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THE PHYSICAL SIGNS OF CELLULAR ŒDEMA OF THE LUNG, CONSIDERED IN THEIR RELATION TO THE PATHOLOGICAL CHANGES.[1]

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In the year 1886 I drew the attention of the Medico-Chirurgical Society of St. Louis to a form of disease of the upper air-passages and the lung that developed a history and symptoms entirely foreign to those forms of disease known in that locality. The typical form of throat disease was a solid œdema of the mucous membrane, and the pulmonary affection showed varying degrees of consolidation or infiltration of the lungs.

In the pulmonary form of the disease the mortality was very great, far exceeding the ordinary forms of pneumonia. The symptomatic history of the disease was an unusual one, and the combination of the physical signs in the different cases was at once contradictory and perplexing, resembling, in certain cases, a croupous pneumonia, in others a bronchial pneumonia or bronchitis. Still the symptoms and the physical signs were at variance with those which are usually recognized as peculiar to these conditions.

In 1887 I began the study of the post-mortem appearance of these lungs, and published a report of the throat cases in a paper read before the American Laryngological Society, in June, 1889 (*New York Medical Journal*, August 16, 1889); also, one on the lung affection in March, 1890 (American Journal of the Medical Sciences).

In this paper I drew attention to the peculiar cell-infiltration of the lungs, and claimed that this condition was the local effect of a general septic infection of the system. As the symptoms of the disease bear a striking resemblance to the symptoms of the influenza as described by Graves, or la grippe, of Valliex, I surmised that these might be cases of influenza. The history of 1890, 1891, and 1892, with the prevailing epidemic, proved the correctness of this view, and the cases may now be recognized simply as sporadic cases of influenza, which preceded the epidemic by several years, in the same way that sporadic cases have followed it, to the present time. My studies of this disease have been

[1] Read before the Eleventh Annual Meeting of the American Climatological Society.

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chiefly clinical, aided by the macroscopic appearance in the dead-house and microscopic sections. The limited bacteriological study that has been made for me has shown the streptococcus to be the prevailing micro-organism.

Cellular œdema of the lung has been generally called in this country the grippe pneumonia. I would consider the name "pneumonia" as improper, as only a certain number of the cases bear a resemblance to a bronchial pneumonia, and the existence of inflammatory action is very uncertain. I would consider it to be essentially an œdema of the lung in which the cell-elements, largely leucocytes, are poured out very quickly, in greater or less numbers, from the lymph and bloodvessels, producing a partial or complete infiltration of the lung. The fever and other symptoms which occur in the disease I believe to be strictly dependent upon the sepsis, and the symptoms and the amount of the infiltration of the lung will be determined by the intensity of the streptococcus poisoning. That this condition of the lung is only the local expression of a general systemic disturbance will be seen from the changes in the other organs. In fatal cases there is some change in the liver; it is usually enlarged and softened: the spleen is pulpy, giving the appearance of a septic spleen; and the heart-muscles are in many cases so softened that they can be readily torn: the kidneys are generally hyperæmic.

This conclusion is the result of a study of thirty-four post-mortem examinations made between the years 1886 and 1894. In twenty-one of these the typical appearance of the lung was alone present. In thirteen it was complicated with other conditions. On the post-mortem table the lung, in a condition of cellular œdema, presents a swollen, congested appearance. It is generally brown-black in color, and interspersed over the lungs are areas of a lighter brown or gray. These areas may be of limited extent or they may include the greater part of the lobe. The boundaries are irregular and they are scattered over different portions of the lung. If we take up a portion which is brown-black in appearance, we will find that on pressure it crepitates with difficulty. The crepitation is apparent, but the "feel" of the lung presents a degree of solidity foreign to healthy lung tissue, and the crepitation is produced only with more pressure. When placed in water it quickly rises to the surface. The cut surface is smooth and moist. If this section of the lung is placed under the microscope, it will be seen that the alveolar walls are infiltrated with numerous cell-elements, but the interior of the air-vesicles remains free. The physical signs of a lung in this condition are such that they can be fully explained by the pathological conditions. To the inspiratory sound is wanting the free flowing breath-sound of normal lung expansion. The lungs seem to be extended with decided effort, and the inspiratory sound is delayed and prolonged. In quality the sound

may be slightly harsher than the pure vesicular breathing. No change, or only a slight lengthening of the expiratory sound, is apparent. The percussion-sound may be so slightly changed as to appear normal, or show only a slight shortening of the normal resonance.

Another area of the lung presents a dark-brown color. To the hand this gives a feeling of solidity. When cut it appears as a solid substance, like a piece of beef. There are no crepitations on pressure, but when placed in water it floats on the surface. Examined under the microscope we find the alveolar walls densely infiltrated with cell-elements, with a partial filling of the vesicles. Some of the vesicles are more filled than others, but in the greater part we recognize the existence of air-spaces. The amount of infiltration varies in different sections and portions of the sections, but the presence of a large number of partially filled vesicles may be recognized in all. In a cut section of the bronchus the walls may be seen thickened by the same cell-elements.

The physical signs produced by this condition of the lung depend largely upon the amount and extent of the infiltration of the bronchial mucous membrane, together with the partial infiltration of the vesicles. The percussion-sound is always deadened, giving to the finger an increased sense of resistance. In minor degrees of infiltration through the deadened sound a slight degree of pulmonary resistance may be appreciated. When the infiltration is greater we find the percussion-sound very dull; even there may be a perfect flatness. In some cases we find an exaggerated full percussion sound, such as we find in emphysema, and it may be even tympanitic in character; but even if this is the case, the sense of resistance is still always somewhat increased. It is difficult to explain this resonance of the percussion-sound under such conditions, and we can only believe that it is either due to the confined air in the vesicles, through the complete obstruction of the bronchii, or it may be the result of some gas-producing micro-organism which it has been claimed has been found in these lungs. Occasionally we find an interchangeability between the dull percussion-sound and the exaggerated resonance. The same place may be dull to-day and to-morrow may be hyper-resonant, and this may again be replaced by dulness.

The auscultatory signs of this degree of cellular œdema are very perplexing and contradictory. In cases where the œdema of the bronchial mucous membrane is sufficient to thicken the membrane, rendering it dry, we find the normal respiratory murmur changed into a harsh respiration, in which the inspiratory sound is lengthened, sharp, and of a high pitch, and it is immediately followed by a prolonged expiration of lower pitch. In some cases the expiratory sound is absent, and the prolonged harsh inspiration is alone heard. In cases where the œdema narrows the bronchii to a still greater degree we shall have the sibilant râles. They may be inspiratory or expiratory, or both. They

resemble asthmatic râles in a measure, but they are shorter in time, the inspiratory râle rarely covering the whole time of the inspiratory act; a distinct interval of silence may or may not be followed by the expiratory râle. Occasionally we will have large-sized sub-crepitant râles, largely expiratory. They resemble dry crackling sounds, and are probably formed by the passage of the air through the viscid secretion of the bronchii. The dry and the moist râles are not, as a rule, continuous phenomena: they vary from day to day, and may entirely disappear for a time, to be again heard in varying degrees of intensity.

Where the œdema is so great that it completely occludes the bronchii we will find a complete silence over the lung. This silence is even greater than that formed over pleuritic effusion, where more or less distinct breathing may often be heard. Vocal and pectoral fremitus is wanting. This silence of the lung may continue for an hour or for weeks. It may be broken, first, by a gush of mucous râles, which can be heard during the inspiration. These râles may again disappear, and no respiratory sound may be heard. Later, they have a tendency to become permanent, and may continue as large-sized inspiratory râles for months. In other cases we have a gradual return to normal breathing without any appreciable râles.

The portions of the lungs which are of light brown, or which may appear as steel-gray, give all the evidences of a consolidated lung. They are dense and firm. The cut surface is dry, smooth, and glistening, and, when placed in water, sinks instantly. Under the microscope we find the alveolar walls and the vesicles densely filled with the cell-elements. The physical signs of this condition are the ordinary signs of consolidation (bronchial breathing and increased pectoral and vocal fremitus). The percussion-sound is slightly flat. Occasionally the bronchial breathing is replaced by amphoric breathing. This was true where, on the post-mortem, the apex of the lung was of a light-gray color and completely consolidated. The tendency of this consolidation is to continue for a long time. It remains after all the other portions of the lung have become normal. The disappearance of the abnormal signs and the reappearance of healthy lung action is gradual. Cellular œdema of the lung seems to present a most fertile soil for the growth and development of the tubercle bacillus, and I believe we find here the origin of a large number of our present cases of acquired tuberculosis.

Occasionally we find signs of cavities. On post-mortem examination we find the cavities to be irregular in form, with soft walls, giving the appearance as if they were worm-eaten, with an entire absence of the fibroid tissue which is usually seen about tubercular cavities.

I would describe the different degrees of infiltration as types and not as stages of the disease, for I have not found them to merge from one

into the other. A type which occurs in the beginning in a certain portion of the lung continues throughout the attack. The infiltration is sudden, and resolution may occur as quickly.

NOTE.—The treatment of *cellular œdema* of the lung which has given excellent results in the hands of the writer may be thus summarized: Fifteen to twenty grain doses of benzoate of sodium with half-drachm doses of solution of acetate of ammonium should be given every two hours. In addition, twenty-five drops of the muriated tincture of iron should be taken every four hours. Whiskey or champagne may be given freely, and digitalis is almost always required through the whole course of the disease. Frequent counter-irritation over the lung is desirable.