

SLADE (D. D.)

PRIZE ESSAY.

“TO WHAT AFFECTIONS OF THE LUNGS DOES
BRONCHITIS GIVE ORIGIN?”

“DISCERE ET AUDIRE.”

BY DANIEL DENISON SLADE, M.D.

OF BOSTON.



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Extracts from the Records
OF
THE MASSACHUSETTS MEDICAL SOCIETY.

At a Meeting of the Councillors, Oct. 7, 1857,—

The Treasurer announced, that, through the liberality of one of its Fellows, the Massachusetts Medical Society is authorized to offer the sum of One Hundred Dollars to the author of a Dissertation which may be adjudged worthy of a prize by a Committee appointed by the Councillors of the Society, on the following subject, viz. :—

“To what affections of the lungs does bronchitis give origin?”

*At a Meeting of the Councillors, Oct. 7, 1857, the following gentlemen were appointed
the Prize Committee, viz. :—*

Dr. CHARLES E. WARE.

Dr. OLIVER W. HOLMES.

Dr. HENRY I. BOWDITCH.

Dr. WILLIAM W. MORLAND.

Dr. CHARLES E. BUCKINGHAM.

At the Annual Meeting of the Society, May 25, 1859,—

Dr. WARE, Chairman of the Prize Committee, reported that only one Essay had been received, and that one had been unanimously adjudged worthy of the prize; and handed to the President the envelope bearing the motto of the Dissertation. On breaking the seal, the author was found to be Dr. DANIEL DENISON SLADE, of Boston, Mass.

ESSAY.

BEFORE entering directly upon the consideration of the subject proposed, it may be well to establish definitely, what we would understand by the term, Bronchitis.

We restrict the application of this term, to inflammation of the mucous membrane lining the bronchial tubes. This inflammation admits of being divided nosologically into two forms, the distinction referring to difference in seat. In one form, the disease is confined to the larger bronchi, and constitutes what is known as ordinary bronchitis. In the second form, the inflammatory action is confined to the smaller air-tubes, or, as is more commonly the case, it affects them and the larger ones also.

Another division of bronchitis is based on the duration and degree of the inflammation; hence it is commonly divided into acute and chronic.

The inflammation may be developed in the bronchi, either as a primary, idiopathic disease, or it may occur as secondary to some general disease. It is with the first of these that we have to deal in the following pages.

The general symptoms of bronchitis are too familiar to require of us any detailed description. We shall therefore proceed at once to discuss—*First*, the direct and primary results of bronchitis, comprising, under this head, the ordinary effects of this disease upon the mucous membrane of the air-tubes, and their contents; their connection with the

auscultatory and other signs—and, *Secondly*, the secondary and more permanent lesions of the pulmonary organs, which result from the former under certain circumstances. We shall devote attention more especially to the pathological states of the lung, connected with bronchitis—not presuming, however, to offer anything which does not already belong to science.

Bronchitis is essentially an inflammation of the mucous membrane lining the bronchi and their ramifications in the lungs. The first effect of inflammation of a mucous membrane is an increase in the quantity of blood ramifying immediately beneath the surface, with frequently some serous effusion into the areolar tissue, but a diminution, rather than an increase in the secretion of the membrane itself. Now, the effect of this state of things upon the bronchial tubes must be to produce a swelling and dryness of the lining membrane, accompanied with heat and pain, together with more or less tenderness; effects which fully account for the sensations felt in the chest in the early stage of acute bronchitis.

Again, the effect of this inflammatory process upon the lining membrane, is to produce certain changes in the auscultatory phenomena of the chest. As regards the sounds elicited by percussion, we know that the quantity of air in the cells of the lungs is not necessarily affected by this condition of the mucous membrane of the tubes, and consequently we should not expect to find any particular alteration in the resonance of the chest—and experience shows that such is the case. Not so, however, with the sounds of respiration. Here the changes brought about by inflammation are for the most part distinct and well marked.

Owing to the irregularly narrowed calibre of the air-tubes, the result of the thickened membrane, we have the well-known musical sounds, differing in degree as they proceed from the large or small bronchi. In other words, we

have the bronchial dry sounds of respiration, the sibilant and sonorous râles of the early stages of the disease.

So soon, however, as the inflammatory secretion begins to show itself, we have a decided alteration in the stethoscopic signs, the sounds which attend the respiration being of the *moist* kind, and differing according to the size of the tubes, and according to the changes in the secretion itself.

The varieties in the character of the secretion from the diseased mucous membrane are very great; and in order that we may satisfactorily understand the peculiarities to which they may give rise in the auscultatory phenomena, it will be well to give them a brief consideration.

In the earlier stages of bronchitis, the mucus thrown out is profuse, thin, watery and mixed with air-bubbles of various sizes. At this period it contains but few microscopic elements, a few altered epithelium cells being alone visible. At a later stage the mucus has assumed a yellowish tinge, is more tenacious and viscid, and contains numerous pus corpuscles, and more of the altered epithelium cells.

When the disease has existed for some time, and is nevertheless the result of an acute attack, and in those cases where there has been a recurrence of active inflammation, we usually find, on incising the bronchi, numerous yellowish pellets of a somewhat curdy consistence, which float in the more recent and thinner mucus. These masses are no doubt formed by the evaporation of the more fluid parts of the mucus, in consequence of the constant passage over it of dry air; the inspissated materials clinging to the walls of the air-passages, until detached and washed away by renewed exudations from the mucous membrane. Such a mass is not unfrequently seen plugging the whole calibre of one of the larger or smaller bronchi, in such a position as to leave no doubt "that in the act of inspiration it must have acted the part of a ball-valve," completely preventing the access of air to the part of the lung involved, by falling

back upon the orifices of the smaller bronchi, into which its size would prevent it from entering.

When bronchitis has existed for some time in a comparatively mild form, portions of the secretions become inspissated in the form of a thick, glairy, tenacious, somewhat transparent substance, sometimes resembling the raw white of an egg.

The effects of this increase of the secretion, and its morbid accumulations in the bronchial tubes, are easily recognized by auscultation, giving rise to the familiar fine and coarse mucous râles. Now, as long as the secretion is thin and watery, there is no serious impediment to the passage of air, which, as the râles just spoken of indicate, finds its way through the fluid into the minute ramifications of the bronchi and into the pulmonary vesicles. Not so, however, when the secretion within the tubes has become inspissated; the mucus having either become purulent and formed itself into the tenacious pellets before mentioned, or assumed the tenacious, stringy consistence, which we meet with in the chronic form of the disease. When, under these circumstances, expectoration is interfered with, either from the tenacity of the mucus, the debility of the patient, or from any other cause, the tubes become obstructed, the sound of vesicular respiration is much diminished at some points, and perhaps altogether lost, and the bronchial râles are correspondingly modified; the ordinary mucous râles being in part superseded by certain sounds which are well described by Dr. Williams.*

It sometimes occurs, where the inflammation is very intense and very extensive, that the swelling of the lining membrane which occurs at its commencement is sufficient to prevent the access of air to the minute vessels ramifying in the cells of the lungs, and thus speedily cause death by

* Diseases of Chest, Appendix A.

apnoea, before any secretion has been poured out into the tubes.

A more common mode, however, of the fatal termination of bronchitis, is by obstruction to the respiration, and consequent death from apnoea caused by the inflammatory effusion in the tubes preventing the access of air to the pulmonary cells. This may occur at any period of the disease, and arises either from the intensity of the inflammation and its rapid extension to the minuter tubes causing a profuse secretion to be poured into them, or from inability of excretion arising partly from its quantity and partly from the failure of the powers of life. This constitutes the suffocative catarrh of some writers—the capillary bronchitis of others; a form of the disease which generally runs its course with great rapidity, especially in children—and involving the pulmonary vesicles themselves, gives rise through a considerable extent of the lungs to those appearances known under the names of vesicular pneumonia and vesicular bronchitis.

Again, death may occur very suddenly and very unexpectedly in those cases where the disease is idiopathic, and where it has not appeared severe, by the accidental plugging up of one or more of the principal bronchi, by a mass of inspissated mucus. Andral relates two instances of this kind, in the "*Clinique Médicale*,"* in each of which the respiratory murmur became completely suppressed in the upper part of one lung, the patients having been previously affected with moderate bronchitis. The autopsy showed the signs to be due to an obstructive accumulation in the bronchi leading to the upper lobe of the lung; the absence of respiratory murmur combined with clearness on percussion had led, during life, to the diagnosis of emphysema.

It is hardly necessary for us to remark that the morbid

* See Appendix B.

anatomy, as far as it goes, is in perfect accordance with what we learn from the physical signs. Patients seldom die in those cases where the disease is confined to the larger bronchi, and which constitutes acute ordinary bronchitis; and in the other forms, we have much more frequent opportunities to observe the morbid appearances produced by inflammation of the air-tubes, in children.

An increased degree of redness of the mucous membrane of the bronchi, is almost constantly observed, varying much in degree and extent. It may be diffused, or in patches, and may vary in color from a bright crimson to a brown red. In many cases, no other change may be observed in the mucous membrane, but we frequently find it thickened and softened, sometimes to a most unusual extent.

Ulceration of the mucous membrane, which is occasionally met with in the bronchitis of adults, is extremely rare in children.

The inflamed bronchi contain a more or less abundant viscid, transparent, or opaque yellowish mucus. Traces of blood are rarely observed in the secretion.

In capillary bronchitis, the alterations of the mucous membrane of the capillary tubes do not always reveal the existence of the disease. That membrane is sometimes pale in the minute ramifications, and exhibits morbid changes only in those of medium size. The alterations of the membrane consist in redness, which is made up either of fine points seated in the membrane itself, or of arborizations seated both in the membrane and the cellular tissue beneath. It sometimes presents a granulated appearance, and it may be more or less thickened.

The bronchi are usually filled, and almost, in some cases, obliterated, by a substance of a yellowish-white color, non-aerated, and composed of a thick muco-pus. Portions of false membrane are sometimes also found mixed with the secretions just mentioned, while occasionally false mem-

branes alone are found in certain tubes. This false membrane may exist in patches, or it may constitute a lining to the whole extent of the bronchial ramifications. It is usually soft and but slightly adherent.

Not only are the contents of the air-tubes altered in character, but the tubes themselves often undergo a marked alteration in their calibre, becoming dilated. This dilatation is usually observable from the secondary bronchi to the minutest air-tubes, and, in the adult, sometimes becomes fusiform.

It was once supposed that this dilatation of the bronchi was due to the purely mechanical effect of the accumulations of the secretions within them. There is, however, no constant relation between the quantity of the liquids within the bronchi, and the degree of their dilatation. We must therefore look to two other circumstances as being the primary causes of the occurrence. These are, the weakening of the muscular fibres of the bronchi, by the inflammatory action, and the loss of the ciliary epithelium, which lines the air-tubes in their normal state, and which contributes, by the incessant vibration of its cilia, to keep them free for the access of air. These two circumstances lead to certain important changes in the pulmonary tissue, of which we shall speak presently.

Thus far, we have spoken of the immediate effects of inflammation upon the bronchial tubes, and upon their contents.

We have seen that not unfrequently, under certain circumstances, the bronchi become to a greater or less extent obstructed by mucus of a certain degree of tenacity. We have now to advert to that condition of the pulmonary texture which springs more directly than any other from this obstruction of the bronchi, and which is now known and described as collapse of the lung.

By collapse of the lung, is meant the return of that or-

gan to its foetal or unexpanded condition. It is in fact a state of atelektasis, or imperfect expansion of its vesicular structure. The terms, collapse, or *post-natal* atelektasis, are employed to contra-distinguish it from *congenital* atelektasis, the former being applied to imperfect expansion as it occurs in lung tissue after previous expansion, and the latter to the same condition as it exists in children, who have never expanded certain portions of the pulmonary substance.

The true nature of collapse of the lung was never understood, and its practical importance never appreciated, until the year 1844, when MM. Legendre and Bailly published in the "Archives Générales de Médecine" their researches on the subject. Since then, various observers have repeated the investigations of these gentlemen, and thrown new light upon the subject.

Among those who have contributed particularly to our present knowledge on this point, we may mention, besides MM. Legendre and Bailly, MM. Hardy et Behier, of Paris; Dr. W. T. Gairdner, of Edinburgh; Dr. West, and MM. Rilliet et Barthez.*

This new discovery in pathology is one of very great value, not only because it renders our knowledge upon diseases of the lungs more exact than it ever was before, but because it explains certain anatomical changes in these organs, never before understood.

Certain appearances, particularly in children, which were supposed to be the result of pneumonia, had long attracted the attention of pathologists by the wide differences which they presented from those which were produced by the same disease in the adult. Although it had been observed that children under five or six years of age died after presenting some of the usual symptoms of pneumonia, such as cough, dyspnœa, together with more or less extensive dul-

* See Appendix C.

ness of the chest on percussion, and some of the auscultatory signs of solidification of the lung, it happened not unfrequently that both the febrile and pneumonic symptoms very much diminished before any signs of approaching death appeared—and yet these very cases presented these supposed anatomical evidences of pneumonia in a very marked degree. So, again, with children who appeared to die worn out from various causes, and during whose lifetime no indications of inflammation of the lungs had existed.

So frequently were cases of this description observed, that it was assumed that pneumonia was an extremely frequent concomitant of almost all the diseases of infancy and early childhood; that this inflammation of the lungs, owing to certain unexplained causes, did not manifest its existence by its usual symptoms, and gave rise to alterations in the lung very different from what was observed in the adult. One of the principal peculiarities of this supposed inflammation of the lungs in childhood, was, that it did not attack any large portion of the lung, but was confined to isolated lobules, and sometimes even to a single lobule, the boundaries of which could be distinctly traced; whence it derived the name of Lobular Pneumonia. These isolated lobules presented a dark color, were solid, often depressed below the surrounding parts, which appeared healthy, and sank if thrown into water. In some cases, the affection was limited to a single lobule, the boundaries of which could be distinctly traced; while in others, although it happened that a cluster of lobules was thus dark and solid, still there was no gradual shading off from the darker to the lighter parts, so that it was evident, that however the disease extended, it was certainly not by any continuity of tissue merely.

The course of the disease and the results of medical treatment only seemed to add to the difficulties which presented themselves, when it was attempted to refer these

anatomical peculiarities to the same category of affections with the pneumonia of the adult. The usual antiphlogistic routine pursued in the case of the latter, seemed certainly to aggravate symptoms in childhood and to hasten death, while, on the other hand, a stimulant treatment, practised even through the entire course of the disease, seemed often to be followed by most satisfactory results.

Then, again, the rapidity of the changes that took place in the lung, was another perplexing and obscure character of the disease. For where air was heard freely entering on one day, none would be perceptible on the morrow, and on percussion there would be complete dulness over that part of the chest. Again, just the opposite would sometimes be observed; the breathing became distinctly audible, where on the previous day nothing could be heard, and the dulness was succeeded just as quickly by resonance on percussion.

That this condition of which we are speaking, should so long have been described by writers as Lobular Pneumonia, shows most forcibly the influence of a mere name, particularly as it was very evident, from the concurrent testimony of every one, that neither in its results, nor in its progress, was it even similar to inflammation of the lungs in the adult. Having once, however, been called Pneumonia, it continued to be called so, in spite of its peculiarities.

Even the almost exact resemblance which the lung in this condition presented to foetal lung, or to those portions which are characteristic of atelektasis, was noticed and remarked upon by many observers, without the slightest suspicion that these were identical.

Then, again, while these peculiarities of lobular pneumonia were thus commented on, it seems to us now very strange that no one should have thought for a moment of inflation as a means of solving some of the difficulties which surrounded its nature. This seems still more extraordinary, when we remember that this very means had already been

put into use, and had cleared up many doubts concerning certain appearances in the lungs of new-born infants, which had formerly been supposed to be the result of pneumonia in the fœtus, or of some arrest of development.

At length, however, the idea of inflation happily occurred to those indefatigable observers, MM. Bailly and Legendre, and by them the experiment was carried into effect.*

Thus, by this very simple means, more light has been thrown upon the pulmonary affections of infancy and childhood, than can well be appreciated by us who enjoy the results to which this experiment has led.

We have said that obstruction of the bronchi was one of the most direct causes of collapse of the lung, whether this obstruction be the consequence of bronchial inflammation as it is in the great majority of cases, or whether it is the mere natural secretion of these tubes, accumulated for the want of power to throw it off. We must add to this another cause, viz., deficient respiratory power.

It has been found in the case of children, that collapse seldom occurs to any very great extent except in those who are exhausted and debilitated. The debility may be congenital, it may be the result of some wearing disease, or it may be due to unwholesome and enfeebling hygienic conditions. Now it is easy to understand that a child must necessarily, if placed under these circumstances, lose some portion of the muscular power by which alone a complete and efficient dilatation of the thoracic cavity can be produced, and, that when this is the case, the inspirations must be short and imperfect, and that portions of the lung most distant from the primary air-passages not being reached by the inspired air, will remain in an unexpanded or collapsed condition.

* Nouvelles Recherches sur quelques Maladies du Poumon: Archives Générales de Médecine. Jan., Fév., Mars, 1844.

Whether a mere deficiency of inspiratory force alone, without obstructing mucus in the bronchia, will give rise to collapse, is a somewhat doubtful point. MM. Legendre and Bailly are of the opinion that it is often due to the inspiratory power having been inadequate to overcome that natural elasticity of the lung which opposes a full dilatation of the organ. With this opinion Dr. West also agrees. Dr. Gairdner, however, does not believe that mere debility, apart from any obstruction in the tubes, is a sufficient cause for collapse in the child. He remarks that the very fact of the lesion being usually more or less *lobular*, appears certainly to indicate special circumstances of a local kind, as having a decided influence on the production of this affection.

* "It is sufficiently evident, *a priori*, that the occurrence of the *état fœtal* or of the collapse of the pulmonary air-cells in the distinctly limited lobular form in which it is so frequently observed, is not to be adequately accounted for by any influence, acting through the general system (such as constitutional debility), or by a force such as the elasticity of the lung, which not being subject to variation, and being quite equally distributed, cannot well be conceived to affect certain pulmonary lobules to the exclusion of others. The theory of MM. Legendre and Bailly is, as we have seen (and as Barthez and Rilliet have also pointed out), defective in attributing the *état fœtal* principally to the pulmonary elasticity, aided by everything which tends to obstruct the respiratory function. But although their rationale of the lesion is undoubtedly inadequate, we shall find that the observations of MM. Legendre and Bailly give a prominent position to one circumstance in connexion with the *état fœtal*, which has also attracted the notice of other observers. They state that the production of the *état fœtal* is *favoured*

* On Bronchitis. Gairdner, L. and E. Monthly, 1851.

by the accumulation in the bronchi of thick mucus, and in proof of this opinion, they assert that while they have met with this condition in eight cases (in enfeebled infants), independently of all pulmonary inflammation, it has in thirty-nine cases been found connected either with what they call catarrhal pneumonia (capillary bronchitis), or with bronchial catarrh; and in twenty-seven of these thirty-nine cases, it was the pulmonary affection alone which caused death. These details certainly present strong evidences of an intimate connexion between collapse of the pulmonary air-cells, and bronchial mucous accumulations."

After having given the observations of several eminent authors upon this point, Dr. Gairdner goes on to say—"From all these separate observations, converging as it were to a point, it follows clearly enough, we think, that the collapse of the air-cells, when occurring in a lung that has once been expanded, is, in all probability, a secondary lesion, and dependent, in the majority of instances, on a catarrhal condition of the bronchial tubes. This is indeed the most important conclusion arrived at by Dr. Fuchs in the memoir formerly reviewed by us."*

The main advance in the point of view assumed by Fuchs, as contrasted with that of Legendre and Bailly, consists in his having kept distinctly before him the fact of the connexion of the collapse of the air-cells with pulmonary catarrh, and treated this connexion throughout, not as accidental or of secondary importance, but as one involving the theoretical view of the dependence of the pulmonary condensation mainly upon the obstruction of the bronchi by mucus. He gives many cases in illustration of the effects of infantile bronchitis; but by far the most valuable facts which he brings to bear on the subject are from the experi-

* Die Bronchitis der Kinder. Leipzig, 1849.

ments of Mendelsohn and Traube, which leave scarcely anything more to be desired.

Among the most striking of these experiments were the following:—Tracheotomy being performed on a rabbit, a shot or slug (the size is not mentioned) was inserted into the trachea and impacted into the left bronchus by means of a probe. The animal died in two days. "The right lung was large and emphysematous; the left collapsed; the lower lobe in great part red, void of air, as also the upper lobe in some portions, in the midst of which were emphysematous parts. The whole lung could be inflated from the trachea (of course after the removal of the obstacle)." In other instances paper balls and solutions of gum were employed, with results in a great measure the same.

Traube's experiments were very nearly the same. A rabbit, having a paper plug inserted into the air-passages, died in somewhat more than twenty-four hours.

"The right lung was, in its whole extent, dark red, and uniformly solid to the touch; it had no appearance of air-vesicles on the surface. The lower lobe was completely distensible, and had, after insufflation, all the properties of the normal tissues. The upper lobe was left undistended for further examination of its physical condition. Superficial incision made perpendicularly into its substance showed a smooth, glancing surface, from which even upon pressure no blood flowed out, except where considerable vessels were divided. Portions of the lung sank completely in water; there was no fluid either in the trachea or bronchi."

The above experiments leave, as we think, no doubt as to the very considerable influence of bronchial obstruction in producing that physical condition of the pulmonary texture variously called collapse, lobular pneumonia, carnification, &c. The mechanism of this lesion, as produced artifi-

cially in the experiments above related, may be open to discussion; but no doubt can exist as to the fact that the existence of solid or fluid obstructions in the air-passages tends, in an eminent degree, to the production of pulmonary collapse, and that large portions of the lung may even be emptied completely of air in the course of a few hours if the obstruction be considerable.

We have already remarked that an inability to cough and to expectorate, and thus to remove the obstructing mucus, may be considered as one of the causes of collapse. Laennec supposed the expiratory force of respiration to be weaker than the inspiratory. He says—"The mucus secreted into the bronchi in consequence of pulmonary catarrh, must, especially if it is very viscous, present a great resistance to the free passage of air in inspiration and expiration; and we shall show, in speaking of the rôle, that this resistance often goes the length of producing complete, though momentary obstruction of a part of the bronchial ramifications. Now, as the muscles which subserve inspiration are strong and numerous, while expiration is produced only by the elasticity of the parts and the weak contractions of the intercostal muscles, it must necessarily happen that the air which has been forcibly driven beyond the obstruction in inspiration, will not be able to overcome it in expiration, and will be in a manner imprisoned, by a mechanism not unlike that in the condenser of an air-gun."*

Dr. Gairdner shows the fallacy of Laennec's theory, and refers to the experiments of Hutchinson and Mendelsohn, which prove that though *ordinary* inspiration is more of a muscular act than *ordinary* expiration, yet the residual effective force for overcoming adventitious obstruction is very considerably greater in expiration. "The *forced* or *muscular* expiratory act is in fact about one third more powerful as

* De l'Auscultation Médiante.

measured by its effect upon a pressure gauge, than the extreme force of inspiration, and it is this force which is thrown into action when obstruction in the tubes is to be overcome."*

In the act of coughing, the air in the vesicles is brought to bear upon the obstructing substance within the bronchia, at a maximum amount of outward pressure, and with the additional advantage of a sudden impulse, so that the practical efficiency of the expiration in forcing air through obstructions, must be far greater than that of inspiration. It is easily seen, therefore, that if the secretions in the air-tubes be so abundant as to interfere materially with the entrance and exit of air, they must necessarily occasion collapse, either partial or total, of the parts beyond them, since not only does the air enter with difficulty, but being expelled with greater force and in larger quantity than it can be drawn in, the amount remaining in the vesicular structure must gradually diminish. Again, when the muscular force of respiration is diminished by debility, this effect of obstruction will be still more remarkable, for then the inability of the inspiratory act to replace the air driven out by expiration will be yet more marked than when the muscular powers of the body retain their full force.

There is still another mechanical condition which tends to produce collapse from obstruction, to which Dr. Gairdner refers. This condition is to be found in the form of the bronchial tubes.

The bronchi are a series of gradually diminishing cylinders, and if a plug of any kind, but especially one closely adapted to the form of the tube, and possessing considerable tenacity, be lodged in any portion of such a cylinder, it will move with more difficulty towards the smaller end, and in doing so, will close up the tapering tube much more

* On Bronchitis. Gairdner. L. and E. Monthly, 1850.

tightly against the passage of air than when moved in the opposite direction. If such a plug be placed over a bifurcation, it will, even if freely moving in the larger space in which it lies, be of sufficient bulk to fall back upon one or other of the subdivisions during inspiration, in the manner of a ball-valve, upon the orifice of a syringe, and thus completely to occlude it. From this arrangement of the parts it will happen that at every expiration a portion of air will be expelled which in inspiration is not restored, owing in part to the comparative weakness of the inspiratory force, and in part to the valvular action of the plug.

If cough should supervene, the plug may be entirely thrown out from its position, and the air of course be thus freely admitted into the obstructed portion; but if the force of expiration is only just strong enough to displace the obstruction sufficient to allow of the outward passage of the air, the inspiration will return it to its former position—and if this process is repeated for any length of time, it must finally end in perfect collapse of that portion of the lung supplied with air by the obstructed branches.

In summing up the causes which tend to produce collapse, they would seem to resolve themselves into these. First—the existence of mucus in the bronchi, which is more liable to produce obstruction according as it is tenacious; Second, weakness or inefficiency of the inspiratory power, however it may be caused; Third, inability to cough and expectorate and thus to remove the obstructing mucus.

Bronchitic collapse of the lung occurs in two different forms—the diffused, and the limited or lobular. Of these, the latter variety is the more characteristic, while the diffused form is the most common. The only real difference, however, between the two forms is in the number of lobules affected. Both present the same fundamental changes of the pulmonary tissue, which is generally of a dark violet

color, externally, and internally of a more or less deep brownish red, or mahogany tint.

But it may be much darker in color, when it is much engorged with blood. Its consistence is always changed, being more or less condensed; this condensation may amount to a mere diminution of the crepitation, or to a total absence of it, in which case portions thrown into water sink rapidly.

These portions are both more flaccid, and much less friable than the pulmonary tissue when in a state of red hepatisation. When cut into, the surface is seen to be smooth and uniform, having somewhat the appearance of muscle and presenting no granulations. It yields on pressure or to the knife, only a semi-transparent bloody serosity. Close examination shows that the organic elements of the tissue, the vessels, bronchia, cellular tissue, &c., can still be distinctly traced. If inflation be employed, the condensed portions can be distended, and their natural physiological characters can be more or less completely restored.

MM. Rilliet and Barthez, in the second edition of their work,* treat particularly of congestion of the lung as a very constant accompaniment in the state of collapse. They regard this congestion as being connected almost always with bronchitic inflammation, and as being not merely a passive state, but as exhibiting phenomena, which in many instances prove it to be an active condition. And at the same time that they acknowledge that a state of debility, prolonged dorsal decubitus, and the obstruction to the circulation thus occasioned, facilitate the production of this condition, and give to it the appearance of a simple passive congestion, they believe that there also exists (frequently if not always) a really active and even inflammatory movement. They formed this opinion chiefly upon these facts—

* *Traité des Maladies des Enfants.*

that they have not unfrequently seen the texture of the affected parts softened, and easily torn by the finger; that the tissues exhibit a swelled and turgid condition, and that, on pressure, a sero-sanguineous liquid escapes; and that there is present a serous exudation around the pulmonary vesicles, while the interior of the vesicles appears to be healthy.

It is this combination of bronchitis with congestion and collapse, which was formerly described by them under the names of lobular and generalized lobular pneumonia. The alteration to which the term Carnification is applied, they regard as different from the above, and as consisting in a simple collapse of the lung tissue, without the active or passive congestion which exists in the first form. The principal causes of this condition are, according to them, debility and catarrh.

Our observations thus far have been directed more especially to collapse of the lung, as it is a consequence of bronchitis in childhood. But there is certainly no such peculiarity in the structure of the lung in childhood, which should render it then exclusively liable to a morbid process, from which it is exempt at all other ages. In fact, it is not a little curious that so clear a distinction should have been drawn between pulmonary affections in infancy and early childhood, and those of the other periods of life. If we consult almost any of the authors who have written upon the peculiar and characteristic lesion of pulmonary collapse, termed lobular, we shall find that it is spoken of as an affection quite peculiar to childhood. Some of the earliest writers on the subject even distinctly state, that of the two forms of pneumonia, the lobular, and the lobar, the former is alone to be found in early infancy, and the latter alone beyond that age. The latest authorities in some instances maintain the same opinion, and even such an author

as Fuchs still asserts that true pneumonia does not appear to present itself in children below the age of five years, and this he asserts in spite of the distinct observations of Rilliet and Barthez, Legendre, Bailly and others as to the occurrence of lobar hepatization in young infants. Again, on the other hand, Fuchs describes the lobular collapse as a disease of infancy, and, like almost all the other writers, says nothing as to its occurrence at other ages. Of course, it is not to be supposed that there is any truth in the suggestion which is thus advanced—that there is a complete revolution in pulmonary pathology after the fifth year of life. We must, therefore, consider that any such idea is based rather upon imperfect observation, than upon any organic difference between the infantile and adult lung.

The identity of the affection in the adult with that in the child is now fully established, by several well known observers. This condition of the lungs is mentioned as having been of frequent occurrence in adults during the epidemic fever in Edinburgh, in 1847. Dr. Baly communicated the particulars of several cases of collapse of the lung in the adult, to Dr. West, one of which is so carefully and exactly told, as to leave nothing wanting in the description of lobular pneumonia.

“No effusion, lungs healthy, except in lower and posterior fourth of right inferior lobe, which is of a dark purple color, is depressed somewhat below the level of other parts—does not crepitate, feels solid, but flexible and tough almost like leather, and sinks quickly in water; the part having these characters is distinctly defined by boundaries of lobules. The whole lung being inflated, the part just described receives air with greater difficulty than the other parts, but at length becomes distended, lobule by lobule, and assumes the same pale red color as the rest of the lung. On cutting through the lungs and tracing the bronchi, it is found that the ramifications of those tubes which go to the

dark, contracted and condensed parts, are filled up with tough mucus, from which those going to other parts are free."

Rilliet and Barthez pointed out the resemblance between the infantile collapse and the "carnification" of the adult, described by Laennec as the result of pleurisy. Legendre and Bailly refer to the catarrhal affections of old persons, and admit that the diseases of the lungs in the two extremes of life greatly resemble each other. Dr. West enters more fully into the subject, and by a reference to the researches of MM. Hourmann and Dechambre, establishes the re-appearance of the phenomena of pulmonary collapse at the period of "second childhood," and he concludes from the description of typhoid fever given by M. Louis, that in certain diseases, which are attended with much depression of the vital powers, collapse of the lungs is by no means an unusual concomitant.

We might cite the observations of others were it necessary. So far, then, as these conclusions go, we may safely infer that collapse of the lung is by no means an uncommon lesion in the adult, whether in the lobular or diffused form. Moreover, that in all essential particulars, it is the same in children and adults, and that, in both, a certain amount of pulmonary collapse may be almost invariably found as a concomitant of fatal bronchitis. Again, that this condition of the lung bears so obvious a relation to obstruction of the tubes, that we are unavoidably led to infer the dependence of the former upon the latter.*

There is another condition of the lung, which occurs so frequently, in connexion with bronchitic collapse, that it requires notice. We refer to what is termed bronchial abscess.

We have already remarked that dilatation of the bronchia

* Appendix D.

was not unfrequently found upon examination of fatal cases of bronchitis. This alteration may affect either the length of the air-tubes, or only their extremities. In the former case the tube continues of the same size, or becomes gradually larger from one of its principal sub-divisions until it reaches the surface of the lung—in the latter case, a section of the lung presents an areolar appearance, from the presence of a multitude of little rounded cavities communicating with each other and with the bronchia, of which they seem to be a continuation.

The fact of these cavities being in reality dilatations of the bronchia, has been called in question by several medical writers. Among these, Dr. Gairdner states his opinion to be, that almost all the so-called bronchial dilatations, and all of those presenting the abrupt sacculated character which has been described by him, are in fact the result of ulcerated excavations of the lung communicating with the bronchia. These he designates as bronchial abscesses, corresponding to what the French writers term "Vacuoles." Dr. Gairdner says that in the centre of the collapsed lobules of a lung affected with acute bronchitis there are found, not unfrequently, small collections of pus, varying in size from that of a hemp-seed to double or treble that volume. These small abscesses present on section an appearance so much like that of softening tubercles, as to be very readily mistaken by many persons for these bodies; and the resemblance is all the greater, on account of the peculiar limited form of the condensations by which they are generally surrounded, which when felt by the touch from the exterior of the lung is exceedingly deceptive. In their interior, however, these little abscesses contain, in the recent state, a very fluid pus; moreover, they are often met with as acute lesions produced by a few days of illness, and without a trace of tubercle in any other organ. When the pus is pressed out of these abscesses in their recent

form, they are found to be lined with a fine villous membrane, while in other instances they are not abruptly limited, but the pus appears to lie in contact with the surrounding pulmonary tissue.

When the bronchi leading to the lung so affected are carefully incised, they are found much inflamed; their mucous membrane vascular, thickened and covered with pus; and some of them can be observed to communicate with the purulent collections, the mucous membrane having been, at the point of communication, destroyed by ulceration, and either stopping short abruptly, or becoming gradually incorporated with the false membrane lining the abscess. Sometimes these abscesses communicate not only with the bronchi, but also with each other; but more commonly they remain of limited size, preserving perfectly the direction and relations of the bronchial tubes.

These abscesses occur both in the diffused and lobular form of condensation from collapse of the lung, and sometimes both forms are met with in the same lung. Dr. Gairdner states that they unquestionably arise from the accumulation of pus primarily in the extreme bronchial tubes of the collapsed lobules. This view, which is also supported by the observations of MM. Barrier, Legendre and Bailly, seems to us much the most reasonable.

MM. Rilliet and Barthez regard these cavities as simple terminal dilatations of the bronchi, while MM. Hardy and Behier consider them as a lesion of a complex nature, partaking both of dilatation of the bronchi and of pulmonary emphysema. In Dr. West's lectures, they are described as a *true* lobular pneumonia, the result, however, of bronchitis.

"It does, however, happen now and then, that the lung is found in a condition which may justly be called *lobular pneumonia*, as the result of the extension to the surrounding tissues of inflammation beginning in the air-tubes. Patches of lung will then be interspersed through the sur-

rounding pulmonary tissue of a vivid red color, of various sizes, from that of a pea to that of an almond, irregular in shape, and not circumscribed exactly by the margins of lobules, as is the case with portions of carnified lung. This process going on in a number of different situations, the affected parts may at length coalesce, and a pneumonia, at first lobular, may thus eventually become generalized. Or, though this should not occur, the inflammation may yet go on in the isolated portions of lung to the infiltration of pus into its substance, or the actual destruction of its tissue, when a portion of the lung will appear riddled with small distinct abscesses, seldom larger than a pea, irregular in form, and communicating more or less evidently with a minute air-tube. They may be distinguished from the vomicæ produced by softened tubercle, partly by the absence of tubercular deposits in other parts of the body, and by their being almost always limited to a single lobe of one lung. Their own characters, however, are sufficiently well marked, for they are altogether destitute of those solid walls which the tubercular deposit forms around a phthisical cavity; though the yellow lymph which often lines them may be mistaken by the inattentive observer for tubercle."* It is not difficult to explain the mechanism of these abscesses.

When pus accumulates in the central bronchi of a collapsed lobule, its evacuation is prevented in two ways—first, by the absence of the expiratory *vis a tergo*; and secondly, by the thickened mucous membrane and its secretion closing up the air-tube in front. The coats of the minuter bronchi gradually become softened and give way to ulceration, and the pus thus formed soon begins to be surrounded by a false membrane similar to that of any other abscess in any part of the body. This false membrane gradually be-

* Diseases of Children.

comes intimately blended with the bronchial mucous membrane, becoming as it were a part of it.

This lesion which we have just described, although not so frequently met with in adults, is a very common consequence of intense bronchitis in children.

Before proceeding further, it may be well to consider what evidences we have during life of the presence of bronchitic collapse of the lung; in other words, what are the diagnostic symptoms.

The diagnosis of this condition of the lung must always be more or less uncertain, when it is of the lobular form, inasmuch as the collapsed lobules being irregularly disseminated through the pulmonary tissue, afford no physical sign by which we can detect their condition. The presence of this form ought, however, to be suspected, whenever in a chronic disease, and especially in the course of a bronchitic attack occurring in a feeble and debilitated person, particularly in a child, the breathing becomes excessively quick and labored, the pulse small and feeble, the skin pale and coolish, accompanied with a degree of prostration which the amount of bronchitis present would not seem to explain.

In cases of collapse, where a considerable or the greater part of a lobe is affected, the diagnosis is more clear and satisfactory than in the lobular form, in which we are obliged to depend almost entirely upon the rational symptoms. In the diffused form, we have some useful physical signs. These are, the existence of more or less dulness on percussion; feeble respiratory murmur, prolonged expiratory sound, and occasionally bronchial respiration, which, when they occur in connexion with bronchitis, are usually sufficient to render the diagnosis easy.

The only diseases, with which collapse of the lung presenting the same physical signs, could be confounded, are, pneumonia and pleurisy. On careful observation, however,

we shall note a difference in the character of the physical signs. Though we have dulness on percussion in collapse, it is not so absolute as that of pleurisy with large effusion, or that of confirmed pneumonia. The bronchial respiration in collapse is indistinct, distant, instead of being clear, metallic and near, as in pneumonia, and moreover is heard much more in the expiration than in inspiration.

Collapse is also distinguishable by the slight severity of the re-actional symptoms, by the absence of acute pain, by the greater severity of the bronchial symptoms, and by the fact that it rarely occurs except in enfeebled subjects, or in those laboring under severe bronchitis.*

Collapse of the lung from bronchial obstruction, being established, is there a probability that the portion or portions of the organ thus affected may be restored to their normal condition? In answer to this, we say, that there can be little doubt that such a condition, when recent, may be completely removed. For the inflation of the lung after death not only proves at once that there is no organic change, but that a sufficiently strong inspiratory force to overcome the opposing obstacle, was all that was required to allow the free entrance of air. The collapsed lung, however, labors under certain disadvantages as compared with that which is normal, in its power to remove such obstacles. In the case of the former, the inspiratory force alone can be brought into action, which would seem in many cases rather to increase the difficulty by driving the cause of the obstruction still further inwards—while, in the latter, on the contrary, the strong expiratory force, which when aided by the impulsive effort of coughing is by far the most powerful agent in removing bronchial obstructions, may be rendered available.

* Appendix E.

When we consider the frequent occurrence of attacks of bronchitis, and that too in the same individual, without any appreciable change which is permanent, and when we know that post-mortem examinations teach us that a very moderate amount of accumulation is sufficient to produce a certain degree of collapse of the lung, we are necessarily forced to believe that there is some inherent power in the bronchi themselves by which to get rid of obstructive mucus, inasmuch as we have seen the expiratory forces, under such circumstances, are thrown out of action.

It is not unreasonable to suppose that this power lies in the slow contraction of those circular fibres, the muscular character of which has been demonstrated by Reisseisen, and whose physiological properties have been illustrated particularly by the experiments of Dr. Williams.*

By these, it is shown that the contractility of the bronchi resembles more that of the intestines or of the arteries, than that of voluntary muscles or of the œsophagus, the contractions and relaxations being gradual and not sudden—a contractility in fact similar to that which empties the arteries of their blood after death, or which facilitates the passage of a calculus along the gall-bladder or ureters. The experiments to which we have referred, appear to prove that the contractility of the air-tubes is readily excited by mechanical and chemical stimuli, to the mucous membrane, showing conclusively, we think, that the bronchi have a most important power of removing obstructions, independent altogether of the forces of respiration. “When these forces are in active operation, indeed, the tonic or slow contraction will be in abeyance, or very slightly manifested, as the air-tubes will then be dilated to their full extent at each inspiration and expiration. But, according as the admission of air to any part of the lung becomes less

* Diseases of the Chest.

from obstruction, the detrusive action of the bronchial muscles will increase, being thus called into effective action precisely at the period when most required. Perhaps, also, the slighter contractions of these muscles may be in almost constant operation in the normal condition, to aid, by a kind of peristaltic movement, the outward passage of the physiological secretion. This secretion, comparatively small in quantity as it is, would almost necessarily tend to accumulate in the air-tubes (seeing that no efforts of coughing or forced expiration are made for its removal); and this would take place particularly in the smaller bronchi, which we know to be especially subject to mechanical obstruction, and in which the ciliated epithelium, so abundant in the cartilaginous bronchi and trachea, gradually gives way to transition forms, not constantly furnished with cilia."*

We have already remarked that it is now the generally received opinion among medical authorities, that the condition of the lung which has so long been known and described under the titles of lobular pneumonia, generalized lobular pneumonia, pseudo-lobar pneumonia, marginal pneumonia, and, by some, as carnification, is, in fact, bronchitis associated with congestion and collapse of the lung. But we may ask, does not bronchitis also lead to inflammation of the parenchyma of the lung—to *true* pneumonia—lobular as well as lobar? Unquestionably it does, but inasmuch as the term lobular should now, we think, be restricted to the lesion which is due to bronchial obstruction, we prefer to substitute for it, that of *partial*, which is the term employed by M. Legendre, and by others at the present time. So that we have the two forms, partial and lobar. Of these, the latter is far the most frequent, although it was formerly

* On Bronchitis. Gairdner. L. and E. Monthly, 1851.

supposed that what was termed lobular was the more common, simply from the fact that bronchitis with collapse is more often met with, especially in children, than either partial or lobar pneumonia.

MM. Trousseau and Lasegne describe the pneumonia of children under the terms catarrhal and lobar. "Catarrhal (or lobular) pneumonia is a disease as distinct from simple (lobar) as variola is from erythema. This is seen in their respective mortality. Of twenty children who have been admitted into the hospital clinique, suffering from *simple* pneumonia, in six months, all have recovered; of nearly thirty who were attacked with *catarrhal* pneumonia, not one survived. Most of the first class of cases exhibited an excessive degree of acuteness which burnt out like a fire of straw; while several of the second, notwithstanding their fatal termination, commenced with very mild symptoms.*

Simple pneumonia hardly ever affects a child under two years of age, and rarely those of two or three, but becomes of more and more frequent occurrence as the child approaches adolescence. Its cause and symptoms resemble those of the adult, with some modifications. In the mild form of the disease, recovery takes place rapidly and in large proportion; but in its grave form, many cases are lost, whatever the mode of treatment.

Catarrhal pneumonia commences with a catarrh, which rapidly extends to the small bronchi, and then we hear numerous and small sub-crepitant râles disseminated over both lungs, and especially posteriorly. These râles may persist for four, six, eight, or fifteen days without any *souffle* becoming manifest; but sooner or later we hear a *souffle*, the resonance of the cries or the voice, or at least a prolonged respiratory murmur. While these latter sounds, common to simple and catarrhal pneumonia, are thus manifesting

* L'Union Médicale, 1851.

themselves, we find by the sub-crepitant râles that the capillary catarrh is still persisting in the rest of the lung. The disease has extended from the mucous membrane to the parenchyma of the organ. As more and more of the parenchyma becomes implicated, the fever becomes more continuous and intense, and the respiration more difficult, until the child dies exhausted.

In other cases in which the bronchial inflammation is very intense from the commencement, the lung becomes rapidly invaded over a large extent, and death takes place with great rapidity.

M. Trousseau compares these two affections to erysipelas and phlegmon. Erysipelas traverses the surface like the catarrh, and when it persists too long, it induces ulcerations of the skin, furuncles, &c.—just as the capillary catarrh induces suppuration of the lobules, and small abscesses of the lungs. Simple pneumonia, on the other hand, progresses like simple phlegmon, violent in its febrile re-action, but terminating rapidly.

We do not think it necessary, in this connexion, to give the physical and rational symptoms of pneumonia, which are so familiar to every one, but we shall proceed to describe briefly the anatomical lesions of *partial* pneumonia. These occur under two conditions. In one, the alterations are precisely the same as those of the lobar form, only, in the partial, the hepatization affects distinct patches of the pulmonary substance, thus producing hard nodules, scattered through healthy tissue. These nodules are irregular in form, and not perfectly circumscribed, but present, like the lobar form, the three stages of the inflammation—engorgement, red and gray hepatization. In the other variety of partial pneumonia, we find patches of hepatization, varying in number, and in size from that of a hemp seed to that of a pigeon's egg, more or less spherical in shape, hard to the

touch, and exactly limited. M. Legendre states that these hepatized parts become changed into a grayish, rough substance, of a fibrous appearance, a change which takes place at different points of the diseased mass, sometimes in the centre only, sometimes in their whole extent, and at others on their circumference.

Inflation of the lung after death has been much employed for the purpose of distinguishing between pneumonia and collapse. For while this process distends and restores more or less completely that portion of the organ which is completely collapsed, it fails almost entirely to have any influence on parts affected with true pneumonia—particularly if the disease has reached the stage of hepatization, and this of course is owing to the tissues being so agglutinated and indurated by the deposit of plastic lymph.

Inasmuch as pleurisy, to a greater or less degree, exists in a large proportion of the cases of pneumonia, we may regard bronchitis as giving rise indirectly to this affection. It does not, however, call for any special mention here.

HAVING discussed at some length the primary and direct effects of bronchitis, particularly in its relation to collapse of the lung, we shall now pass on to a consideration of the secondary and more permanent lesions, the result, for the most part, of this pathological condition.

In this part of our subject, we must especially acknowledge our indebtedness to the researches of Dr. Gairdner, who by his admirable memoir* on bronchitis, and by his

* On the Pathological Anatomy of Bronchitis. By W. T. Gairdner. Edinburgh, 1850.

various contributions to several journals, has done much towards the elucidation of pulmonary pathology.

Collapse of the lung, if it becomes permanent, gives rise to a peculiar form of pulmonary atrophy, which bears the same relation to the atrophy which surrounds retrograde tubercles, or which results from chronic pneumonia, that collapse of the lung does to recent hepatization. In the atrophy from tubercular disease, we find the tissue indurated, dark colored from carbonaceous deposit, and often charged with the remains of former exudation in the air-vesicles and their walls. But in the atrophy from collapse of the lung, none of these characters are present; the pulmonary tissue has simply disappeared, leaving a small amount of fibrous tissue, and occasionally a few specks of carbonaceous matter in its place.

The transition stages between collapse, which is removable by inflation, and permanent atrophy have been carefully observed in the bronchitic lung by Dr. Gairdner, in his memoir. They are also noticed as the result of congenital atelektasis by Hasse.

We transcribe the remarks of Dr. Gairdner on this point.

“In *simple atrophy of the lung*, the result of uncomplicated bronchitic collapse, the affected parts usually present somewhat different characters from other forms of pulmonary atrophy. They are, in part, reduced to a lax fibrous or areolar tissue inclosing the remains of bronchi and vessels; perfectly flaccid, free from all induration or abnormal exudation, and very frequently, in the purest form of the lesion, free even from that excessive deposit of carbonaceous pigment, which is so apt to accompany all chronic affections of the lungs; and in more recent specimens of emphysema, the anatomist will generally be able to trace several of the stages which I have indicated above, as intervening between collapse and atrophy. The atrophied lobules at the edge of the lung correspond to the indentations and grooves between the emphysematous parts.

“Simple atrophy, like the lesion which gives rise to it, occurs in the lobular and diffused form. The latter is chiefly found in the posterior portions of the lungs near their root. In diffused, simple atrophy, the lung is rarely entirely condensed, generally retaining a certain degree of crepitation, but being dense, tough, and fibrous; sometimes dark slate-colored, at other times not so, and in the most marked and exaggerated examples, crossed in every direction by fibrous processes or septa of considerable thickness and density, corresponding to numerous irregularities on the surface of the lung.

“Such lungs will always be found, when a fresh section is inspected with or without a lens, to present the most remarkable varieties in the size of the air-vesicles, some of which are entirely obliterated or very small, and others greatly expanded beyond the normal volume; the latter condition prevailing, of course, towards the anterior margins in the most emphysematous parts.”*

Hasse says, “When atelektasic infants die a day or two after birth, it is generally possible to dilate artificially the undeveloped parts. The depressed lobule is then seen to rise by degrees to the level of the rest, and to assume the color, permeability and other characters of sound lung. Up to this point, had other circumstances been favorable, perfect recovery might have taken place. When, however, the little patients have survived for weeks or months, this inflation seldom succeeds—or only imperfectly. At this juncture, the unexpanded pulmonary cells are for the most part coherent, a remarkable fact, seeing how long the lungs continue unexpanded in the fœtus, without adhesion ever taking place. What ulterior transformations go on in the diseased parts it is not yet satisfactorily determined; it is, however, more than probable that not a few indurations

* On Bronchitis. Gairdner. L. and E. Monthly, 1851.

and depressions, especially the small calcareous concretions sometimes occurring without any obvious cause at particular spots within the lungs (generally at the back of the inferior lobes), are referable in some measure to the above source. At all events, it may be observed generally, that in atelektasis, the boundary line between the diseased and the healthy substance becomes less and less distinct, in proportion as life is prolonged."*

The obliteration of the air-cells leading to atrophy of the lung has been received as a consequence of bronchitis by Dr. Stokes, and we believe the merit of first suggesting this, belongs to him.

In his work on "Diseases of the Chest," he says, "Atrophy of the lung has been recognized in a variety of diseases, such as tubercle, pneumonia, cancer and pleurisy, but its direct connection with bronchitis has not been sufficiently examined."

As might be expected, from the great frequency of pulmonary collapse, the lesion to which we have thus referred must occupy an important place in the chronic affections of the lung. Although it has long been customary to refer the various depressions, cicatrices, and slight indurations so commonly met with, indiscriminately, and as we must confess, rather vaguely, to pulmonary tubercle as the cause, there have been many observers who have expressed their doubts on the subject.

Dr. Gairdner remarks, "We have found cicatrices, *presumably tubercular*, to exist in between 40 and 50 per cent. of the hospital patients examined by us, nearly a third of the whole number examined being cases of phthisis pulmonalis—over and above this number, however, a large proportion of lungs contain more or less marked indications of partial atrophy, and often cicatrices and depressions of pervious parts of the

* Hasse. Pathological Anatomy.

surface. Sometimes we have even found calcareous concretions in considerable numbers, and so disposed, as to lead us to ascribe them to some other cause than tubercle. We cannot, with the evidence before us, doubt for a moment that many of these atrophic lesions have their origin in bronchitis and collapse of the lung; sometimes complicated with those local ulcerations which to distinguish them from tubercular ulcerations, have been called 'vacuoles' by the French pathologists, and by us, 'Bronchial abscesses.' The distinction between the tubercular and non-tubercular cicatrices is, however, we are ready to admit, often difficult, and not always possible. We find, accordingly, a considerable amount of variation in the results arrived at by those who have repeated Laennec's observations as to the cure of tubercle. M. Rogée, of Paris, finds evidence of the healing tubercle in 51 per cent. of the bodies examined by him in hospitals. M. Bondet, on the other hand, gives 86 per cent.; Dr. Bennett, of Edinburgh, not more than 40 per cent., probably a smaller proportion even than that indicated by Laennec."*

The pulmonary concretions which are frequently found in the midst of atrophied and indurated portions of pulmonary tissue, have been described by Bonetus, Morgagni, and by almost all the pathological anatomists, as often connected with asthma. The tendency to follow Laennec in considering these concretions as the result of cured tubercle, is now giving way to views of their origin which have been advanced principally by Dr. Gairdner, and which seem to us the more rational.

Laennec was the first to protest against the opinion that these pulmonary concretions were necessarily attended by symptoms. While he notices their frequent occurrence with or without the accompaniment of other lesions, and remarks

* On Bronchitis. Gairdner. L. and E. Monthly, 1851.

that they are frequently found in the centre of tubercles, he does not, however, deny that the osseous and cretaceous concretions may be developed independently of tubercle, although this must be very rare.

Now, while we admit that the healing of tubercle is a fact perfectly consistent with what is taught us by daily observation, we are of the opinion that the doctrine of Laennec, which includes all or nearly all pulmonary cicatrices and concretions under the designation of healed or obsolete tubercle, should at least be subject to some limitations. We therefore perfectly acquiesce in the conclusions arrived at by Dr. Gairdner, who says—" These lesions *are probably tubercular*, if they occur exclusively or chiefly at the apices and back part of the upper lobe of both lungs at once; or in the apex of one lung only, without trace of a lesion elsewhere; or generally diffused throughout both lungs, but chiefly in their upper lobes, and especially at their back part and apex; or, in any case, in company with characteristic traces of tubercular lesions in other organs.

" These lesions *are probably non-tubercular*, if they occur in one lung in a generally diffused form, without traces of tubercle even obsolete, in the other lung; or in the lower lobes to the exclusion of the upper; or at the edges of the lung in both lobes and not at its apex; or at the root of the lungs only; being in all these cases unaccompanied by tubercles or the traces of tubercles elsewhere."

At the same time that we are convinced of the accuracy of the above conclusions, we must in fairness state that cases will constantly occur in which no distinct opinion can be formed.

We come now to the consideration of another secondary and permanent result of bronchitis, viz., vesicular emphysema, a condition which, as we shall show, depends essentially upon collapse of the lung, and pulmonary atrophy, as its cause.

This lesion, mentioned by Bonet, Morgagni, Baillie, Floyer, and others, was first accurately described by Laennec, and by him considered as a consequence of bronchitis, an opinion which has been concurred in by the majority of pathological authorities since his day.

Emphysema of the lungs was described by Laennec, as consisting of two varieties—the one being a dilatation of the air-cells, and finally a rupture of them one into another by removal of their septa; the other a rupture of the air-passages directly into the inter-lobular areolar tissue. The distinction between vesicular and inter-lobular emphysema is too familiar to every one to require of us any description. We can only say that the modern means of investigation have scarcely added anything to the morbid anatomy of emphysema as known and recognized by Laennec.

Emphysema, then, is an unnatural distension of the pulmonary tissue with air; and the fact that artificial inflation of the lungs to an undue amount exactly imitates the appearances to which emphysema gives rise, shows incontestably that this is a mechanical lesion. Moreover, all the subsequent structural changes implied in the gradual removal of the septa and obliteration of the capillaries, are readily explained by the mechanical effect, of distension, as shown by the beautiful experiment of M. Poiseuille.

An instrument being adapted to the pulmonary artery of an animal, by which a given quantity of liquid was propelled with a given force through the capillaries of the lung, he found that this was effected in the normal condition in 29 seconds. M. Poiseuille now inflated the lungs so as exactly to fill the cavity of the chest; the time was still 29 seconds. On distending the lungs, however, further, so as to produce the appearance of a partial emphysema, the time required for the passage of the fluid became lengthened to 62 seconds; when it pervaded the whole lung in consequence of excessive distension, 129 seconds were required, and the fluid

returned from the pulmonary veins mixed with some bubbles of air.*

The results of this experiment show us that whenever the air-cells are abnormally distended, the flow of blood through the ultimate capillaries of the lung must be retarded and even obstructed; a condition which readily accounts for the structural changes, the absorption of the walls of the air-cells, and the obliteration of vessels observed in the latter stages of emphysema.

A great number of conflicting theories have been advanced to account for the development of pulmonary emphysema. Let us examine a few of these.

The theory of Laennec ascribes emphysema to mucus in the bronchi, and accumulation of air behind the obstruction; that it is produced in the act of expiration, and is the result of violent efforts of coughing or other forcible expiratory acts. Now, in answer to this, we would say, that what we have already, as we think, satisfactorily shown in the preceding pages, proves clearly that obstruction of the bronchi has precisely the opposite effect to emphysema, giving rise to *emptying of the air-vesicles and collapse of the lung*.

Every one who has studied the anatomy of this pathological condition, knows that the emphysematous portions of a lung can in most cases be inflated from the bronchi with the greatest ease, whereas in collapsed lung very considerable resistance is often opposed to its inflation from the air-tubes—conclusively showing that the emphysematous parts of the organ are free from obstruction, while the collapsed parts are not.

Again, the usual seat of emphysema leads to an inference directly opposed to the theory of Laennec—for while this condition is usually found to occupy the anterior edges of

* Bulletin de l'Académie Royale de Médecine, Vol. viii.

the lung, the accumulations of mucus in bronchitis generally take place at the posterior and lower parts of the organ, which are, especially in the adult, the principal seat of bronchitic collapse.

With regard to that portion of Laennec's theory, which conceives that emphysema is dependent upon expiration, and that it is produced by repeated forcible expiratory acts, our evidence would seem to be somewhat contradictory. The most serious objection to this appears to be that the expiratory act is mechanically incapable of producing distension of any portion of the lung. The act of expiration tends entirely towards emptying the air-vesicles by the uniform pressure of the external parietes of the thorax upon the whole pulmonary surface; and even when the air-vesicles are maintained at their maximum or normal state of fulness, by a closed glottis, "any further distension of them by the expiratory force is as much out of the question as would be the further distension of a bladder blown up and tied at the neck, by hydrostatic or equalized pressure applied to its entire external surface." The air-vesicles can sustain no distending pressure from the column of air *within* the tubes, as that air only becomes compressed in virtue of a force acting on the *exterior* of the lung, which opposes exactly as much resistance without as it creates pressure within.

Have we any direct proof that cough, however violent, can in itself produce emphysema apart from the other accidents of bronchitis? In croup, laryngitis, and some other affections, we have cough even more violent than that of bronchitis, and yet we are not aware that these are known to cause emphysema. Laennec asserts, and it is a prevailing idea, that this condition of the lung is of frequent occurrence among players of wind instruments. But this assertion is devoid of proof, and rests, so far as we can discover, solely upon Laennec's statement. Even if it could

be shown that such was the fact, we must also take into consideration the great tendency to other pulmonary affections in this class of individuals before the question could be decided on such grounds.

Thus, so far as former experience and observation go, it would seem to be impossible that emphysema could be produced by any act of expiration, however forcible.

We have now, however, what would seem to be direct proof in favor of this theory, in the extraordinary case of M. Groux. In this individual, the lung is distinctly seen thrown forward and distended at every forcible expiration, such as is produced by ordinary coughing, or by any other sudden expiratory act, forming a well-marked bulging out of the parietes of the fissure. This certainly seems strong evidence in favor of the expiration theory, and as such we must receive it. We shall at least be safe in saying that emphysema *may* be produced by the expiration-force.*

We have next the inspiration theory, which refers emphysema to an increase in the force with which the air penetrates into those portions of a lung which are healthy, the remaining portions being occluded. This theory is supported by Dr. Williams and by others of authority, and is based upon facts, but still does not cover the whole ground; moreover, it is inconsistent with these facts—that the inspiratory power of the chest is exactly limited by its capacity; and that even when a portion of lung is impervious to air, the inspiratory force can no more distend the air-cells to the degree observed in emphysema, than it can do so in the normal state. There is clearly, then, another condition necessary, besides mere occlusion of the air-vesicles in a part of the lung, to the mechanical completeness of the inspiration-theory of emphysema. And this is *partially diminished bulk*;

* See Appendix F,

—in other words, pulmonary collapse, or permanent atrophy of some portion of the lung.

Dr. Gairdner, in his Memoir on Bronchitis, gives an analysis of forty cases of emphysema collected from a series of 502 miscellaneous cases of disease, and without any special reference to this inquiry—all but two of these cases are noted as presenting some form of condensation of the pulmonary texture, such as collapse, lobular or diffused atrophy, with induration, concretions, hepatization or tubercle. An analysis of these cases of emphysema gives the following result—that the atrophic lesions alone stand in any special relation to emphysema, while hepatization and tubercle are found among emphysematous, in nearly the same proportions as among mixed cases.

“It is therefore clear that direct observation points, in a manner not to be mistaken, to partial atrophic disease as the invariable accompaniment of emphysema of the lung.

“But even had this not been so clearly shown in the manner above indicated, it might have been fairly enough argued, from the mechanical conditions by which the lung is confined within certain limits of size. The emphysematous lung generally appears, as a whole, more voluminous than natural; but a moment's consideration will show that this is entirely from the fact of its not collapsing, like the normal organ, on being removed from the chest. The lung is, in fact, as was well known to Laennec, extremely restricted as to real enlargement by the bony case in which it is confined, and which does not admit of expansion beyond the capacity to which it is brought by a full inspiration. Were the emphysematous lung really increased in volume, as a whole, even to this amount, it is quite clear that there could be no respiratory movement; the thorax being maintained by the lung in a state of perpetual distension, instead of being itself the cause of the expansion of the lung. But as in most emphysematous lungs, the air-cells are individually in-

creased in size to a very marked extent, it is clear that in a case of very general or 'universal' emphysema, such as is described by Louis and others, the thorax could not, by any conceivable amount of yielding of its parietes, give space for the hypertrophied organ, except upon the supposition that the enlargement of the emphysematous parts is accompanied by a nearly corresponding diminution of bulk in others. This conclusion becomes still more evident, when exact measurement is applied to the emphysematous, as compared with the healthy chest; we have, in fact, found, that so far from the chest being enlarged to any considerable extent in this form of disease, there is great reason to believe that it is usually smaller than natural, the arching of the front and the increase of the antero-posterior diameter being more than counteracted by a diminution in all the lateral diameters, particularly at the base of the chest."*

Both on the ground of personal observation, and of inference from the mechanical conditions, which prevent enlargement of the whole lung, Dr. Gairdner asserts, and as we think with truth, that in emphysema, the increase in size of the affected portions of the lung is always accompanied by diminution in the volume of other portions, frequently in the form of simple atrophy or of atrophy with induration.

From these facts it may be safely assumed that emphysema is a lesion occurring from mechanical causes in those parts of atrophied and collapsed lungs to which air has the most free access; in other words, it is produced by atmospheric pressure in the comparatively sound portions of such lungs; that it is an *increase in volume* of those portions of the lung permeable to the air, to supply the place of diminished volume in those parts from which it is excluded. It is produced by the expansion of the chest in inspiration, and is dependent simply upon the normal expansion-force being

* Brit. and For. Med. Chi. Review, 1851.

exercised under the abnormal conditions to which we have alluded. It cannot be produced in health, by *any* amount of inspiratory violence, because the lung admits of being readily and easily expanded, without straining any of its air-cells, to the full volume permitted by the expansion of the thoracic walls. It cannot even be produced in disease, except when the volume of the lung is directly diminished in relation to the space which it has to fill in inspiration. "Emphysema, therefore, is never found in connexion merely with pleuritic effusion, or with hepatization, or with tubercle. It cannot be produced when large cavities exist in the lung, even in connexion with atrophy, if they have very flaccid walls, and be distributed through all its lobes; for under such circumstances, the cavities are expanded by the inspiration-force more readily than the air-vesicles can be forced beyond their normal maximum. Hence the comparative rarity of emphysema in connexion with rapidly advancing tubercle, while it is generally observed to be the accompaniment of the retrograde or contracting stages of that affection."

The various changes impressed upon the form and movements of the chest by the sequelæ of bronchitis, form a marked illustration of the doctrines which we have put forth regarding the supervention of emphysema on pulmonary atrophy and bronchitic collapse. These have been made the subject of special investigation by Messrs. Rilliet and Barthez, by Dr. Sibson, as well as by Drs. Gairdner and Rees. The limits of our essay will only allow us to allude briefly to this portion of our subject, and to offer a few of the observations of Dr. Gairdner.

Many irregularities in the form of the chest are undoubtedly owing to bronchitic attacks in infancy and early childhood, either modifying the expansion of the lung or producing subsequent partial collapse of its tissue. To this source may be traced many of the disorganizations of the

lung which are revealed by morbid anatomy in subsequent years.

That the respiratory motions of the chest may be seriously interfered with at certain points in the adult during bronchitis, although not to so great an extent as in childhood, owing to the greater solidity and firmness of its walls, is well known to every medical man, and has been particularly demonstrated by the observations of Dr. Sibson.

It is evident to the eye, that while even in the severer forms of bronchitis, the chest on the whole expands both in its upper and lower zones, the movement of the latter is much more restricted than the other, and that while the lateral expansion of the thorax is circumscribed, the anterior movement or projection of the sternum and costal cartilages is usually even exaggerated. The modification in the form of the chest, which supervenes on this condition, is well known as the "emphysematous chest," being marked by increased fulness and prominence of the whole anterior portion, by a diminution in the lateral and by a relative increase of the antero-posterior diameter of the thorax.

The relations between these thoracic changes and the existence of vesicular emphysema have never been called in question, so far as we know, by any writer on the subject. The observations of Dr. Gairdner would, however, seem to lead him to different conclusions from those usually maintained. While he admits that the permanent modification of form is the consequence of the peculiarly altered movements of the chest, and that the diminished lateral motion is the direct effect of the diminished expansion of the lung in consequence of bronchitic accumulation with partial collapse and atrophy of its tissue—he thinks that to ascribe the increased movement and consequent deformity of the anterior portion of the chest to the production of emphysema would be an error of logic and observation.

He believes that whatever be the relation of pulmonary

emphysema to the emphysematous chest, it is not directly nor indirectly the cause of that deformity. He thinks it susceptible of demonstration, that the abnormal motion of the chest always precedes both the deformity and the emphysema; that the emphysema frequently precludes the deformity, but in its more chronic and exaggerated forms generally follows in its wake; that a certain amount of emphysema may exist without deformity, and a certain amount of deformity without marked emphysema. He arrives at the probable conclusion, that both emphysema and the emphysematous chest depend on the altered respiratory movements in bronchitis, and on the exaggerated respiration necessary to overcome the tendency to bronchitic collapse of the lung.

From these observations, then, we may safely deduce the following conclusions, in regard to the relation of emphysema to the emphysematous chest.

In order to overcome the bronchial accumulation, the result of bronchitis, forced respiration is thrown into action, by which the breathing, instead of being diaphragmatic, becomes to a high degree costal and thoracic. This peculiar state of the respiration serves to bring about decided changes, both in the chest and in the lungs. Those parts of the chest which are acted upon by the most powerful muscles, acting too at the greatest mechanical advantage, take upon themselves the principal function, while the motions of the other portions tend to fall into disuse. So those portions of the lungs which are in contact with the most movable parts of the chest, which are the anterior and upper, as well also as the lower edges, are subjected most directly to the respiratory force, thereby tending to the development of emphysema; while the root of the lung, and its lateral and posterior surfaces, receive the inspiratory impulse secondarily or in greatly diminished ratio, which leads to bronchial accumulation, and to consequent pulmonary collapse and atrophy.

These irregularities of movement of the thorax tend ultimately to affect its form, producing, in both the child and the adult, an increase of the antero-posterior diameter of the chest relatively to the lateral, and of the upper zone relatively to the lower. "The deformity of the chest usually accompanying emphysema of the lungs is neither a cause nor an effect of that lesion, but both emphysema and the 'emphysematous chest' depend on the altered respiratory movements in bronchitis and the exaggerated respiration necessary to overcome the tendency to bronchitic collapse of the lung."

A certain amount of emphysema of the lung is of very frequent occurrence in the aged. This was first pointed out by Magendie, and this form of the lesion has since been described by many pathologists as a peculiar one, constituting a kind of senile atrophy of the pulmonary tissue. However, there is little doubt that this lesion is the concomitant of chronic bronchitis which is so constantly met with in individuals advanced in life, and in itself is hardly entitled to the name of a disease distinct from the other evidences of decaying nature.

We have yet to speak of certain pathological alterations of the bronchi, as among the secondary results of bronchitis. These consist of their permanent contraction and obliteration, as well as their dilatation. The first of these is slightly alluded to by Andral, as a consequence of bronchitis, having been entirely passed over by Laennec. As an independent affection it has been made a subject of special attention by a French observer, M. Reynaud, in 1835*—and the conclusions at which he arrives have been adapted to the subject of bronchitis by Dr. Stokes—with more or less modification.

* *Memoir De l'Académie Royale de Méd.*, Vol. iv., 1835.

Reynaud was led by his observations to the opinion that obliterations of the bronchi were quite frequently met with. Hasse, however, suggests that he may in some instances have confounded simple obstruction produced by the presence of exudation of lymph in plastic bronchitis, and acute obliteration arising from organization of the exudation or adhesion of the walls of the tubes. The forms of obliteration and contraction described by Reynaud are numerous. Thus the lesion may be continuous, extending either over a single tube or a series, or even in some cases over all the tubes of a lobe—or again, the tubes may be narrowed or closed at one or more points, as if a ligature had been applied. More or less dilatation of some portions of the same tube or bronchial division may accompany contraction and obliteration of other portions. Thus a small bronchus may be dilated into a pouch-like cavity, just before the point of the obstruction.

In some cases this dilatation terminates all traces of the bronchus.

Again, a tube may show a succession of marked irregular dilatations through its whole length, at some parts having a sacculated character, at others being irregularly cylindrical.

Whatever may be the particular variety of the lesion, it is very obvious that it must tend to induce other physical changes in the pulmonary organs. And herein is where the lengthy memoir of M. Reynaud is singularly deficient—for he has failed entirely to connect these alterations of the bronchi with those of the pulmonary tissue with which they are constantly associated. The extent of these consecutive changes will of course depend on the size of the bronchial tube or tubes which are contracted or obliterated, as well as on the amount of obstruction. Whatever may be the amount of the obliteration, more or less atrophy and collapse of the tissue will be invariably found in connection with it, and this may be either simple bronchitic collapse,

or some of the more complex varieties which proceed from other lesions, such as tubercle or chronic hepatization.

The immediate local causes of diminished calibre of the tubes are situated within or exterior to the bronchi. Within the tubes, they consist of plastic exudation upon the mucous surface; of a tuberculous deposit; certain morbid excrescences; hypertrophy of the lining membrane; sub-mucous deposits of serum or lymph. The causes situated exteriorly act by producing pressure on the tube or tubes. Among these we may enumerate enlarged bronchial glands, various kinds of tumors, pleuritic effusions, &c.

If the obliteration of the bronchi has received but very little attention from pathologists, it is far different with the opposite condition, the dilatation of the bronchi, which, since its description by Laennec, has been almost universally admitted to be at least connected with, if not produced by bronchitis. We have already alluded to this lesion in the early part of our essay, but it requires of us here a more extended notice.

Dilatation of the bronchi was scarcely known to pathologists prior to the researches of Laennec—not certainly on account of its rare occurrence, but rather because this condition was overlooked at autopsical examinations. The mode in which it is produced is an interesting point of pathological inquiry.

We have already remarked that it was formerly supposed that this condition of the air-tubes was due to the mechanical effect of the accumulation of the secretion within them. This was the opinion entertained by Laennec, but the explanation is now deemed inadequate, and the accumulation is regarded rather as the effect than the cause of the dilatation. A morbid condition of the walls of the air-tubes, impairing their elasticity, and rendering them less resisting to dilating forces, is probably pre-requisite; the result of

long-continued inflammation. This was first pointed out by Dr. Stokes.

With regard to the immediate causes of this condition, they are not in all cases the same. Thus the obstruction of a bronchus by an enlarged bronchial gland, or by other causes preventing the exit of air and mucus, may bring about sufficient distension behind the obstruction to lead to permanent enlargement. But in the great majority of cases there is reason to believe that this dilatation depends on a prior morbid condition of the pulmonary parenchyma.

It is observed, in the case of dilated tubes, that the pulmonary tissue lying in immediate contact with them is usually, if not always, impermeable by air; that it is in a condition of fibrous atrophy, generally without marked induration. It was this circumstance which called forth the theory of bronchial dilatation by Dr. Corrigan*—to which he gave the name of "cirrhosis of the lung," from an apparent resemblance to the affection of the liver, known by that name. Dr. Corrigan conceived that a peculiar contractile fibrous tissue was formed in the interstices of the bronchi, which led to atrophy and obliteration of the pulmonary cells, and in some instances even to a contraction of the entire lung. Under these circumstances, two active forces, according to his views, are combined in producing bronchial dilatation. One is the pressure of the atmosphere from within the tubes in an outward direction to fill the vacuum caused by the diminution of the bulk of the surrounding parenchyma. The other is the traction exerted on the bronchial walls in consequence of the adventitious fibro-cellular deposit becoming attached to the longitudinal fibres of the tubes, so that dilatation in this way results from the shrinking of the surrounding tissue. Now, although we do not think that there is sufficient evidence that any

* Dublin Journal, May, 1858.

new or peculiar tissue is formed in this affection, yet there is no doubt that his observations in other respects are correct. We must at least accord to him the merit of being the first to notice and describe the morbid alteration and obliteration of the air-cells in connexion with dilated bronchi.

Following Laennec, subsequent writers have described three varieties of dilatation. One variety consists of a cylindrical and nearly uniform enlargement of a tube with more or less of its branches. A second variety consists in a spherical, sacculated, or pouch-like dilatation, occurring usually in the third or fourth sub-divisions, forming, in effect, a cavity which may attain the size in some cases, according to Rokitansky, of a hen's egg. Another variety consists in a series of globular dilatations along the course of a tube, the calibre of the intermediate portions retaining the normal size; the tube thus presenting an appearance not unlike a string of beads.

What, may we ask, is the true cause of dilatation of the bronchi? The explanations given by Laennec and others, which attribute it to violent coughing, to distension by accumulations of mucus, &c., do not seem to be entirely satisfactory, for the same reasons which have already been indicated as applying to emphysema. It seems, therefore, much more reasonable to ascribe these dilatations, as Dr. Corrigan has done, to the expansive forces of inspiration acting upon the bronchi of atrophied lung. But how shall we explain the occasional partial character of the lesion, the expansion of one portion of a tube into a sacculated globular enlargement, while portions of the same tube and adjoining ones retain their normal size?

Dr. Gairdner has, we think, given the most plausible solution of this question—that almost all the so-called bronchial dilatations, and all of those presenting the abrupt sacculated character to which we have just alluded, are in

fact the result of ulcerative excavations of the lung communicating with the bronchi.

As we have devoted sufficient space to the consideration of Dr. Gairdner's remarks on this point, when speaking of "bronchial abscesses," we need not discuss the subject farther.

The relations which exist between asthma and bronchitis demand a brief consideration.

The term asthma, like most terms in medicine, which have come down to us from antiquity, has been applied to a great many different affections. In popular language, every chronic shortness of breath or dyspnoea is still termed asthma, and this application of the term has had the sanction of many systematic writers, at least up to a comparatively recent period.

In this connexion, we have to do only with that variety of disordered respiration, which is denominated spasmodic asthma, and which is dependent upon causes acting through the nervous system upon the muscular structure of the bronchial tubes. Whether attacks of the disease are related or not to the existence of appreciable organic lesions, asthma is a complaint in which the spasmodic element prevails over all others.

What we term pure asthma frequently comes on without any appreciable exciting cause, without being related to any organic lesion susceptible of demonstration. But in the great majority of cases, we shall find that there are certain predisposing as well as exciting causes.

One of the most common predisposing causes is hereditary transmission—but a still more frequent predisposing influence is to be found in the inflammatory affections of the bronchial membrane—or perhaps it would be more correct to go still farther back, and say that it consisted in an extraordinary susceptibility to the impression of cold, rather

than in the bronchial inflammation which is the consequence of this susceptibility.

Under the head of the exciting causes, are to be comprehended all such as are known to bring about an asthmatic paroxysm, whether in the predisposed or not. Without denying the influence of many exciting causes which act upon the nervous system and thus produce an attack of asthma, such as fatigue, physical exhaustion, sudden or violent mental emotion, gastric irritation, &c., we must say that inflammatory affections of the bronchial membrane act most frequently as the stimulus, or as the source of irritation, to the production of the paroxysm.

Sir John Forbes says, "out of the immense majority of the cases met with, of asthma from other causes, nine-tenths are complicated with some form of catarrh, or at least with a morbid susceptibility of the bronchial membrane to be affected with cold."

According to statistics by Wunderlich*, true genuine asthma is extremely rare. It would be foreign to our purpose to enter into the pathology of this disease, which has lately been very ably discussed,† but we would remark that it seems impossible to avoid referring its most obvious symptoms to some kind of irregular action of the muscular apparatus of the air-tubes.

We have already spoken of the de-obstruent function of the bronchial tubes. It is now generally admitted that the muscular fibres of these tubes are undoubtedly perfectly passive as regards the respiratory act, and that their contractility is intended for the expulsion of obstructive mucus; that according as the admission of air to any part of the lung becomes less from obstruction, their detrusive action will be increased.

* See Appendix H.

† The Pathology of Asthma, by Hyde Salter, M.D. Brit. and For. Med. Chi. Review, July, 1858.

If now we apply these views to an explanation of the phenomena exhibited by asthma, particularly by that kind which is termed humoral, we shall be able to throw some light upon what is otherwise obscure.

The copious expectoration with which attacks of this kind are commonly terminated, and by which they are relieved, would seem to indicate that an undue accumulation of mucus had been taking place; while the absence of almost all catarrhal symptoms, in many cases, appears to show that this accumulation is directly connected with the spasmodic derangement which brings on the paroxysm. The connexion of these two phenomena is not difficult to understand; for if the removal of this mucus depends, in a healthy condition, upon the regular peristaltic contraction of the bronchial muscular fibres, it is very obvious that its accumulation must accompany any derangement of that action — and the paroxysm ceases when the normal action is restored. “The aggravated asthmatic paroxysm always occurs during sleep, when the energy of the nervous system is at the lowest, and the comparatively quiescent condition of the respiratory function favors the accumulation of mucus. It seems probable that the asthmatic paroxysm is attended with more or less of pulmonary collapse, the consequence of the accumulation in the bronchi, but I have not had an opportunity of direct observation on this point. It is certain, however, that this accumulation must seriously contribute to the production of the most distressing symptoms of the paroxysm.”*

Finally—however doubtful and difficult may be the pathology of this disease, we shall be safe in saying, that the catarrhal element plays its part, as we have before remarked, most frequently in its production; at least, it is the exciting cause of the asthmatic paroxysm in the great proportion of cases.

* On Bronchitis. Gairdner. L. and E. Monthly, 1851.

Does bronchitis give rise to pulmonary phthisis?

Perhaps no one opinion has ever been more generally admitted both by the profession and the public, than the direct relation of bronchitis to pulmonary consumption as cause and effect. Upon what does this opinion rest?

It is very certain, at the outset, that no affection so commonly appears to precede tuberculous phthisis as bronchial irritation. But this may be accounted for in two ways. The pulmonary mucous membrane of tuberculous subjects is undoubtedly very susceptible to the impressions of those causes which produce congestion and inflammation, such as atmospheric changes, &c. Then, again, tubercles often prove a source of bronchial irritation some time before their presence is indicated by other symptoms.

The opinion of Louis upon this point is well known. He says, in his work on Phthisis, that he has arrived at the following conclusions — “that phthisis is equally frequent in individuals liable to bronchitis, as in those where no such liability exists; it cannot therefore be considered as a consequence of the latter, no evident relation existing between them.”

Again — “Not only, then, is the influence of pneumonia, pleurisy and bronchitis in the development of phthisis not demonstrated, but our observations induce us to suppose its existence imaginary, or at least restricted within very narrow limits; from what has preceded, we think that we have proved that in one-twelfth part of our cases, pulmonary tubercles were developed independently of all inflammation, either of the substance of the lung, pleuræ or bronchia.”

“There is also a circumstance not less certain than those on which we have hitherto insisted, and which might indeed be substituted in their stead, viz.—that the bronchia are, in general, healthy in the vicinity of either unsoftened tubercles or masses of gray, semi-transparent matter; that the redness and thickening of those which communicate with tuberculous

excavations, seem the result of the constant passage of the contents of the latter, and that in cases fatal from some other affection, but with crude tubercles or gray granulations in the lungs, the bronchia are almost constantly healthy, both as to color and thickness."*

"Perhaps in the history of phthisis no one opinion was more universally admitted than that bronchitis was amongst the most frequent and active causes of pulmonary tubercles; this cannot, however, be any longer supported, and its accuracy is rendered still more problematical by what has been advanced in the notes on 'Diagnosis' as to the situation of simple bronchitis at the *base of the lungs*. *Chronic* inflammation of the bronchial mucous membrane does not appear more influential. In eleven cases of dilated bronchia, where the general symptoms of phthisis had not been present, and the duration of the affection was from two to six years, the mucous membrane was intensely red, thickened and granulated, while tubercles existed only in three, and were neither numerous nor softened. It is not intended to deny that bronchitis may and does occasionally hasten the development of tubercles (acting like all other causes which tend to weaken the sum total of health), but that it has no *direct* specific influence."†

Andral observes—"What ought never to be lost sight of, is this, that in order that inflammation of the mucous membranes of the air-passages shall be followed by the production of pulmonary tubercles, it is necessary to admit a predisposition. This being admitted, we can easily conceive how in one individual very slight bronchitis is sufficient to produce tubercles, whilst others do not become phthisical from the most severe and long-continued pulmonary catarrh."‡

* Louis on Phthisis. Bowditch's Edition.

† Bowditch. Appendix E. Louis on Phthisis.

‡ Clinique Médicale, t. ii., p. 32.

According to some statistics prepared with great impartiality and care by Dr. James Pollock, Physician to the London Hospital for Consumption, in relation to the effect of the season of the year upon the commencement of phthisis, and consequently in relation to the influence of bronchitis on the production of the disease, the following results were arrived at. The history of 487 cases of well-marked tubercle being satisfactorily obtained, it was found that

In the spring quarter — } there were 154 commenced.
 March, April, May, }

In the summer quarter— } there were 75 commenced.
 June, July, August, }

In the autumn quarter— } there were 141 commenced.
 Sept., Oct., November, }

In the winter quarter— } there were 117 commenced.
 Dec., Jan., February, }

According to this result, it will be seen that the spring and autumn originated by far the largest number of cases, then the winter quarter, and finally the summer. Now, the winter quarter, which ranks *third* in the list, is the period of the year at which most cases of bronchitis commence, when in fact the influences of the weather and its changes have most effect. The spring and autumn, however, seem to hold the pre-eminence, and the summer stands at the bottom of the list.

Therefore the commencement of phthisis at the period of the year when bronchitis is most commonly at its minimum, as shown by the above tables, is certainly opposed to the view that bronchitis leads to phthisis.

These observations, which were made with reference to English climate, are equally well adapted to our own.

Dr. James Clark remarks that repeated attacks of bronchial inflammation, or the long-continued application of mechanical irritants to the membrane of the bronchi, may

prove the exciting cause of tubercles, when the constitutional predisposition exists.

Dr. Wunderlich,* in his remarks on phthisis, says that frequent attacks of acute bronchial catarrh seem to predispose towards phthisis, or at all events to hasten the eruption of its symptoms; while, on the other hand, chronic bronchial catarrhs seem to keep off pulmonary tuberculosis.

“Where a real predisposition prevails, tubercles become developed in the lungs under a variety of circumstances. In some instances the tubercular development advances obviously under the exclusive influence of exciting predisposition—so slowly, and its ulterior stages follow so insensibly, that the mischief escapes ordinary observation until a very late period. For the most part, the disease is traceable to a catarrh, which after a first attack leaves perhaps but a slight cough behind, but on frequent repetition gradually and irretrievably lapses into confirmed phthisis. Generally speaking, chronic phthisis is wont to be interrupted by tolerably frequent breaks of seeming recovery, so that a limited number of tubercles may, under favorable circumstances, remain dormant for months, and even for an entire year, until an attack of catarrh or bronchitis, slight pleurisy or a peripneumony, operates upon the already diseased parts, so as to revive the quiescent diathesis, exciting it to increased productiveness, and thus hurrying on the disease uninterruptedly to its fatal issue.”†

In an analysis of 136 cases of phthisis observed at the Royal Infirmary for Diseases of the Chest, by Dr. Leared (no case which was considered in the least doubtful being taken into consideration), in more than two-thirds of the whole number of cases a definite cause was assigned for the disorder. The statements were almost invariably made

* Handbuch der Pathologie und Therapie. Stuttgart.

† Hasse. Pathological Anatomy.

with great positiveness; 29 of the whole number assigning causes, attributed the onset of the disease to a "cold" or to repeated "colds," the cause of neither of the latter, however, being stated; while a particular cold has been itself traced to its source or connected with something else in 26 other cases. We have thus a total of 55 attributing their disease to cold or catarrh. Making therefore every allowance for the liability to confound with it the earlier stages of phthisis, and admitting that a fair proportion of the first named 29 cases were in reality involved in this error, it would perhaps seem probable that in this instance, as well as in others, there may be a shadow of reason upon which to found this popular notion. However, Dr. Leared agrees with the majority of authors, in the main. "The general fact seems to be, that in the predisposed habit phthisis is not uncommonly developed by the operation of causes producing symptoms recognized as 'cold.' In rejecting the explanation of the origin of the disease given by certain humorists of a by-gone age, we have, it appears to me, gone too far in the opposite direction, since catarrh by some is quite ignored as an exciting cause."

According to statistical researches by Dr. Briquet, at the Hopital Cochin, Paris, of 109 phthisical patients, a third were more subject to bronchitis than other persons, and were more sensible of cold.

A tabular view is given by Dr. Gellerstedt, of the amount of previous disease of all kinds in 119 patients who ultimately died of phthisis. From this it will be seen that 44 out of 119 had previous hæmoptysis, and 42 had bronchitis. Dr. G. considers bronchitis to be a frequent cause of the rapid development of phthisis in those already laboring under the tubercular diathesis, and he believes that acute affections are at all times peculiarly perilous to such constitutions.

	In 119 Cases.	Per cent.	In 191 Cases.	Per cent.	In 310 Cases.	Per cent.
Bronchitis	42	35.29	37	19.37	79	25.43
Hæmoptysis	44	36.97	84	43.97	128	41.29
Pneumonia	22	18.48	51	26.70	73	23.54
Pleurisy	17	14.28	28	14.65	45	14.51
Nervous Fevers	10	8.40	12	6.02	22	7.09
Ague	27	23.52	49	25.65	76	24.51
Dyspeptic symp.	10	8.40	32	6.02	22	7.09
Diarrh. & Dysen.	23	19.32	33	17.27	56	18.06

The figures in the first, third and fifth columns denote the number of individuals who had suffered from these antecedent maladies: first, of those 119 who died of phthisis, then among 191 persons affected with this disease; and lastly, among these two classes conjointly, while the per-centage of each affection is given in the second, fourth and sixth columns.

We have thus brought forward sufficient authority (more might easily be added, were it deemed necessary) to show that the popular notion that tubercular phthisis is the common and direct result of bronchitis, or, in common parlance, of a "cold," is not founded upon fact. Under the circumstances, it is not a little curious that such an idea should have prevailed so extensively, and become as it were a matter established beyond question.

In our opinion, the great mistake made by most writers on the etiology of phthisis, consists in the constant endeavor which they make to find out some special and uniform influence by which to originate the malady. For it must be borne in mind that the origin of phthisis is not local, but connected with the most deeply seated vital processes; that it is a constitutional affection with local developments, rather than a local disease with sympathetic disorder of the system. We are thus naturally drawn to the conclusion, that bronchitis is to be considered as a cause of phthisis, only when a predisposition exists.

Although great advances have been made at the present day in our knowledge and treatment of pulmonary con-

sumption, it undoubtedly stands at the head of the list of the causes of death. It would seem, therefore, as if the hereditary predisposition to the disease must be very strong amongst us as a nation, and that our best means of staying its ravages must depend upon the attention given to invigorating the constitution of the young. Much interest has been awakened, of late years, as regards the importance of manly sports and out-door exercise for our youth; still there is necessity for even greater attention to this point. We have especially to guard against the too long confinement of our children in the school-room, and the over-taxation of their brains at the expense of their physical wants.

In this way alone can we hope to overcome that predisposition which in so many cases, lying dormant, is ready to break forth whenever excited by the most trivial causes.

In the preceding pages, we have endeavored to point out the immediate and remote effects of bronchitis, as shown more particularly by the *pathological* states of the lung. We first directed attention to the effect of inflammation upon the lining membrane of the bronchi, and their secretions, and the consequent effects upon the auscultatory phenomena of the chest. We then spoke of death from apnœa, the result of sudden and abundant effusion of the inflammatory secretion, or of the plugging up of one or more of the principal bronchi. We proved that all the phenomena exhibited by the physical signs of bronchitis were in perfect accordance with the anatomical appearances, which we described.

Next, we considered the direct effects of the obstruction of the bronchi upon the adjacent pulmonary tissue, leading to that peculiar condition, collapse of the lung, a lesion which has but lately been properly understood, having been heretofore considered and described as a form of pneumonia. The history of this affection we discussed at some

length—commenting upon the light which a knowledge of it had thrown upon the pathological conditions of the lung, particularly in childhood.

We next considered the causes of this lesion—and whether obstruction of the bronchi, without some deficiency in the respiratory power, was sufficient to bring it about. We gave the views of several observers on this point, and the results of experiments on animals—and having discussed the relative effects of inspiration and expiration, in their power to get rid of bronchial obstructions, as well as the mechanical condition, conducing to the production of collapse, to be found in the air-tubes themselves, we arrived at the following conclusions:—

That the production of collapse of the lung is due—first, to the existence of mucus in the bronchi, which is the more liable to produce collapse in proportion as it is tenacious. Second, to weakness or inefficiency of the inspiratory power, however it may be caused. Third, to inability to cough or to expectorate, and thus remove the obstructing mucus.

Bronchitic collapse of the lung occurring under two different forms, we gave the anatomical appearances which they present, observing, also, that the disease offered the same characteristics in the adult as in the child.

The question of bronchial abscess next occupied us. We considered its pathology and relation to bronchitic collapse, and the views of several observers on this point.

The diagnostic symptoms of collapse having been given, we considered whether, this condition being once established, the lung could be restored to its normal condition—a consideration which led to the question of the function belonging to the muscular fibres of the air-tubes.

Does bronchitis give rise to true pneumonia, lobular as well as lobar? We presented the views of several authors on this point—as also the anatomical appearances of partial (lobular) pneumonia.

We next passed on to the secondary and more permanent lesions of the lung—the result, for the most part, of bronchitic collapse. We said that this pulmonary lesion led to atrophy of the lung—which we fully considered, giving the observations of Dr. Gairdner, and others, on this point, and on the pulmonary concretions which are not unfrequently found in the midst of atrophied lung.

Next in order, as secondary effects of bronchitis, we discussed the important subject of vesicular emphysema. Describing the nature of this lesion, we considered at some length the conflicting theories which have been offered to account for its development. We endeavored to show that the theory of Laennec and others, which would ascribe the origin of emphysema to forced *expiration*, could not be supported, reasoning on the mechanical incapability of the act, but that the experiment of M. Groux, upon himself, would seem to decide otherwise; so that we are forced to admit that vesicular emphysema *may* be produced by the expiratory act.

The *inspiratory* theory of Dr. Williams and others, we attempted to prove, approached the truth, but did not cover the entire ground. Reasoning from the fact that the inspiratory power of the chest is exactly limited by its capacity, it is obvious that the inspiratory force can no more distend the air-cells so as to produce emphysema, than it can do so in perfect health. Another condition is therefore necessary to the perfection of the theory, and this is to be found in partially diminished bulk, or, in other words, pulmonary collapse or permanent atrophy of some portion of the lung.

These observations are based not only upon what the anatomical appearances teach, but upon the peculiarities which are presented by the “emphysematous chest,” and the relation which it bears to pulmonary emphysema.

Certain pathological alterations of the bronchi, the contraction, obliteration, as well as the dilatation of these ves-

sels, as secondary results of bronchitis, were next attended to. The forms, causes and changes in the lung due to these conditions were spoken of.

The nature of asthma, and its relation to bronchitis, were discussed. Having again spoken of the de-obstruent action of the air-tubes, we said that attacks of asthma, especially that which is termed "Humoral," were undoubtedly owing in many cases to a spasmodic derangement of the muscular fibres of the bronchi, whereby a great accumulation of mucus took place. Connecting these two phenomena, it is obvious that if this removal of the mucus depends in a healthy condition upon the regular action of these fibres, its accumulation must accompany any derangement of that action.

In our remarks upon the relation of bronchitis to phthisis, we said that observation and experience have now conclusively shown that this affection is to be considered as a cause of phthisis *only when a predisposition exists*; that there was in reality very little or no foundation for the widely-spread opinion that bronchitis is a frequent and direct cause of phthisis.

We closed with some general observations upon the etiology and prophylaxis of the disease.

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APPENDIX

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Case Reports by Asst. Prof.

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A P P E N D I X .

A—(Page 224.)

“There is another rhonchus, which may be called the dry mucous, because it is produced by a pellet of tough mucus obstructing a tube and yielding to the air only in successive jerks, which cause a ticking sound like that of a click-wheel. When the air is driven very fast, these click-sounds pass into a continuous note, and constitute the sonorous rhonchus. Sometimes, again, particularly in inspiration, the click-sound suddenly stops, the tough mucus being forced into a smaller tube, which it completely closes, and may not be dislodged again, but by dint of forcible coughing.”*

B.—(Page 226.)

CASES REPORTED BY ANDRAL.†

Chronic Bronchitis—Obstruction of a Bronchus by Mucus—Death from Asphyxia.

A mechanic, aged 53, entered the hospital of La Charité, for articular rheumatism. He had had besides, for nearly two months, an obstinate cough, with thick and tenacious expectoration. On percussion, the sounds of the chest were normal. On auscultation, the respiration was perfectly distinct throughout the whole left side, with some râles at the superior and middle portions of the right lung. There was no dyspnoea. Suddenly, one day, in the midst of a severe paroxysm of coughing, the patient was seized with a great difficulty in respiration. This was followed very soon by symptoms of asphyxia, livid countenance, cold extremities, small pulse. The sonorousness of the chest

* Dr. C. J. B. Williams. Diseases of the Chest.

† Clinique Médicale. Tome 3d.

had not altered. The respiration was puerile throughout the left side; behind, on the right side, there were mucous râles, but in front, from the clavicle to a little below the nipple, there was entire absence of all respiration, although the thorax was forcibly expanded.

We diagnosed the existence of pulmonary emphysema. The patient soon died.

Autopsy.—The viscera of the cranium and abdomen presented nothing abnormal. The heart normal. The lungs in part infiltrated, presented other portions which floated in water. Larynx and trachea healthy. Nothing as yet explained the absence of respiration at the superior part of the right lung, which had been recognized during life and had been referred to emphysema. But on incising the bronchi, we discovered, at the commencement of one of the principal bronchi, a mass of concrete mucus, which closed up the tube precisely like a cork. It was this bronchus which supplied the upper lobe of the lung.

Chronic Bronchitis—Obstruction of a Bronchus by Mucus—Dyspnoea and Death.

A coachman, 50 years old, had been a patient in La Charité several times for obstinate pulmonary catarrh, with slight dyspnoea and fusiform expectoration. Every time he went away relieved, but not cured. On both sides of the chest could be heard all the varieties of bronchial râles. In one spot, the column of air which penetrated the bronchi imitated the snoring of one in a deep sleep; in another spot it was like a dull and prolonged groan; in a third, a sound resembling that made by bellows; and in a fourth, the cooing of a turtle dove was exactly imitated. On the last occasion of his entering the hospital, his respiration was still tolerably free. One morning he was found in a state of unusual anxiety. In the middle of the night, after a violent paroxysm of cough, his breathing, he said, had suddenly become very much oppressed. It was discovered, on auscultation, that no air penetrated into the upper lobe of the right lung; yet that part sounded well on percussion—even louder than the corresponding part of the other side, which was morbidly dull. The difficulty of breathing increased, and the man soon died.

Autopsy.—Besides other marks of disease in the lungs, the primary bronchus leading to the upper lobe on the right side was closed up completely by tough mucus, and exhibited the appearance of a full cylinder. All the other organs were healthy.

C.—(Page 228.)

To advert a little more fully to the authors who have described collapse of the lung, as a state distinct from pneumonic consolidation.

Laennec seems to have described the diffused form of collapse only in connection with pleuritic effusion and compression. However, that he did not entirely overlook the lobular form is evident from his speaking of meeting with carnified portions, of the size of an almond, in the midst of very crepitant pulmonary tissue, and which he ascribes to "a slight inflammation in the first stage, the resolution of which, hastened by compression of the lung perhaps, has taken place in an irregular and imperfect manner."*

In 1829, M. Louis described the condition of the lungs in a variety of cases of typhoid fever, which descriptions were republished in 1841, and the state of the lungs in fevers compared with that found in other diseases. The result of these inquiries was a description of collapse of the lung, as a condition altogether different from pneumonia. M. Louis, however, does not account for the origin of the affection.†

Dr. Alderson,‡ in a monograph on the "Pathology of Hooping Cough," in 1830, pointed out the distinction between the lobular condensation observed in that affection and true hepatization of the lung.

In 1832,§ Prof. Jörg, of Leipzig, published an account of a morbid condition found in the bodies of newly born children, analogous to that considered as characteristic of lobular pneumonia. To this condition he gave the name of atelektasis. Jörg was the first to point out the effects of inflation in this form of pulmonary disease, as contra-distinguished from pneumonia.

MM. Barthez and Rilliet, in 1838, published an article on infantile pneumonia. While they accurately described the appearances of carnified lung, they failed to see its true condition, although they came near the truth, as will be seen by the following remark. "Bronchitis, especially of the smaller bronchi, is a frequent concomitant of the pneumonia of children, *especially the lobular form.*"

In 1844, were published the very important researches of MM. Bailly and Legendre, which fully demonstrated the identity of the lobular pneumonia of children with the atelektasis of Jörg.

Since this period, this pulmonary lesion has been recognized and commented upon by numerous writers—among whom we may mention Hasse, Dr. West, Dr. Fuchs, of Leipsic, and Dr. W. T. Gairdner, of Edinburgh, as the latest authorities.

* De l'Auscultation Médiante. Vol. I., ch. v.

† Med. Chi. Transact.

‡ Sur la Fièvre Typhoïde.

§ De Pulm. Vit. Org. Leipsic, 1832.

D.—(Page 241.)

The statistical returns of the "Salpêtrière," at Paris, furnish, as has been shown by M. Valleix, in a paper on this subject, ample proof of the frequent occurrence of what has hitherto been considered as "pneumonia" in the aged, but which more accurate observation has now undoubtedly established to consist of collapse of the lung, and its results.

M. Piorry, in his first researches into this affection, as it occurred in this very hospital, the Salpêtrière, was much struck with the extreme frequency of this "pneumonia," both as a cause of death, and as a curable disease, accompanying almost all the diseases of old men. These researches satisfied him that this affection was a peculiar disease, distinctly to be traced by auscultation and percussion during life. To this condition, M. Piorry has applied the term congestion (engorgement), a condition, according to him, having a great tendency to pass into inflammation, but not necessarily in itself inflammatory. Accordingly, he advises general bleeding—the consequences of which practice (if the "pneumonia" or collapse of the lung is due in these cases, as it undoubtedly is, to general debility), may be guessed at by the data given by MM. Hourmann and Dechambre. They state that pneumonia was by far the most frequently fatal disease in Salpêtrière, and that its frequency amongst those affected primarily with other diseases and recovering, was also very great; but that the immense majority (37 to 53) among those who recovered, were either latent affections, or at least presented no symptoms of acute pneumonia, but only of "congestion."*

E.—(Page 246.)

Cases illustrative of Collapse of the Lung—as a result of Bronchitis and Bronchial Obstructions.

CASE I.

Mary A—, aged 10 months. Weak and feeble constitution. Parents both dead. About four weeks ago was suddenly seized with bronchitic symptoms, which have continued up to this time. Has lost much flesh. On examination, find chest "pigeon-breasted." Respiration is almost entirely abdominal; the ribs being very yielding, are drawn in by the diaphragm. Cough not very urgent.

A few days before death, breathing became much oppressed, and cough very severe. The dyspnœa increased rapidly, the cough became

* Archives Générales de Médecine. Tome xii.

less frequent. On auscultation, large mucous râles could be heard. Five or six hours before death, the lips grew livid, respiration 85 in a minute, abdominal muscles acted violently, but chest was scarcely at all expanded.

Autopsy.—No tuberculous deposit in any organ. Large portions of both lungs presented the undilated condition, which disappeared on inflation. Considerable mucus in bronchi. Right side of heart greatly distended with coagulated blood.

In this case collapse took place not only from bronchial obstruction, but also from imperfect respiration, the result of great debility.

CASE II.

Catherine K—, aged 2 years. Has always been in good health, until two weeks ago, when she was seized with symptoms of acute bronchitis with suffocative cough, coming on in paroxysms—and sometimes followed by the rejection of a muco-purulent fluid. Has lost flesh and strength. Some stimulating expectorants were given, with some temporary benefit. Symptoms continued very much the same, until very suddenly coldness, faintness, and exceedingly labored respiration came on, under which symptoms she died in the course of twenty-four hours.

Autopsy.—A few recent adhesions were found on both sides of chest. The trachea contained a large quantity of muco-purulent matter, and the bronchial tubes were also filled with it. Some congestion of the lungs posteriorly. The upper and posterior part of the upper lobe of left lung, nearly the whole of the middle lobe, and the lower posterior edge of the lower lobe, were dark, solid, and non-crepitant. The same condition was found in the whole lower third of the upper lobe of the right lung. On inflation, most of these portions were restored to a natural condition, although some patches remained a little less dilated than the others. The other organs were comparatively healthy. Collapse of the lung, in this case, was the result of the sudden supervention of the muco-purulent secretion, which the organs were unable to throw off.

CASE III.

Adelaide K—, aged 5 years. Died with marked symptoms of hydrocephalus. The condition of the lungs was not noted during life. At the autopsy, besides certain lesions discovered in the brain and its appendages, the lungs presented the following appearances. The root of both organs was occupied by enlarged bronchial glands, infiltrated with crude yellow tubercle. The left lung was healthy, with the exception of partial collapse of some parts of its tissue. The enlarged bronchial glands in the right lung pressed upon some of the bronchi going

to the lower lobe, so as to distinctly diminish their calibre, as could be ascertained by passing a probe. In the upper margin of the lower lobe, was a somewhat rounded portion of condensed lung, of the size of a walnut, which, on being incised, showed a cluster of yellow tubercles. The anterior extremity of the lower lobe was completely collapsed, and evidently sunk below the rest of the lung. On inflating the right lung, it was found that the collapsed portions could be restored to their natural condition by using considerable force.

In this case, besides there being some general bronchitis, the collapsed portions were owing to the encroachments upon the bronchi by the enlarged bronchial glands.

CASE IV.

George S——, aged 19. Had been long affected with necrosis of the tibia, and also with disease of liver. Finally died, worn out and very much emaciated.

There had been no pulmonary symptoms to attract notice.

Autopsy.—The lungs were generally healthy in appearance. But on closer examination, there were found two or three places, of about an inch in diameter, which were plainly circumscribed by abrupt margins, and which crepitated imperfectly. The bronchi leading to these portions yielded on pressure a thick, tough, gelatinous substance, which had the ordinary appearance of mucus under the microscope.

In this case the collapse was evidently connected with the bronchial obstruction. For the portions affected and the bronchi leading to them could be perfectly isolated, and, moreover, there was no trace of any inflammatory action in any part of the pulmonary tissue.

CASE V.

Catherine C——, aged 30. Died of dysentery. No particular notes of case during life.

Autopsy.—Body much emaciated. On opening thorax, both pleural cavities contained a small quantity of fluid, with some adherent soft lymph. The lungs presented marked variations in density. The anterior edges were somewhat emphysematous, but between the parts thus affected, could be felt numerous condensed portions, which when on the surface had a collapsed appearance. Condensed portions were also found at posterior part of lungs.

On cutting into the pulmonary tissue, throughout the condensed portions there were found numerous small yellow spots resembling softened tubercles. These, when examined closely, were found to be bronchial tubes, or small cavities filled with pus. Except at these points, the

condensed tissue yielded only a little sero-sanguinolent fluid. Heart normal. Several dysenteric ulcers were found in the intestine.

This case presented quite extensive collapse of the lung, accompanied with bronchial abscesses, as also with emphysema.

CASE VI.

Charles P—, aged 9. On visit was found suffering under an attack of measles. On percussion, chest clear, but numerous mucous râles heard on auscultation. Four days after, chest symptoms much aggravated, with a good deal of fever. Crepitating râles heard all over chest, but no dulness on percussion. A day or two after this last visit, there was much dyspnœa, quick and feeble pulse, dulness over both sides of chest, behind. Died.

Autopsy.—The lungs collapsed imperfectly, on opening the thorax; at their anterior edges they were emphysematous, with some irregularities of surface. In the midst of the emphysematous portions, condensed lobules could be felt in various parts. On cutting into the lower lobes, there were found several yellowish deposits, irregularly formed, the largest of the size of a small nut, perforated in many places by bronchial tubes, and not so distinctly circumscribed as tubercles.

F.—(Page 42.)

Dr. Jenner lately read a paper before the Royal Medical and Surgical Society, the object of which was to show, in opposition to the views advanced by Dr. Gairdner, and others, that the force called into play by violent expiratory effort is by far the most common and efficient cause of acute vesicular emphysema, and of the chronic form which accompanies chronic bronchitis. Dr. J. denies that during expiration every part of the lung is equally supported, and equally compressed, and he affirms that the apex, the anterior margin, the margin of the base, and some parts of the root of the lung, are at once imperfectly supported, and comparatively little compressed only during expiration. The thoracic parietes covering those parts of the lung which are the least supported and compressed, are those which are seen, when a person makes a powerful expiratory effort with a closed or imperfectly open glottis, as in whooping cough, croup and hypertrophous emphysema, to be driven outwards. These same parts are the most common seats of emphysema. Three cases are detailed by Dr. Jenner in support of his position.

Dr. Edward Smith remarked that the author's theory was not so opposed to that ordinarily received as the antithesis of the terms in-

spiratory and expiratory, and the author's denunciation of the inspiratory theory, seemed to imply; since on both theories, the disease is produced by the forcible entrance of air into the cells. The disease is not produced by or during expiration; since it is essential, on the author's theory, that the glottis should be closed; and hence, when expiration is permitted, the production of the disease cannot occur. The occurrence of the disease with cough, that is, under the conditions mentioned by the author, has long been admitted by the advocates of the inspiratory theory; and since the essential act in the induction of the disease is the introduction of air into the cells, he would regard the author's theory simply as a modification of the inspiratory, notwithstanding that the power employed is that which, with the glottis open, would be expiratory. The author's theory is one of compression and dislocation of the contained air, and not of expiration.

G.—(Page 53.)

Chronic Bronchitis—Emphysema—Asthma.

CASE I.

Samuel J—, aged 22. A hearty, robust sailor—cook to a vessel.

He says that three months before our visit, when at sea, he first began to suffer from cough, attended with expectoration and shortness of breath, which symptoms have continued to increase up to the present time. On examination, the thorax anteriorly is unusually arched from above downwards. On percussion, there is everywhere a loud resonance, especially in front. On auscultation, the expiration is much prolonged, and accompanied by sibilant and sonorous râles, louder and more general on the right side. Cough is frequent and prolonged, with copious frothy mucous expectoration; great dyspnoea on any exertion, and occasionally coming on in paroxysms without any obvious cause. Sometimes vomits after a severe fit of coughing.

In addition to the dry râles heard at our first examination, copious coarse moist râles soon appeared posteriorly and inferiorly on both sides of chest. These continued pretty constant, the dry râles undergoing several variations both in intensity and in situation. About a month after we first saw patient, he was attacked with severe and long paroxysms of dyspnoea. These attacks generally terminated by violent cough, expectoration and vomiting, after which he always felt relieved. After exposure to cold, a short period after the asthmatic attacks commenced, he was seized with sore throat, followed by laryngitis and partial aphonia which greatly aggravated the asthma. These symptoms

were much relieved by the application of nitrate of silver to the larynx.

When we last saw him, the chest was still unusually resonant on percussion, and many of the auscultatory signs were still present.

CASE II.

Jane D—, aged 20. Servant.

About fourteen months ago, after exposure to damp and cold, patient was seized with a severe pain in the chest, accompanied with cough. The pain in the chest soon disappeared, but the cough remained and became much more severe—attended with considerable dyspnœa.

On percussion, resonance is very loud both anteriorly and posteriorly. On auscultation, expiration is everywhere prolonged. Sibilant and sonorous râles accompany inspiration and expiration on both sides anteriorly and posteriorly. Vocal resonance everywhere diminished. Cough and dyspnœa are paroxysmal. Expectoration is moderate, and consists of frothy fluid mixed with tough gelatinous mucus. Sounds of heart healthy. Bowels generally constipated. Some appetite.

After treatment for a few days, the symptoms generally improved, but shortly after this, the dyspnœa again became distressing, and she was nightly attacked with asthmatic paroxysms. Anodyne draughts generally afforded relief. As the summer approached, the fits became less frequent, gradually diminishing in severity and duration.

The above was a case of chronic bronchitis, with emphysema, and severe paroxysms of asthma.

H.—(Page 54.)

According to statistics by Dr. Wunderlich, genuine asthma is an extremely rare disease. Out of 10,000 clinical patients, only three, according to him, presented at all the character of asthma. He says, "The pathological condition of asthma is very unsatisfactory and imperfect. Among the causes in connection with the respiratory organs which are enumerated by writers, are congestion, extensive tubercular and cancerous deposits, emphysema, œdema, chronic bronchitis, catarrh, affections of the glottis, &c." These are often complications, but he doubts whether they ever give rise to asthma.

