A Septic and Unusual Form of Lung Disease Existing in the Mississippi Valley During the Years 1886-7-8-9-90.

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A SEPTIC AND UNUSUAL FORM OF LUNG DISEASE EXISTING IN THE MISSISSIPPI VALLEY DURING THE YEARS 1886, 1887, 1888, 1889, 1890.[1]

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During the past three years an unusual form of lung disease has existed in the city of St. Louis and in many parts of the Mississippi Valley. In some cases it bears a striking resemblance to croupous pneumonia, although dissimilar in many of the symptoms and physical signs; in others the signs and symptoms would point to broncho-pneumonia; and in still others the diagnosis bronchitis would be made by a superficial observer.

The great prevalence of acute pulmonary disease and the increased ratio of mortality during the years 1886, 1837, and 1888 are shown clearly by the records of the Board of Health and of the City Hospital. Through the courtesy of Health Commissioner Dudley, I can present a tabulated statement of the number of deaths, and the ratio of deaths from acute pulmonary disease to that of the general mortality that occurred in the city during the winter months of 1886, 1887, and 1888. From these tables we see the great increase of mortality, both in the number of cases and the increased ratio to the general death-rate, from the winters of 1885 and 1886 to those of 1887 and 1888. In January and February of 1886 we find two hundred and nineteen deaths, which is about nine and one-half per cent. of the total mortality; in January and February of 1887 we find two hundred and fifty deaths, about ten per cent. of the total mortality; and in January and February of 1888 three hundred and eighty-three deaths, about equal to sixteen per cent. of all deaths reported.

The records of the City Hospital, obtained through the politeness of Dr. H. N. Dalton, Superintendent, show the number of cases and the death-rate of the so-called pneumonias during the years of 1885 and 1886 as one hundred and thirty-five cases and thirty-nine deaths; 1886 and 1887, one hundred and thirty-six cases, fifty-four deaths; 1887 and 1888, ten months, fifty-four cases, twenty-five deaths. Dr. Dorsett

[1] Read before the Medico-Chirurgical Society of St. Louis, Jan. 7, 1890.

of the Female Hospital, reports a death-rate of five cases out of eleven. From these figures we will see the increase in the number of cases and the death-rate to be especially great in the winters of 1887 and 1888.

In 1887 the unusual and unexpected mortality of cases which I had classed as croupous pneumonia directed my attention to the subject, and I began to investigate this class of cases. Since that time I have seen one hundred and ninety-four cases, and from them I have obtained twenty-nine post-mortems. The greater number of these cases have been seen in the City Hospital, and in the St. Louis Mullanphy Hospital, and the post-mortem examinations have been made almost exclusively at the City Hospital by the internes of that institution.

In the earlier investigation, when the clinical signs and symptoms of the disease were still unsettled and undetermined, twelve of these post-mortems showed the ordinary pathological changes of pneumonia, croupal or lobular, and tuberculosis, whilst seventeen alone gave pathological evidence of an unusual form of disease. In all of these seventeen cases the pathological appearances corresponded in a striking manner, and varying only in a degree that would correspond with differences in the intensity of the disease. The difference in the macroscopical appearance of the organs from that seen in the ordinary forms of pneumonia —croupous, lobular, and embolic—was very apparent.

Post-mortem examination showed in all cases that the greater part of both lungs was in a condition of infiltration by a morbid material, and certain limited portions were in a consolidated condition. In the least infiltrated parts the lung was of a black-brown color; on pressure a firm, sodden feeling was given to the fingers, the normal pulmonary elasticity was wanting, still it crepitated with resistance, and when placed in water did not sink. In other parts of the lung the color was of a dark brown, the lung crepitated slightly, and on pressure gave a firm, resistant feeling not unlike a piece of beef; it would not sink in water. In still other parts the color of the lung was of a light brown, being in many cases of a steel gray; here the parts affected were absolutely solid, no crepitation on pressure, and the piece would sink instantly when placed in water. In all cases the greater portion of both lungs was of a dark or dark brown color, whilst the light brown or grayish color was found in isolated parts. The consolidation occurred in irregular areas in the middle or lower border of the upper lobe. The apex was frequently found consolidated, and in three cases was converted into a solid mass presenting a light gray appearance. These areas of consolidation were always found in each lung, varying from the size of a walnut to that involving the greater part of a lobe. The areas were irregular in shape and presented no strictly defined limit such as we see in croupous pneumonia. *The inferior part of the left upper lobe posteriorly was the most frequent site of the consolidation.*

In all cases the liver was enlarged and softened; the spleen was of a light brown color, not much enlarged, but the substance was almost always softened. In two cases it was so pulpy that it could be taken out with a spoon. The heart was often softened, and in some cases could be torn easily; in three cases it was tightly contracted in systole. The kidney showed pathological changes; they were usually congested. and many were in a condition of parenchymatous nephritis. The venous system was invariably engorged with blood. This was especially seen in the portal system. The absence of serous effusion in the cavities, which should be expected from the condition of the venous system, was noticeable in a certain number of cases. In three cases a purulent effusion was found in the pleura, and in one a purulent pericarditis. Numerous microscopic examinations of these lungs have been made by Dr. E. M. Senseny, and he reports:

In the darkest portions, which swim in water, and where crepitation can be felt, the alveolar walls are seen infiltrated with leucocytes, the alveolæ themselves remaining free. In the dark brown parts, which crepitate sluggishly, and which swim in water, besides infiltration of the alveolar walls, he finds a partial filling of the alveolæ with leucocytes, white blood-corpuscles, epithelial débris, and occasionally some red blood-corpuscles. These cling to the walls of the alveolæ, leaving the centre more or less open. In the lighter portions, which do not crepitate and which sink instantly in water, he finds an infiltration of the alveolar walls and a complete filling of the alveola. The pathological condition of the lung corresponds to that described as existing in broncho-pneumonia.

Clinically the diseases may be divided into what may be called the parenchymatous and the bronchial type of cases—the parenchymatous where the areas of consolidation are extensive and rapidly developed, and where the symptoms show the greatest intensity of the disease; the bronchial type where areas of consolidation are wanting or limited, and where the disease hardly passes beyond what may be called the first and second degrees. In these cases the symptoms are essentially different from those seen in the former, and were in some cases of so trivial a nature that the lung disease would have been overlooked unless attention had been drawn to it.

In the clinical study of the *bronchial* type we meet with a variety and a want of constancy in the symptoms. These may be said to depend on what may be called the intensity of the disease; still there is a certain similarity, and certain symptoms will be found to correspond closely to the presence of certain physical signs in such a way that the diagnosis cannot be doubtful. The disease usually commences suddenly in previous good health, with a severe chill or chilliness; this is followed by fever, which may only last from twelve to twenty-four hours. The

fever may assume a remittent or an intermittent character, and may be interchangeable with hot flushes and chilly sensations. The tongue is usually clean, very rarely coated or furred. There may be a violent headache, with pains in the limbs and back; the pains in the back are especially about the coccyx, radiating into the pelvis and thighs. In some cases the pain is in the lumbar region; there is a stiffness of the joints and a soreness and tenderness of the muscles, with a general condition of great lassitude and prostration. Great dejection, low spirits with drowsiness, is a frequent symptom. The headache is often first frontal, then following the longitudinal sinus to the occiput; at times it is rather a feeling of fulness than pain. The body is often covered with profuse perspiration during the high fever; cold sweats alternating with hot flushes are very often seen. The superficial veins are swollen and engorged with very dark blood; this was especially noticeable in the tortuous, swollen temporal vein and the veins of the forearm. There is a cough, usually of a violent paroxysmal character, and a profuse discharge of a clear, viscid fluid. This fluid is so viscid that in some cases the cup may be inverted and the secretions will hang as a string. In some cases we find, instead of a paroxysmal cough, an incessant hacking cough, with a thick, viscid secretion that is expectorated with great difficulty. Marked inspiratory stridor, resembling that of spasmodic laryngitis, or whooping-cough, is often seen, the paroxysm ceasing with retching and the expectoration of viscid fluid. The cough is frequent at night, especially on lying down, and in the early morning hours. In children the secretion is often wanting, and the cough has a deep, brassy tone. Pulmonary hemorrhage is often one of the earliest symptoms. This may be a simple discoloration of the sputa, or it may be pure blood; it is usually recurrent, at intervals of several days. In other cases the hemorrhages recur at intervals of weeks or months.

Physical Signs.—On examining the lungs we find the changes in the respiratory sounds characteristic and pathognomic of the condition of the lungs. *The inspiration is lengthened, sharp, and of a high pitch, and immediately followed by a prolonged expiration of a lower pitch.* This respiratory sound greatly resembles that heard in the first stage of asthma; the pitch, however, is higher.[1] In the earlier stages the inspiration is very prolonged, suggestive of a difficult lung expansion, with a short expiration of a lower pitch; there is an absence of the normal susurrus, and it is harsher and sharper than normal respiration. Later the inspiration becomes harsh, high-pitched, and shorter, followed by an expiration which is softer and of lower pitch. In some cases the harsh inspiration is alone heard, the expiration being absent. This

[1] William C. Glasgow: The Etiology and Mechanism of Asthma. American Journal of the Medical Sciences, July, 1887.

peculiar respiration I believe to be due to the swollen, infiltrated condition of the bronchial mucous membrane—the same solid œdema of the mucous membrane that we see in the fauces in similar cases. Where the areas of consolidation exist, the harsh respiration gives place to the true bronchial respiration. In all cases we find this type of breathing present. In many cases we find the respiratory sound covered by the sonorous and sibilant râles. In some cases the whistling râles and the wheezing with the oppression resemble an attack of asthma. These are always transient in character; variations occur from hour to hour. Occasionally we find mixed with the inspiratory sound large-sized, subcrepitant râles of a dry character. These râles are heard in only a certain number of cases, on expiration as well as on inspiration. The rapid and striking modifications and changes in these respiratory sounds are noticeable features of the disease. The râles come and go, and the respiratory sounds vary in different degrees of harshness and softness. Striking and rapid changes may take place within a few hours. In some cases respiratory sounds are scarcely audible—in fact, there is a complete silence. This may continue for hours, when the characteristic breathing will be heard.

Percussion gives a peculiar wooden or deadened percussion sound, in which the pulmonary resonance is still recognizable, but the sense of resistance is greatly increased. In some cases the wooden percussion note has a distinct tympanitic character. If areas of consolidation are present, the complete dulness of consolidation will be apparent over those areas. There is always increased vocal resonance, or, rather, *vocal shock*, with *increased pectoral fremitus*, over that part of the lung where the deadened percussion sound is found.

The abnormal physical signs may be summarized as follows: a deadened percussion sound, increased vocal and pectoral fremitus, a harsh inspiration of high pitch, followed by an expiration of lower pitch, with the occasional presence of the dry and moist râles.

In comparing the physical signs of this condition with those of an ordinary bronchitis, we find this difference:

Bronchitis.	*Bronchial Type.*
Percussion normal.	Percussion deadened; increased resistance; occasionally tympanitic.
Vocal fremitus and pectoral fremitus normal.	Vocal and pectoral fremitus increased.
The inspiration may be harsh, often feeble, but with no change of expiration.	Inspiration harsh and high-pitched, with prolonged expiration of lower pitch.
Sonorous and sibilant râles heard both on inspiration and expiration.	Râles largely inspiratory.
Constancy of abnormal sounds during attack.	Sudden and frequent variations of the abnormal sounds.

These characteristic sounds may continue from a few hours to several weeks. The usual time is between two and four days; when they continue into the third week it is probable that the tubercular process has been added to the original disease. Resolution usually takes place rapidly, and the abnormal physical signs are quickly replaced by the normal signs. There is often, however, a noticeable tendency toward relapse, and we see often after a few days, weeks, or months, a return of the abnormal physical signs with the presence of more or less of its symptoms.

In the *parenchymatous* type the disease usually commences with a chill, followed by fever. The thermometer usually ranges between 101° to 103° or 104°; in some cases reaches as high as 105°. The fever continues with morning remissions and evening exacerbations, but there is no strict regularity in its course. In some days we will find the highest temperature during the midday hours, or it may vary from hour to hour or day to day without any regularity. In fact, this want of a systematic regular course of the fever is one of the striking characteristics of the disease. The general height of the temperature varies greatly every few days without any appreciable cause. It will continue high for three or four days; then a remission of two or three degrees will take place, and again a high temperature will commence. During the remissions there may be repetitions of the chilly sensations, or hot and cold flushes. The fever continues to the end of the disease, and ends gradually by lysis. There is a marked absence of the crisis seen in croupous pneumonia.

In the first days of the attack the patient has a quiet and placid look, and the absence of subjective symptoms is very noticeable. The tongue is remarkably moist, healthy, in some cases slightly covered with fur in the middle; the dry typhoid tongue is a rarity. There may be no cough and no pain in the side; headache, with a feeling of great lassitude, may be the only subject of the complaint. In some cases, rheumatoid pains in the limbs and in the back are present. The breathing is quiet, although the respiration is invariably increased. The ratio between pulse, respiration, and temperature shows a constant change and irregularity. Transient attacks of a feeling of oppression or suffocation come on, but these rarely last for any length of time. Later, the patient may experience great pain and tenderness in the lower border of the chest, about the upper border of the liver and spleen. This pain in some cases is very intense, and may last for twelve hours; it then disappears to reappear again at the same or some other place. I have known this pain to recur at irregular intervals during convalescence. It is entirely dissimilar from the nipple pain seen in croupal pneumonia. It is a myalgia not a pleural pain, and in some cases is widely diffused. In some cases it seems essentially neuralgic. During the whole course of the disease the

same expression of content and well-being may continue in the face. There is no cyanosis or noticeable action of the accessory muscles of respiration. The skin is of a dirty white color, and, in some cases, tinged with yellow. The pulse is generally accelerated; not, however, to a marked degree. It usually varies from 100 to 110; in some cases it has risen to 120. It is full, or half full, and very compressible. I have noticed a marked slowness in several cases, and this continued up to the time of death. In one case where I happened to be present as the patient expired I could feel the slow, half full, very compressible pulse to the last. When it ceased at the wrist it could still be felt for a few seconds in the carotids, while the patient remained conscious and expired with a few gasping respirations. Profuse perspirations are very marked. In some cases they occur during the height of the fever, and again are seen like cold sweats.

On the second or third day a cough comes on with the expectoration of a viscid, ropy secretion. In some cases this occurred in great quantities; in others it was scanty, and expectoration consisted of rounded, isolated masses of a substance resembling mucine. These masses were usually expectorated with great difficulty. The cough is, as a rule, a full, free cough—what may be called a tracheal cough, and is caused by the effort to expel the accumulating secretions. Occasionally it occurs in paroxysms. In some cases blood was mixed with the expectorated secretion, and it had a strong resemblance to the rusty colored sputa peculiar to croupous pneumonia. The color, however, is rather darker, and pure blood is almost always present in these cases. In some cases free hemorrhage has been seen; in fact, it has occurred on the onset of the disease. The blood is usually dark, and is often mixed with black clots. When the hemorrhage occurs it is generally recurrent, appearing at intervals of one, two, or more days. The hemorrhage may be in amount from half a drachm to a pint. In one case, an infant of seven months, a large hemorrhage produced death. The child had been ill two months with a pneumonia contracted in California. It is a noticeable fact that the viscid secretion always ceased with the appearance of a pure hemorrhage, and again reappeared later. In the later stages the viscid sputa shows a purulent character, this continuing for days or weeks. The purulent sputa may, however, disappear for a time, to be again replaced by the clear, viscid sputa. As the disease progresses, during recovery, a muco-purulent secretion is observed. The history and varying changes in the character of the sputa are most interesting and remarkable characteristics of the disease. The causes of these changes are unknown, as there is nothing in the clinical history that can account for them. In fatal cases, in the last days there is an absence of the cyanotic condition seen in croupous pneumonia. The

death is quiet and peaceful; it may occur at the most unexpected times, and at times when the patient shows most favorable symptoms.

Physical Signs.—On percussion, even in the first days of the attack, a deadened or a wooden percussion sound is generally apparent over parts of both lungs; over irregular areas of either one or both lungs we find a more complete dulness, almost flatness. The most frequent site of the flatness is the posterior portion of the lower part of the left upper lobe, immediately under the lower edge of the scapula. Next to this, the left upper apex was most frequently affected. As a rule, several areas of flatness were found in each lung. The pectoral fremitus was invariably exaggerated over those portions of the lung giving the wooden percussion sound; over those portions giving the flat sound it was greatly increased. On auscultation over the flat areas bronchial breathing could be heard; and over those portions of the lung which gave the wooden percussion sound a harsh, high-pitched inspiration, with a prolonged expiration, was always heard with varying degrees of intensity. Vocal resonance, or, rather, the vocal shock, was always increased over the flat areas, giving a true bronchophony; and over the deadened areas a marked increase in the vocal shock was present. As the disease progressed the only change in the vocal sounds was seen in the occurrence of the adventitious râles. Over the consolidated areas very frequently small mucous râles could be heard mixed with the bronchial breathing. This occurred both on inspiration and expiration. The sonorous and sibilant râles were also heard from time to time in the other portions of the lung. In no case was the true crepitant râle heard. The physical signs of abscess of the lung were found in two cases, which made a complete recovery. At times, in some cases, there would be a complete dulness on percussion, with increased pectoral fremitus and vocal shock, and a complete absence of the respiratory sound over that portion of the lung. In a few hours the bronchial breathing would be heard. As resolution advanced there was a rapid change in the physical signs. The dulness gave place to normal percussion sound, while the increased vocal shock and resonance disappeared, and the bronchial breathing gave place to a rather harsh vesicular breathing. The râles disappeared later than the other physical signs. When resolution was delayed the physical signs continued. In many cases amphoric breathing would be heard. In two cases this was heard at the apex and in one at the angle of the scapula. The post-mortem in these cases showed a complete consolidation of lung tissue at these points. During the course of the disease attacks of palpitation are quite frequent, with a tendency to heart failure.

It will be interesting and instructive to compare the clinical history and the signs of this disease with those found in croupous pneumonia.

In certain points there is a great similarity, and in others the difference is so great that a differential diagnosis would be made.

Croupous Pneumonia.	*The Parenchymatous Type.*
The chill not repeated.	The chill repeated, with occasional hot and cold flushes.
Fever of a regular type, terminating most frequently by crisis.	Fever irregular, terminating by lysis.
Sharp, lancinating pain about the nipple, disappearing after a few days.	No pain in the commencement, but later through the lungs, and especially over the lower lobes.
Cough in early stage constant, painful, and suppressed.	No cough at first; later cough on expectoration.
Face distressed in early stage of the disease, with red spots on cheeks, face becoming cyanotic and livid as the disease progresses.	Face has a calm, restful look, usually somewhat pale, with rather a dirty white color.
Pulse hard, rapid, small, becoming very much accelerated toward the end.	The pulse full, compressible, rarely over 120, in some cases very slow.
Respiration in earlier stages quickened, suppressed, and shallow; in later stages deep, labored,and heaving.	Respiration not visibly accelerated. Ratio between pulse, respiration and temperature irregular. In the last stage respiration often quiet and slow.
On percussion a dulness, usually following the line of the lobes, most frequently the lower lobes. Respiratory sounds mostly normal over remaining parts of lung.	On percussion a flatness in irregular areas, affecting most frequently the middle and upper lobes; deadened percussion sound over other parts of lung, with increased vocal and pectoral fremitus.
On auscultation the crepitant râle heard in early stage; a râle redux in later stages.	The crepitant râle absent; the râle redux not recognizable.
Bronchial breathing, gradually becoming broncho-vesicular and vesicular as resolution takes place.	Respiration is bronchial over the dull area, and over the remaining portion of the lung a harsh, high-pitched inspiration, with a prolonged expiration of lower pitch on resolution changing rapidly to the normal respiratory murmur.
Sputa small in quantity, always viscid, almost always rusty colored.	Sputa may be profuse, a clear, viscid fluid often mixed with blood—becoming later purulent.
Death agony prolonged, with labored breathing and a general condition of cyanosis.	Death sudden, with a few gasping respirations; peaceful.
A sero-fibrinous or fibrinous pleurisy occasionally found.	Purulent effusions in pleura, pericardium, and other cavities.

In the Mullanphy Hospital three cases during convalescence from pneumonia died in a state of coma, with paralysis and symptoms of meningitis, and one case on post-mortem showed the rupture of a softened artery and a blood-clot in the brain. A mild delirium is sometimes seen with hallucinations. One case during convalescence had facial erysipelas with abscess of the eyelids, and later enlargement and suppuration of the maxillary glands. In the other, erysipelas preceded pneumonia, and the patient died during the latter. In one case an attack of pneumonia was followed by peritonitis, to which the patient succumbed. In the City Hospital in many cases the symptoms of acute nephritis have been added to those of the lung disease. On December 27, 1889, a patient entered the Mullanphy Hospital with acute nephritis, suppression of urine, and general anasarca. On the second day after admittance he began to expectorate quantities of viscid sputa mixed with blood, and on examination the physical signs of broncho-pneumonia were found. Gastro-intestinal disturbance has been the initial symptom in several cases. Two cases were admitted into the City Hospital with such violent vomiting and purging that poisoning was suspected. The following day the signs of broncho-pneumonia were well developed. Skin eruptions with glandular enlargement have been frequently observed. The herpetic eruptions and purulent bullæ have been most frequent. In one case an erythematous blush over the whole body somewhat resembling scarlet fever was observed. Numerous cases have complained of violent otalgia, and in many of these there has been an œdematous, swollen condition of the external ear. This occurred frequently during convalescence. Pericarditis has occurred in several cases. The parenchymatous consolidation may continue with the usual symptoms and physical signs from five to thirty days, and still resolution with recovery may take place.

That these two classes of cases are types, rather than stages, of the same disease is evidenced by the fact that clinical observation has proven that the typical condition, as observed, remains through the whole course of the disease. In the cases under my observation the bronchial type never merged into the parenchymatous, and the parenchymatous type, with its extensive consolidated areas, is observed on the first days of the illness. Observation of a certain number of cases of both types during the past two years has conclusively proven that when the process continues a certain length of time the result in both classes of disease is tubercular disease. The period when the tubercular process is added to the bronchial pneumonia is still uncertain, but from my observation I can safely say that when the symptoms and the physical signs continue for one month there is a strong probability that the bacillus tuberculosis will be found in the sputa. In a few cases careful and repeated examinations of the sputa were made by Dr. E. M. Sen-

seny in the Mullanphy Hospital, and he finds that the bacillus appears about the third week of the disease. The appearance of the bacillus coincides in many cases with the change of the sputa from the clear, viscid fluid to the viscid, purulent secretion. At this time the expectoration may become of the typical tubercular type, showing the minute white particles, and a microscopic examination reveals the bacillus tuberculosis in enormous numbers. I can, however, mention two cases where the consolidation continued six weeks; both these patients recovered; no bacillus could be found in the sputa. Some have claimed that the bronchial type of cases are simply cases of tubercular disease, but repeated examinations of sputa by Dr. Senseny, and numerous microscopic examinations with cultures and inoculations of the lung tissues made by Dr. Bremer, have positively proved the absence of the tubercular bacillus in the early stages of the disease. It seems to be pretty well established that the condition of lung tissue in this disease proves a most fertile soil for the development and growth of the tubercular germ. This may account for the fact which I have observed during the past few years that the number of persons showing tubercular disease has greatly increased, and this is true of persons in whose families no hereditary disease or disposition can be found.

The question of the contagiousness of this disease is still an open one, but many facts have occurred that seem at least to suggest it. Last winter, when a ward in the Mullanphy Hospital contained four cases of this disease, several other cases with different forms of disease began expectorating the viscid, gelatinous sputa similar to that of the pneumonic cases, and the viscid sputa did not entirely disappear from the ward until the walls had been washed and cleaned.

I am informed by Dr. Senseny that in the female ward it was noticed that the patients occupying the adjoining beds to the pneumonic cases seemed to contract the same disease in the mild form.

In the City Hospital many cases have occurred in the surgical ward, where the patients had long been occupants of the hospital, and confined to bed by their injuries.

That the disease is a septic disease cannot be questioned after a consideration of the post-mortem appearance of the organs. That it is primarily due to some change in the blood, possibly the presence of a microörganism, seems most probable from a clinical study of the disease. The microscope shows the presence of broncho-pneumonic infiltration of the lung tissue, but that this is a secondary process is self-evident. No local inflammatory disease could be associated with such a disorganized condition of the other vital organs, these conditions being essentially that produced by sepsis.

The microörganism causing this septicæmia I must leave others to investigate. In this age of bacteriology, with the modern improved

methods of research and investigation, the solution of the problem cannot long be delayed.

In a paper read before the Eleventh Congress of the American Laryngological Association, held in Washington in June of last year, I read a paper entitled "An Œdematous Form of Disease of the Upper Air-passages."[1] In this paper I described a peculiar condition of the upper air-passages, accompanied by symptoms showing a close similarity to those seen in the disease of the lungs described in this paper. The similarity was especially marked in the nature of the viscid fluid which was expectorated, and in the venous engorgement and the character of the symptoms. I claimed in this paper that the throat disease was only a manifestation of a general constitutional condition, and that a similar disease existed in the bronchi and the parenchyma of the lung. I was also justified in this conclusion from the fact that in a large number of these cases of lung disease the palate and uvula will be found in the condition of solid œdema described in my former paper, and I could only reasonably conclude that the diseases of the upper air-passages—the bronchi and the lung parenchyma—are all different manifestations of the same process, varying only in different degrees of intensity.

In my former article I suggested that probably this disease, from the similarity of the symptoms, might be the "influenza," so graphically described by Graves, or the "grippe" of Valleix; but in the absence of any definite description of the pathological condition found in influenza, I was not justified in so naming it. At the time this paper was written, April, 1889, there was no notice of the presence of influenza in any part of the globe, and it seems strange that a disease which has always occurred in waves, spreading rapidly over large portions of the world, should show itself as a sporadic form of disease. However, the throat disease described in my former paper and the lung disease the subject of this article were not confined to this part of the country. I saw one of these cases of septic broncho-pneumonia originating on the Mexican Plateau, one in Colorado, one in El Paso, Texas, one in California, and one in Galveston, Texas. Cases of the throat disease originated in Boston, Brooklyn, New York, Philadelphia, Pittsburg, and New Orleans. At the meeting of the American Medical Association in Cincinnati, 1888, Dr. Carl Seiler, of Philadelphia, was the only physician of many whom I consulted that had recognized the peculiar throat affection. In May, 1888, Dr. R. P. Lincoln, of New York, described to me an obscure case of lung disease he had seen with Dr. Loomis, and from the description I recognized one of the cases described in this paper. If this disease is the influenza, we have had numerous cases in this country three years

[1] W. C. Glasgow, M.D.: An Œdematous Form of Disease or Septic Œdema of the Upper Air-passages. New York Medical Journal, August 10, 1889.

preceding its appearance in Europe. During the winters of 1886, 1887, and 1888 they were numerous, and during the past summer and autumn a number have been seen in the hospitals of this city. The number of cases began largely to increase during December of 1889, and at the present time there are a large number in the City and Mullanphy Hospitals. During the month of January, 1890, while the epidemic of influenza prevailed, large numbers of these cases appeared in St. Louis. The greatest number have been of the bronchial type. They have occurred as a relapse of the influenza one, two, three, four, and six weeks after the initial attack.

Three years ago, when the parenchymatous type of this disease was considered as croupous pneumonia, it was treated by the stimulant plan usual in this disease. The sudden and great mortality proved the uselessness of this form of treatment. From the success attendant on the use of benzoate of soda in the œdematous disease of the upper air-passages, I was led to use it in this condition of the lung, and the success and the greatly diminished mortality were so striking that I am convinced it is the best remedy for this class of cases. My individual experience is in accordance with that of the gentlemen in charge of the City Hospital, who have found a greatly diminished mortality since the benzoate of soda treatment was introduced. I usually give large doses of the benzoate of soda with acetate of ammonia, and supplement this with large doses of muriated tincture of iron, with a moderate use of stimulants. The excessive use of stimulants is injurious, as at the critical periods the heart cannot respond. The addition of the salicylate of soda in some cases to the benzoate seems to be useful. In certain cases large individual doses of quinine will be necessary to bring down the temperature when it becomes too high, and as there is a constant tendency to heart failure the use of digitalis and the other heart tonics and diffusible stimulants will often be necessary. *Careful and constant attention is absolutely necessary. Many patients die who might be saved by the timely administration of a stimulant in threatened heart failure.* Antifebrin has also been used with a certain amount of success in the City Hospital.

In conclusion, I would publicly express my thanks to Dr. Dalton, Superintendent of the City Hospital, who has placed the large material of that hospital so freely at my disposal, and also to the physicians in attendance at that institution during the past two years. Their interest in the investigations of this disease, their post-mortem examinations, and their confirmation of the symptoms and physical signs as embraced in this paper will strengthen the views I have advanced. My thanks are also due to Dr. L. Bremer, Professor of Physiology and Bacteriology in the Missouri Medical College, who has assisted at most of the post-mortems.

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