphilitic adhesion of the palate to the posterior wall of the pharynx; sometimes total; either with the preservation of the uvula, and then, with absence of all communication between the upper and lower portions of the pharynx (that is to say, between nose and larynx), or after loss of the uvula, and then with slight, sometimes more extensive communication; at other times the adhesion is partial, the modifications in form, extent, locality, etc., being very various.

The *tonsils*, whose surface is usually simultaneously affected in the above-mentioned forms, frequently exhibit alterations in their parenchyma, the amount of which does not always bear any relation to that at the surface. These are mostly wanting only in those instances in which the tonsil is in an advanced state of atrophy. These alterations are: hyperæmia, serous infiltration, parenchymatous inflammation, syphilitic infiltration, with consecutive ulceration or without it, and with lobulation of the surface. In the last instance the organ is composed almost entirely of connective tissue, traces only of the follicular tissue being still present.

The immunity of the soft palate from syphilis in one of the forms mentioned, especially also from syphilitic cicatrices, while syphilitic affections show themselves in the lips, tongue, remaining mucous membrane of the mouth, and the pharynx, is more frequently met with in women than in men. The latter appear to be more disposed to syphilitic affections of the palate by indulgence in smoking, etc.

Treatment.

The treatment of syphilitic affections of the palate is both local and general. The local treatment consists in abstinence from injurious practices (the use of irritating food and drink, smoking, etc.), and in the employment of cleansing or disinfectant astringent mouth-washes, gargles, etc. The general treatment is the well-known mercurial treatment.

VII. Morbid Growths of the Soft Palate.

Concerning hypertrophy, of the tonsils especially, and concerning syphilitic morbid formations, see pp. 972 and 978.

Besides those mentioned, almost all the morbid growths known are observed on the palate, a circumstance readily explained by the multiplicity of the tissues of which it is composed, and the various injurious influences to which it is subject. The morbid formations are primary, or secondary—extensions from contiguous tissues. They are circumscribed or diffuse. Some of them are altogether unimportant or especially of surgical interest. Others, on the contrary, especially those which are diffuse, are particularly of clinical interest. Most of the latter pass into ulceration, and frequently first come under observation in this stage.

Neoplasms of the soft palate are not frequent, if we except hypertrophy and syphilis, and are little known clinically or pathologico-anatomically. In what follows, only those which interest the medical practitioner will be more particularly described.

Clinically considered, morbid growths are important, by their local action in impeding swallowing, etc.; by their extension upon the contiguous parts; and by the fact, peculiar to some of them, of simultaneous occurrences taking place in other organs and tissues. *Etiologically*, nothing is known concerning them. *Therapeutically*, internal medication is entirely fruitless; surgical assistance only is indicated[1] (extirpation, hot-iron, galvano-cautery).

[1] Consult *Passaquay*, Tumeurs des Amygdales. Paris. 1873.

1. *Neoplasms of Connective Tissue and Vessels, of Fatty Tissue, and of Muscular Tissue.*

Diffuse connective-tissue new formations, in the form of *hypertrophy*, either of the uvula alone, or of the entire soft palate, and sometimes, also, of the tongue, etc., have occasionally been observed in entirely healthy subjects, as well as in cretins. But they are very rare, and are almost always congenital.

Very rarely *fibromas* occur in the form of small tumors in the palate and in the tonsils.[1]

Myomas likewise occur very seldom.

I have seen one small flattened myoma, the size of a small pea (myoma striocellulare), sharply circumscribed, in the submucosa of the posterior surface of the soft palate.

Lipomas are also infrequent.

Lambl (Aus. d. Franz-Josef-Kinderspit. 1860, p. 181) describes a pedunculated lipoma the size of a pear, *lipoma pharyngis cum indumento dermoidali*, which probably sprung from the posterior surface of the palate of a female child six months of age, and which became detached spontaneously.

Primary *sarcomas* appear to be exceedingly infrequent in the soft palate. Somewhat more frequently they spread from the contiguous tissue, especially from the fissures and cavities of the neighborhood on to the lateral portions of the palate.

Papillomas possess characteristics sometimes more like those of warts, sometimes more like those of a chondyloma. They are the most frequent of the circumscribed morbid growths, usually smaller than peas, seldom larger, and have broad or narrow pedicles. Their favorite seat is on the uvula; they are less frequent on the remaining portions of the soft palate and on the tonsils. Most frequently they exhibit no symptoms of their existence; occasionally they give rise to slight symptoms, and still less frequently to severe symptoms.[2]

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[1] *Curling*, The Lancet. 1858. No. 6.

[2] Consult the case of *Herzfelder* (Wbl. d. Z. d. Wien. Aerzte. 1856. No. 30), in which hysterico-epileptic convulsions always set in on lying down, in a patient nineteen years of age. After extirpation these symptoms disappeared.

2. *Lymphatic Morbid Formations.*

In *typhoid fever* new formations, similar to those occurring in the bowels, etc., occur but very unfrequently.[1]

In *leukæmia*, the lienteric as well as the lymphatic form, lymphomas sometimes occur in the mucous membrane of the soft palate and the pharynx; the tonsils are transformed into medullary tumors of different sizes. These conditions, when strongly developed, are visible during life.[2]

In *lepra* similar nodes occur on the palate, etc., as on the skin.

Lupus of the soft palate occurs almost always simultaneously with lupus of the nose or of the face. It sometimes affects the palate only, sometimes the pharynx and upper portion of the larynx at the same time. It usually has the form of *lupus exulcerans*, and then either proceeds to cicatrization without great loss of substance, or it may destroy the palate in varying extent, after which, likewise, cicatrization or adhesion to the posterior wall of the pharynx, etc., may ensue. The *treatment* is similar to that for lupus of the skin.

Simultaneous lupus of the face and of the palate is not infrequent. The mucous membrane of the mouth may be normal, or there may be lupous morbid growths upon the lips and the gums. When lupus affects the palate alone, its differentiation from some syphilitic infiltrations and ulcers is the more difficult, because some forms of lupus are without doubt of syphilitic nature; at least they heal promptly under the use of mercury and iodine, even when they have existed for months and years. The cicatrices of healed lupus are characteristically striated like those of the external skin. The tonsils, in such cases, are often hypertrophic.

Tuberculosis of the soft palate occurs only simultaneously with advanced tuberculosis of other organs, especially of the lungs, usually of the larynx also, especially its upper portion, of the root of the tongue, and of the pharynx. At first there is

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[1] Concerning the alterations in the tonsils in typhoid fever, hypertrophy of the follicles, etc., see *C. E. E. Hoffmann*, Unt. üb. d. path-anat. Veränd. der Org. beim Abdominaltyphus. 1869. p. 158.

[2] Consult *Mosler*, Virch. Arch. 1868. XLII. p. 444.—*Idem*, Die Leukämie, 1872, p. 181.—*Valtat*, in Passaquay, l. c. p. 41. The tonsils healed similarly in a case of lienteric and at the same time lymphatic leukæmia that I observed myself.

a tolerably uniform thickening and infiltration of the mucosa and submucosa, usually of the entire soft palate, frequently including the tonsils. This renders the surface pale. Frequently a suppuration of the epithelium occurs, which gives the surface an appearance similar to that of a flat croup deposit. Tuberculous masses, from the smallest size to that of peas, occur but seldom in addition to the uniform flat infiltration. Furthermore, numerous shallow ulcers usually occur, which coalesce at a later period, and produce fissures of various depths on the surface of the tonsils. It seldom amounts to extensive and deep ulceration in the soft palate, while these conditions are more frequent in the pharynx. The lymphatic glands of the jaw are likewise tuberculously infiltrated, but generally only to a slight degree. Microscopically tuberculosis of the palate exhibits nothing special. The deeper tuberculous ulcers almost always show the muscles likewise infiltrated with tubercles.

Clinically the diagnosis is easy, from the symptoms mentioned, and from the existence of extensive tuberculosis of the lungs, etc. The disease is most frequently confounded with some syphilitic affection of the palate. The variation in the painfulness of the two is also common to both classes of affections, only tuberculosis appears to be painful more frequently than syphilis, and sometimes causes very serious difficulty in swallowing.

Tuberculosis of the palate is much less rare than one would surmise from the sparse pathologico-anatomical and clinical descriptions. Consult *Weber*, in Pitha, Billroth's Chir. 1866. III. 1. Abth. pp. 337 and 360.—*B. Wagner*, Arch. d. Heilk. 1865. VI. p. 470.—*E. Wagner*, ibid. 1870. XII. p. 5.—*Oulmont*, Gaz. des Hôp. 1866. No. 43.—*Trélat*, Arch. gén. Jan. 1870.

The so-called *scrofulous ulcers* of the palate and pharynx, *angina scrofulosa*, are not yet sufficiently studied to admit of any general exposition. The cases referred to this category, which usually affect the pharynx at the same time and to a greater degree, are perhaps real scrofulous ulcerations similar to those of the skin; or they are secondary, or extensions of previous tuberculous ulcerations, or they have a lupous, leprous, or syphilitic character.[1]

[1] Consult *Hamilton*, Dublin Journ. 1845. XXVI. p. 282.—*Isambert*, Gaz. hebd. 1871, No. 47. L'Union. Nos. 3–6.

3. *Epithelial Morbid Formations.*

Epithelial thickenings (so-called psoriasis oris) on the surface of the tongue, on the lips, and on the inner surface of the cheeks, occur most frequently in habitual smokers, occasionally as the result of other causes, or even in women.

I have only seen them on the most anterior portion of the soft palate and simultaneously upon the hard palate also, but always only to a very slight extent. In those cases which I saw, the same affection existed at the time upon the skin of the hands, nose, and ears, and showed an inclination partly to the formation of crusts and partly to the development of callosities or warts. In the palate they were regularly pierced by the mouths of the mucous glands.

Mucous polypi, *adenomas*, are likewise very seldom found in the soft palate, and then most frequently on its borders as well as at the base of the uvula. They are usually single, seldom double, and when double they are sometimes symmetrical, with a broad base, more rarely pedunculated, and of a size which may reach that of a walnut or even a hen's egg. They have the known characters of these tumors, and sometimes contain cysts in places; sometimes they contain smaller or larger calculi. They occur more frequently in youthful subjects. I saw one tumor, the size of two peas, in a child three weeks of age.[1]

4. *Cancer.*

Cancer of the soft palate and of the tonsil occurs but rarely. The most frequent method of its occurrence is by spreading from contiguous parts; it is more rarely primary, and most rarely secondary, appearing then in the form of one or more little nodules. Cancer may extend upon the palate from all portions of the surrounding structures, most frequently from the base of the tongue and from the pharynx, less frequently from the

[1] Consult *Rennes* and *Robin*, Gaz. des Hôp. 1855. No. 41.—*Nélaton* and *Robin*, ib. 1856. No. 143.—*Anselmier*, L'Union. 1856. No. 128.—*Langenbeck*, Deutsches Klinik. 1859. No. 48.

larynx, the mucous membranes of the cheeks, the nose, the parotid gland, and the base of the skull. Primary cancer occurs more frequently in the tonsils than in the other portions of the soft palate.

Both primary cancers and cancers from extension are most frequently epithelial in character, less frequently of the soft connective tissue variety (so-called encephaloid). The first occur almost exclusively in elderly persons; the latter at all ages, often in young subjects. The first give rise in general to slighter infiltrations, and usually have a slower course; they are sometimes very markedly papillary. The latter usually form larger, rapidly-growing tumors, more frequently starting from the tonsils. Both forms are greatly disposed to ulceration.

Secondary cancer of the palate, under certain circumstances, may be of diagnostic interest. I once saw small secondary nodules in the closed follicles of one palatine arch and the root of the tongue in a case of primary encephaloid of the root of the lung.

Some diffuse morbid growths in the palate and pharynx, recently described as lymphoma, lymphadenoma, etc., are encephaloid.

The *diagnosis* of cancer is usually easy, because it does not often come under observation in its earlier stages. The most important diagnostic indications are: the presence of a new formation, which extends upon all the surrounding parts; its slow course, as compared with that of an inflammation; its ulceration with or without previous or simultaneous hemorrhage; and the usual existence of swelling of the lymphatic glands of the jaw.

The *local disturbances* are very various, according to the extent and volume of the cancer, but are not distinguishable from those dependent on ordinary causes. The affection may be altogether painless. The *general symptoms* vary according to the nature of the local symptoms, the existence of hemorrhage, the extension upon contiguous organs, etc.

The regular *termination* is in death; usually by starvation,

hemorrhage, suffocation, etc.; less frequently by secondary cancer.[1]

5. *Cysts.*

Cysts of the palate are mostly retention cysts, rarely new formations. They originate but rarely in the mucous glands, and may form circumscribed tumors reaching the size of cherries, similar to the like occurrences in the lips. They take their origin much more frequently in the lacunæ of the tonsils, and then usually have the characters of atheromas (see p. 969). *A new formation of cysts* is very infrequent.

In a women eighty years of age, I saw on the posterior surface of the soft palate above the uvula, a cyst the size of a half pea, with fatty, curdy contents, enveloped with a single layer of ciliated epithelium.

Echinococcus has been observed in the palate only a few times.

VIII. Nervous Affections of the Soft Palate.

The nervous affections of the palate will only be treated of concisely here, because they will receive a detailed consideration in a later volume of this series. They affect mobility, sensibility, and nutrition.

1. MOTOR DISTURBANCES.

A. *Paralysis of the soft palate.*

Paralysis of the muscles of the soft palate either occurs alone, or simultaneously with paralysis of other muscles, most frequently those supplied by the facial nerve. There may be central paralysis, paralysis of conduction, or peripheral paralysis. Paralysis following diphtheria, the form most frequently encountered, probably belongs to the latter class. It may be complete or incomplete (paralysis or paresis). It may be simple or

[1] *O. Weber*, Chir. Beob. p. 364.—*Poland*, Brit. and For. Med.-Chir. Rev. 1872. XLIX. p. 477.

associated with disturbance of sensibility or of nutrition. The paralyses occurring simultaneously with catarrh, phlegmonous inflammation, morbid growths, etc., are thrown out of consideration here.

a. *Isolated paralysis of the palate* may affect individual muscles only, or all the muscles collectively.

a. Paralysis of the azygos uvulæ muscle may be unilateral or bilateral.

In *unilateral paralysis* the uvula is bent towards the opposite side. Swallowing and speaking are not impeded.

In *bilateral paralysis* the uvula is elongated, and cannot be retracted spontaneously nor by local irritation (touching it, etc.). Deglutition and articulation are not impeded. When the uvula is so long that it touches the root of the tongue, in the standing and sitting postures, involuntary movements of deglutition occur not unfrequently. If it touches the posterior wall of the pharynx, adhering to it at times, frequent hawking is produced.

Paralysis of the uvula alone has no significance in determining the seat of the nervous disease. A distortion and an elongation of the uvula, with slight power of retraction, or none at all, also occurs very frequently after repeated anginas, in chronic angina, as a consequence of cicatrization, etc.

β. The levator and the tensor veli palatini muscles are almost always paralyzed simultaneously, and almost always both sides are affected—sometimes, however, in unequal degrees. The soft palate is depressed to a varying degree, and appears relaxed; it is but little or not at all elevated in breathing. If the soft palate, the uvula especially, is touched, no movement is produced in pure cases of complete paralysis. If the tensor is not completely paralyzed, the palate is made somewhat tense, but is not raised. It is usually drawn somewhat downwards and backwards by the pharyngo-palatine muscles. Swallowing is impaired to a high degree; the food taken, especially if liquid, is regurgitated in greater part through the nose; but not completely, if the superior constrictor muscle of the pharynx remains normal. Speech is nasal, indistinct, especially in the palate sounds. Sucking, gargling, blowing a light out, and forcible distention of the cheeks, are impossible.

γ. *Paralysis of the pharyngo-palatine muscles* occurs extremely seldom as an independent affection. In one case of Duchenne's, irritation of the uvula did not produce any mutual approximation of the posterior palatine arches, and no formation of a division-wall between the upper and middle portions of the cavity of the pharynx. Swallowing was somewhat impeded, but speech was not implicated.

Paralysis of the glosso-palatine muscles has not been observed as an independent affection.

δ. *Paralysis of the muscles of the soft palate collectively* often occurs. The soft palate is depressed, and does not move in breathing or on local contact with a foreign body. Swallowing is very difficult, or altogether impossible, etc.; all the other relations are the same as those mentioned under the headings α, β, and γ.

b. *Paralysis of the muscles of the palate and of other muscles.*

α. *Simultaneous paralysis of the palate and of the facial muscles.* The causes of this affection reside in the nervous centres (the hemispheres, the pons, and most frequently the medulla oblongata), or in the course of the facial nerve (caries of the temporal bone, pressure of the forceps in artificial delivery), or in the periphery (rheumatic and traumatic causes), or they are unknown (diphtheritic paralysis). The paralysis is almost always unilateral, extremely seldom bilateral. It most frequently affects one-half of the uvula, and one levator veli palatini. In slight cases the uvula is normal, or it moves on contact towards the healthy side only; the palatine arch is somewhat flatter. In more pronounced cases the uvula is drawn towards the sound side, and the palate hangs lower. (Various observations, differing from these, may be found elsewhere.)

β. *Simultaneous paralysis of the palate and the corresponding half of the body* occurs in some cases of hemiplegia from cerebral hemorrhages, etc.

γ. *Simultaneous paralysis of the palate and the motor portion of the trifacial nerve* affects the tensor veli palatini and one or more of the muscles of mastication. The cause is usually central.

δ. Paralysis of the palate in *progressive glosso-pharyngo-labial paralysis* is usually but little pronounced, so far as can be ascertained by inspection, but is more strongly indicated by destruction of function.

ε. Paralysis of the palate in *progressive muscular atrophy.*

λ. Paralysis of the palate in *acute ascending paralysis* (Landry).

B. *Spasm of the soft palate.*

Spasm of the soft palate is almost entirely unknown. Even the behavior of the affected muscles in spasms proceeding from the facial nerve, and in those proceeding from the motor filaments of the tri-facial, is unknown.

In advanced cases of *paralysis agitans* movements occur in the soft palate similar to those in the muscles of the exterior. In a patient, with constitutional syphilis and paralysis of one-half of the body (without involvement of the palate), I saw twitching movements, synchronous with the pulse, in the left and sound half of the palate.

2. SENSORY DISTURBANCES.

Anæsthesia of the soft palate, mostly with diminished reflex irritability, is found in insane patients, and also in consequence of the influence of some substances upon the periphery (ice, bromide of potassium, morphine, lye, etc.). In diphtheritic paralysis there is almost always paralysis of sensation likewise.

Hyperæsthesia of the soft palate occurs as well with the maintenance of a normal appearance of the parts, as in the various disturbances of circulation and the inflammations. Characteristic neuralgias, analogous to those of other branches of the trigeminus, have not been accurately observed.

Türck (Wien. allg. med. Ztg. 1862. VII. No. 9) describes a neuralgia and hyperæsthesia of the pharyngeal entrance; his description is based upon some six cases (two in males, four in females), most of them unilateral, but always more severe on one side. The symptoms were various—sometimes very severe pains, at other times only difficulty in swallowing, a sensation as of a tumor or foreign body, or of dryness in the parts. The duration varied from six weeks to six years. Treatment: Cauterization with nitrate of silver; resection of the lingual nerve.

3. DISTURBANCES OF NUTRITION.

These are still less known than the disturbances of sensation. The palate and uvula are sometimes atrophic in *hemiatrophia facialis*, which mostly affects the left side of the face.

Appendix.

Diseases of the muscles of the soft palate alone are not known. Where they occur simultaneously with diseases of other muscles or of all the muscles of the body, *i. e.*, in the fatty metamorphosis from poisoning by phosphorus, etc., and in the trichinous disease, the participation of the palate is devoid of practical interest.

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