

## **A case of status lymphaticus / by J.H. Musser and J.T. Ullom.**

### **Contributors**

Musser, John Herr, 1856-1912.

Ullom, J.T.

Royal College of Surgeons of England

### **Publication/Creation**

[Detroit] : E.G. Swift, 1904.

### **Persistent URL**

<https://wellcomecollection.org/works/b8dw4es2>

### **Provider**

Royal College of Surgeons

### **License and attribution**

This material has been provided by This material has been provided by The Royal College of Surgeons of England. The original may be consulted at The Royal College of Surgeons of England. where the originals may be consulted. The copyright of this item has not been evaluated. Please refer to the original publisher/creator of this item for more information. You are free to use this item in any way that is permitted by the copyright and related rights legislation that applies to your use. See [rightsstatements.org](https://rightsstatements.org) for more information.

p.c. 5

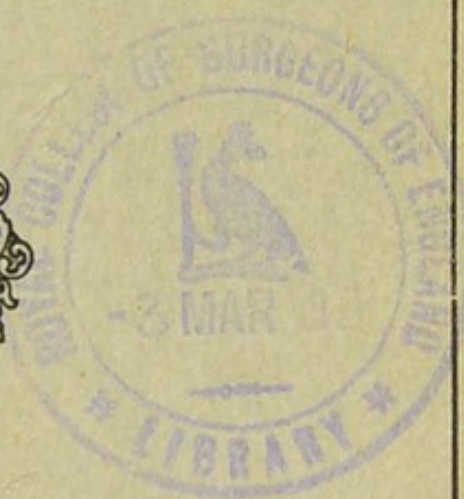
2

# A CASE OF STATUS LYMPHATICUS

BY

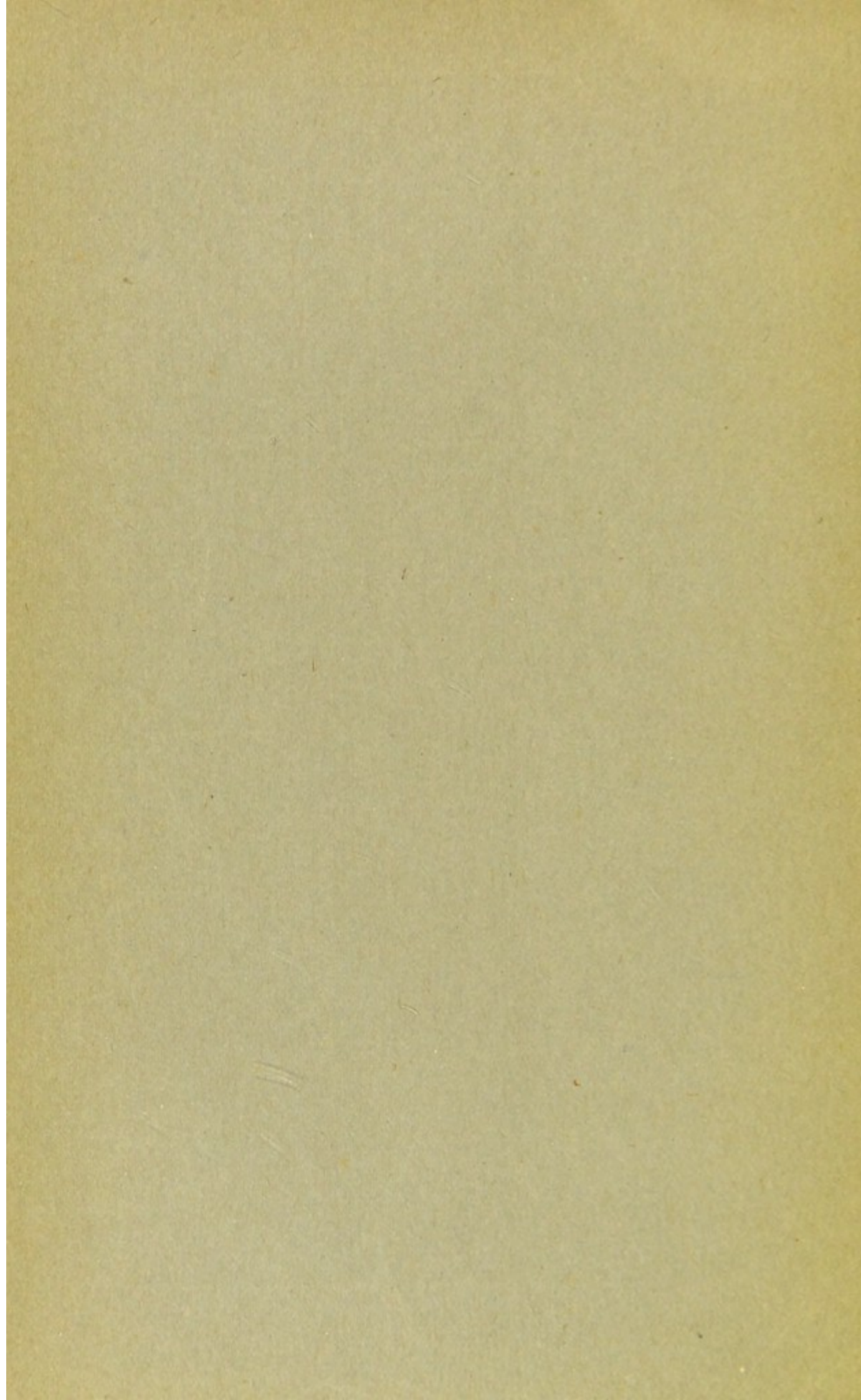
J. H. MUSSER, M.D., and J. T. ULLOM, M.D.

PHILADELPHIA, PA.

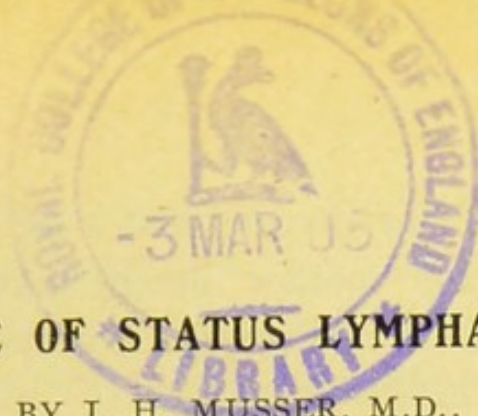


REPRINTED FROM MEDICINE  
E. G. SWIFT, PUBLISHER  
MAY, 1904









## A CASE OF STATUS LYMPHATICUS.

BY J. H. MUSSER, M.D.,

AND

J. T. ULLOM, M.D., PHILADELPHIA, PA.

T. D——, aged 5 years and 11 months, when about 18 months old had a protracted enterocolitis which ran a prolonged febrile course; had also a discharging ear as a resultant of an attack of influenza; and had had occasional slight febrile attacks attributed to digestive errors. After his enterocolitis he was very anemic and emaciated, but never had rickets. Took on flesh rapidly and grew rapidly, and at the time of his fatal illness was very well nourished and large for his age.

Family history: Singularly prone to cardiovascular disease both on the maternal and paternal side.

Present illness: On the evening of November 10 he became hoarse, and the hoarseness continued throughout the night, but on November 11 he seemed much better and played as usual all day. He was seen about 6.30 or 7 P.M. of that day by the family physician, and his temperature was  $100^{\circ}$  and his pulse 100. The pulse seemed a little rapid for the temperature, but it was usually out of proportion to any fever, and little attention was paid to it.



He went to bed as usual and slept soundly, but at 2 A.M. his mother noted that he looked pinched, and that there was a great deal of rattling and bubbling apparently in his throat. No alarm was felt, however, until about 4.30, when, though still sleeping, it was noted that he was very much cyanosed in his face and hands, and that his breathing was rapid and short.

The physician saw him about 5.30 A.M., and at this time he was in a state of shock and had some difficulty in breathing, respirations 45 to 50, pulse 160, quick and weak, some cyanosis of the face and hands. The bubbling and rattling in throat and chest was very evident. He was asleep, but when aroused his mind was perfectly clear. His voice sounded hoarse. At 7 o'clock he was much the same, his temperature being  $102^{\circ}$ , pulse 150, respirations 50. He was extremely restless, tossing about in his bed. His voice was at turns hoarse, and again would become perfectly clear. At 9 o'clock he was about the same. He was actively stimulated with strychnine, whiskey, and atropine since the last observation. His pulse was 150 to 160, respiration about 60. His voice was clear and hoarse at intervals, as before noted.

Physical examination: Boy, very large, apparently older than five years. Clear complexion, the skin being very clear and the veins very distinct. He was very sturdily built, being



what is ordinarily called a "fat boy." He was very dyspneic, and his face and hands were somewhat cyanosed and pupils dilated. Waxy pallor of the hands and face was most apparent. There was bubbling and rattling in the throat and chest, such as is heard in ether anesthesia. There was retraction of the interspaces and epigastrium on inspiration, with labored inspiration and expiration. You were impressed with the effort required to breathe. He was very restless, and even during the physical examination tossed all over the bed. There was great movement of the larynx. His pharynx and larynx showed no membrane, but were congested.

Palpation: Practically negative.

Percussion: Revealed no areas of dulness, indeed a hyperresonant note all over the chest, although the observations were made with difficulty.

Auscultation: Only a confused bubbling and rattling could be heard, no bronchial breathing. There seemed to be apparently mediastinal pressure, from the labored breathing.

A diagnosis of probable hypertrophy of the thymus or of status lymphaticus in the course of some infection was made, and as the possibility of a laryngeal or tracheal diphtheria was thought of, he was given 3000 units of antitoxin, and was heroically stimulated. His pulse now was 160 and very weak, and respira-



tions 60 to 70. After the stimulants there would be some response, but after a few minutes the pulse would be as weak as before. His condition grew progressively worse, and at 12.30 he had a slight convulsive seizure. He seemed now to be entirely oblivious to his surroundings, although until this time he had spoken to those about him and recognized them. These slight convulsive seizures continued at short intervals (five or ten minutes). He was placed in a mustard bath, and responded to this very well, his pulse becoming stronger and less rapid, about 140, and his convulsions ceasing temporarily. About 1 P.M. his temperature, taken in the axilla, was  $105.6^{\circ}$ . His stimulation had been carried on regularly throughout all this time, but he went rapidly from bad to worse, and died peacefully at 2.30 P.M. His urine, obtained by a catheter at 1 P.M. and examined after death, was loaded with albumin but showed no casts. Autopsy performed at 5 P.M.

*Autopsy:* Made by Dr. W. T. Longcope. The body is that of a young lad about 120 centimeters in length. Rigor mortis present. Moderate post-mortem discoloration over dependent parts. Pupils equal and dilated. No edema of extremities. Subcutaneous fat present in good amount, of orange-yellow color. Muscles dark.

Abdominal cavity: Omentum contains much



fat, and entirely covers the intestines. Peritoneal surfaces are everywhere pale, smooth, and glistening. The small intestines are slightly distended with gas. Appendix lies over the brim of the pelvis, is patent throughout and appears normal. Diaphragm on right side reaches the third rib, on left side to third interspace.

Thorax: On removing sternum, lungs collapse slightly. The left pleural cavity is free from fluid and adhesions. A few cubic centimeters of turbid fluid of a yellowish color are found in the right cavity and over the posterior portion of the lung. The parietal and visceral pleura are adherent by a few delicate fibrinous adhesions. The superior mediastinum is almost entirely filled by the thymus gland. The general appearance is of three lobes; a middle lobe which lies, as far as can be seen, just below the sternal ends of the clavicles and reaches almost from one clavicle to the other, being about one centimeter nearer the left than the right. What appears as the lateral lobes extend down into the mediastinal fat, between the pleura and pericardium. The right lobe measures six centimeters in length and extends to the upper margin of the third costal cartilage. These lateral lobes are more or less fused above the pericardium, but as they descend they separate and apparently extend far down between the pericardial sac and lungs. Lower down the phrenic



nerve is in intimate connection with the gland on both sides.

The pericardial cavity contains a slight excess of fluid, clear and straw-colored. The serous surfaces are smooth and glistening.

Heart: The heart is of moderate size. Epicardium contains a slight excess of fat. Over the tip of the left ventricle is a soft, raised, milk patch about the size of a five-cent piece. The heart contains firm, red, post-mortem clots and a small amount of fluid blood. All the valves are thin, delicate, and normal. The heart's flesh is firm, brownish-red, and rather dark in color. The aorta is smooth.

Auricular appendages free from thrombi.

Lungs: The left lung is of medium size, pleura is smooth and glistening, free from adhesions, and most of the upper lobe and anterior portion of lower lobe is pale pinkish-gray, rather puffed up and crepitant. In the posterior portion of the lung, and most extensive in the lower lobe, there are many irregular firm areas which are of a deep purplish-blue color, and sharply defined from the gray crepitant lung. The smaller ones measure one-half to one centimeter in diameter, and are slightly depressed. The larger ones average from four to five centimeters in diameter, are raised, and have a slightly lobulated appearance. On section the general cut surface is deep pink, moist, somewhat irregular, but crepitant. Here and



there in the anterior portion of the lung small, deep purplish-black nodules are seen, which are raised, well defined, and measure from three to five millimeters in diameter. When they occur near the surface they correspond to the depressed bluish areas. In the posterior part of the lobes there is a very irregular and extensive patchy consolidation of a purplish-black color. These consolidated areas are raised and have a smooth, slippery surface, which is irregular and appears somewhat lobulated. Scattered everywhere through the consolidations are seen small gray or yellowish points, the largest measuring from two to three millimeters in diameter; some of them exude drops of yellow pus. The smaller bronchi in these areas frequently contain pus, which can be squeezed out in thick, yellow drops. The bronchi at the root are deeply congested and contain a thick, yellow, mucopurulent material, which is most plentiful in the smaller ramifications. The vessels at the root are clear. The bronchial glands are not specially enlarged; they measure from three to eight millimeters in diameter and are pinkish-gray in color. The right lung is of medium size. The middle lobe and most of the anterior portion of the upper lobe is pale pinkish-gray, dotted with a few small, deep purple areas, but is generally insufflated and crepitant.

On the under surface of the middle lobe along the extreme margin there are several



large air bullæ. The lower lobe is very heavy, entirely solid, and of a deep purplish color. It has a rather nodular feel. In the posterior portion of the lobe there is a rounded, particularly raised area about five centimeters in diameter. This has a mottled, reddish black, gray, and yellow appearance, while the pleura over it is roughened by a delicate film of fibrin. For some distance about this area the pleura is filled by a fine granular exudate, but elsewhere is smooth and glistening. On section the upper lobe and middle lobe show irregular areas of deep purple consolidations studded with minute yellow points, and in general the cut surface appears much like the section through the left lung. The cut surface of the lower lobe is deep purplish-red in color, but has a somewhat nodular look and feel, due to small raised masses from three to eight millimeters in diameter which are of a yellow-gray color.

The general consolidated surface is smooth and slippery, but the raised nodules have a more or less granular look and seem to be made up of many small, discrete, and confluent granules. Beneath the area over which the pleura shows a fibrinous exudate, the section exposes a large raised granular patch  $2\frac{1}{2}$  centimeters in diameter which is thickly sprinkled with minute yellow abscesses. The smaller bronchi, in many places, are entirely plugged by thick, yellowish pus. The bronchi at the root



are deeply congested and contain a small amount of the same material. The vessels are clear. The lymph glands at the root are somewhat larger than those on the left side, but have the same general appearance.

The tonsils are apparently normal. The epiglottis is edematous and deeply congested. It is covered by a good deal of mucus and a soft, yellowish, purulent material which is readily wiped off. The larynx is also deeply congested, being dark purplish-red in color and very edematous, so that the ventricles are almost closed, but on pressure a small amount of their purulent fluid wells up over the swollen vocal cords.

Small flakes of mucopurulent material cover the mucous membrane of the larynx, but there is no ulceration and no false membrane. The trachea shows the same condition as the larynx and the epiglottis, and this extends down into the bronchi. The right lobe of the thyroid gland and isthmus appear normal. The left lobe of the thyroid gland measures about four centimeters in length. Attached to its lower border is a smooth, rounded mass which measures about two centimeters in diameter, and appears about the size of a Lima bean. On careful dissection this does not appear to be in direct connection with the thyroid gland.

On section the cut surface is rather firm and has a slightly yellowish-gray color, being some-



what translucent. It is dotted with minute points, opaque and of yellowish hue.

The thymus gland appears very large, and is more or less divided into three lobes. The upper or middle lobe arises  $1\frac{1}{2}$  centimeters below the lower edge of the thyroid gland, and measures  $5\frac{1}{2}$  centimeters in length and  $2\frac{1}{2}$  centimeters in width. The left lower lateral lobe measures 9 centimeters in length and 4 centimeters in width. The right lateral lobe measures 7 centimeters in length and 4 centimeters in width at its widest point. These two lateral lobes extend down in a wing-like manner from the upper and central lobe.

The thymus weighs 40 grammes. On section it is of a pale pinkish-gray color, dotted with minute opaque yellow points. The consistency is rather firm.

Spleen: Weight 50 grammes. Size  $8 \times 5 \times 3\frac{1}{2}$  centimeters. The spleen is of medium size, decreased in consistency, and of a pale lilac color. The capsule is smooth and free from adhesions. On section the pulp is soft, of a dirty-red color, and pliable rather than friable. Here and there small hemorrhagic points are seen. Malpighian bodies are large and well defined. The trabeculæ are not increased.

Liver: The liver is of normal size, regular in outline, decreased in consistency, and dark brownish in color. The capsule is smooth and free from adhesions. On section the cut sur-



face is smooth, dark brownish-red in color, and oozes much blood. The lobules are fairly well marked and have deep-red centers.

Portal connective tissue not increased.

Gall-bladder is distended with dark-green bile. Bile-ducts patent.

Kidneys: Both alike; combined weight 100 grammes. The right kidney measures  $8 \times 4\frac{1}{2} \times 3\frac{1}{2}$  centimeters. It is of medium size and decreased in consistency. The capsule strips readily, leaving a smooth, dark reddish-gray surface, covered everywhere with injected stellate veins. On section the medullary pyramids are pale about the papillæ, but deeply injected about the peripheral zone. The cortex averages from 5 to 7 millimeters in thickness, is grayish-red in color, and cloudy; striæ are fairly well marked; glomerule injected; pelvis and ureter are apparently normal. Pancreas, stomach, and esophagus apparently normal. Aorta smooth. Intestines apparently normal.

*Anatomical Diagnosis.*—Acute mucopurulent laryngitis, tracheitis, and bronchitis; acute diffuse bronchopneumonia; hypertrophy of thymus gland; congestion of liver and kidneys; acute splenic tumor, and cloudy swelling of viscera.

*Histology.*—Heart muscle shows edema, fragmentation, absence of cross-striation of fibers and marked granulation of muscle cells.

Lungs show extensive bronchopneumonia of



hemorrhagic type. Collections of staphylococci are scattered through the consolidated parts, and above them the lung tissue and exudate in air cells is necrotic.

The vessels show no especial changes. Bronchi show marked alterations, consisting in thickening of mucous membrane with infiltration of polymorphonuclear leucocytes, small round cells and epithelioid cells, while the superficial portions are necrotic, and epithelium is partially or wholly desquamated. There are many cocci in groups, found in sections stained by Weigert-Gram, which are scattered through the necrotic mucous membrane and seen in the lumen.

The trachea and larynx show practically the same condition as the bronchi. Mucosa is partially necrotic and covered with masses of cocci in groups. There is practically no formation of fibrin, and no pseudomembrane. No bacilli can be found. Small lymph glands surrounding the trachea are infiltrated with polymorphonuclear leucocytes, and one gland is converted into an abscess. The bronchial and cervical lymph glands show hyperplasia of the lymphoid follicles with swelling and necrosis of the germinal centers. There is endothelial proliferation, and the lymph sinuses are large and packed with polymorphonuclear leucocytes, epithelioid cells, red blood cells, fibrin, and coagulated serum.

The thymus gland shows very little change



from the normal, microscopically. The centers of the lobules are perhaps slightly enlarged, and the lymphadenoid tissue about the periphery hyperplastic. The spleen is congested, and the pulp is rich in lymphoid cells and epithelioid cells. The Malpighian bodies are swollen and the germinal centers are extremely large. There is extensive necrosis of the cells of the germinal centers.

Liver shows congestion accompanied with cloudy swelling and fatty degeneration.

The kidney shows marked cloudy swelling and congestion. There are no proliferative changes.

*Microscopical Diagnosis.*—Extensive pneumonia with areas of necrosis. Acute inflammation and superficial necrosis of the mucous membrane of the larynx, trachea, and bronchi, acute cervical and bronchial adenitis, simple hyperplasia of thymus gland, congestion of spleen with central necrosis of Malpighian bodies, parenchymatous degeneration of liver and kidney.

*Bacteriological Examination.*—From lung and mucous membrane of larynx, staphylococcus pyogenes aureus. No other organism. Cover-slips from larynx and lungs showed only cocci in groups. No other bacteria. No growth of bacteria was obtained from heart, spleen, liver, kidney, or thymus gland.

In taking up for consideration this very in-



teresting condition, our attention turns to its etiology, pathology, diagnosis, and cause of death.

The condition has received such a limited amount of study, and the cases reported are so few, that little regarding its etiology can be gained from an analysis of the cases, and theories as to its cause must, at best, be vague and uncertain. It is considered a disease of childhood, and probably it is usually found in early life, but that it does occur in adults is without any doubt, for of eighteen cases available we found six occurring in patients between 27 and 55.

Rickets is supposed to be a predisposing factor, or at least to be usually coincident, but in these cases we failed to find one with a history of rickets. A careful analysis of eighteen cases shows twelve males and six females, and the ages vary from 18 days to 55 years. There was negative family and personal history in thirteen, and of the others, one, a male of 3½ months, had a history of alcoholism in the mother, and both grandfathers died of phthisis. One, a male of 40, had a personal history of alcohol. One, a male of 30, had had syphilis, rheumatism, and smallpox, and used alcohol to excess. One, a lad of 13, had a history of dyspnea on exertion; and one, a boy of 5, had a history of Bright's disease and organic heart disease and tuberculosis on the father's side,



and Bright's disease on the maternal side, and a personal history of enterocolitis.

Of the eighteen cases, nine died suddenly without apparent cause—five of anesthesia and one of complicating tetanus, one after an illness of several months in which dyspnea and aphonia were the principal symptoms, and for which tracheotomy had been done, and in whom at autopsy an aneurism of the thoracic aorta was found which exerted no pressure. One died of a condition diagnosed as laryngismus stridulus of a few hours' duration, and this probably belongs in the class of cases dying suddenly without apparent cause. One case, the one reported, died with complicating bronchopneumonia.

In the nine cases of sudden death, six were children, and were found dead in bed after having gone to sleep in apparent health. One was a male of 30, who became unconscious while having his boots blacked, and died on the way to the hospital. One was a lad of 13, who while in apparently the best of health had a few slight convulsive seizures and died. One was a woman of 30, who died suddenly twenty-two hours after a normal labor. There were five cases from anesthesia, the anesthetic not known, the operations being thyroidectomy, circumcision, repair of vicious non-union of humerus, supravaginal hysterectomy, and operation for necrosis of jaw-bone. The death in



each case seemed due to sudden cardiac and respiratory failure.

Kundrat reports ten deaths from chloroform in which the thymus was enlarged.

*Pathology.*—The pathological findings in these cases were practically constant, and consist of an enlarged thymus, spleen, lymph glands, Peyer's patches, tonsils, and pharyngeal lymph tissue. Not all these are found in every case, the enlarged thymus, spleen, and some of the lymph glands being found constantly. Cloudy swelling of liver and kidney also is a constant lesion. Histologically we find hyperplasia and proliferation of the lymphadenoid tissue of the thymus, lymph gland, and spleen, with focal necrosis of the germinal centers of these organs.

The question arises, does this case conform to the pathology of the disease? We think it does. The one thing lacking is the focal necrosis and marked hyperplasia of the germinal centers in the thymus. But we think this can be overlooked when there is marked enlargement of the thymus, and the changes in spleen and cervical and bronchial glands are identical with those found in other cases. The pneumonic process, the changes in the heart muscle, and the condition of the air-passages we will take up under the causation of death.

*Diagnosis.*—The diagnosis of the disease can be made, but an opportunity is rarely given.



Dyspnea, dulness over the manubrium, and enlargement of the superficial lymph glands are the factors on which we may base our diagnosis. Suddenness of onset, a transparent skin with prominent veins, and the absence of other explanations of the symptoms, are minor factors in the conclusions.

Ewing has claimed a lymphocytosis in the disease, and offers in support a case in which the lymphocytes small and medium were 84 per cent of the leucocytes present, but the evidence of one case is hardly convincing. Hoarseness was noted in our case, which was transient, and in another case, a man of 30, there was complete aphonia. In our case the hoarseness was due to the laryngitis, and its cause was probably to be found in the infection and not in the disease under consideration.

*Cause of Death.*—The cause of death is probably the most interesting point in this rare condition. There has been a great deal of debate in the matter, one set of observers assigning pressure by the thymus, another toxemia, as a cause of death.

Schule claims that it requires 1000 grammes to compress the trachea, and there is no case on record where the weight of the thymus was above 150 grammes. In twelve cases where the weight was given, the average weight was 54 grammes, the highest 150 grammes.

The thymus normally, according to Biedert,



weighs at birth 14 grammes, at 9 months 20 grammes, and at 2 years 27 grammes. After the second year it remains about constant till puberty, after which it atrophies.

It seems indisputable that the thymus does cause pressure on the trachea, as witness the case of Koenig and Purruicker, in which operation relieved the dyspnea. Lange reports a case where the trachea was compressed from left to right and from behind forward until one diameter was only one-third of the other. That it may cause dyspnea by pressure on the phrenics or pneumogastric may be possible, but has never been proven. The cases of Koenig and Purruicker were both in young infants, one 3 weeks and one  $3\frac{1}{2}$  months.

The other set of observers claim death to result from toxemia induced by the thymus or lymphatic apparatus. This opinion is not without very plausible evidence in its favor.

Lochte, in some experiments on hyperthymization of the blood, found that this caused, first, a lowering of blood-pressure due to paralysis or depression of the vasoconstrictors; secondly, acceleration of pulse due to direct influence on the heart; thirdly, an overdose killed, showing results at autopsy suggestive of asphyxia.

Flexner in some experiments with mydaxtoxins and cytotoxins found that their introduction caused a hyperplasia of the lymphoid tissue



of the lymph glands and spleen with focal necrosis of their germinal centers.

Blumer has called attention to the similarity in the lesions found to those of status lymphaticus. There is of course no change noted in the thymus, but the changes in the spleen and lymph gland are practically identical.

It is hardly conceivable that in those cases dying very suddenly, without any previous evidence of disease, the thymus could swell to sufficient bulk within a very short time to compress the trachea or cause dangerous pressure on the phrenics or pneumogastric, and none of the autopsies would further this view. On the other hand, that there is more or less of an autointoxication present, and that a sudden accession of toxic material is sufficient to disturb the physiological equilibrium and cause sudden death, is not an unwarrantable proposition.

It might well be asked what gives rise to this sudden accession of toxic material, and in most of the cases we must confess we are unable to answer. Whether there is a sudden cumulative effect or not, with some unknown factor as a cause, we find it impossible to say.

In our case there was no evidence of compression of the trachea, while there was a sudden onset and a coincident bronchopneumonia. There was cardiac failure from the beginning, and there can be little question that this was



the main factor in the cause of death. The heart muscle was degenerated, and we naturally seek the cause of this condition. There was an infection of the larynx, trachea, and pulmonary tissue, with consolidation of one lobe and small patches throughout the lung. Our alternative is a toxemia, and we have two possible sources for that—one the infection, and the other the toxin, the product of the lymphatic system. While the source of the toxemia might have been the infection, yet the experiments of Lochte in hyperthymization proved that there was a direct depressant and paralyzant effect on the heart muscle, and until there is a further study on the subject no one can deny the plausibility of a theory of an autointoxication with its source resident in the lymphatic apparatus. In fact all our clinical experiences show that an infection of the type we had to deal with would not have caused such toxemia in a few hours.

The question arises as to whether the condition of status lymphaticus causes a lowered vital resistance, thus rendering the organism more subject to an infection, or whether the infection was a mere coincidence and became a factor in causing death.

Two positions are open to us. We can say that the infection destroyed the vital equilibrium and permitted the toxemia due to the thymus to kill, or we can say that the lowered



resistance due to the thymus permitted the infection to kill more quickly than it otherwise would. Longcope has found that in chronic Bright's disease and cirrhosis of the liver there is a decrease in the serum-complement or alexin in the blood, and this fact permits of an easy infection, in most cases a terminal one. It is not hard to trace a similarity between such diseases and this one of status lymphaticus. The question is one, however, that requires a more complete study and observation of a large number of cases. The condition in the larynx unquestionably gave rise to the hoarseness, and the laryngitis was only a part of the staphylococcus infection.

#### REMARKS BY DR. MUSSER.

The practical point was this: A child apparently robust, with signs of laryngitis and bronchitis, but without high temperature, is after an illness of a few hours evidently dying, rapidly overwhelmed by some cause not readily demonstrable. Death was evidently imminent because of some toxic agent spending its force upon the circulation—a cardiac death. We could not realize it was due to an infection of the pneumococcus or streptococcus type because of the clinical course, the low temperature, and the physical signs. That bronchial diphtheria was present was more than likely because of this low temperature, the rapid course, and the



profound toxemia, although we believe, if diphtheria, an albuminuria should have been present with the first examination of the urine. Moreover, no etiological facts supported this assumption. Some other agent than that of an infection had to be involved, and so we concluded, because of the lymphatic type of the patient, because of the rapid onset of shock in the course of a mild infection, because of the seemingly manifest signs of mediastinal pressure and the absence of uremia, the case was one of lymphatic constitution with enlargement of the thymus gland, in which status lymphaticus had taken place. In the light of the autopsy, although we did not realize such great infection of the lymphatic system, we were supported in the contention. We must confess, from the character of the respiration, we thought there was occlusion of the bronchi from intrathoracic pressure. Because of the age, the physical appearance of the patient, the sudden onset, and the absence of pleural effusion, we thought the thymus gland was enlarged and pressed upon the bronchi. Perhaps had we remembered the aphonia would come and go, we might have excluded pressure.

On percussion the high degree of secondary emphysema and circular shape of the chest permitted only a highly tympanitic note to be heard.

While the autopsy disclosed a thymus gland



fully one-third larger than normal, there was no gross indication of pressure upon the other structures. It is possible with the agonal emptying of vessels any signs of marked pressure might have disappeared. Even though obstruction of the air-passages from within or without was present, death was overtaking the patient because of shock, and cardiovascular failure, we believe, due to the lymphatic toxemia. The diagnosis during life was that of status lymphaticus or lymphatism with enlarged thymus gland, in the course of which an infection causing laryngitis, tracheitis, and bronchitis occurred.



