Indurative mediastino-pericarditis / by Thomas Harris.

INDURATIVE

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THOMAS HARRIS, M.D., F.R.C.P.

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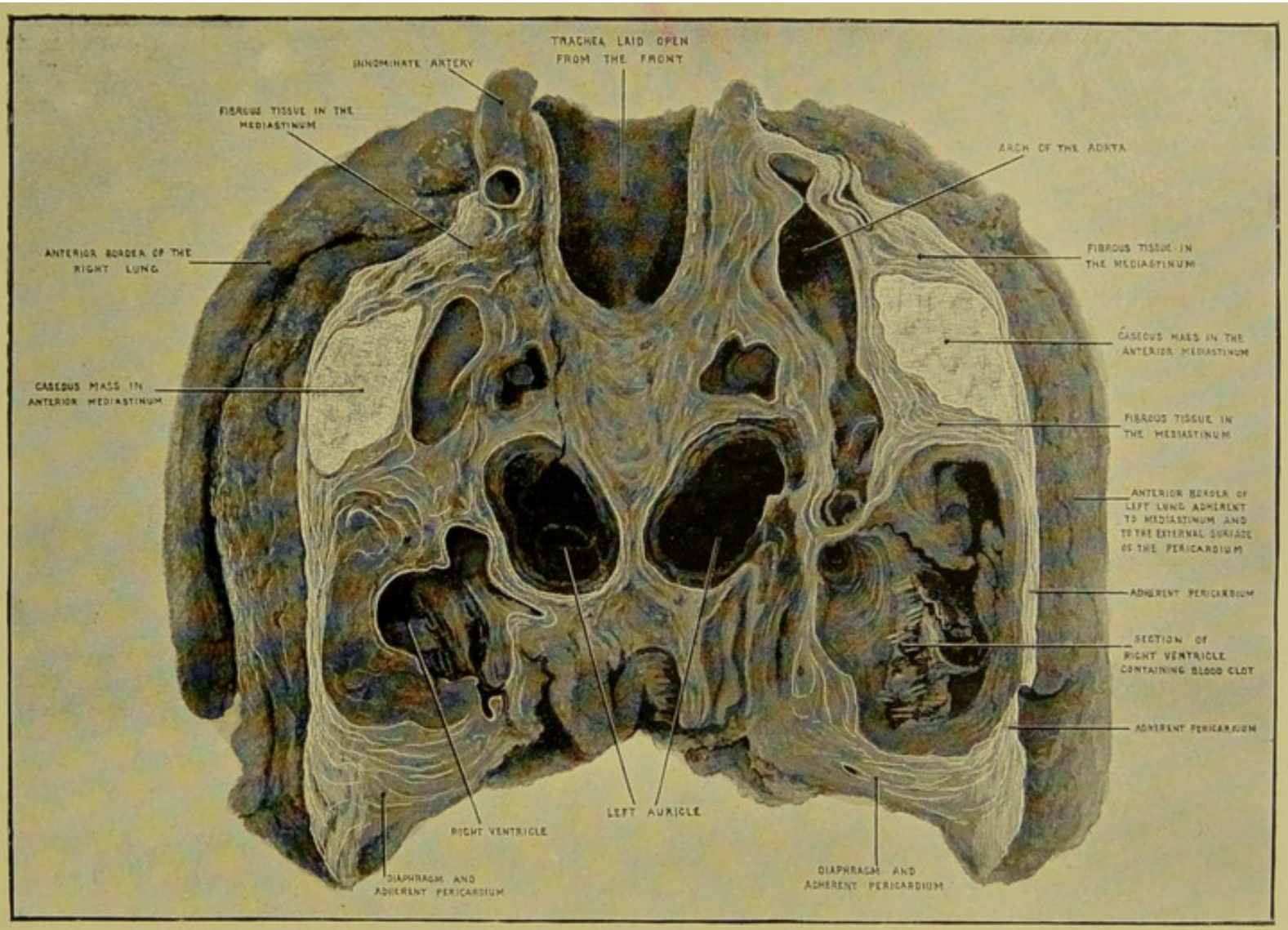

INDURATIVE MEDIASTINO-PERICARDITIS WITH CASEOUS MASS IN THE MEDIASTINUM.—From CASE 1.—J. L.

Explanation of the Plate.—The illustration represents the parts which were exposed by an antero-posterior section, made in the following manner, through the pericardium, heart, and mediastinum:—

After the thoracic organs had been removed *en bloc* from the chest, a single median incision was made from before backwards through the pericardium, heart, arch of the aorta, and the mediastinum until the trachea was laid open. The parts so divided were then brought into view by separating the cut surfaces, like widely opening a book at its middle; the parts at the front being thus widely separated, whilst the posterior wall of the trachea and the tissue in the posterior mediastinum, being uncut, represent, so to speak, the binding of the book. (Each half, right and left, of the illustration would, therefore, have been a *facsimile* of the other, were it not that a small slice was removed from the right half of the specimen for microscopical purposes before the drawing was made.)

INDURATIVE MEDIASTINO-PERICARDITIS.

BY

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INDURATIVE MEDIASTINO-PERICARDITIS.

Cases are occasionally observed which clinically often present great difficulties of diagnosis, and where at the autopsy is found an adherent pericardium with an increase of fibrous tissue in the mediastinum; the increased mediastinal tissue is united to the external surface of the pericardium, and both it and the pericardium are united to the left, and may be also to the right lung. Such cases have been described by various observers, notably by Kussmaul,* to whom the profession is indebted for being the first to bring the subject ably and prominently before its notice, under the term of Indurative Mediastino-Pericarditis. They are not commonly seen, but I think cannot be so rare as is frequently supposed. Well-marked examples of the affection present a very interesting clinical history, and one which is sufficiently distinctive to allow of the recognition of them before they reach that place of accurate diagnosis, the post-mortem department. I do not propose to consider the acute affections of the pericardium, nor the condition of acute mediastinitis. I desire to refer only to the chronic inflammatory states of these parts, and at the outset ought to explain that I employ the term *chronic* in the sense that the cases run a chronic or prolonged course, and at the post-mortem examination fibrous tissue is the chief morbid product found. Such cases may or may not have an acute onset, just as we see a case of acute pericarditis terminate in firm pericardial adhesions, and it is in the sense in which we speak of an adherent pericardium being a chronic case that the word chronic is here employed.

Pathologically, but probably not clinically, we may recognise three classes of cases of chronic inflammatory conditions in relation to the pericardium and the mediastinum.

Class I. comprises cases where there is an adherent pericardium with marked increase of fibrous tissue in the mediastinum, not infrequently associated with a caseous affection of the lymphatic glands of the mediastinum, and where there is adhesion of the exterior of the pericardium to surrounding parts; a condition which is accurately termed *indurative mediastino-pericarditis.*

* Kussmaul (Prof. Dr. A.). *Berliner klinische Wochenschrift*, Jahrgang X., 1873, S. 433.

Class II. comprises cases of an adherent pericardium with thickening of the sac and adhesion of the exterior of it to surrounding parts (sternum, costal cartilages, and lungs), but with very little and sometimes no general mediastinitis, a condition which has been termed *pericarditis externa and interna.* Cases of this nature are probably more common than those belonging to Class I.

Class III. comprises those rare cases where there is an increase of fibrous tissue in the mediastinum without any internal pericardial adhesions. To cases of this class the term *chronic mediastinitis* is appropriate.

These cases, as I have said, represent three classes of chronic mediastinal and pericardial affections, which may be separated by the pathologist, but which the physician will have, in the present state of our clinical knowledge, much greater difficulty in separating during life. We may, probably, during the life of our patient, separate cases belonging to the third class from those belonging to the other two, but I think even the most experienced physician will have great difficulty in separating cases belonging to Class I. from those belonging to Class II., so as to be able, for instance, to predict that the pathologist will find the appearances of pericarditis externa and interna, and not those of indurative mediastino-pericarditis when the case terminates fatally. It appears also, from a consideration of the records published by various observers, that some of the cases described as representatives of indurative mediastino-pericarditis really belong not to Class I. but to Class II., and would be more accurately described as cases of pericarditis externa and interna.

During the past five years I have met with cases belonging to each of the above three classes, and three cases, one belonging to each of the three classes, I have been able to follow to the pathological department. I propose, therefore, to first relate each of the cases in their clinical and pathological aspects, to offer a few comments on each, and finally to review the subject of indurative mediastino-pericarditis from the experience gained by the study of my own cases, and of those which have been recorded by other observers, only referring to cases which have been followed by a post-mortem examination. In a subject so difficult of accurate diagnosis as this, it is not advisable to discuss the records of cases, however probable it is that the diagnosis was correct, where no examination after death has been made.

Case 1.—*Orthopnœa; cyanosis; pulsus paradoxus; engorgement of cervical veins; increased precordial and mediastinal dulness. Pleuritic effusion; enlargement of the liver. Later, general anasarca and ascites and death from the effects of cardiac dilatation.*

J. L., a boy, aged 14 years, first came under my observation in July, 1890. He was admitted to the Manchester Hospital for Consumption and Diseases of the Throat and Chest, on July 12th, 1890. He stated that he had enjoyed good health until the previous Christmas (1889), about which time he began to feel unwell and to suffer from pain in the left side. This continued until the beginning of the following March (1890), when he became more acutely ill, and had to take to his bed for the first time. He remained in bed for five weeks, during which time he was seriously ill. Dr. Yeats, who attended him at this time, tells me that he remembers the case very well, and that the boy had acute pericarditis and pneumonia. After the acute symptoms had subsided he was sent to the seaside for a month, and as

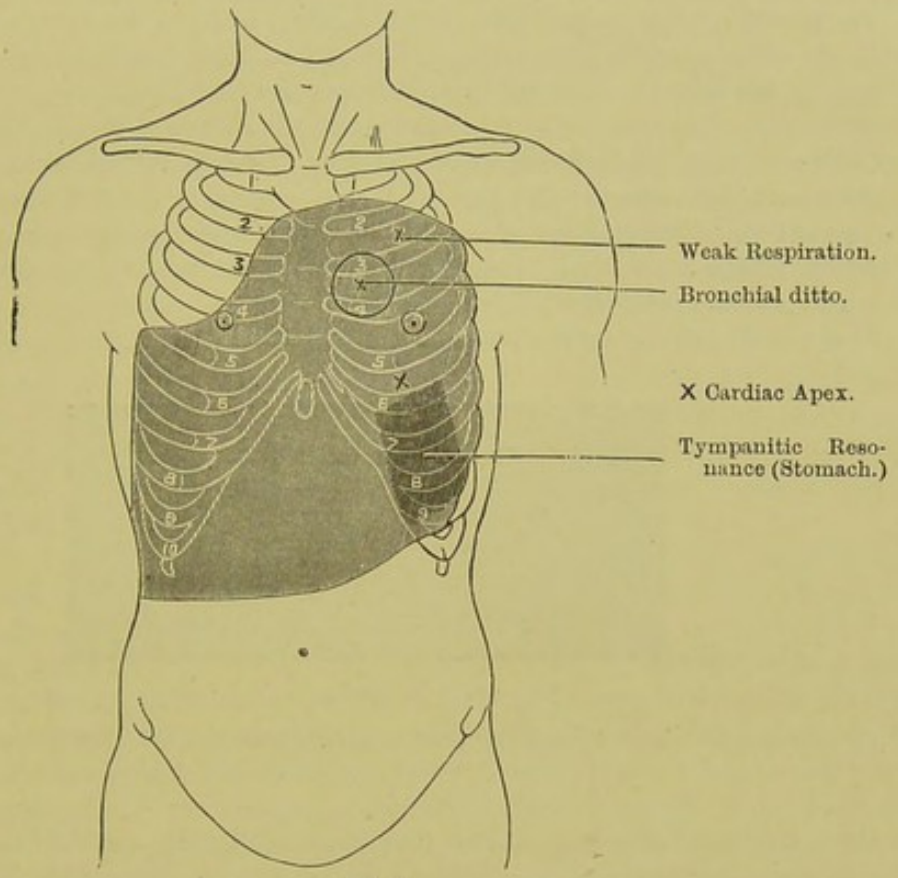


Fig. 1.

Case 1.—J. L., æt. 14. July 13th, 1890.

Diagram showing limits of percussion dulness (cardiac, mediastinal, pleuritic, and hepatic).

he did not seem to get strong, was brought to the Consumption Hospital on July 12th, 1890.

On July 13th, 1890, the following was his condition:—He was a small boy for his age, poorly nourished, and weighed 77½lbs. He complained chiefly of shortness of breath, especially on slight exertion, and pain on the left side of the chest. His lips were extremely livid, and also his ears, but less so than the lips. The veins in his neck were prominent, and a deep inspiration seemed to have very little effect on them, certainly they did not become more prominent during a full inspiration. There was no anasarca and no evidence of ascites; the feet and hands were blue and felt cold, although it is noted that the temperature of the ward at the time was 60° F. There were no enlarged glands anywhere to be felt. Temperature normal.

Respirations, 30 per minute. Slight cough; no expectoration. Chest well shaped; right moved better than the left side. In front there was dulness over the left side of the chest, with the exception of the clavicular supra-clavicular, and the upper part of the infra-clavicular regions. (See diagram, Fig. 1.) The dulness extended across the sternum opposite the second and third costal cartilages nearly to the right mammary line. The upper border of the liver dulness immediately outside the right mammary line was in the fourth intercostal space. There was good resonance over the greater part of the manubrium sterni, the right supra-clavicular and clavicular regions, and over the outer part of right infra-clavicular and upper part of the mammary regions. (See diagram, Fig. 1.) Posteriorly there was good resonance over the upper part of the chest on both sides, but there was dulness at both bases which extended nearly as far as the spine of the scapula on the left side, whilst on the right side it only reached to a little above the inferior angle of the scapula. On auscultation in front there was vesicular respiration over both supra-clavicular regions, whilst adjoining the left border of the sternum over third costal cartilages and the third intercostal space there was perfect bronchial breathing. More to the left over the dull area at the outer part of the left mammary region, the respiratory murmur was weak. In this latter region also were audible either fine crepitations or a fine friction sound, we could not be certain whether the adventitious sounds which were there audible were of pleural or pulmonary origin. No adventitious sounds over the right side or elsewhere over the front of the chest. Posteriorly

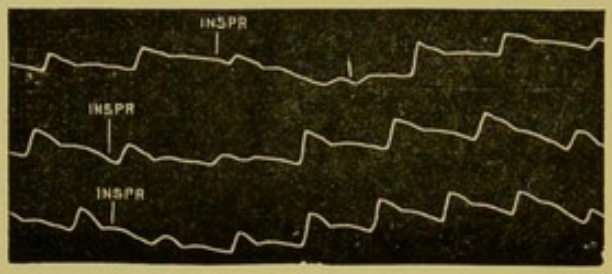


Fig. 2.

Sphygmographic tracing showing the effect of the respiration on the pulse (pulsus paradoxus) in the case of J. L., æt. 14. Case 1. Inspr. = period at which a deep inspiration was made.

there was weak respiration over the dull area on either side, and over that of the left side were a few very small moist râles. An exploring needle was introduced into the left side near the inferior angle of the scapula and a small quantity of clear yellow fluid withdrawn.

Pulse 120, small. A sphygmographic tracing showed that it was markedly affected by deep inspirations. When a deep inspiration was taken the two subsequent pulse waves were very much smaller than the preceding or subsequent ones, and the phenomenon was manifested on each succeeding deep inspiratory act. (See sphygmogram, Fig. 2.) The cardiac apex beat could not be seen or felt, but from auscultation it appeared to be in the fifth intercostal space just internal to the left nipple. The cardiac dulness could not be defined from the dulness over the left side of the chest previously mentioned. On auscultation the heart sounds appeared feeble, but unaccompanied by any murmur.

The liver dulness extended from the fourth intercostal space immediately outside the right mammary line to a point about 1½in. below the costal margin, and in the median line the lower limit was just above the umbilicus. The surface of the liver appeared smooth and the organ was slightly tender on palpation.

There was no indication of disease of any other organs. The spleen could not be felt below the left costal margin. The limits of the organ could not be ascertained by percussion, on account of the existence of the pleuritic effusion at the left base.

The dyspnœa and the blueness of the lips increased, and as the fluid showed no signs of diminution, on August 4th, 1890, 44 ounces of clear yellow serous fluid was removed by aspiration from the left side of the chest. The dulness on the left side in consequence diminished to a small area at the left base behind, and also the bronchial respiration over the third and fourth left costal cartilages vanished, being replaced by weak vesicular breath sound. Fluid re-accumulated, and as it did so and reached its previous limits, the bronchial breathing again appeared in its former position.

On October 5th, 1890, the left side was again aspirated and 40 ounces of clear yellow fluid were withdrawn. Fluid again accumulated, and although it had not become purulent, it was decided to try if making an incision and introducing a drainage tube would prevent any re-accumulation of fluid taking place. Accordingly on November 18th, 1890, a drainage tube was inserted in an intercostal space in the left lower axillary region. After three days the tube was removed and the wound allowed to close. No harm resulted from the operation; on the contrary, the fluid did not again accumulate, and the general condition of the boy, as shown by the lessened dyspnœa and the diminished blueness of the lips, improved.

The temperature during the first three months of the boy's stay in hospital was, with a few exceptions, sub-normal both morning and evening. A few days before the operation of draining the chest it became slightly elevated. On four occasions about this period it reached 101° F., but usually, at this period, it varied between 98° and 100° F.

He left the hospital on December 28th, 1890, his general condition being improved, but he was still troubled with slight dyspnœa on exertion and had slight blueness of the lips.

Four months later, on April 27, 1891, he was admitted to the Manchester Royal Infirmary on account of shortness of breath. He stated that since he had left the Consumption Hospital he had enjoyed fairly good health, except for being rather short of breath, which difficulty had lately increased.

On admission to the Infirmary he had much dyspnœa amounting to orthopnœa. The lips were very livid; there was no anasarca nor ascites. The temperature was normal, and the pulse 108, small, and of the "pulsus paradoxus" type. The external jugular veins were full, and there was marked venous pulsation at the root of the neck. There was, however, no inspiratory swelling of the veins in the neck. No cardiac apex beat could be felt, but from auscultation it appeared to be in the fourth intercostal space internal to the nipple. At that point the second sound resembled the first, both being feeble and of a short ticking character. There were no murmurs.

Respirations were 42. There was marked orthopnœa. Troublesome cough. Expectoration of about 2oz. of muco-purulent matter in the course of 24 hours. Breathing was mainly abdominal, the chest generally moving but slightly though better on the right than on the left side. When the boy took a full inspiration, the lower ribs were drawn slightly inwards. The area of dulness over the front of the chest at this date is shown in the accompanying diagram (Fig. 3). Auscultation over the front of the chest revealed the following:—Left supra-clavicular region, weak vesicular respiration accompanied by sonorous rhonchi. Right supra-clavicular region, ditto, Left infra-clavicular region: Outside the area of

dulness vesicular respiration obscured by numerous fine bubbles; in the same region over the area of dulness, between the left sterno-clavicular joint and the second rib expiration was of bronchial type, and accompanied by sonorous rhonchi; below the second costal cartilage the bronchial character was lost, the breathing being vesicular and accompanied by a few fine bubbles. Right infra-clavicular region: Exaggerated respiration accompanied by sonorous rhonchi. Posteriorly (see diagram, Fig. 4): On the right side there was absolute dulness at the base to a point one finger's breadth above the inferior angle of the scapula; above that there was fairly good resonance, but, perhaps, somewhat impaired. On the left side there was absolute dulness to the inferior angle of the scapula, but above that point up to the

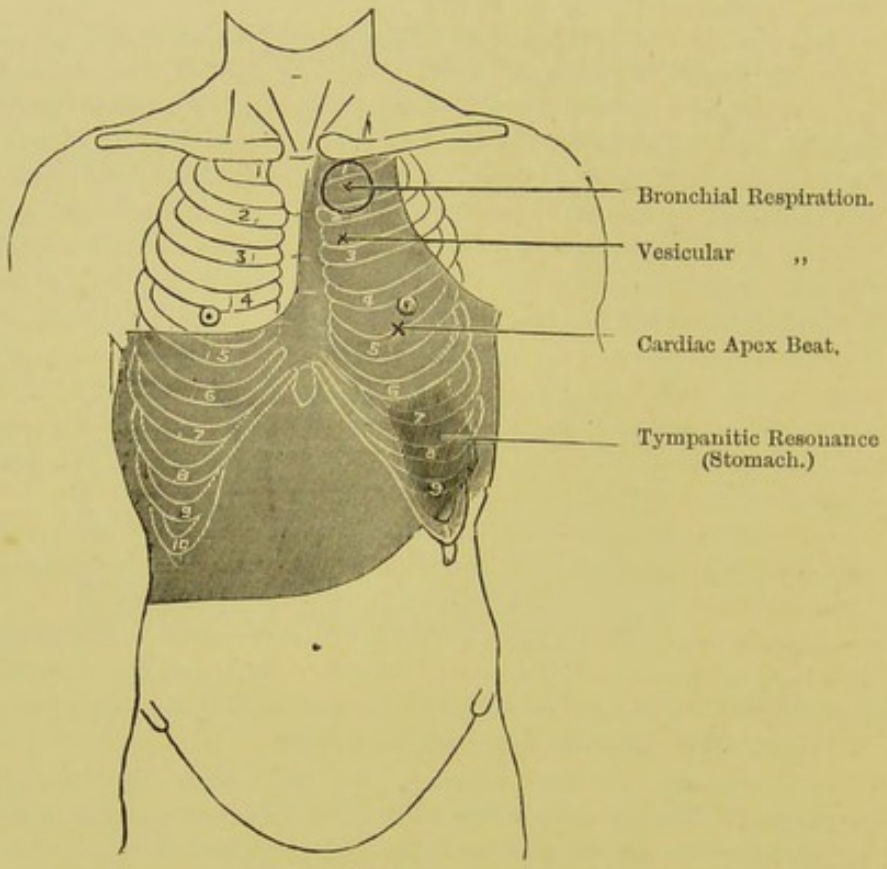


FIG. 3.

CASE 1.—J. L., æt. 14.

Diagram showing area of dulness (cardiac, mediastinal, pleuritic, and hepatic) on April 28th, 1891.

apex of the lung the left side showed only impaired resonance. Auscultation behind revealed as follows:—Right supra-scapular region: Bronchial respiration accompanied by sonorous rhonchi. Left supra-scapular region: Bronchial breathing, with a few fine bubbles. Right supra-spinous region: Vesicular respiration and sonorous rhonchi. Left supra-spinous region: Weak, distant bronchial respiration, and sonorous rhonchi. Right inter-scapular region: Opposite the spine of the scapula bronchial respiration and sonorous rhonchi, below that point in the inter-scapular region respiration was vesicular. Left inter-scapular region: Opposite the spine of the scapula bronchial respiration with fine bubbles. Right sub-scapular region: Vesicular respiration in many parts; at others, however, breathing was

bronchial in type and accompanied by fine bubbles. Left sub-scapular region: Bronchial respiration accompanied by fine bubbles. Vocal fremitus at the left base greater than at the right base. Also over the left mammary region it was greater than over the corresponding region of the right side.

Liver: Dulness commenced above in the right mammary line at the upper border of the fifth rib and extended below the costal margin, measuring 6in. in this direction. In the median line the lower limit of the liver dulness was 1½in. above the umbilicus. (See diagram, Fig. 3.) Slight tenderness to pressure over the liver.

Urine scanty (16oz. passed during the previous 24 hours), acid, sp. g. 1015, contained a small quantity of albumen, no sugar.

He stayed in the Infirmary two months, and during that time his cough

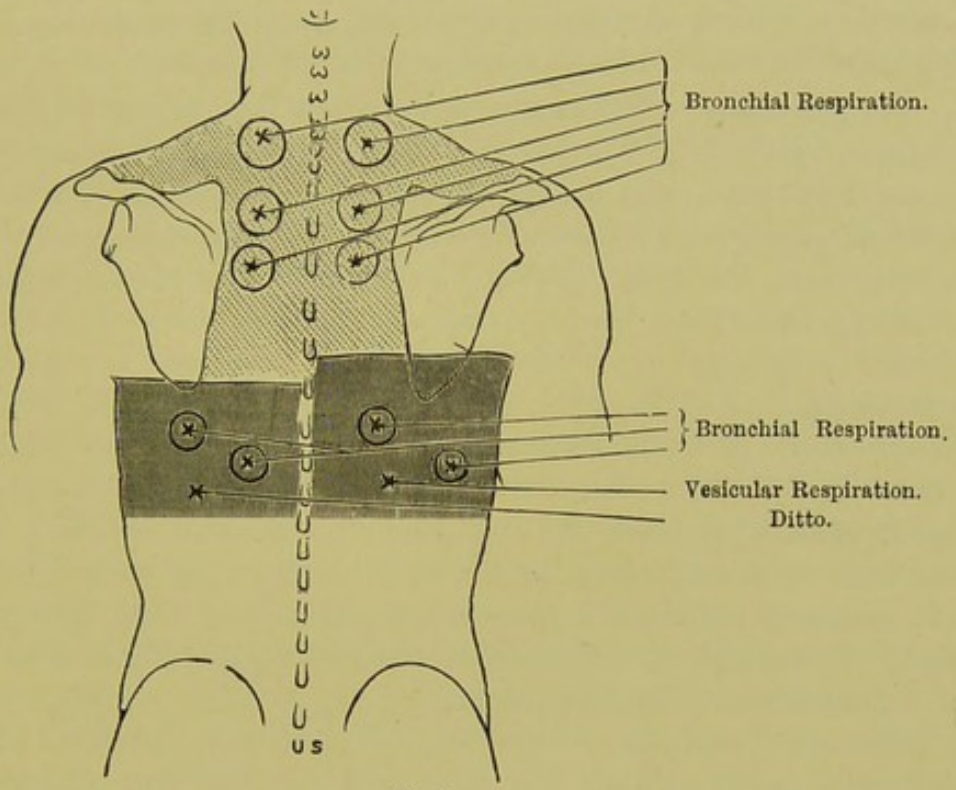


FIG. 4.

CASE 1.—J. L., æt. 14.

Diagram showing area of dulness at the base of the chest behind and the character of the breath-sounds at various parts on April 28th, 1891.

became less troublesome and his dyspnœa much diminished. The chief change in the physical signs over the chest was in the diminution of the adventitious sounds. During his stay in the Infirmary his temperature never rose above 98° F. As it was felt that there probably was a rise of temperature above the normal at some period of the 24 hours, a 4-hours' chart was kept during the first fortnight he was in the hospital, but no rise beyond the normal limit was ever found. On May 26, 1891, an exploring needle was introduced into the right pleural cavity, and a small quantity of clear serous fluid was withdrawn. On June 1st, 1891, he was sent to the Convalescent Hospital.

On his admission to the Convalescent Hospital on June 1st, 1891, he had considerable dyspnœa on exertion, and his cough was somewhat troublesome. There was marked blueness of the lips and ears; no anasarca or ascites. There was much

enlargement of the liver, and some effusion in the right pleural cavity. The urine contained a small quantity of albumen. About a week after admission he became worse, he was troubled with sickness and took very ittle food. The cyanosis and dyspnœa became more marked, and the fluid in the right side of the chest increased in quantity. On July 14, 28oz. of pale, clear fluid were removed by means of an aspirator from the right pleural cavity. This appeared to relieve the dyspnœa for a time, and the cyanosis became less marked. On August 3rd, however, it was necessary to again puncture the right pleural cavity, and on this occasion 42½ oz. of clear fluid were removed. This was not followed by so much relief as the previous aspiration, and the boy gradually sank and died on August 12, 1891. During the last fortnight of life a considerable amount of anasarca of the lower extremities and ascites appeared. The amount of albumen in the urine increased. On August 10th, two days before death, it was noted that the urine was acid, sp. gr. 1018, contained about one-eighth albumen, was free from sugar, and no casts could be found.

The post-mortem examination was conducted, on August 12th, in my presence, by Dr. J. E. Platt, at that time the Resident Medical Officer to the Institution, who has kindly furnished the following report :— *Adherent pericardium; indurative mediastinitis with caseous affection in mediastinum. Thickened left pleura; effusion in right pleura; dilatation of heart; venous engorgement of liver and kidneys; anasarca and ascites.* Marked cutaneous hypostasis; considerable anasarca of the lower extremities, none of the upper. Left side of the chest slightly retracted; an old cicatrix of small size in the left lower axillary region. [This was the result of the operation of draining the left pleural cavity, performed twelve months previously at the Consumption Hospital.—T. H.] Rigor mortis was well marked in the lower extremities; less so in the upper. The abdomen contained a considerable quantity of clear, pale yellow fluid. There were no adhesions in the peritoneal cavity, and no signs of inflammation of the peritoneum.

Pleuræ: There was a large amount of clear pale yellow fluid in the *right* pleural cavity. The right lung was free from adhesions to the chest wall, except a few old adhesions over the apex. There were no adhesions of the right lung to the diaphragm, but old, tough, firm adhesions united its inner aspect to the mediastinum and to the external surface of the pericardium. The anterior edge of the right lung was about three-quarters of an inch to the right of the median line. The left pleural cavity was obliterated by firm, old, tough adhesions, the left lung being united to the chestwall, diaphragm, and mediastinum. The parietal layer of the left pleura over the lower half of the chest was very much thickened; in places it was half an inch in thickness.

Pericardium: The cavity was obliterated by firm, old adhesions, which closely united the visceral with the parietal layer.

Mediastinum: In the anterior and middle mediastinum, immediately above the heart, and extending from there to the upper part

of the sternum, was a yellowish-white, fairly firm, caseous-looking mass, which measured one and a half inches in length and two inches transversely. It was surrounded by firm fibrous tissue, bands of which, with the caseous mass, occupied the whole anterior and middle parts of the mediastinum, and extended backwards to the trachea and œsophagus. There were no signs of any recent activity around the periphery of the caseous-looking mass, or in the surrounding fibrous tissue. Both the caseous-looking mass and the fibrous tissue were evidently of very old standing. The caseous mass and fibrous tissue of the mediastinum were very firmly adherent to the sternum in front, to the lungs on either side, and to the outer aspect of the pericardium. No other caseous mass existed in the mediastinum except the one above described.

Lungs: Both congested, but otherwise presented nothing abnormal. No signs of any disease in either apex.

Liver: Large, but not enlarged to an extreme degree. Surface smooth. On section it presented a marked nutmeg appearance.

Spleen: Normal.

Kidneys: Congested, but otherwise healthy.

Heart: An antero-posterior section was made through the heart and the mediastinum, so as to demonstrate the lesions in the latter part. As the preparation was required for museum purposes, no further dissection of the heart was made. (See plate.) As far as could be ascertained, however, there was no valvular disease, and the muscular substance of the organ was of a healthy colour, though somewhat soft.

Microscopically the caseous mass in the mediastinum presented a homogeneous, granular appearance, and would not stain with logwood or carmine. A few isolated nuclei here and there among the granular material were the only structures which stained at all. A number of sections taken from the periphery of the mass were stained and examined for tubercle bacilli, but with negative results. Microscopical examination of sections made from the liver showed very marked venous congestion, but no increase of fibrous tissue at any part.

Remarks on the case of J. L.—The total duration of this boy's illness appears to have been at the most about one year and nine months. For the first three months his symptoms were very indefinite, as he only complained of feeling unwell and of pain in the left side. It was not until these indefinite symptoms had existed for about three months that he became seriously ill, the illness being apparently acute pericarditis and pneumonia. Between four and five months after the onset of this acute illness he first came under my observation. At that time his chief complaint was shortness of breath. He then had marked evidence of venous engorgement, the lips and ears being very blue, and

the veins of the neck engorged. There was evidence of pleuritic effusion on the left side, and there was dulness over the sternum and to the right of the lower part of that bone, which could not be accounted for simply by assuming a displacement of the heart by the pleuritic effusion. This dulness, as the post-mortem examination proved, was due to the increase of the tissue in the mediastinum, to the caseous mass which was present there, and to the enlargement of the right side of the heart, secondary to the adherent pericardium. The liver at this time was enlarged in consequence of the venous engorgement, but there was no ascites nor anasarca. Although the veins of the neck were much engorged they did not become more swollen during inspiration, as has been seen in some cases of mediastino-pericarditis ; but another feature of that affection was present—viz., the pulsus paradoxus. Pleuritic effusion of the left side accumulated again and again after tapping, until a drainage tube was inserted into that side, with the result of causing a union of the parietal with the visceral layer of the pleura, and consequent obliteration of the left pleural cavity. Relief followed this operation for a time, but subsequently pleuritic effusion developed on the right side, the heart became more dilated and feeble, anasarca and ascites ensued in consequence of cardiac dilatation, and the boy succumbed, chiefly from the effect of the condition of the heart.

Ascites, which sometimes forms a prominent feature of the cases of mediastino-pericarditis, was absent in this patient until a late stage of the illness, and was then clearly a part of the general dropsy, which resulted from the venous engorgement consequent upon the cardiac dilatation. Inspiratory swelling of the veins of the neck was also not present. The pulsus paradoxus, which is frequently present in such cases, but as we know occurs in other affections also, was marked in the above case.

Another physical sign which was present is well worth a few considerations : This sign is the bronchial breathing which was observed in front to the left of the sternum, and the same type of breath-sound which at a later period of the boy's illness (April, 1891), was so marked in the upper part of the inter-scapular region of both sides. The bronchial respiration present, when he first came under my observation, to the left of the sternum, was clearly associated with the accumulation of fluid in the left pleural cavity, and the displacement of the left lung brought about thereby, because, when the fluid was removed by aspiration, weak vesicular respiration replaced the bronchial breathing, and as the fluid re-accumulated and reached its previous limits, the breath sounds again became bronchial at the same spot.

The bronchial respiration which was present at a later period of his illness (April, 1891), and heard over the inner extremity of the first intercostal space, at the left border of the sternum, was most probably conducted from the trachea, as it may be found in this position occasionally in healthy individuals. A similar explanation applies to the bronchial respiration which was audible at the same period over the upper part of the interscapular region of both sides. Having regard to the appearances presented at the post-mortem examination, I do not think that the bronchial breathing, heard at the posterior and upper part of the chest, could have been connected with the mediastinal affection, but that it was probably simply a pronounced example of a type of breathing which is not uncommon in this region, especially in children, who present neither an affection of the lungs nor of the mediastinum. [See also the report and comments on Case 2.]

The following case is an illustration of Class II., which comprises a group of cases, where, on post-mortem examination, the pericardial cavity is found to be obliterated by adhesions, and the exterior of the pericardium is adherent to surrounding parts, whilst there is little or no increase of fibrous tissue throughout the mediastinum generally; a condition which has been termed "Pericarditis interna and externa."

CASE 2.—*Dyspnœa; cyanosis; tubercular disease of the elbow joint; cardiac dilatation; engorgement of the veins of the neck; enlarged liver and ascites; very slight anasarca of the lower extremities. Repeated paracentesis of the abdomen. Bronchitis and catarrhal pneumonia, and death from gradual cardiac failure.*

[From notes by Dr. R. W. Marsden.]

J. M., aged 8 years, was admitted to the Manchester Royal Infirmary, on April 7th, 1893, complaining chiefly of great distension of the abdomen. His mother gave the following account of his illness: The boy had been quite well up to 5½ years of age, *i.e.*, 2½ years before he was admitted into the Infirmary. The only illness which he had had before that time was scarlet fever. At 5½ years of age he was noticed to be looking ill, to be getting thinner, and to have a slight cough. At that time, however, he had no swelling of the abdomen or elsewhere. Three months later the abdomen commenced to enlarge, and after another period of three months had elapsed he was admitted to the Manchester Clinical Hospital. During the past two years the mother stated that the abdomen had been tapped 23 times, but Dr. Railton, under whose care he was, has kindly looked up the records of the case for me, and from these we only have evidence that during the past two years the boy had been tapped 13 times. About 12 months previous to his admission into the Manchester Infirmary, signs of disease in the right elbow joint appeared, suppuration had subsequently ensued, and the joint had been drained.

On admission the most prominent feature of the case was the enormous distension of the abdomen, which measured 33in. in circumference at the umbilicus,

and 32½in. midway between that point and the xiphoid cartilage (see Fig. 5.) The boy was fairly well nourished, had somewhat rosy cheeks, somewhat prominent eyeballs, and there was no blueness of the lips or of the extremities. There was a mere trace of œdema in the neighbourhood of the ankles. The right elbow joint presented a fusiform swelling, and two drainage openings led into the joint, and through them exuded a little thin purulent discharge. One or two slightly enlarged glands could be felt in each axilla, more marked in the right than in the left. There was no enlargement of the glands in the neck or in the groin. The temperature on the morning of admission was subnormal, in the evening of the same day it was 99°. During the following week it was very irregular, on some days it was normal both in the morning and evening, but usually, whilst it was subnormal in the morning it rose in the evening to about 100°F. The pulse was 108, regular, and showed no variations in size during inspiration. The external jugular veins were very much distended and pulsated markedly. They showed, however, no increased distension

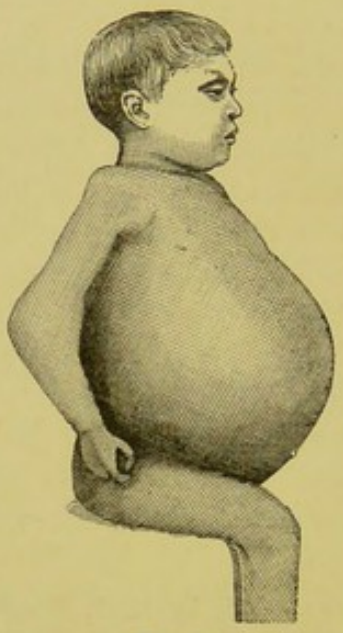


FIG. 5.

CASE 2.—J. M., æt. 8.

From a photograph showing the distended condition of the abdomen, and the fusiform swelling (tubercular) of the right elbow.

during inspiration. There was no appreciable change in the size of the veins during inspiration or expiration.

The respirations were 30. There was a somewhat troublesome cough, but no expectoration could be obtained during his stay in the hospital, although the boy was repeatedly urged to spit out and not to swallow any phlegm which might come up. There was slight orthopnœa, but he did not complain of shortness of breath. The chest was well shaped—no flattening and no bulging of any part. There was no absolute dulness over the front of the chest apart from the precordial dulness. The resonance over the left infra-clavicular region was less marked, however, than over the right, but that region was not absolutely dull. Posteriorly: There was no dulness, except in the right supra-scapular and upper part of the right inter-scapular regions, whilst the adjoining part

of the right supra-spinous region showed good resonance. The impaired resonance in the regions indicated was very marked, and was repeatedly confirmed by subsequent examinations. Over the right and left infra-clavicular regions there was vesicular respiration accompanied by sonorous rhonchi and a few simple bubbles. Posteriorly: The respiration was vesicular and accompanied by sonorous rhonchi and a few bubbles, except over the dull area at the right apex where the breathing was distinctly bronchial in character. Over the outer part of the right supra-spinous region it was, however, of the vesicular type, and not at all bronchial. The rhonchi and

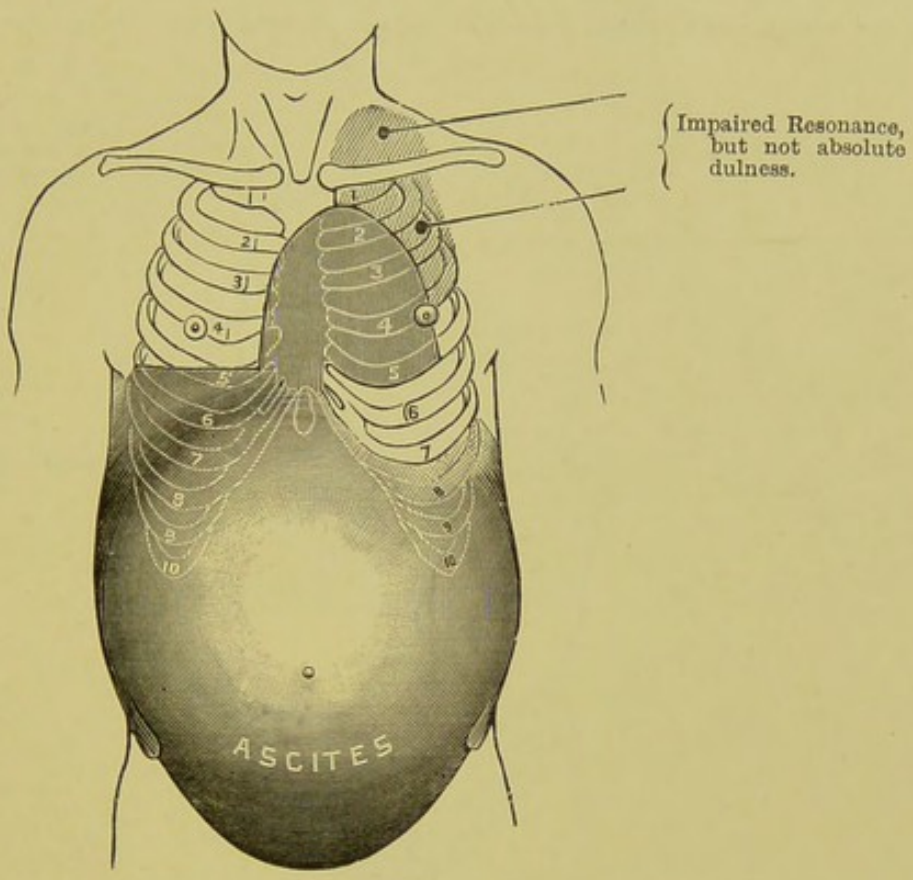


Fig. 6.

Case 2.—J. M., æt. 8.

Diagram representing the results of percussion of the chest and abdomen on April 8th, 1893. The pyramidal shaded area represents the deep cardiac dulness. The difference in the depth of the shading over the abdomen does not represent any difference in the percussion note. It has been so shaded to give the rounded and distended appearance which the abdomen presented at this date. There was dulness all over the abdomen, the cavity being apparently full of fluid.

bubbles were much more marked at the lower than the upper part of the chest (see diagram, Fig. 7). The cardiac apex beat could be seen and felt in the fourth intercostal space. The deep cardiac dulness is represented in the accompanying diagram (Fig. 6). As will be seen, it was very much increased, and reminded one of the dulness of pericardial effusion or of some cases of enormous distension of the right side of the heart. On auscultation, the heart sounds were clear and distinct. The second sound was more accentuated at the third left than at the second right costal cartilage. There were no murmurs.

As regards the digestive system, the appetite was good and there was no sickness. The bowels were regular. The distension of the abdomen caused slight discomfort, but no acute pain. The abdomen measured 33in. in circumference at the umbilicus. It was quite free from tenderness, and a very marked thrill could be obtained by placing the hand on one side and tapping the opposite side. The distension was a distinctly uniform one. The veins of the abdominal walls were only slightly more marked than usual.

The liver dulness commenced above at the level of the fifth rib in the right mammary line, but the lower limit of the organ could not be ascertained on account of the ascites. The splenic dulness could not be ascertained for the same reason.

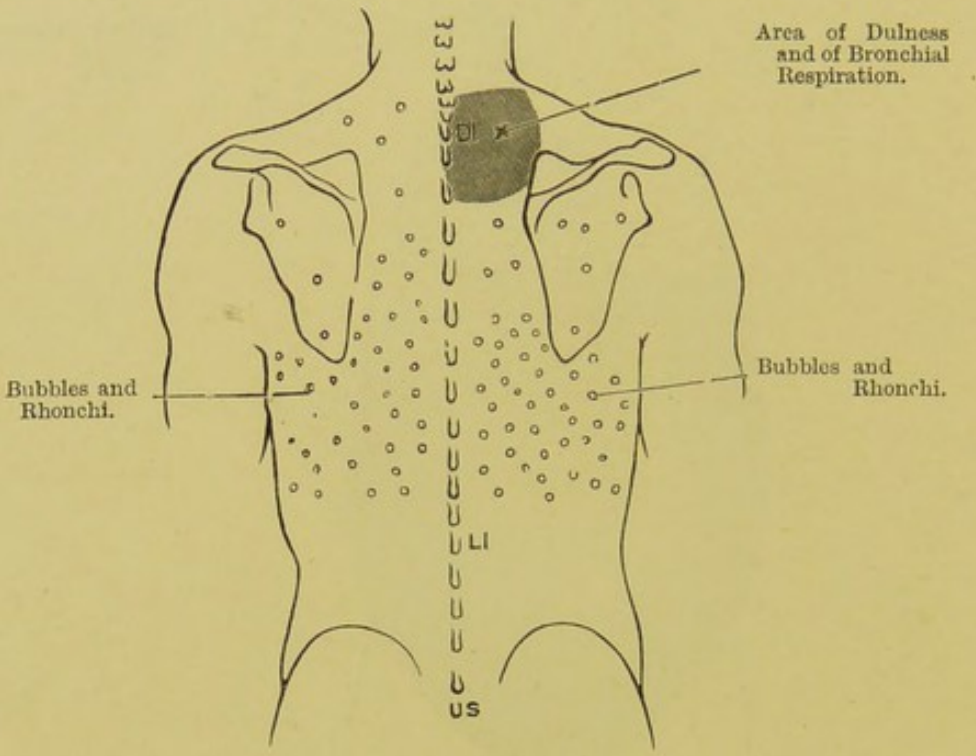


FIG. 7.

CASE 2.—J. M., æt. 8.

Diagram representing the area of dulness over the right supra-scapular and the upper part of the right interscapular regions. Also representing the bubbles and rhonchi which were audible over the posterior part of the chest on April 8th, 1893.

The urine was acid, of specific gravity 1022, and contained neither albumen nor sugar. The nervous system presented nothing unusual.

Five days after admission the abdomen was punctured, and 167 ounces of clear slightly yellow fluid were withdrawn. After removal of the ascitic fluid, the lower margin of the liver could be felt 1¾in. below the costal margin in the right mammary line, and the surface of the organ felt smooth and firm. The upper limit of the liver dulness after the tapping was in the fifth intercostal space. The upper limit of the precordial dulness also fell to the lower border of the second left costal cartilage, and the transverse measurement of the cardiac dulness was considerably less than before the tapping. There still remained a small area of dulness over the lower part of the abdomen, due to a

small quantity of ascitic fluid (see diagram, Fig. 8). The cough became much better, and the adventitious sounds disappeared from the chest. The impaired resonance and bronchial breathing at the right apex remained, however, as before. The abdomen filled up again, and on May 2nd 140oz. of clear fluid were removed by the trocar. The boy was able to leave the hospital on the 4th of the same month.

He was re-admitted again on June 7th, 1893, an account of the re-accumulation of the ascites and the discomfort caused thereby. On this occasion he stayed in the Infirmary only nineteen days, during which time the abdomen was tapped twice, on the first occasion 197 ounces, and on the second 120 ounces of fluid being withdrawn.

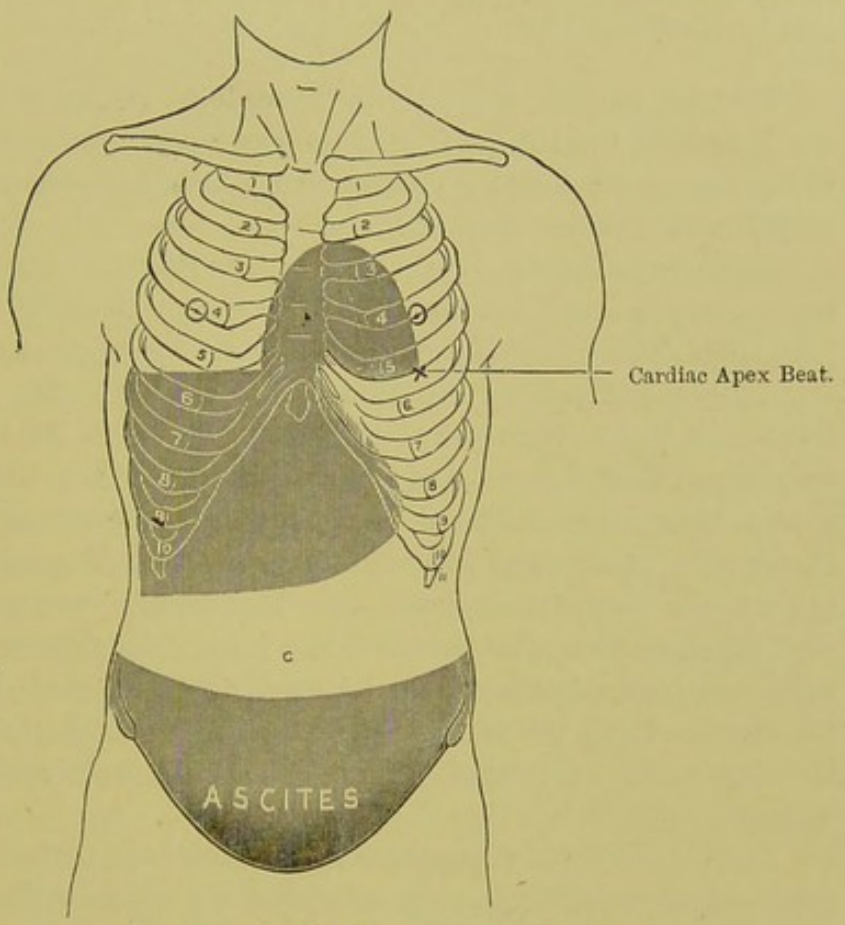


FIG. 8.

CASE 2. –J. M., æt. 8.

Diagram representing the precordial, hepatic, and ascitic dulness the day after 197 ounces of fluid had been withdrawn from the abdomen.

After this period the boy was repeatedly re-admitted to the Infirmary in order to have the abdomen tapped. Between April 7th, 1893, the date of his first admission, and February 17th, 1894, we tapped the abdomen seventeen times ; therefore, including the occasions on which it had been tapped in the Clinical Hospital, it had been tapped no less than on thirty occasions during the preceding three years.

It is not necessary to enter fully into the details of the boy's condition on each of the occasions when he was under observation in the Infirmary. Usually he was able to leave the hospital a few days after the tapping, but in November, 1893, he developed marked signs of bronchitis and catarrhal pneumonia, and from that time his appearance and general condition changed much for the worse. From being rosy, and of fairly

healthy appearance, he became more and more livid. He recovered, however, from the pulmonary attack, and was able to return home for a short time, but he had to come again and again for the ascitic fluid to be removed. He was admitted for the last time on February 5th, 1894, when he was in a more serious condition than on any previous occasion. The abdomen was, as usual, distended, and the boy was very livid. He had a troublesome cough, and there were numerous râles and rhonchi audible over the chest. The precordial dulness was similar to that above recorded when he first came under observation. The elbow-joint and the enlarged axillary glands showed no appreciable change. The jugular veins were very much distended ; the cheeks, lips, and ears became more and more cyanotic. The extremities became cold, the boy passed into a drowsy semi-conscious condition, and died on February 22nd, 1894.

The post-mortem examination was made on February 23rd, 1894, by Dr. Kelynack, the Pathological Registrar to the Infirmary. Permission was not obtained for the examination, which consequently was not so complete as it otherwise would have been.

Adherent pericardium ; adhesion of the exterior of the pericardium to surrounding parts ; dilatation of the heart ; nutmeg liver ; chronic peritonitis and ascites ; slight anasarca of the lower extremities.

External.—The body was that of a spare boy. The abdomen was prominent, and evidently had been distended with fluid. Very slight anasarca of both feet, none of any other part of the body. Lips and cheeks blue. Rigor mortis well marked in both upper and lower extremities. Cutaneous hypostasis dorsal. Eyeballs prominent, but the eyelids just met over the eyeballs. Right elbow presented a fusiform swelling, with sinuses on the outer and inner sides of the olecranon, and a little thin pus oozing therefrom. A small cicatrix existed over the front of the elbow. The joint was ankylosed. An enlarged gland could be felt in the right axilla. On opening the abdomen 35oz. of turbid yellow fluid was removed.

Internal.—Thorax : On removing the operculum the lungs and the pericardium were seen to be closely adherent to the posterior surface of the operculum by old inflammatory matting. The pericardial and mediastinal area at the level of the second costal cartilage measured 2in. across ; at the level of the third costal cartilage it measured $2\frac{1}{2}$in. across ; at the fourth, $3\frac{1}{3}$in. ; at the fifth, $3\frac{1}{2}$in. ; at the lower border of the sixth, 4in.

Pleuræ : Firm fibrous adhesions on both sides, almost completely obliterating the pleural cavities ; a small space, however, existed at the upper portion of the right cavity, which contained a small quantity of straw-coloured serous effusion, and another small space also remained at the lower part of the same cavity. The pleuritic adhesions were evidently of old standing. They could not be separated by the fingers, but had to be detached with the aid of the knife. The pleuræ were also closely adherent to the pericardium and to the diaphragm.

Pericardium: Immensely enlarged, as indicated by the above measurements; but its cavity obliterated by old adhesions. The external or fibrous coat was closely adherent anteriorly to the back of the sternum and to the anterior portion of the costal cartilages. Laterally it was closely adherent to the pericardial layers of the pleura.

The Heart, Lungs, and the structures in the Mediastinum, together with certain of the abdomina viscera, were removed *en masse*, and a single vertical incision was made, through the heart and mediastinum from front to back so as to ascertain the condition of the mediastinal tissue and the structures therein contained. Mediastinum: There was no enlargement of the lymphatic glands of the mediastinum, with the exception of one gland situated immediately below the bifurcation of the trachea between the two bronchi. This gland was slightly enlarged, measuring ¾in. in length by ½in. in thickness, and was the seat of the caseo-calcareous change. There was possibly a slight increase of the fibrous tissue in the mediastinum surrounding the arteries, veins, and adjacent structures; but this was not marked, and the chief evidence of mediastinitis was afforded by the firm adhesion of the external surface of the pericardium to the pleura and to the sternum and costal cartilages. As previously stated, the anterior part of the mediastinum was adherent to the posterior aspect of the sternum.

Heart: The interior of certain of the cavities of the heart was exposed by the above described median incision through the mediastinum. The cavities contained dark-red recent blood clot. The cavities were dilated, but the valves, as far as could be ascertained from the incision made, appeared normal. As the specimen was required for museum purposes, the heart was not further dissected.

Lungs: At the apex of the right organ there was slight puckering and a small area of fibroid induration, apparently indicative of old apicial pulmonary tubercle. Both lungs congested and much compressed by the extensive chronic pleurisy. The fibrous tissue forming the thickened pleura not only completely surrounded them both, but in places extended slightly inwards into the pulmonary tissue. Although increased in consistency, there was no complete consolidation anywhere, and no indication of any recent tubercular focus. The only sign of tubercle in the lungs being the old obsolescent affection of the apex of the right organ. Vertical measurement from base to apex of the right organ 4½in., of the left 4¼in.

Abdomen: Peritoneal cavity contained 35oz. of turbid yellow fluid. The parietal layer was generally thickened; it was smooth and had a white porcelain-like appearance. The visceral layer was also densely thickened, and over the liver and spleen the opaque white porcelain-like appearance was very pronounced. The peritoneum forming the serous

coat of the intestines was somewhat thickened, and presented numerous closely packed rounded white spots. These at first sight had much the appearance of tubercles, but were not so raised, and were evidently localised spots of thickening of the serous coat of a fibroid character. There were no adhesions of the coils of the intestine to each other, but there was much peritoneal matting in the neighbourhood of the spleen, which was firmly fixed by such adhesions to the under surface of the diaphragm. A few similar adhesions united the upper surface of the liver to the diaphragm.

Liver: Not markedly differing in size from what one would expect in a child, but the edges were very rounded, this apparently being caused by the contraction of the previously described thickened capsule. The capsule was everywhere much thickened, and of the opaque white porcelain-like aspect previously mentioned. The organ was adherent to the under surface of the diaphragm. On section the organ was extremely congested, presenting a typical myristicated appearance, but apparently little or no cirrhosis. No evidence of tubercle or of old caseous foci. The inlet of the hepatic veins into the hepatic portion of the inferior vena cava did not appear to be mechanically obstructed by direct fibrous compression, and there was no narrowing of the inferior vena cava at its entrance into the right auricle. The measurements of the liver were as follows: Vertical, 4in. anteriorly, and $4\frac{1}{8}$in. posteriorly, transverse, $8\frac{1}{2}$in. The weight of the organ cannot be given, as it was desired to leave it attached to the thoracic organs and diaphragm for museum purposes.

Spleen: Congested; not, however, markedly enlarged; capsule thickened, of dense opaque white porcelain-like aspect, but this not to so marked a degree as is the case with the capsule of the liver.

Kidneys: Both markedly congested, but not otherwise presenting any abnormal appearances.

Retroperitoneal lymphatic glands: Not enlarged. No signs of caseation or calcareous change in any of the glands. Brain not examined for reasons previously stated.

Microscopical examination of the liver showed immense venous engorgement; the capillaries of the central portion of the lobules were enormously distended, and a great part of the lobule presented simply a network of engorged capillaries with no liver cells. A little more remote from the centre of the lobule the vessels were still engorged, and the liver cells between them atrophied and pigmented. At the periphery of the lobules the liver cells were fairly healthy, except that some of them contained a large amount of fat. The periportal tissue showed very little change; it was nowhere markedly increased, but was possibly slightly in excess of the

usual amount. The thickened fibrous capsule did not send prolongations of fibroid tissue into the substance of the organ, the thickening being limited to the peritoneal covering of the liver.

Remarks on Case 2.—The boy was under my observation between ten and eleven months. On no occasion had it been observed that there was any swelling of the veins of the neck during inspiration, nor a pulsus paradoxus, although attention had been repeatedly directed to both these signs. The temperature had been variable. Often for days together, and sometimes for weeks together the temperature charts show that there was no rise of temperature above normal, but frequently there was a slight evening rise, whilst the morning temperature was normal or sub-normal; the evening temperature on such occasions rarely reaching 101°F., and more commonly it was below 100°F.

The most marked feature of the case was always the ascites, associated with only very slight œdema of the lower extremities. The abdominal dropsy needed frequent and repeated tapping, but the relief afforded on each occasion was of short duration, fluid rapidly accumulating, and it was chiefly on account of the ascites that he sought hospital treatment. During the period he was under observation he also presented marked evidence of cardiac dilatation, as shown by increased area of precordial dulness. There never were any cardiac murmurs. The veins of the neck were always immensely distended, and although at first there was no cyanosis of the face, this became marked at a later period of his illness. Generally, he had a slight cough which frequently became troublesome for one or two weeks, during which time he presented signs of bronchitis and of catarrhal pneumonia. He also presented well-marked signs of tubercular disease of the right elbow and slight enlargement of the axillary glands, but otherwise there were no signs of a tubercular affection of any part of the body.

The onset of the illness had been a gradual one, the distension of the abdomen having been the most prominent symptom even before he came under our observation. No history could be obtained of the child having been acutely ill, of having presented any signs of acute pericarditis or other acute malady. The total duration of the illness was about three and a half years.

The post-mortem examination revealed an adherent pericardium with a dilated heart, general pleuritic adhesions, a certain amount of indurative mediastinitis, chronic peritonitis, and venous engorgement of the liver and kidneys.

The enlargement of the heart was evidently the result of the adherent pericardium, and the venous engorgement of the veins of the neck, which had been such a prominent sign during life, was probably the result

chiefly of the dilated heart, but possibly the mediastinitis might have assisted and rendered the distension more marked. The engorgement of the liver and kidneys was also the result of the cardiac dilatation.

The ascites, which had been one of the most prominent features of the case during life, was clearly the result of the chronic peritonitis. The exact pathology of the chronic peritonitis is one of the most interesting points of the case, but one about which it is impossible to be dogmatic. From the history it is clear that the ascites was of early occurrence, and was one of the first signs noticed by the friends. It therefore is a question worthy of consideration whether it can be brought into association with the cardiac condition, and explained as a secondary affection to it, or whether it was an independent disease.

Chronic peritonitis in children is so commonly tubercular, that considering the fact that we had to deal undoubtedly with a tubercular subject, as shown more especially by the affection of the elbow-joint, it must be regarded as *possible* that the chronic peritonitis in this case was tubercular. Against this, however, is the very long duration of it, the absence of any signs of tubercular abdominal disease, except the accumulation of fluid in the abdomen and the simply fibroid thickening presented by the peritoneum at the autopsy, without any signs of tubercle in the peritoneum or lymphatic glands. We know that recovery may ensue in even acute tuberculosis of the peritoneum, and although tubercular foci may result in the formation of much fibrous tissue, a general fibroid thickening of the peritoneum, such as existed in this case without any signs of caseation at any part, is not likely to be the result of peritoneal tuberculosis. For these reasons, together with the consideration of the fact that we may find a similar condition of the peritoneum in undoubtedly non-tubercular cases, I am inclined to think that the chronic peritonitis was not of tubercular origin.

It is, however, possible that the ascites was indirectly due to the cardiac dilatation in this case. In some cases of valvular heart disease with cardiac dilatation, instead of getting a general dropsy affecting both the peritoneal cavity and the extremities, we have very little or no anasarca of the lower extremities, but marked ascites, the peritoneal dropsy being out of all proportion to the anasarca of the extremities. Of course, in many such cases there is independent hepatic cirrhosis, due to its most common cause, alcohol. In the cases, however, here referred to, there is often no history leading us to suspect such independent hepatic cirrhosis, and the peritoneal dropsy is probably connected with the cardiac lesion. Of course, it is quite possible that congestion of the liver—the result of cardiac dilatation—may set up a secondary periportal cirrhosis of the liver, and so account for the ascites of such cases preponderating over the anasarca. From my own

experience, such secondary hepatic cirrhosis is not common; in fact, I cannot recall a single case where such cirrhosis has been so produced, and where it has not been probable that the cirrhosis has been due to another cause—most commonly alcoholic excess—and been independent of the venous stasis caused by the co-existing heart lesion. I have, however, seen a few cases, some of which have been followed to the pathological department, where it was probable that the ascites was due to a chronic peritonitis, which had occurred as a complication of the cardiac lesion, and most probably produced by the venous congestion of the peritoneum. In the cases of this kind in which I have had an opportunity of making post-mortem examinations, the appearances presented by the peritoneum have been almost identical to those presented in the second case above recorded. The peritoneum has been found thickened, and the thickening has been usually most marked and evident over the liver and spleen, the capsule of those organs appearing of an opaque, white, porcelain-like appearance, and very much thickened. I think, therefore, that the most probable explanation of the peritoneal dropsy in this case was that it was the consequence of a simple chronic peritonitis, which was either an independent affection or was secondary to the venous congestion of the peritoneum caused by the cardiac dilatation. We shall have occasion to refer again to this question of the pathology of the ascites in some cases of mediastino-pericarditis, when considering the symptoms and signs which have been observed in this affection.

The slight anasarca of the lower extremities observed in this case would be the result of the venous stasis caused by the cardiac dilatation assisted by the pressure of the ascitic fluid on the inferior vena cava. The dulness and bronchial breathing, noticed on repeated examinations, over the right supra-scapular and upper part of the right inter-scapular regions is also worthy of remark. There was no sufficient cause found to account for these signs at the post-mortem examination. The puckering and slight fibroid consolidation of the apex of the right lung was not sufficiently extensive and marked to account for the signs. During life, it was thought that it might possibly be due to the condition of the mediastinum, especially to the presence of enlarged and probably caseous mediastinal glands, between the trachea, or right bronchus, and the posterior part of the chest wall; such a condition, however, was not found to be the case at the post-mortem examination. We are, therefore, compelled to conclude that both the dulness and the bronchial breathing at the right apex behind were simply an exaggeration of a condition which may be normally found in that position. Certainly the breath sound opposite the spine of the scapulæ is not uncommonly of a faint bronchial character; and in some cases, especially in children, it is markedly so. Probably, also, it more frequently presents this character

on the right than on the left side, opposite the spine of the scapula and also above in the supra-scapular region. The case in this respect illustrates how careful one has to be, especially in children, in interpreting even well-marked bronchial respiration at the right supra-scapular and upper part of the right inter-scapular regions, and that such breath sounds may be present in a marked form independent of any enlargement of the bronchial or mediastinal lymphatic glands. The little value that can be attached to the presence of bronchial breathing in the regions indicated is quite in accord with the experience of many German observers, although much greater importance has been attached to the sign as indicative, especially of tubercular disease of the bronchial glands, by Austrian and French physicians.*

The following case is an illustration of Class III., where we have increase of fibrous tissue in the mediastinum without any internal pericardial adhesions. The case was one of very chronic tubercular phthisis, probably beginning in the apex of the left lung, which resulted in a marked thickening of the pleura over the left apex, and subsequently a chronic fibroid change in the upper part of the mediastinum.

Case 3.—*Chronic tubercular phthisis of both apices; difficulty in detection of tubercle bacilli in the expectoration; inoculation experiments with guinea-pigs; nephritis; general anasarca and later limitation of dropsy to the left arm; paralysis of the left vocal cord; marked orthopnœa.*

P. J., a big stout and muscular man, by occupation a lithographer, aged 40 years, was admitted to the Manchester Royal Infirmary on June 6th, 1894, with great dyspnœa and slight anasarca of the lower extremities. Patient was a very intelligent man, and gave the following history: He stated that 12 years ago, when he was 28 years of age, he was operated upon for fistula in ano. At the time of the operation he had a slight cough, and this had persisted ever since and somewhat increased in severity, but at no time had it been very troublesome. His chief complaint during the past 10 years had been shortness of breath, which during the past 12 months had been much worse than formerly. He had been able to attend to his work, however, which was of a light nature (the superintending of the lithographic department in a large firm) up to 10 days before admission, at which time the swelling of the feet commenced. He had had no dropsy before that time. During the past 12 years he had never been confined to his bed or to the house with any acute pulmonary affection. The dyspnœa had always been the most troublesome complaint, and he frequently had to rest on his way to and from his work, and to go upstairs very slowly. This difficulty had been especially marked during the past 12 months. Many years ago, exactly how many he could not say, he brought up a very little blood, but never any considerable amount. He admitted having taken rather freely of spirits, but did not impress us as having been a heavy drinker. He stated that he had not only not lost any flesh during the past 12 years, but was much heavier now than 13 years ago, which, considering his history and the distinct physical signs of phthisis which were present, and which will be directly related, was remarkable and scarcely

* Neumann (Dr. H.). "Ueber die Bronchialdrüsen-tuberculose und ihre Beziehungen zur Tuberculose im Kindesalter."—*Deutsche medicinische Wochenschrift*, 1893, S. 376.

credible. I therefore very carefully enquired into this point, and he stated that he was quite sure of the fact, because up to 27 years of age (13 years ago) he was an amateur boxer and weighed 10st. 8lb., which was his usual weight at that time, and he was classed in those days in competitions as "a middle-weight." Shortly after that time he gave up boxing and began to increase in weight, which he attributed to better living and lighter work, as he was made, about that time, manager of his lithographic department. For fully the last eight years he had been 12st., and affirmed most strongly that during his illness he had gained rather than lost in weight. On the day after his admission into hospital he weighed 12st. 5lb. in his shirt, slippers, and a dressing gown.* The hoarseness came on only a week ago, and although he states he has occasionally been hoarse before, it never lasted so long as seven days.

The family history was as follows : Father died when about 40 years of age, the cause of death being given as "some chest complaint;" mother alive, 68 years old, in good health ; two brothers and sisters living, aged 27 and 36, in fairly good but not very robust health ; two brothers and sisters had died, one at 21 years of age, the cause of death being believed to be consumption, and the other at 25 years, of acute inflammation of the bowels after her confinement.

Condition on the day after admission (June 7th, 1894) : Patient is a big muscular man, with marked orthopnœa. He has great difficulty in lying down in bed, and sits propped up, breathing somewhat noisily, distinct bubbling sounds being audible, and which are apparently produced in the throat. He keeps blowing out his lips with each respiration, making a noise very similar to what many grooms do when cleaning a horse. His cough is somewhat but not very troublesome. There is very little expectoration, only two or three small purulent masses being in the spittoon, which was emptied about 14 hours ago. He is decidedly hoarse. Respirations, 40 per minute. Pulse, 114 per minute, regular, but small. Temperature last night, 99·8° F. ; this morning, 98·8° F. (Further details of pulse, respirations, and temperature are given in the appended table and by the sphymographic tracings.) The face is pale and the lips not markedly blue. The fingers are, however, decidedly clubbed, and the bed of the nails slightly livid. No fulness of the veins of the neck. There is slight anasarca of the lower extremities, which readily pit on pressure. No ascites. No œdema of the upper extremities.

The chest is a broad, well-shaped one. There is possibly slight flattening of the left infra-clavicular region, but the difference between the two sides is very slight. There is very little movement of either side of the chest, the respiration being chiefly abdominal. Percussion : In front there is dulness all over the left side of the chest from the supra-clavicular to the infra-mammary region, the latter region not being absolutely dull but giving impaired resonance on percussion. The dulness extends across the median line to the right border of the sternum. The right supra-clavicular, clavicular, and upper part of the right infra-clavicular regions show decidedly impaired resonance, but are not absolutely dull. (See Fig. 9.) The vocal fremitus over the left infra-clavicular region is less marked than over the corresponding region of the right side, but it is not absent. Posteriorly, there is dulness over the left supra-scapular, supra-spinous, infra-spinous, and inter-scapular regions, the dulness being more absolute above than below. Below the inferior angle of the left scapula the resonance is impaired, but there is not absolute dulness, and on the right side there is dulness over the supra-scapular, supra-spinous, and upper part of

* In reference to this question of weight, it is to be remembered that he had on admission *slight* anasarca of the lower extremities.

the inter-scapular regions as far as the spine of the scapula. Below that point there is good resonance over the right side. (See Fig. 10.) The vocal fremitus is less marked over the left side behind than over the right, but the difference between the two is not marked. Auscultation: In front over the left supra-clavicular, infra-clavicular, and mammary regions, there are large consonating bubbles obscuring the character of the breath sounds and appearing to the ear as very coarse crackles. The consonating character of these bubbles appears most marked over the left infra-clavicular region, and there the bubbles give the impression of being of very large size. Over the

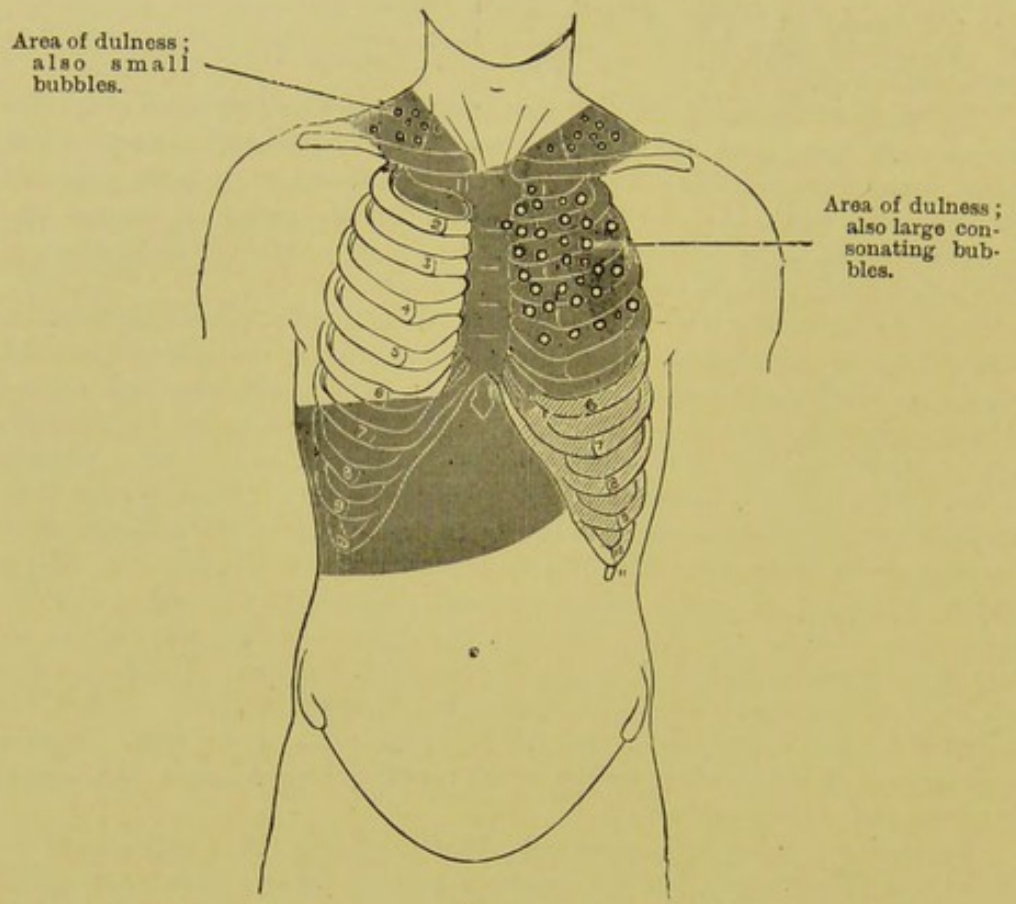


FIG. 9.

CASE 3.—T. J., æt. 40. June 7th, 1894.

Diagram representing the area of dulness over the front of the chest on the left and on the right side, and the hepatic dulness. The more deeply shaded part over the upper regions of the chest on the left side indicates that the chest was less resonant than below, where there is lighter shading.

right supra-clavicular region, and immediately below the clavicle, there are a few small bubbles; all over the front of the right side of the chest are numerous sonorous rhonchi; sonorous rhonchi are also to be heard over the left side at some places in addition to the bubbles. Posteriorly: Over the left supra-clavicular and supra-spinous regions, marked bubbles; and over the inter-scapular region, opposite the spine of the scapula, very marked bronchial respiration, together with large consonating bubbles. Below that point, simply sonorous rhonchi are audible, until at the extreme base a few bubbles can be heard. Over the right supra-scapular and supra-spinous regions some

simple non-consonating bubbles obscuring the breath sounds, and opposite the spine of the scapula, in the right inter-scapular region, distinct bronchial respiration, but of a somewhat less marked type than over the corresponding region of the left side. Below that point numerous sonorous rhonchi are audible. The sputum was examined at this time for tubercle bacilli, but none were found. The examination of the larynx by means of the laryngoscope was difficult on account of the retching which the examination caused, and also on account of the patient's dyspnœa, but I thought from the glimpse I got that the left vocal cord did not move. On the following day I was able to make a more careful examination, and found that the left cord was paralysed,

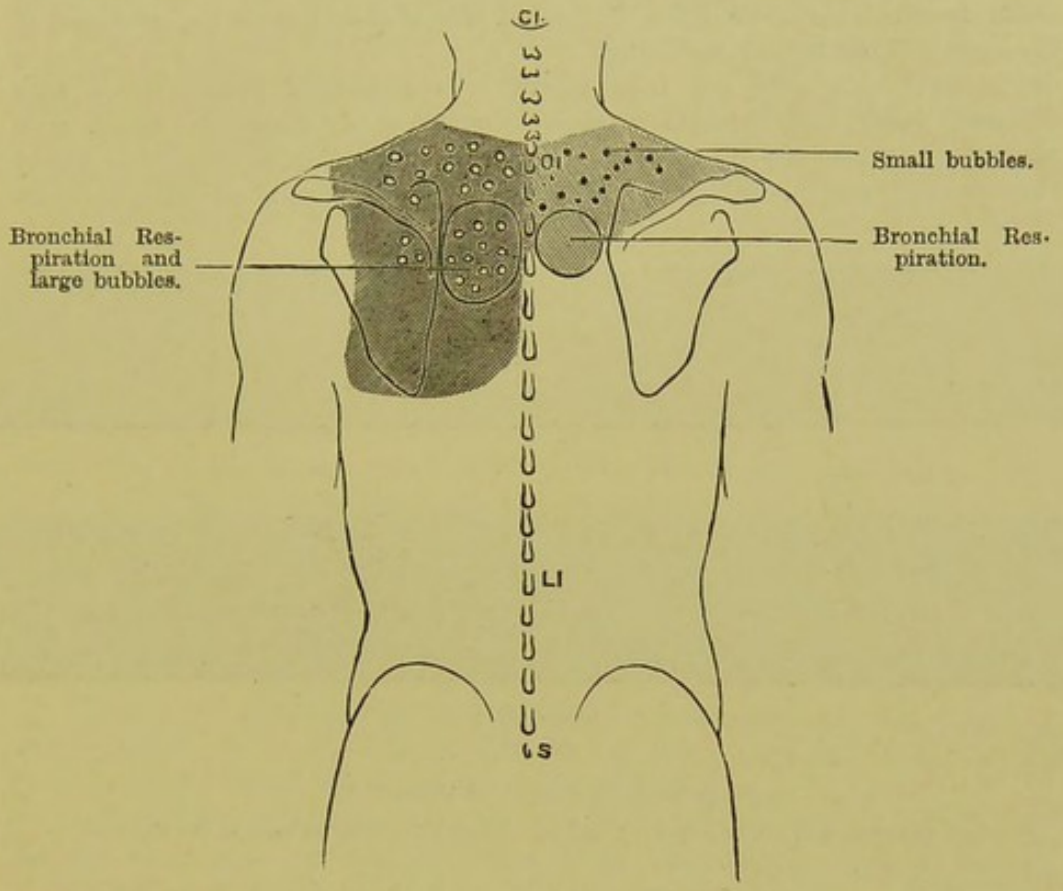


FIG. 10.

CASE 3.—T. J., æt. 40. June 7th, 1894.

Diagram representing the area of dulness over the posterior aspect of the chest. The lighter shading over the apex of the right lung indicates that the chest was there more resonant than over the corresponding region of the left side, where there was absolute dulness.

and did not move at all. There was no laryngitis nor any swelling of the laryngeal mucous membrane.

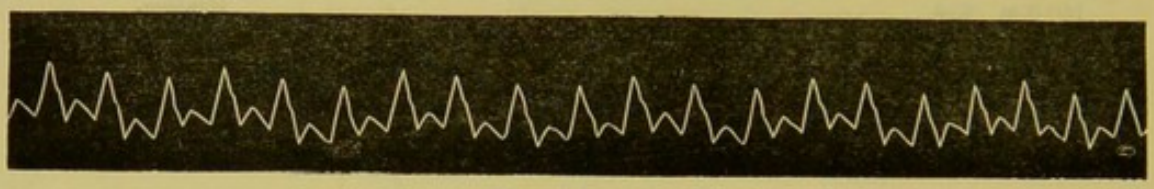
Heart: Apex beat indistinct, can be just felt to the right and below the left nipple in the fifth space. The cardiac dulness cannot be percussed out on the left side on account of the pulmonary affection, opposite the fourth and fifth ribs it extends a little to the right of the right border of the sternum. (See diagram, Fig. 9.) The heart sounds present no appreciable deviation from the normal. The second is not more accentuated over the second right as compared with that sound as heard over the

third left costal cartilage. No murmurs. Pulse 114 ; to the finger it appeared regular, but small. The sphygmograph showed, however, that it was slightly irregular in force and rhythm and of low tension. No difference between the radial pulse of the two sides. (See sphygmographic tracings taken June 8, 1894, Fig. 11). There is no unusual pulsation in the supra-sternal notch nor over the upper part of the front of the chest. No tracheal tugging. Pupils equal and of medium size.

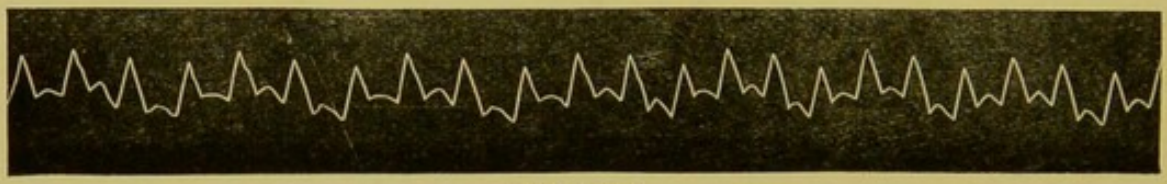
Liver dulness commences above, at the sixth rib, in the right mammary line, and extends downwards to fully two inches below the costal margin. Patient is too stout for any statement to be made as to the character of the surface of the liver.

Spleen cannot be felt below the costal margin. The impaired resonance which exists over the lower costal region of the left side of chest renders the percussion of the organ difficult and unsatisfactory.

Urine : 14oz. only has been passed in the last 24 hours. It is high coloured, 1025, acid, deposits a copious quantity of urates, is loaded with albumen, but contains no sugar. (For the amount of urine which was passed subsequently, see the subjoined table). The large quantity of lithates which



Right Radial.



Left Radial.

FIG. 11.

CASE 3.—T. J., æt. 40. June 8th. 1894.

Sphygmograms of Right and Left Radial. Pulse 114. Respirations 40 per minute.

the urine deposited rendered the search for organic elements in the deposit very difficult. Some recently passed urine was therefore mixed with an equal volume of potassium acetate and chloroform solution,* in order to prevent the deposition of urates, and the following day the deposit was examined. Epithelial casts small, and medium sized hyaline casts were found, but no fatty or opaque granular casts. Bowels have not been moved since patient was admitted.

The nervous system presents nothing unusual, except that for a hospital patient the invalid is an unusually intelligent man.

June 11, 1894.—The dyspnœa, with pallor of the face, remains the prominent feature of the case. The only noteworthy change is that the œdema has quite disappeared from the lower extremities, but the left arm has become considerably œdematous along its whole length. This œdema is unaccompanied by any pain in the arm, the limb simply feeling unusually heavy and full. There is no evident assign-

* HARRIS (THOMAS). "A method of collecting and preserving urinary casts and other organic urinary sediments."—*British Medical Journal*, Vol. I., 1894. p. 1356.

able cause for this. From enquiries made it is clearly not due to any more dependent position of that arm during sleep. The dyspnœa causes him to be very restless, and the face is pale and perspires freely. About one ounce of expectoration has been brought up every 24 hours since admission. It is very purulent and very suggestive in its appearance of phthisis, but considering the extensive area over which the auscultatory signs are audible, and their character, the amount is exceedingly small. The sputum has been three times examined (once by myself and twice by the house physician, Dr. Ainsworth,) for tubercle bacilli, but in each instance with negative results.

June 12, 1894.—Two guinea-pigs were inoculated in the left thigh with the expectoration on this day, the specimen of sputum from which that for inoculation was taken having been examined for tubercle bacilli, with negative results.

June 15, 1894.—The dyspnœa has slightly improved. The cough and expectoration remain about the same as before. The left arm is the only one which is œdematous, and that is less so than it was three days ago. The physical signs over the chest remain as before. Sonorous rhonchi are audible all over the chest, in addition to the other adventitious sounds over the apices, as described in the note of June 7, 1894.

June 28, 1894.—Patient shows very little change except that he has not only œdema of the *left* arm, but dropsy of the legs has reappeared. There is no œdema of the *right* arm. He has still marked orthopnœa and some cough. The expectoration is very purulent, but still only about 1oz. in 24 hours is expectorated, which small amount still remains remarkable, considering the extensive pulmonary disease which is manifest ; no tubercle bacilli can be found in the expectoration. The physical signs over the chest remain much the same as before, except that immediately below the left clavicle and over the left supra-scapular region the percussion note is of shallow tympanitic quality, and is not dull as was previously the case. I made the following note on this day :—"Although the physical signs are so extremely like those seen in a case of phthisis, the appearance of the man is not at all like that seen in a phthisical case. He is a big, well-nourished man, with a full face, sitting up in bed with evident dyspnœa."

Urine : 37oz. passed during last 24 hours ; has now become clear ; does not deposit urates ; it is of fairly good colour, not being very pale ; is acid ; 1017 sp. gr. ; contains fully ½ albumen ; epithelial and hyaline casts ; no blood.

July 11, 1894. The general condition has somewhat improved. He feels better, and the dyspnœa is less marked. There is still well-marked œdema of the legs and *left* arm, none of the right arm. The physical signs over the chest remain as before. The expectoration is about the same in amount, and presents the same character. It has been examined 16 times since patient's admission, on some occasions by myself, sometimes by the Medical Registrar, Dr. R. T. Williamson, and also by the house physician, Dr. Ainsworth, but always with negative results as regards tubercle bacilli. Urine, 64oz. passed during the last 24 hours, is somewhat paler than normal urine, sp. gr. 1013, albumen ¼. No blood. The temperature since a week after admission and, therefore, for the past month, as shown by the daily chart, has been generally subnormal, only on one or two occasions has it been above normal, but never since June 11th has it reached 99°. The temperature therefore has been taken every four hours since July 5th in order to ascertain if there is any rise of temperature at other times than the usual ones at which it is customary to take the temperature of the patients (8 a.m. and 8 p.m.), but only on one occasion during this time has it been above 99° (July 6th at 4 p.m. it was 99·4°), and on six other occasions during this time it has been at points between 98·4° F. and 99° F., at other times normal or subnormal. (See record of every four hours' temperature.)

TABLE SHOWING MORNING AND EVENING TEMPERATURES, THE NUMBER OF RESPIRATIONS AND PULSE BEATS PER MINUTE, THE DAILY QUANTITY OF URINE, AND THE ACTION OF THE BOWELS.

Date.	Temperature		Pulse.	Respirations.	Urine—Ounces in 24 hours.	Bowels—Motions in 24 hours.
	8 a.m.	8 p.m.				
June.						
6	98·6	99·8	—	—	—	—
7	98·8	98·2	114	40	14	—
8	98·6	98·4	102	40	8	3
9	98·0	97·8	114	38	14	—
10	99 2	97·0	108	32	20	—
11	97·0	99·4	102	32	18	4
12	97·4	98·0	96	32	16	1
13	97·6	98·0	108	26	29	1
14	97·0	98·0	84	24	24	1
15	97·8	97·6	108	32	47	1
16	98·4	97·6	96	38	50	2
17	98·0	98·6	102	32	57	—
18	97·2	98·2	96	36	51	4
19	97·6	98·0	114	36	49	—
20	98·6	98·0	114	40	60	1
21	97·8	97·4	102	34	41	1
22	97·0	98·4	114	36	50	2
23	97·0	98·0	102	32	37	2
24	98·0	98·2	102	44	38	2
25	98·0	98·6	108	36	50	—
26	97·4	97·0	108	32	44	1
27	97·8	98·6	102	32	34	1
28	97·4	97·0	108	30	37	—
29	97·4	98·0	114	36	39	4
30	98·4	97·6	108	32	41	1
July.						
1	97·4	98·0	108	36	31	1
2	98·2	98·0	114	38	36	2
3	98·0	98·2	96	30	22	2
4	97·6	98·0	102	32	38	2
5	98·2	97·6	102	32	35	2
6	98·2	98·0	108	34	31	2
7	97·0	97·6	120	40	34	1
8	97·8	97·4	108	32	58	1
9	98·0	98·6	108	32	81	1
10	98·0	98·8	108	34	62	2
11	97·6	98·4	110	32	64	1
12	98·0	97·0	102	28	77	2
13	97·6	97·0	108	32	76	1
14	97·0	98·0	102	36	64	2
15	97·0	98·0	102	32	54	1
16	97·0	98·4	60	36	74	1
17	98·0	97·0	102	28	51	1
18	97·4	98·0	100	34	70	1
19	97·4	97·8	102	30	51	1
20	97·0	98·0	108	36	34	2
21	97·4	97·0	108	36	44	2
22	97·0	97·0	108	34	27	1
23	97·6	98·0	102	32	24	2
24	97·8	97·6	96	32	31	2
25	97·4	98·4	108	28	34	2
26	97·4	97·0	108	32	42	2
27	97·0	97·0	108	28	24	2
28	98·0	97·0	108	32	44	2
29	97·0	97·8	114	36	34	2
30	97·0	98·0	102	36	20	2
31	97·4	98·4	108	36	26	2
August.						
1	97·0	97·0	108	40	19	2
2	97·6	97·0	102	32	26	1
3	97·0	97·0	102	30	33	1
4	97·0	98·6	96	34	38	1
5	98·0	97·0	96	36	37	1
6	98·0	97·8	120	30	16	2
7	97·0	98·2	108	36	8	1
8	97·6	98·0	96	30	16	2
9	97·2	97·8	96	28	13	2
10	97·0	97·0	102	28	9	2
11	98·4	97·6	100	34	31	2
12	97·6	97·8	102	32	29	2
13	97·0	98·0	96	34	24	2
14	97·0	98·4	102	32	34	2
15	97·8	98·4	96	32	50	2
16	97·0	98·4	102	32	36	2
17	98·0	98·4	96	32	36	2
18	97·4	98·4	96	32	37	2
19	97·0	97·8	114	32	31	2
20	98·2	97·6	108	36	35	2
21	98·2	97·4	96	32	37	4
22	98·6	97·2	120	44	9	2
23	97·0	—	—	52	7	1

RECORDS OF TEMPERATURE, TAKEN EVERY FOUR HOURS, BETWEEN JULY 5TH AND JULY 18TH, 1894.

Date.	12 noon.	4 p.m.	8 p.m.	12 midn't.	4 a.m.	8 a.m.
July						
5	97·6	97·0	97·6	98·6	98·6	98·2
6	98·2	99·4	98·0	98·4	98·0	97·0
7	98·0	97·6	97·6	98·6	98·2	97·8
8	98·2	97·0	97·4	98·0	98·2	98·0
9	98·4	97·8	98·6	98·8	98·0	98·0
10	97·6	97·0	98·8	98·0	97·6	97·6
11	98·6	99·0	98·4	98·6	97·0	98·0
12	98·8	97·4	97·0	—	—	97·6
13	98·2	98·2	97·0	97·6	97·6	97·0
14	98·6	97·4	98·0	98·0	98·4	97·0
15	97·8	97·0	98·0	98·0	98·6	97·0
16	97·0	98·4	97·6	97·8	98·0	98·0
17	97·6	97·0	98·0	97·4	97·4	98·2
18	97·0	98·0	98·4	97·2	97·4	—

July 13, 1894. Patient is now 12st. 8lbs. in weight as compared with 12st. 5lbs. on admission. (See note of weight and the condition of anasarca on July 30th, 1894.) One of the two guinea-pigs inoculated on June 12th, 1894, although apparently in good general health, was killed yesterday by the administration of chloroform. The post-mortem examination of this animal showed :—

A small thickened patch forming a small nodule at the seat of inoculation. Two of the superficial inguinal glands of the left side were enlarged. One of these was firm, but just beginning to caseate. The other was diffluent with a creamy caseous material.

The left sub-lumbar gland was enlarged, but not caseous. The corresponding gland of the right side was not affected. There was also no affection of the other lumbar glands. Spleen large, and contained a few distinct tubercles.

Liver, kidneys, heart, lungs, and other lymphatic glands other than those mentioned above healthy.

Microscopical examination of the caseous material from the larger of the two left inguinal glands showed the presence of a large number of tubercle bacilli.

July 30th, 1894.—The dyspnœa is somewhat better, and the expectoration even a little less than formerly. Since the 9th inst. patient has passed more urine than before that time, and although for the past ten days the quantity of urine has somewhat diminished, the bowels have been more frequently moved. (See table.) The anasarca has quite disappeared from the legs, but the *left* arm is distinctly larger than the right, although no pitting can be obtained. There are marked silvery (distension) marks at the bend of the left elbow, showing previous distension of that arm. Weight, now that all anasarca has gone, is 11st. 11lbs., showing a diminution of 9lbs. since admission, when anasarca of the lower extremities was present. The urine is now high-coloured and clear ; it contains one-third albumen, and a large number of hyaline casts. The physical signs over the chest remain

much about the same. In the left infra-clavicular region there is still a very shallow tympanitic note. The bubbles are audible, as before, over the upper part of both lungs, being especially loud and large over the left organ. The sonorous rhonchi are also still audible all over the chest. The sputum presents the same features. The records of the examination for tubercle bacilli are interesting. It has been examined twenty times with negative results. In order to eliminate errors from carelessness, etc., it was examined by different methods of staining, and not only by myself, but, at different times, by the House Physician, the Medical Registrar, and the Pathological Registrar. The 21st examination was made by Biedert's sedimenting method. One cover glass was then stained by Ehrlich's and the other by Gabbett's method. On the examination of the cover glass stained by the first method no tubercle bacilli could be found ; on that stained by the second method I found an organism which was pink, about the size of a tubercle bacillus, but which, even under one-twelfth oil immersion, looked more like a chain of cocci than a bacillus. As I could not find any other organisms at all like tubercle bacilli, I naturally felt doubtful as to whether the above was one of those organisms. The following day (22nd examination) I examined the sputum again, and found a few undoubted tubercle bacilli, and Professor Delépine, who also kindly examined independently the same specimen of sputum, found a few scattered groups of the same organisms. The organisms did not present anything unusual in their form. They could be regarded as typical tubercle bacilli.

August 1, 1894.—The second guinea-pig, which had been inoculated on June 12th, 1894, was killed this day by chloroform administration. There was a distinct ulcer, with sharp punched-out edges at the seat of the inoculation, with a thickening of the subjacent tissues. There was marked enlargement of the left superficial inguinal glands, which were full of soft caseous material. Left sub-lumbar gland markedly enlarged. Right gland not affected. Spleen large, with several groups of typical miliary tubercles therein, the organs being more affected than in the animal killed on July 12th. Lungs, heart, liver, and kidneys healthy. Lymphatic glands, other than those mentioned above, normal.

Shortly after the above date I left for my autumn holiday, and had not another opportunity of examining the patient. On my return I learnt that he had died somewhat suddenly on August 23rd, 1894. A few days before his death the dyspnœa became very distressing, and the nurses had great difficulty in persuading him to keep his bed, as he desired and was allowed to sit in a chair. On August 23rd, about 1-30 p.m., he had just been persuaded to get into bed, when he suddenly died.

The treatment during his stay in hospital had consisted in the administration of brandy, and at various times of a mixture of ammonium carbonate, nux vomica, and senega, of an inhalation of turpentine and eucalyptus, and occasional doses of a solution of ethyl nitrite and powders of caffeine. No creasote, or any drug likely to affect the virulence of the tubercle bacilli, was ever given.

The post-mortem examination was conducted by Dr. T. N. Kelynack, the Pathological Registrar to the Infirmary, on August 24th, 1894, at 10·30 a.m., and to him I am indebted for the following report :—

Chronic tubercular (fibroid) phthisis, thickening of the pleura over the apices of the lungs; chronic mediastinitis; compression of the left recurrent laryngeal and of the left pneumogastric nerves; dilatation of the heart; slight anasarca; venous congestion of the liver and kidneys, with (? secondary) nephritis.

External.—Body that of a pale, anæmic, well-built, and well-nourished middle-aged male. Hair dark. Face plump, and not at all like that of a tubercular subject. No enlarged cervical glands. Slight œdema of the lower extremities. Rigor mortis well marked. Dorsal hypostasis.

Internal.—Mediastinum and Pleuræ: On removing the operculum extensive fibroid induration about the upper part of the pleuræ and lungs, with extensive matting in the upper part of the mediastinum, is seen. This is so marked as to at first suggest the presence of an intra-thoracic tumour. The anterior borders of the lower part of both lungs are somewhat emphysematous. The heart is evidently large, and the area seen from the front is extensive. There is extensive chronic fibroid pleurisy over the upper lobe of both lungs. The thickening is excessive and especially well marked at the posterior wall of the chest, where the lungs are closely and firmly attached to the chest wall. The limitation of this pleurisy is remarkable, for while the pleural cavities are practically obliterated at their upper parts, there is very little involvement at the base and lower portions, where only a few fibrous adhesions exist. The thickening of the pleura over the upper parts is so firm that the lungs can only be removed with considerable difficulty and by cutting with the knife. The attachment is so close that the possibility of malignant growth at once suggests itself. The fibroid pleurisy has apparently extended to the mediastinal tissue at its upper part, closely uniting the lungs to the matted and indurated mediastinal tissues and also to the upper part of the pericardium. The left vagus nerve is closely surrounded by matted mediastinal tissue from which it can only be dissected out with difficulty. The left recurrent laryngeal nerve is also surrounded and compressed by the same fibrous tissue. Pericardium: Presents no internal adhesions, and is normal, except that there are some adhesions externally, at its upper part, uniting it to the matted mediastinal tissue.

Heart: Weight, 16oz.; considerably enlarged; all its cavities dilated, and the auricula-ventricular orifices enlarged; segments of the mitral valve slightly indurated; aortic cusps healthy.

Lungs: The left lung is the one most extensively affected. The whole of the upper lobe and upper part of the lower lobe is the seat of a chronic fibroid induration. The formation of fibrous tissue is extensive, and the upper part of the

lung consists of little more than deeply pigmented cicatricial tissue surrounded by a much thickened pleura, and having embedded in it large dilated bronchi which, towards the upper part, form distinct bronchiectatic cavities. The pleural involvement over this part of the lung has evidently been secondary to, or at least associated with the chronic fibrosis of the lungs and is not a primary condition. The lower portion of this left lung is more or less air containing, but in parts presents a certain amount of fibroid induration. No evidence of caseation or of recent tubercles. Weight of left lung, 1lb. 10oz. The right lung is very similar in general appearances to the left one, but is not quite so extensively affected. The upper part of the upper lobe is in a condition of fibroid induration with bronchiectatic cavities. In one or two places there is a very small focus of caseation. In the upper part of the lower lobe there is also slight fibrosis, whilst the greater part of this lobe is extremely congested and presents evidence of more recent tubercular involvement. Weight of right lung, 2lbs. 2oz. Bronchi of both sides much congested, slightly thickened, and dilated into bronchiectatic cavities in the midst of the fibroid parts of the lungs. No bronchiectatic cavities in the lowermost parts of the lungs.

Larynx and trachea congested, but otherwise presenting no abnormal appearances.

The condition of the lungs and bronchi above described is peculiar, in that the upper part of both lungs is converted into firm, tough, pigmented and coarse fibrous tissue; but there is little or no macroscopic evidence to show that the fibrosis is of tubercular origin. There is little or no ulceration in the form usually seen even in cases of chronic tubercular phthisis, and practically no caseation. The bronchi embedded in the fibroid lung are much dilated, and in one or two instances enlarged, and form distinct pouch-like cavities. These bronchiectatic cavities are lined with a distinct smooth membrane, like a mucous membrane, and contain a certain amount of secretion. There appears to be no ulceration of their walls, and no distinct evidence of tubercles in connection with them can be detected. If the fibroid condition were limited to the lower parts of the lungs, and not, as it is, especially seen in the upper parts, the appearances would be exactly what is seen in the rare cases of chronic non-tubercular fibroid phthisis with bronchiectasis. There can, however, be no doubt that the fibroid change is of tubercular origin when the condition of the lungs as a whole is considered, because (1) the fibroid change and bronchiectasis is specially seen in the apices of the lungs and on both sides; (2) in some portions, after making several sections into the lung parenchyma, small caseous foci can be detected; (3) at the lower part of the fibrous areas distinct fibrous tubercles are present singly, in patches, and

running along lines suggestive of vascular or lymphatic extension. There is, however, no indication of any recent tuberculous pneumonia nor of the formation of acute tubercles.

The aorta presents slight evidences of atheroma.

Abdomen: Peritoneum normal.

Stomach and intestines congested.

Liver: Considerably enlarged; weight, 5lb. 6oz., soft and friable, and, on section, of typical nutmeg appearance. Gall-bladder: Normal.

Spleen: Slightly enlarged; weight, 5oz., soft, friable, and congested.

Kidneys: Both enlarged; each weighs 8oz.; capsules peel off readily; surface smooth and of mottled congested appearance. On section, both organs much congested, and much blood drips from the cut surface. Slight increase of the cortex, but no distinct evidence of fibrosis. There is evidently considerable parenchymatous degeneration, the change being probably dependent upon the passive congestion. Ureters normal. Bladder and testicles normal.

Joints and bones present no abnormal appearances. The muscles of the body generally are not appreciably wasted.

Remarks on Case 3.—During life this case presented many features of clinical interest. The very long history, during which time dyspnœa, rather than cough or expectoration, had been the most prominent feature, together with a gain rather than a loss of flesh, was not what we expect to find in phthisical cases. His appearance too was not the least suggestive of phthisis, but very much so of a thoracic aneurism or other intra-thoracic tumour. In favour of the latter diagnosis was also the paralysis of the left vocal cord, and the occurrence of œdema of the left arm. Except, however, for the two last named signs no others, except his age and general appearance, could be cited in favour of the diagnosis of a thoracic aneurism. The physical signs of phthisis were, however, so distinct that had it not been for the great difficulty of finding tubercle bacilli in the expectoration, we should have had no hesitation in deciding upon the existence at least of one lesion, viz., tubercular phthisis. But at this time, after the repeated examinations of the sputum by different stains and by different persons, I was inclined to regard the case as possibly one of aneurism with secondary affection of the lungs, due to the pressure of an aneurismal sac. When, however, the guinea-pigs showed unmistakable signs of tuberculosis, the result of inoculation, and when later a few tubercle bacilli were found in the expectoration, we could no longer escape the diagnosis of chronic tubercular phthisis, but whether there was an aneurism in addition we could not say. Paralysis of the left recurrent laryngeal nerve is so uncommon in cases of phthisis that with the recurrent œdema of the left arm I felt we could not exclude the possible presence

of either an aneurism or a mediastinal growth in addition to the phthisis. The clinical diagnosis which we did arrive at, and which was sent down on the usual abstract of the case to the post-mortem department, was "Chronic tubercular phthisis—paralysis of the left vocal cord—nephritis," and the paralysis of the vocal cord we attributed to thickening of the pleura over the apex of the left lung, just as we see occasionally, paralysis of the right vocal cord in phthisis of the apex of the right lung.

I must admit that, although I have been for years interested in the subject of mediastino-pericarditis, and was at the time this case occurred actually engaged in writing a part of this communication, I never even thought of chronic mediastinitis as the cause of the œdema of the left arm and the paralysis of the left vocal cord.

The result of the inoculation experiments with the guinea-pigs shows the value of such experiments for diagnostic purposes. The animals were manifestly tubercular, and tubercle bacilli had been found in the inguinal gland of one of the animals before we found tubercle bacilli in the expectoration of the patient. I am perfectly aware that many will be inclined to say that we did not perform our examinations of the sputum carefully enough. That is probably true to this extent, that a few tubercle bacilli were in the expectoration which we examined, but that on account of their scarcity we overlooked them. Too many examinations were made, and with the actual knowledge or strong belief that the case was one of tubercular phthisis, for us to admit that carelessness was the real cause why we did not find the organisms in the sputum before the twenty-first examination.

As to whether the dyspnœa was entirely to be attributed to the general bronchitis and to the cardiac dilatation, or to any effect which the chronic mediastinitis had on the vagus or other nerves, I cannot say.

The pathological appearances of the lungs show how closely a fibroid lung of tubercular origin may resemble a fibrosis of simple inflammatory origin, and how difficult it may be to distinguish the two unless regard be paid to the position of the fioroid affection and the condition of the parts of the lung surrounding the old fibroid parts.

The pathological history of the case was probably that we had a tubercular phthisis which ran a very chronic course; that this was associated with thickening of the pleura over the tubercular lung, and that the inflammation extended to the upper part of the mediastinal tissue and produced there the fibroid mass found at the post-mortem examination. This fibroid mediastinal tissue and the thickened pleura compressed the left recurrent and left vagus nerves and the left innominate vein, thus accounting for the paralysis of the left vocal cord, and for the œdema being so marked and at one time limited to the left arm. Dilatation of the heart subsequently followed, probably from muscular degeneration

rather than from the obstructed pulmonary circulation. This was followed by an enlarged liver, anasarca of the lower extremities, congestion of the kidneys and secondary nephritis, the immediate cause of death being cardiac failure.

As previously stated, the subject of indurative mediastino-pericarditis was first brought prominently before the notice of the profession by Kussmaul[1] in 1873. Widenmann's case[4] from Griesinger's clinic had been at that time apparently forgotten, if not entirely overlooked, by the majority of physicians. Although we are indebted to Kussmaul for his careful and detailed account of the clinical features of three cases which he had observed, and for describing in detail the pathological appearance found after death in two of the cases, it should not be forgotten that, in 1871, two years before Kussmaul's communication appeared, Dr. Samuel Wilks wrote in the *Guy's Hospital Reports*[5] for that year, when referring to pericardial adhesions, as follows: "I would especially draw attention not only to the fact that the chronic inflammatory process very often involves the vessels entering the heart within the pericardium, but that, where the latter is adherent, the thickening and induration proceeds, as would a growth, to involve the cellular tissue in the mediastinum, and even creeping upwards to surround the veins in the neck. I have thus seen, in two or three cases, a tough areolar tissue surrounding the vena cava and brachio-cephalic veins in connection with the old pericarditis." Dr. Wilks then relates six cases of adherent pericardium, five of which belong to the affection now under consideration. Four of them are well-marked examples of the condition known as pericarditis interna and externa, and one is a most typical instance of an adherent pericardium, associated with marked increase of the tissue in the mediastinum. Certainly, there appears to be a great deal in a name. Wilks' cases were recorded chiefly to illustrate the effects of an adherent pericardium upon the heart, and although the majority were instances of one variety of mediastino-pericarditis, that term was not employed. Kussmaul, on the other hand, recorded his cases not only as examples of mediastino-pericarditis, but drew prominent attention to the occurrence of a special form of pulse, the pulsus paradoxus, and to inspiratory swelling of the veins of the neck in connection with that affection.

Subsequent observations and increased experience do not, I think, allow us to attach that importance to the pulsus paradoxus in the diagnosis of mediastino-pericarditis, which Kussmaul appeared inclined to attribute to the phenomenon. It was, nevertheless, that physician's communication which created an interest in the subject, and brought forward the publication of a number of cases of indurative mediastino-pericarditis.

TABLES

GIVING AN ABSTRACT OF CASES OF INDURATIVE MEDIASTINO-PERICARDITIS AND OF CHRONIC MEDIASTINITIS (NON-SUPPURATIVE) WHERE POST-MORTEM EXAMINATIONS HAVE BEEN MADE.

These tables include only such cases where the diagnosis has been confirmed by a post-mortem examination. As before stated, it is useless to abstract the cases believed during life to be cases of mediastino-pericarditis, but which, however probable it was that the diagnosis was correctly made, have not been confirmed by a subsequent post-mortem examination. The tables have been made as complete as possible. In the majority of cases the original records have been consulted, and in the few instances where the original papers have not been accessible, and reliance has had to be placed upon an abstract from some other publication, the reference has been marked with a †. The author fears that, although he has tried to avoid omitting any published case, he has probably inadvertently failed, as even with the aid of the various Year Books, Dictionaries, and Medical Digests, it is almost impossible, considering the numerous medical journals now published, to avoid some omissions even in a comparatively small subject like the one under consideration.

I. Case No.	II. Author.	II. Where Recorded.	IV. Age.	V. Sex.	VI. Previous History and Mode of Onset.	VII. Duration.	VIII. Variety.	IX. Pathological Appearances.	X. Chief Symptoms, Physical Signs, and Cause of Death.
1	Widenmann, A. (Griesinger's case.)	† "Dissert. inaug." Tübingen, 1856. "Beitrag zur Diagnose der Mediastinitis," by A. Widenmann. See also a detailed account in Kussmaul's communication in the *Berliner klin. Woch.*, 1873, S. 434; and "Ziemssen's Cyclopædia," English translation, Vol. VI., p. 649.	22	M.	Previously healthy. Affection probably of traumatic origin. Somewhat acute onset, with pain in chest, cough, and hæmoptysis.	Nine Weeks.	Mediastino-pericarditis, but rather acute than chronic and indurative.	Fibrinous mediastinitis, with fibrino-purulent pericarditis. Stiff, stringy exudations were found in the connective tissue, in which the large vessels were imbedded after their exit from the pericardium. Some of the venous trunks were constricted, as was also the aorta in its ascending portion and arch. It was also indented and twisted about its axis.	Dyspnœa, cyanosis, and general dropsy, which began with ascites. Increased cardiac dulness. Heart sounds weak but pure. Pulsus paradoxus. No marked distension of cervical veins. Urine became highly albuminous. Died comatose, the dyspnœa, cyanosis, dropsy, and fever having gradually increased.

2	Wilks, S.	"Guy's Hospital Reports," 1871. Third Series. Vol. XVI., p. 196. "Adherent pericardium as a cause of cardiac disease."	21	M.	Had been ailing from boyhood, although there was no history of any distinct illness. He used to get short-breathed, livid in the face, and his hands became blue and cold. About three years before his death all these symptoms became aggravated, and occasionally his ankles would swell. All these symptoms were more severe in the cold weather. During these three years he was several times in the hospital, and came under the notice of all the physicians in turn. The diagnosis was some congenital affection of the heart, but although they repeatedly examined the heart, they could find no evidence of disease of it.	? 3 years under observation, but ailing from boyhood.	Mediastino-pericarditis (Pericarditis externa and interna).	General dropsy. In right pleura 2 pints of fluid; left lung adherent to chest; both lungs adherent to pericardium. Pericardium universally adherent to heart and immensely thickened. Heart enlarged. Right auricle firmly adherent to the lung behind and the pericardium in front. A linear opening in the fossa ovalis, but there was a valve-like arrangement, so that probably no blood passed through. Pulmonary artery of normal size. Tricuspid, aortic, and mitral valves healthy, muscle of heart thin, pale, and flabby, liver nutmeg, spleen and kidneys healthy.	Dyspnœa and later orthopnœa. Marked cyanosis. Coldness of extremities. General anasarca and ascites. Enlarged veins over chest, abdomen, and legs. Heart-sounds more obscure than natural, but no bruit was discoverable at any part; action often irregular. Liver enlarged. Whilst in hospital, owing to the sudden setting in of cold weather, his breathing became most difficult, and the blueness and enlargement of the veins most remarkable. With increase of the dropsy and blueness, death ensued.
3	Wilks, S.	*Ibid.*	12	M.	Tolerably well until a few months previous to coming under observation, when, after a cold, suffered from a cough and difficulty of breathing. Subsequently some swelling of the abdomen and legs appeared.	Some months. Was 3 months under observation, and had been ailing a "few months" previously.	Mediastino-pericarditis. (Pericarditis interna and externa).	Pericardium universally adherent and thickened. Lungs universally adherent to chest walls and to pericardium. Heart "about the usual size;" valves quite healthy. Lungs healthy. Liver enlarged and nutmeg. "Recent peritonitis."	Dyspnœa; face livid; anasarca and ascites. Enlarged liver; heart sounds normal; urine not albuminous; râles over lungs. Abdomen was tapped twice, and although the legs became very dropsical, it appears, from the brief report of the case, as though the ascites at one time was out of proportion to the anasarca. He sank three days after the second tapping, and died apparently from cardiac failure.

I.	II.	III.	IV.	V.	VI.	VII.	VIII.	IX.	X.
Case No.	Author.	Where Recorded.	Age.	Sex.	Previous History and Mode of Onset.	Duration.	Variety.	Pathological Appearances.	Chief Symptoms, Physical Signs, and Cause of Death.
4	Wilks, S.	*Ibid.*	16	F.	Rheumatism when a child, and occasionally suffered from dyspnœa ever since, and other symptoms indicative of heart affection. She had been able, however, to follow her employment until a few hours before her admission, when, being taken much worse, she came to the hospital. She survived only a few hours. There was much dyspnœa, but no bruit to be heard.	? Was 16 years old when she died and had had symptoms of heart affection since rheumatic attack "when quite a child."	Mediastino-pericarditis. (Pericarditis externa and interna.)	Pericardium very thick, universally adherent, and it encroached upon the heart muscle. Both lungs universally adherent to the chest wall, and closely embraced the heart, to which they were adherent. Heart rather dilated in all its cavities. Muscle of heart pale and soft throughout. Endocardium did not show any marked signs of ever having been inflamed, but the edges of the mitral valve were slightly thickened. It appeared, however, to be an efficient valve. Congestion of the lungs, with some recent pneumonia.	See account in Column VI. Immediate cause of death, as shown by the post-mortem examination, was congestion of the lungs and some recent pneumonia.
5	Wilks, S.	*Ibid.*	26	M.	See Column X.	No definite statement; but certainly over a year, and probably several years.	Mediastino-pericarditis.	Slight œdema of the legs; a little fluid in the right side of the chest. Pericardium universally adherent, much thickened and indurated: it was a quarter of an inch thick in all parts, but at the base anteriorly it was still thicker, and the tissue so tough that the adventitious matter felt more like a tumour. It encroached on the muscles of the auricles, and proceeded upwards along the aorta and the roots of the blood-vessels coming from the arch. Thus the trachea, aorta, mediastinum, etc., were all involved in one hard, tough, fibrous tissue. The heart was enlarged in all its parts. Endocardium and valves quite healthy. Pericardium also firmly united externally to both lungs. Liver large and myristicated.	Was taken to the hospital in a very exhausted state; the dyspnœa was excessive, and coming on in paroxysms, so that it seemed at any moment he would die. Face and hands blue and cold. Heart violently beating against the chest, but no bruit could be heard; the natural sounds were indistinct. On this occasion he died soon after admission. He had been in the hospital a year before with what was believed to be a heart affection, though no abnormal cardiac sounds were detected, but the natural ones were described as obscure. He then had some dropsy, which afterwards disappeared.

6	Wilks, S.	*Ibid.*	24	M.	For many years had had cough, shortness of breath, and expectoration. There was a history of rheumatism several years ago. Was in the habit of coming to London for advice, and on the day of admission into hospital, the weather being cold, was unable to return. Breathing extremely difficult. Venesection was performed, and gave temporary relief, but he died a few days afterwards. No abnormal sounds were heard, to indicate disease of the heart.	Many years.	Mediastino-pericarditis. (Pericarditis externa and interna).	Pericardium much thickened, and universally and closely adherent to the heart. Lungs universally adherent to chest and to the pericardium. They were extremely congested, and in parts the blood was extravasated into the tissue. Bronchial tubes slightly dilated, and contained mucus. Left ventricle of usual size, the right hypertrophied, and walls thickened and tough. The mitral valve quite healthy. The aortic valves efficient, but on their surface a few minute vegetations.	See column VI. Immediate cause of death, as shown by the post-mortem examination, was congestion of the lungs, with hæmorrhage, probably consequent upon cardiac failure.
7	Kussmaul, A.	*Berliner klinische Wochenschrift*, 1873. X. Jahrg. S. 433, 445, 447, and 461. "Ueber schwielige Mediastino-Pericarditis und den paradoxen Puls."	21	M.	Some years previously had enteric fever from which he had apparently recovered. Shortly before coming under observation began to feel unwell, and 7 weeks later there were signs of pleurisy of the left side.	Eight months.	Mediastino-pericarditis.	General dropsy. Pericardium thickened and cavity obliterated by firm adhesions. Externally, sac adherent to the lungs, diaphragm, and chest wall. Compression to a marked degree of the ascending part of the arch of the aorta, of the trunk of the pulmonary artery, and superior vena cava. Enlarged mediastinal glands, some caseous. Recent tuberculosis of the lungs and peritoneum. Liver large; nutmeg; spleen large. Ulceration of ileum.	Dyspnœa; cyanosis; general anasarca and ascites; pulsus paradoxus; inspiratory swelling of the veins of the neck. Slight increase of cardiac dulness. Death from phlegmonous inflammation of the leg.

I.	II.	III.	IV.	V.	VI.	VII.	VIII.	IX.	X.
Case No.	Author.	Where Recorded.	Age.	Sex.	Previous History and Mode of Onset.	Duration.	Variety.	Pathological Appearances.	Chief Symptoms, Physical Signs, and Cause of Death.
8	Kussmaul, A.	Ditto. * See *Berliner klin. Wochenschr*, 1873, page 447, †461.	34* ?32†	F.	Suffered for years with chronic bronchitis and chronic caseous pneumonia. Was not known as to when the mediastino-pericarditis commenced.	? but must have been several years.	Mediastino-pericarditis.	General dropsy. Pericardium much thickened and parietal adherent to visceral layer, but not firmly united. Firm and old adhesions of the exterior of the pericardium to surrounding parts. Increase of fibrous tissues in the mediastinum. Bronchial glands enlarged, and pigmented. Chronic tuberculosis of the lungs. Liver very large, congested. Spleen, 13 cm. long, 7 broad, 4½ thick: seat of tubercles.	Dyspnœa; cyanosis; general dropsy; pulsus paradoxus: increased precordial dulness and distention of jugulars. Death, with gradually increasing dyspnœa, from cardiac failure. Kussmaul relates the clinical features of a third case, but as it was not followed by a post-mortem examination, it is omitted from these tables.
9	Cantalena.	† *Gior. veneto di sc. med.* Venezia: 1874, 3, S., XXI., pp. 37–40. Quoted in "Affections of the mediastinum." By H. A. Hare, B. Sc., M.D. Philadelphia: 1889, p. 95.	22	M.	No statement in the abstract referred to in the second reference.	?	"Callous mediastino-pericarditis."	"Pleuritis; pericarditis and fibrinous exudate which pressed on the veins and produced a hum." (Hare.)	"Pain in chest; bruit in chest; venous hum; oppression." (Hare.)
10	Kipp, F.	"Ein Fall von schwieliger Mediastino-pericarditis." *Inaugural-Abhandlung zur Erlangung der Doktorwürde.* München, 1875. Also referred to briefly in Bauer's paper in *Von Ziemssen's Cyclopædia.* Vol. VI., p. 653.	22	M.	Good health until the 3 or 4 years previous to coming into hospital. During these years had frequent pains in the chest, and two years ago had pleurisy of the left side.	2 years from the attack of pleurisy.	Mediastino-pericarditis.	Adherent and thickened pericardium. Heart very much enlarged. Valves healthy. Adhesion of the posterior aspect of the sternum and costal cartilages, especially of the left side, to the parts beneath. Encapsulated caseous masses beneath the lower costal cartilages of the left side. Upper part of the mediastinum presented very little change. Pleuritic adhesions and a small amount of effusion into both cavities. No bands extending along the course of the main vessels from the heart, such as could cause any narrowing of those vessels. Liver enlarged; nutmeg type. Spleen twice as large as normal. Kidneys large and congested. Hypostatic pneumonia.	Dyspnœa; cyanosis: markedly increased precordial dulness; no cardiac impulse to be felt; weak heart sounds. Pulsus paradoxus. Distension and pulsation of cervical veins, but no mention of inspiratory swelling of those veins. Six weeks before death general dropsy appeared. Enlarged liver. At first urine free from albumen; later it became scanty and albuminous. Death from hypostatic pneumonia.

11	Bauer.	"Cyclopædia of the Practice of Medicine." Edited by Dr. H. von Ziemssen. London, 1876, Vol. VI., p. 653.	"Young Man."	M.	It seems probable from the short account given that the onset was insidious and not sudden and acute. Bauer states that probably a pleurisy several years previous to him coming under his observation had been the beginning and the foundation of the subsequent chronic mediastino-pericarditis.	Not definitely stated. Was under observation about six weeks: but the pleurisy, which was believed to be the beginning of the affection, had occurred "several years before."	Indurative-mediastino-pericarditis.	Obliteration of pericardial cavity. The entire ventricular portion of the heart was imbedded in a "stiff callosity." The formation of the callosities had not extended far above the ventricles. Heart dilated, and muscle fatty degenerated. Encapsulated cheesy masses under the left costal cartilages. Induration of the tissues in the mediastinum, in addition to the cheesy masses. Connective tissue bands capable of drawing the large blood vessels of the mediastinum forward, and causing indentations in them, were not found.	Cyanosis, dyspnœa, and general dropsy. Area of precordial dulness increased. The apex beat could not be felt. Heart sounds were uncomplicated and moderately loud. Area of dulness did not change during deep inspiration. Distended jugulars and venous pulsation. Pulsus paradoxus. Urine scanty, but free from albumen.
12	Weiss.	A case from Bamberger's Clinic, recorded by Weiss. † "Oester. med. Jahrbüchr.," 1876. "Ueber Verwachsungen des Herzens mit dem Herzbeutel." Fairly full abstract can be found in Riegel's article on "Krankheiten des Herzbeutels," in *Gerhardt's Handbuch der Kinderkrankheiten*, Band IV., Abth. 1, S. 231 and 235.	14	F.	See Pathological appearances and chief symptoms, etc. Columns IX. and X.	?	Mediastino-pericarditis (? pericarditis externa and interna).	Chronic inflammation of the great serous membranes (the result of an attack of typhoid), which had led to obliteration of the pericardial cavity, of the pleural cavities, and to chronic thickening of the peritoneum. Weiss considered that the pericardial obliteration and the chronic peritonitis, resulting from the enteric fever, had acted in the following manner: the former had effected a venous stasis which, in consequence of the peritonitis, was first felt by the peritoneum, which was, therefore, a "punctum minimæ resistentiæ," and led to a transudation into the abdominal cavity.	The chief feature was that the dropsical phenomena first showed themselves not in the lower extremities, but as ascites, and existed there for a long time before any other dropsical phenomena showed themselves. Enlargement of the liver and spleen. Heart dulness normal. Cardiac impulse indistinct. Sounds pure and pulse small. (See page 235, as well as 231 of *Gerhardt's Handbuch*, Vol. IV., Part 1.)

I.	II.	III.	IV.	V.	VI.	VII.	VIII.	IX.	X.
Case No.	Author.	Where Recorded.	Age.	Sex.	Previous History and Mode of Onset.	Duration.	Variety.	Pathological Appearances.	Chief Symptoms, Physical Signs, and Cause of Death.
13	Habershon, S. O.	"Transactions of the Pathological Society of London," 1876, Vol. XXVII., p. 79.	37	M.	Good health until 7 years previous to his coming under observation. Onset sudden, with swelling of face and neck.	7 years.	Chronic mediastinitis.	Pericardium contained a little fluid, and there were old adhesions at the base of the heart in the pericardium round the great vessels. Upper part of mediastinum presented dense fibrous tissue, and the superior vena cava was completely obliterated. Various systemic veins in thorax much enlarged. Great distension of the cervical veins. Heart much enlarged and seat of fibroid changes. Deformity of pulmonary valve. Thickened peritoneum from chronic peritonitis, and cavity full of dark yellow fluid. No recent peritonitis. Liver, 59oz.; much altered in shape, and rounded; capsule thick and marked with depressions. Spleen, 5½oz.; very firm, and presenting thick white patches on its surface. Kidneys weighed 10½oz., and were quite healthy. Mesenteric glands healthy.	Dyspnœa; cyanosis; œdema of arms; distension, without pulsation, of external jugular veins. Increased area of cardiac dulness. Ascites. Girth of abdomen, 42in. Ankles œdematous, and several spots of purpura on legs. Urine scanty, loaded with lithates, and slightly albuminous. Paracentesis abdominis performed, but relief only partial, and patient gradually sank and died.
14	Fox, A. W.	*British Medical Journal*, 1877, Vol. II., p. 470.	20	F.	Had had pleurisy when at the age of 19; pericarditis and enteric fever at 20, *i.e.*, about four months before the onset of dropsy and other signs of mediastino-pericarditis.	12-18 months.	Pericarditis, interna and externa. (From the account there does not appear to have been much increase of fibrous tissue throughout the mediastinum).	Thickened and adherent pericardium. External aspect of sac adherent to lungs, diaphragm, anterior chest wall, and lower dorsal vertebræ. Heart a little enlarged. Both lungs adherent by their lower lobes to the chest wall; some effusion in right pleural cavity. Lower lobes of lungs congested and œdematous. Peritoneal cavity contained several quarts of fluid, and the omentum was studded with miliary tubercle. Liver enlarged and of nutmeg appearance. Spleen of natural size, but its capsule thickened. Kidneys healthy.	General dropsy; increased area of precordial dulness; no visible cardiac apex beat. Distended pulsating cervical veins, showing inspiratory swelling, Pulsus paradoxus. Enlarged liver. Cause of death was gradual asthenia, associated with the development of tubercular peritonitis.

15	Desnos.	*Le Progrès Medical*, IX., 1881, p. 323.	20	M.	Had measles when 8 years old, and several attacks of pain in the joints at 15. (?Rheumatism.) Two years later palpitation noticed, with dyspnœa on exertion and precordial pain.	Under observation 3 to 4 months. Had had palpitation for 3 years, but as there was valvular disease of the heart, it is difficult to say when the mediastinal affection began.	Mediastino-pericarditis. (Pericarditis externa and interna.)	Fibroid adhesions outside pericardium, uniting sac to surrounding parts. Pericardial cavity presented false membranes, and was adherent, but the process there appeared to be acute rather than chronic. Valvular disease (stenosis of mitral, etc.). Enlarged nutmeg liver. No mention of spleen. No tuberculosis found.	When first observed dyspnœa with distinct œdema of legs, scrotum, and trunk. No ascites. Enlarged liver. Increased area of precordial dulness; faint systolic aortic and distinct systolic and presystolic murmurs at apex. Pulse irregular; full, but compressible; some beats not reaching radial. Pulsating cervical veins. No mention of P. paradoxus or of inspiratory swelling of the veins of the neck. Œdema increased, and patient passed into a comatose condition and died.
16	Hutton, H. R.	St. Thomas's Hospital Reports, New Series, Vol. XIII., 1883, p. 211. Also "Abstracts of some of the Medical and Surgical Cases treated at the General Hospital for Sick Children, Pendlebury, Manchester, 1883," p. 58.	10	M.	Insidious onset. First noticed swollen veins on the front of the thorax. Later had scarlet fever. Ten weeks later dropsy of the feet appeared, and when seen about this time the face was slightly cyanotic.	About 17 months.	Mediastino-pericarditis.	Adherent pericardium, which was very much thickened and almost cartilaginous. Left lung adherent throughout. Right pleural contained a little effusion; also some old adhesions of right pleura. In the mediastinum several small glands, pale and translucent, none caseous; all matted together with much fibrous tissue, forming a large mass surrounding the trachea and great vessels and compressing these vessels, especially the vena cava. Abdomen contained some serum, but there is stated to have been no peritonitis. Liver much enlarged, capsule thick, intense venous congestion. Microscopical examination of the liver showed, in addition to the marked venous congestion, increase of fibrous tissue round the portal vessels, multiplication of bile ducts, and slight "cirrhosis of the hepatic vessels." Spleen tough and congested (size not given). Kidneys large and congested. Capsules slightly adherent. Cortex not wasted.	Dyspnœa; cyanosis; marked ascites (requiring repeated paracentesis abdominis) and general dropsy. Increased precordial dulness; pulsus paradoxus; enlarged liver. Gradually increasing cardiac failure; convulsions, followed by death.

I.	II.	III.	IV.	V.	VI.	VII.	VIII.	IX.	X.
Case No.	Author.	Where Recorded.	Age.	Sex.	Previous History and Mode of Onset.	Duration.	Variety.	Pathological Appearances.	Chief Symptoms, Physical Signs, and Cause of Death.
17	Hutton, H. R.	"St. Thomas's Hospital Reports," New Series, Vol. XIII., 1883, page 211.	6¼	M.	Six months previous to the time he came under observation for the present affection had scarlet fever, from which he appeared to recover entirely. Three months later swelling of feet was noticed, followed some time later by swelling of the abdomen.	About nine months.	Mediastino-pericarditis.	No details given.	Dyspnœa; cyanosis, ascites, and general dropsy. Enlarged liver. No enlargement of the spleen. No albuminuria. Enlarged pulsating cervical veins. Cause of death not given.
18	Leech, D. J.	Case quoted by Hutton. (See above.)	13	F.	Insidious onset. Formerly good health. Scarlet fever at eight years of age. Ten months before she first came under observation, began with pain in the left side of chest, shortness of breath, cough, and hæmoptysis. Two months later dropsy of arms and legs, followed by ascites, the œdema of the extremities soon passing off.	Seventeen months.	Mediastino-pericarditis.	No details given. Stated that the "post-mortem examination revealed, besides pulmonary phthisis, extensive mediastino-pericarditis, causing obstruction to the return of blood from the liver, and great enlargement of that organ."	Signs of pulmonary phthisis; increased area of precordial dulness; diffuse cardiac impulse felt well to the left of the nipple. Cyanosis, dyspnœa, ascites, and general anasarca. Enlarged liver.* Cause of death was sudden and in convulsions.

* Dr. Leech and Dr. Dreschfeld, who examined sections from the liver from this case, tell me that there was a certain amount of peri-portal cirrhosis present.

19	Jaccoud, S.	"Leçons de Clinique Médicale," 1884-85, par S. Jaccoud. Paris, 1886. Pp. 66-97. "Sur un cas de maladie cardio-artérielle pericardite et médiastinite antérieure."	42	M.	2½ months before admission for present complaint was seized, whilst apparently in the enjoyment of good health, with difficulty of breathing. Continued his work, however, for 5 days, and then had to give it up on account of the increasing dyspnœa. Œdema of the lower extremities also came on previous to admission. No previous illness except that eight years previously had severely wounded the right hand, which had necessitated the ligature of axillary artery, and to this ligature Jaccoud attributed the aortitis from which the man was also suffering.	3½ months.	Mediastino-pericarditis (The pericarditis appeared to be of comparatively recent origin.)	Acute pericarditis, with adhesions which could be easily broken down. Thick fibrous adhesions, external to the pericardium, stretching from the anterior surface of the lower lobe of the left lung to the pericardium, and uniting exterior of pericardium to the sternum and costal cartilages. Fibrinous pleurisy of left side. General aortitis; dilatation of arch of aorta; acute endocarditis of mitral valve; and a focus of myocarditis. No record of condition of the liver or spleen or about ascites.	Dyspnœa. Pain in chest. Anasarca. Patient had to sit up in bed with the trunk markedly bent forward, because, if he assumed the erect posture, and still more the recumbent one, the dyspnœa and intra-thoracic pain became much more marked. Patient had signs of dilatation of the arch of the aorta and aortic incompetence. Increased cardiac dulness and a peculiar undulatory movement of the pericordial region, which Jaccoud considers of value in the diagnosis of an adherent pericardium. No pulsus paradoxus nor inspiratory swelling of the veins of the neck.
20	Claessen, H.	*Deutsch. med. Wochenschrift*, 1892, S. 161. "Ueber tuberculöse, kasig-schwielige Mediastino-pericarditis und Tuberculose des Herzfleisches."	19	M.	Formerly healthy, except for an attack of rheumatism, scarlet fever, and typhlitis, when a child. Insidious onset, with shortness of breath on exertion, and blueness of face. Later, slight dropsy of feet.	Two years from first onset of symptoms.	Mediastino-pericarditis, with marked tubercular disease of various parts.	Adherent pericardium. Marked tubercular foci in heart muscle. One caseous bronchial gland, but other mediastinal lymphatic glands not tubercular. Tubercular mesenteric glands. Miliary tubercle in pleura. Spleen, liver, and kidneys congested.	At first marked cyanosis, like a case of morbus cœruleus. Distension, but no pulsation of cervical veins. Enlarged veins over front of the thorax. Cardiac dulness, when first under observation, normal. Pulse very small, and hyperdicrotic. No sign of a pulsus paradoxus in the sphygmogram given in communication. Enlarged liver and spleen. No fever. Urine free from albumen. Later in the course of the disease, left pleuritic effusion (2,200 cb.c. removed). General anasarca, which, however, is stated to have been at first limited to the left half of the face, left upper extremity, left half of trunk, and left thigh. Southey's trocars were introduced, but through patient scratching the parts where they were inserted, phlegmonous inflammation of the leg ensued, followed by general sepsis and death.

I. Case No.	II. Author.	III. Where Recorded.	IV. Age.	V. Sex.	VI. Previous History and Mode of Onset.	VII. Duration.	VIII. Variety.	IX. Pathological Appearances.	X. Chief Symptoms, Physical Signs, and Cause of Death.
21	Westphalen, H.	*St. Petersburger Imed. Wochenschrift*, XVII *Jahrgang, Neue Folge* IX., *Jahrg.*, 1892, s. 277.	50	M.	History was that for a long time had been subject to bronchial asthma, the attacks having recently become more severe. The diagnosis on his first admission into hospital (June 17, 1889) was bronchial asthma, and an acute colitis, to account for a diarrhœa which was present.	? Several years. Was under observation two years and ten months, but had been subject to asthmatic attacks for a long time previously.	Chronic mediastinitis. (No internal pericardial adhesions.)	Emphysema and chronic bronchitis. Adhesion of both lungs to the chest, to the diaphragm, and to the pericardium. Fibrous mediastinitis. Enlargement of the heart. Venous hyperæmia of spleen, which was large, and kidneys with consecutive moderate chronic interstitial nephritis and cyst formation. Anarsarca and ascites. Liver rather small, border rounded. Perisplenitis and perihepatitis* and fibrous adhesions of various organs in the abdomen to surrounding parts. Brown induration of lung, fresh pneumonia, and œdema of lung.	At first this case was regarded as one of bronchial asthma. Later, dyspnœa became more marked. Sputum contained Curschman's spirals, but no Leyden's crystals or tubercle bacilli. At first no cyanosis, but later it became marked, with anasarca and ascites. Under treatment, œdema of legs disappeared, but the ascites showed too slight a diminution in proportion to the improvement of the congestive phenomena. Abdomen repeatedly tapped. Enlarged liver and spleen. Cardiac apex indistinct. Westphalen says that they could not observe a distinct pulsus paradoxus, nor distinct inspiratory swelling of the veins of the neck, apart from the pulsation associated with the tricuspid insufficiency which existed. Heart often showed galloping rhythm, and at one time a "post systolic murmur" was heard, which was at the time believed to be of pericardial origin. Gradual increase of the evidence of venous stasis, and death from cardiac failure.

* Microscopical examination of the liver showed some fibrous bands extending in from the thickened capsule. [The description given, however, is not at all like that of a cirrhotic liver, nor does it suggest the presence of any extensive fibrous change in the organ.—T. H.]

22	Ashby, H.	"The Diseases of Children, Medical and Surgical," by Henry Ashby, M.D., F.R.C.P. and G. A. Wright, B.A., M.B., F.R.C.S. Second Edition. Edited for American students, by W. P. Northrup, A.M., M.D., New York and London. 1893. Page 311.	2 yrs.		Four months previous to admission into hospital had cough, and a month later his abdomen began to swell, and soon after, his feet; this gradually increased.	Four to five months from onset of symptoms.	Mediastino-pericarditis.	Pericardium thick and adherent. In the middle and posterior mediastinum, enlarged glands and fibrous tissue. Glands caseating, and several had putty-like contents. Heart not enlarged. Lungs not adherent; right and left lung showed pneumonia, but no tubercle. Much yellow fluid in abdomen, and some lymph on liver, spleen, diaphragm, and great omentum. Lymph and tubercle between liver and diaphragm. Liver much enlarged and granular, "one boss," size of a marble, on the anterior surface of the right lobe; on section, liver of nutmeg appearance. Spleen enlarged. "Kidneys pale."	Face puffy. Abdomen distended with fluid, and legs much swollen. Pyrexia, 101°, on admission. Pulse, 130. Respirations, 40. Bubbles over both lungs, and diminished resonance over right upper lobe in front. Cardiac apex beat in third space. Sounds normal. Abdomen greatly distended with fluid. On the following day to that of admission edge of liver could be felt below the umbilicus, and a nodule the size of a marble in the left lobe. Temperature, 105°—106° F. before death, which took place apparently from pneumonia.
23	Harris, T.	*Medical Chronicle*, October, 1894. New Series, Vol. II., p. 1.	14	M.	For details see page 3.		Mediastino-pericarditis.	For details see page 9 *et seq.*	For details see page 3 *et seq.*
24	Harris, T.	*Medical Chronicle*, November, 1894. New Series, Vol. II., p. 87.	8	M.	For details see page 87.	3½ years.	Mediastino-pericarditis (pericarditis externa and interna).	For details see p. 92.	For details see page 88, *et seq.*
25	Harris, T.	*Ibid.*	40	M.	For details see page 98.	? Probably had phthisis 12 years, but how long the mediastinal affection existed cannot be stated.	Chronic mediastinitis.	For details see page 107.	For details see page 99, *et seq.*
*									

* Details of any cases published by Rivalta[25] or by Salinas[26] in their respective communications cannot be given, as the original Italian and Spanish publications are not accessible and there are very few particulars given in the year-books.

Since 1873 cases of chronic or indurative mediastino-pericarditis have been published in various medical journals, and the time seems now to have arrived when we may, with advantage, bring such cases together, and consider what are the most common clinical and pathological features of the affection. It is with that object that I have collected as many cases as the medical publications at my disposal will allow, and have abstracted the principal points of each case. The abstracts of these communications I have arranged approximately in the chronological sequence of their publication. (See pages 42 to 53).

The tables given include three cases of chronic non-suppurative mediastinitis, and such cases would therefore come under our heading of Class III. The remaining 22 cases are representatives of the affection which, up to the present, has been known as indurative mediastino-pericarditis. As before stated, however, we can distinguish *on the post-mortem room table* two varieties of this affection, which differ rather in the extent and degree of the lesion than in any other essential features. One variety is where there is an adherent pericardium together with marked increase of the fibrous tissue in the mediastinum, and in that variety there is an adhesion of the exterior of the pericardium to surrounding parts, a condition which is accurately termed *indurative mediastino-pericarditis* (Class I.). The other variety is where there is an adherent pericardium, with thickening of the sac and adhesion of it to surrounding parts, but with very little and sometimes no general mediastinitis, a condition which has been termed *pericarditis externa and interna* (Class II.). I have little doubt that many cases of the latter variety (Class II.) have been described by authors under the heading of indurative mediastino-pericarditis, and certainly one (see Case No. 5 in the tables) and probably others, which can be considered as well-marked examples of indurative mediastino-pericarditis, have been described by the authors under the simple heading of an adherent pericardium. From the descriptions given by those who have recorded these cases, it is not always easy to be certain as to the exact variety, and I have frequently had a difficulty, when reading the records, in saying whether the case was one of true indurative mediastino-pericarditis or one of pericarditis interna and externa. As, however, it is probable that we cannot in the majority of cases distinguish at the bedside between these two varieties of the affection, it will be better, in considering the above tables, not to attempt to determine how many of the 22 cases belong to Class I. (*indurative mediastino-pericarditis*) and how many to Class II. (*pericarditis interna and externa*).

We will now briefly consider some general features in the clinical history of cases of indurative mediastino-pericarditis which the above records appear to warrant.

Age.—I think many physicians have an impression that the affection occurs more commonly in children than in adults. Such an impression may be correct; but if so, a large number of cases in children must have escaped being placed upon record, or have not been followed by a post-mortem examination. From the above tables, which are comprised solely from cases where the diagnosis has been confirmed by a post-mortem examination, the affection would appear to be somewhat more common in adults than in children. Of the 22 cases, nine occurred in persons under 18 years of age, and thirteen in persons over that age (including one where the patient is indefinitely described as a "young man"). Only two cases occurred in persons over 30 years of age. (The three cases of chronic mediastinitis which appear in the tables all occurred in older people, the youngest being 37 years of age.)

Sex.—Males appear to be more frequently affected than females. Of the 22 cases no less than 17 occurred in males and only five in females.

Mode of Onset and Causation.—In some of the cases there is a history of an acute illness, usually some acute chest affection, occurring some time previous to that at which the symptoms of mediastino-pericarditis manifested themselves. In some instances this acute illness was clearly an attack of acute pericarditis. In these cases it is probable that the acute illness represented the time when the disease commenced, and that the manifestation of symptoms indicative of indurative mediastino-pericarditis at a later date represented the commencement of cardiac dilatation and failure, which was brought about by the pericardial adhesions. In some cases the only acute illness which the person is said to have suffered was one of the acute fevers, most commonly scarlet fever or measles.

In many cases the onset has been perfectly insidious, and careful enquiry may fail to elicit any symptoms of the patient having had any acute chest affection. This was so in one of my own cases (Case 24 in the table) where, although from the results of the post-mortem examination, it is almost certain that he had had, at some antecedent date, acute pericarditis, we could obtain no history from the mother pointing to any such acute affection.

Trauma does not appear to have commonly played a part in the causation of the affection, although the contrary has been stated. (Hare).

Tubercle appears certainly to be not an infrequent associate of the disease, but in many no mention is made either of any tuberculosis of the mediastinal lymphatic glands, or of other parts, being found on post-

mortem examination. In my first case a large old caseous focus existed in the mediastinum, and my second case occurred in an undoubtedly tubercular subject.

Symptoms and Physical Signs.—The symptoms and signs observed in the hitherto recorded cases of indurative mediastino-pericarditis have been chiefly dyspnœa, more or less marked evidence of venous engorgement and cyanosis, cardiac enlargement, increase in size of the liver, and either general dropsy, or only ascites with little or no anasarca of the lower limbs, a pulsus paradoxus, and inspiratory swelling of the veins of the neck. All these symptoms and signs are, however, not necessarily present in every case.

Posture of the patient: Jaccoud (in Case 19 in tables) has laid much stress upon the posture of the sufferer from mediastino-pericarditis. He attributed much importance, as a diagnostic sign, to the fact that his patient had to sit up in bed with the trunk markedly bent forward, because if the sufferer assumed the erect posture, and still more the recumbent one, the dyspnœa and intrathoracic pain became much more marked. But we certainly occasionally see very bad heart cases, other than those where the distress is due to the affection under consideration, assume the posture described by Jaccoud. It is also a fact that the majority of cases of indurative mediastino-pericarditis do not assume the position described. At first they are able to lie down in bed fairly comfortably; later, as the dyspnœa becomes more marked, they may have to sit up and to maintain that posture; but it must be quite exceptional, if the records of the published cases can be trusted, for them to have to assume the unusual position described by Jaccoud.

The dyspnœa has varied much in intensity in different cases, and at different stages of the same case. At first it is usually slight, and only marked on exertion; later on, as the heart becomes more dilated and more feeble, the shortness of breath and the evidence of venous stasis commonly become more pronounced. This was the course of events in two of my own cases.

Physical signs: There is commonly evidence of cardiac enlargement, and dilatation preponderates over hypertrophy. This cardiac enlargement is almost entirely to be attributed to the increased work thrown upon the heart by the adherent pericardium, together with the degeneration of the cardiac muscle, which tends to supervene in such cases. In these cases we commonly get a marked increase of the mediastinal dulness, but such is usually due to the increased size of the heart especially of its right cavities, rather than to the increase of the mediastinal tissue. When the heart is very markedly dilated, and especially when it is pushed up by fluid in the abdominal cavity, the mediastinal dulness may be very extensive, reaching up even to the

lower border of the first rib. This extensive mediastinal dulness may be found where there is very little increase of mediastinal tissue, as shown by my second case. In such instances of extensive mediastinal dulness, the probability will be that the cause of the dulness will be found to be due to the cardiac enlargement rather than to the mediastinitis. In some cases, however, enlarged caseous glands, together with the increase of the fibrous tissues in the mediastinum, may contribute, apart from the enlarged heart, to produce a dulness over the upper part of the sternum (Case 23 in the table). The heart sounds may be natural, weak, or if there is independent valvular disease, we may have one or more murmurs present.

The increase in the size of the liver is usually due to marked venous engorgement of the organ, which is chiefly the result of the cardiac dilatation, possibly in some cases, assisted by the fibroid change in the mediastinum, obstructing the inferior vena cava. A certain amount of increase of the fibrous tissues of the liver has also been found, probably a secondary effect of the chronic venous congestion of the organ. I think, however, such increase has never been a very marked one, and such as would be sufficient to account for the production of ascites from portal obstruction.

The spleen has usually been found not to be enlarged, a fact of considerable interest and importance when considering the cause of local dropsy of the abdominal cavity in those cases where such dropsy has been found with little or no anasarca of the extremities. The spleen was, however, considerably enlarged in one case (Claessen's).

Anasarca, especially of the lower extremities, is commonly present at some period of the cases of indurative mediastino-pericarditis. It may be a marked feature of the case during a prolonged period, or only slight, and occur late on in the course of the disease. This was so in my two cases above recorded; the dropsy of the lower extremities was never very extreme, and occurred late in the course of the disease. In those two cases it was probably due to the gradual onset of cardiac dilatation, and such is the probable explanation of it in the majority of cases of mediastino-pericarditis rather than any direct narrowing of the inferior vena cava by the fibroid change in the mediastinum.

As a part of the general dropsy we may also have ascites, and in some instances, as was notably so in Case 2 (*i.e.*, Case 24 in the tables), the ascites may be one of the most marked features of the case, and be present with little or no anasarca of the extremities. This point, the occurrence of ascites, with little or no general anasarca, has been already partly discussed in the remarks on Case 2 (see page 26).

The connection of such ascites with indurative mediastino-pericarditis cannot be considered a settled one. We see some cases of this disease,

of which Case 1 (page 9) is a very good example, where we have throughout the whole course of the affection no marked ascites, and only such as is part of a general dropsy, and can be explained as the result of the cardiac dilatation. Other cases, however, are seen, of which Case 2 is a typical example, where the ascites is one of the most prominent features of the case, and where it occurs without any or only slight œdema of the lower extremities. Ascites without anasarca is therefore not present in every case of mediastino-pericarditis; it occurs in some, it does not occur in others. When it does occur we can only explain it as due to the occurrence of a complication to the primary disease, or as due to a lesion independent of the mediastino-pericarditis. There are two lesions either of which may possibly occur as complications to the mediastinal and pericardial affection, and which would explain the ascites in such cases. In the first place, it is conceivable that chronic venous congestion of the liver produced by the cardiac dilatation may in some cases set up a chronic inflammation of the liver, a secondary peri-portal cirrhosis, which like the ordinary alcoholic cirrhosis would obstruct the portal circulation, and produce ascites. We have seen that an increase of fibrous tissue in the liver is found in some of the cases of mediastino-pericarditis, but such a change does not appear to be the rule, and in the cases where it has been found it is questionable whether it has been sufficiently pronounced to produce portal obstruction and ascites therefrom. Furthermore, in one of my own cases (Case 2) where the abdominal dropsy was such a prominent feature of the illness, the ascites was certainly not due to a cirrhotic liver, no marked peri-portal cirrhosis being found on microscopical examination.

The second explanation of the occurrence of the ascites in the cases under consideration is that a chronic peritonitis ensues, and to it the ascites is due. Such chronic peritonitis, in some instances, may possibly be an independent affection, and one not directly connected with the mediastinal or pericardial lesion; in other cases it is conceivable that the chronic venous congestion, due to the interthoracic affection, sets up a chronic peritonitis. Such a chronic peritoneal change was found in my second case, and was clearly sufficient to account for the ascites. That it is the explanation of the ascites in all such cases, of course we do not say. It is quite possible that other complications may in some cases occur which account for the development of ascites as a prominent feature of the case.[7]

As I have stated before, we do occasionally meet with cases of ordinary obstructive heart disease where ascites is present in

[7] Acute tuberculosis of the peritoneum was found in one case of indurative mediastino-pericarditis, where marked ascites was present. Dr. A. W. Fox.—*British Medical Journal*, 1877, Vol. II. p. 470. (Case 14 in table.)

an amount out of all proportion to the œdema of the lower extremities, or even for a time without the occurrence of any anasarca of the limbs. I have seen several such cases, where I had no reason to suspect any independent hepatic cirrhosis or any other disease, such as a tubercular affection of the peritoneum, which would account for the ascites. In four such cases I have been able to make a post-mortem examination, and have found no marked hepatic cirrhosis, but decided changes in the peritoneum. The peritoneum covering the abdominal wall and the intestines has shown comparatively little change at first sight, very little evidence of thickening being manifest in this part of the peritoneum, and there has not been found any marked matting of the intestines together or general peritonitic adhesions. Some adhesions in the neighbourhood of the spleen and liver have been the chief ones present. The capsule of the liver and that of the spleen has shown the most marked change. The surface of these organs has presented a peculiar opaque white porcelain-like appearance, and usually there have been shallow depressions or pits, due to the thickening of the capsules not having been quite uniform. So that the change in the peritoneum in these cases is somewhat peculiar, and not what one usually associates with one's ideas of chronic peritonitis; it is, however, evidently of the nature of a chronic inflammatory process.

This form of chronic peritonitis with capsulitis of the liver and spleen is referred to by the late Dr. Hilton Fagge,[8] and he says the most common cause of it is Bright's disease. Dr. W. Hale White,[9] also, from an analysis of the cases occurring in the pathological registers of Guy's Hospital, came to a similar conclusion, and says of universal perihepatitis, which is to be regarded in the majority of cases as part of a chronic peritonitis, "it is almost invariably associated with granular kidney, and frequently with gout, syphilis, over indulgence in alcohol, and diseases of the heart and lungs, causing backward pressure." I have collected fourteen cases of chronic non-tubercular peritonitis, in which there was also universal capsulitis of the liver, from the records of the pathological department of the Manchester Infirmary. In ten out of the fourteen cases there was obstruction, usually from valvular or lung disease, causing backward pressure, and in these cases the kidneys were in no instance of the granular contracted variety. The kidneys were usually large and congested, and although in some instances they showed very slight increase in fibrous tissue in parts, it was only to such a degree as might be expected from chronic venous congestion. In only one of the fourteen cases was there any marked nephritis present, and in that instance the kidneys presented both marked parenchymatous and interstitial changes, and in that case also the heart was dilated. The remaining three cases presented no heart, lung, or kidney disease, which would account for

the chronic peritoneal changes. We shall have occasion to refer to these cases more in detail in a future communication, but so far as our experience at the Manchester Infirmary bears upon the subject, obstructive heart or lung disease, causing backward pressure and venous stasis, is a more common associate of chronic non-tubercular peritonitis, and capsulitis of the liver and spleen, than is a granular kidney. It would appear, therefore, that the chronic peritonitis with capsulitis of the liver and spleen is found not only in cases of indurative mediastino-pericarditis, but that similar changes in connection with the abdominal viscera are found in various forms of obstructive heart and lung disease, and also that similar changes may sometimes be present independently of such venous stasis.

Rosenbach,[10] in consequence of an observation of his own and a case recorded by Wiess[11] from Bamberger's clinic, calls attention to the possible connection between an adherent pericardium, an enlarged liver, and the occurrence of ascites without general dropsy. He has observed such cases, and followed one to the pathological department. He calls attention to the thickening of the capsule of the liver which is found in such cases, and suggests that possibly the pericarditis spreads along the course of the large veins to the capsule of the liver and results there in the formation of fibrous tissue, or that possibly the process spreads in the reverse direction from the capsule of the liver to the pericardium. He goes so far as to regard the association of an enlarged liver and marked ascites without general dropsy with cardiac symptoms as of some value in the diagnosis of an adherent pericardium.

This view of Rosenbach's does not seem to be on *a priori* grounds a very probable one, and if it were true we ought to meet with the association more frequently in cases of adherent pericardium than is the case. Dr. Kelynack, the Pathological Registrar at the Manchester Infimary, has kindly looked up the records of the post-mortem examinations which have been made at the hospital during the past few years, and we find that out of twenty-five cases of adherent pericardium only one was associated with chronic peritonitis and thickening of the capsule of the liver—a proportion far too small to render Rosenbach's suggestion probable. We also find the peritoneal lesion and the thickened capsule of the liver in obstructive heart disease, such as mitral stenosis, without any pericardial adhesions being present.

I am inclined, therefore, to think that the ascites which sometimes occurs in cases of mediastino-pericarditis, and which is unaccompanied by extreme anasarca of the lower extremities, is due, in some instances, at all events, to the onset of a peculiar form of chronic peritonitis. This chronic peritonitis may be possibly due to venous engorgement of the peritoneum, consequent upon the dilatation

of the heart or venous obstruction in the mediastinum, or it may occur so early in the case as to be probably independent of such venous stasis. In my second case (Case 24 in table) the ascites was a prominent symptom from a very early period of the illness, and probably was independent of venous engorgement, which only appeared as a marked feature some time after the development of the ascites.

The Pulse in cases of mediastino-pericarditis presents different characters in different cases. We may have it differing very little from the normal, but usually it has been found small and frequently also irregular—one form of irregularity was formerly regarded as a pathognomonic sign of mediastino-pericarditis, viz., the pulsus paradoxus, the pulse becoming smaller during the act of inspiration.

> *Pulsus Paradoxus* :—It is stated (O. Rosenbach)[18] that F. Hoppe (1855) was the first to observe the phenomen of the pulse becoming small during the act of inspiration. Hoppe's observation was made in August, 1853, in a person affected with pericarditis, dilated heart, and emphysema, and was published in 1854.[13] The phenomenon had, however, been observed some years previously by Dr. C. J. B. Williams, and published by him in the *London Journal of Medicine* for 1850. At page 464 of the second volume of that periodical (1850), in a article on "The Prognosis and Treatment of Organic Diseases of the Heart," the following may be found: "Advanced stages of pericarditis and adhesions of the pericardium present a remarkable kind of inequality, and even intermission, in the radial pulse, whilst the heart's pulsations are quite regular ; in fact, some of the latter are too weak to reach the wrist, hence the irregularity ; and I have distinctly ascertained that the weak, or deficient beats, are those corresponding with inspiration, whilst the stronger pulses are those enforced by the expiratory act, which thus helps the weak heart, and augments its propulsive power." It appears, however, that Widenmann's case (1854), from Griesinger's clinic, was the first case of mediastino-pericarditis where this form of pulse intermission was detected and published, although it was not until Kussmaul published his cases of mediastino-pericarditis in 1873 that special attention was directed to this form of pulse in that disease.

This form of pulse, however, is seen occasionally where we have no mediastinal affection, and mediastino-pericarditis may exist without causing the production of this peculiar form of pulse (see several of the cases in the table—*e.g.*, Case 19). The first of my cases presented it in a marked and typical form, my second never presented it during the time the case was under my observation, although it was repeatedly examined for. This form of pulse has been observed not only in mediastino-pericarditis but in different forms of pericarditis without mediastinitis [12, 13, 14, 15, 16, 17], in cases of large pleuritic effusions (Bauer[18]), in great cardiac weakness,

[12] Williams (Charles J. B.). "On the prognosis and treatment of organic diseases of the heart."—*Journal of Medicine*, Vol. II., 1850, page 464.

[13] Hoppe (F.). "Ueber einen Fall von aussetzen des Radialpulses während der Inspiration und die Ursachen dieses Phenomens."—*Deutsche Klinik*, 1854, s. 33. [Under the action of digitalis when the pulse became slower and stronger, the intermission disappeared, and reappeared later when the pulse became quicker and weaker.]

in convalescence from long standing febrile affections[19], I have observed it also in a case of mitral incompetence with dilatation of the heart, and it occurs in cases of great dyspnœa from narrowing of the air passages[20], and it is recorded in cases of mediastinal tumour[21]. Experimentally it has been produced in animals by compression of the inferior vena cava at its entrance into the auricle during the inspiratory period[22], and lastly, slight diminution of the pulse has even been observed in sphymographic tracings taken from healthy individuals.[23] *

Although, therefore, the pulsus paradoxus can no longer be considered peculiar to cases of indurative mediastino-pericarditis, the detection of it in any case should direct the physician's attention to the possible presence of that affection.

Engorgement of the veins of the neck is common in cases of mediastino-pericarditis, and such engorged veins may show marked pulsation, as in cases of dilatation of the right cavities of the heart from simple cardiac dilatation, or in cases of dilatation of those cavities secondary to mitral or other valvular disease. A particular form of distension of the right external jugular vein, however, is sometimes seen in mediastino-pericarditis, and has been considered of great diagnostic value. In this form of pulsation the vein is seen to fill and become distended during the act of inspiration, especially when a *deep* inspiration is taken. Unfortunately for its diagnostic value this inspiratory swelling of the right cervical vein is sometimes absent in cases of this disease. In neither of the two

* At the present time there is a coloured man going the rounds of various cities exhibiting himself to medical men as a pathological curiosity. He is, however, a fraud, in that he has not the two hearts, as he asserts. A few days ago he called upon me, and I examined him. He stated that, in addition to other anomalies, he had two hearts, and that he could stop the one in the left side of his chest. Whilst he presumes to do this, he allows you to listen with the stethoscope, over the mammary region of the left side, and, at the same time, to feel his left radial artery at the wrist. During the performance, which he then goes through, I certainly noticed that the pulse at the left wrist was very small, if not quite imperceptible, and the heart sounds were very indistinct. The explanation appeared to be that he took a very deep inspiration and held his breath. As he was a very muscular man, and contracted the pectoral and other chest muscles, so much muscular noise was produced that the heart sounds were not easily heard, and probably by the deep inspiration and holding his breath, possibly also assisted by the violent muscular contraction of the pectorals and upper arm muscles compressing the main vessel to the left upper extremity, he actually did render the left radial pulse small. Both myself and a friend found that by a similar action we also could diminish the volume of our radial pulse, and I have since seen that F. Hoppe (*ibid.*), so long ago as 1854, wrote that any one by an experiment on himself can prove that the radial pulse becomes weaker when the muscles of inspiration are violently contracted whilst the glottis is kept closed, and he also states that this is not to be confused with the disappearance of the pulse from the radial artery, effected by the compression of the subclavian vessels, by forcibly elevating the first rib during a prolonged deep inspiration.

[19] West (J.). "A case of purulent pericarditis treated by paracentesis, etc."—*British Medical Journal*, 1883. [Disappearance of the pulsus paradoxus after the removal of the fluid.]

[20] Boehr (E.). "Ueber einem zweiten Fall von Pulsus paradoxus in Folge von Pericarditis ohne Mediastinitis."—*Berliner klin. Wochenschrift*, 1883, Nr. 13.

cases above recorded was it ever observed, although it was repeatedly examined for, and although the veins were in each case much distended. In many of the recorded cases no mention is made of the presence or absence of this inspiratory swelling of the cervical veins, and in some (*e.g.*, Case 19) it is expressly stated that it was not present. We cannot, therefore, regard it as a constant sign, and one which is always present even in well-marked cases of mediastino-pericarditis. It has also been observed in an uncomplicated case of pericarditis exudativa (Boehr).[15]

It should, however, be stated that more importance in the diagnosis of mediastino-pericarditis has been attached by one observer to the presence of a pulsus paradoxus and to inspiratory distension of the right jugular vein. Schreiber[24] says that the true pulsus paradoxus, as seen in mediastino-pericarditis, differs materially from the inspiratory pulse intermission, which is met with physiologically and under various other conditions. The special peculiarities and characteristics of the pulsus paradoxus, as seen in cases of mediastino-pericarditis are, according to this observer, the following :—

(1) Marked diminution in the volume or complete disappearance of the pulse of all arteries, and not only of that of the radial vessels, during inspiration and especially in the second half or at the end of that act.

(2) The impossibility of replacing the paradoxical pulse by a full pulse by a prolonged and sustained inspiration.

[16] Gräffner. "Pulsus paradoxus bei eitriger Pericarditis und doppelseitiger Pneumonie."—*Berliner klin. Wochenschr.*, 1876, Nr. 27.

[17] Stricker. "Pulsus paradoxus bei Pericarditis tuberculosa, aber ohne Mediastinitis."—*Charité-Annalen*, II. (1875), 1877.

[18] Rosenbach (O.). "Mediastino-pericarditis."—*Real-Encyclopädie der gesammten Heilkunde* Bd. IX., S. 473.

[19] Riegel. "Krankheiten des Herzbeutels in "Gerhardt's Handbuch der Kinderkrankheiten."—Bd. IV., S. 251.

[20] Brockbank (E. M.). "Pulsus paradoxus in acute laryngitis."—*British Med. Journal*, June, 1893. Baumler. *Deutsch. Archiv. klin. Med.*, XIV., 455. Riegel. *Berliner klin. Woch.*, 1876, Nr. 47.

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[23] Summerbrodt. "Gegen die Lehre vom Pulsus paradoxus."—*Berliner klin. Wochenscht.*, 1877, Nr. 42." Riegel (F.). "Ueber die respiratorischen aenderungen des Pulses und den Pulsus paradoxus."—*Ibid.*, 1876, Nr. 26, also *Ibid.*, 1877, Nr. 45 ; and "Gerhardt's Handbuch der Kinderkrankheiten."—Bd. IV., S. 250.

[15] Boehr (E.), *ibid ;* and Rosenbach (O.), *Real-Encyclopädie der gesammten Heilkunde*, Bd. IX., s. 473.

[24] Schreiber (Julius). "Die wirkung des Veränderten Luftdrucks in den Lungen auf den Blutkreislauf des Menchen."—*Archiv für Experimentelle Pathologie und Pharmacologie*, Leipzig, 1880. Bd. XII., 2 Theil B., S. 184.

(3) The pulsus paradoxus does not require for its development the inspiration to be a deep one.

(4) The pulse intermission is accompanied by a regular action of the heart, and the heart shows

(5) No weakening of its action during the period whilst an inspiration is being made (4 and 5 might, however, be modified in cases where there is at the same time endo- or myocarditis.)

Schrieber concludes his reference to this point with the statement that in his opinion if the pulse presents the above-mentioned characteristic points, and especially if associated with them is inspiratory swelling of the cervical veins, that the condition of pulse is so distinctive and differs so much from similar pulse phenomena that it may be regarded as pathognomonic of indurative mediastino-pericarditis.

What are the exact conditions which favour the development of a paradoxical pulse in cases of indurative mediastino-pericarditis we do not at present know with certainty. The view put forward by Kussmaul that it was due to the dragging effect of the adhesions external to the pericardium on the aorta in the thorax will doubtless explain some of the cases, and where there is such marked narrowing of the arteries and veins in the thorax as existed in one of Kussmaul's cases (Case 7 in table), it is easy to conceive how an inspiratory act might produce both a paradoxical pulse and an inspiratory swelling of the cervical veins. But from the numerous observations, above mentioned, of the occurrence of a paradoxical pulse where there were no bands of adhesions in the chest, it is evident that other agents besides such adhesions are capable of producing that form of pulse. Cardiac weakness from many causes appears to have been a not infrequent condition present when a pulsus paradoxus was present, and it is quite probable that such heart weakness, in some way which at present is not clear to us, contributes to, if it is not the actual cause of, this peculiar form of pulse in some of the cases of mediastino-pericarditis, where the arrangement of the adhesious is not such as will account for a narrowing of the aorta or main arteries in the thorax.

The *duration* of cases of indurative mediastino-pericarditis appears to vary considerably. In reviewing the cases which have been published, it is often difficult to say exactly when the affection began. It is clear, however, that the period from the outset of those symptoms which may fairly be attributed to the affection, to the fatal termination, varies from a few months to several years. Probably the duration of the cases depends largely upon the extent and nature of the adhesions both outside and within the pericardium, and the effect which these have in producing cardiac dilatation and heart failure.

The *cause of death* in cases of indurative mediastino-pericarditis varies, but in the majority of cases it appears to be due to gradual cardiac dilatation and heart failure. Bronchitis and catarrhal pneumonia frequently assist in bringing about the fatal termination. Attacks of pleurisy appear to be very common in the course of the affection, and must assist in reducing the powers of resistance of the sufferer. In other cases we may have the developement of acute tuberculosis or the extension of a pre-existing phthisis as the principal cause in producing death.

Treatment.—There is little to be said on the question of treatment of cases of this affection. No special treatment can be of much avail, and the management of such cases is the same as that required for the majority of cases of cardiac dilatation produced by any other cause. Many of the symptoms and complications met with in cases of indurative mediastino-pericarditis are undoubtedly due to cardiac failure, and although we may frequently have to treat such symptoms and complications as they arise, our chief attention has to be directed to support and maintain the strength of the heart and to postpone as long as possible the onset of cardiac muscle failure.

The following references may be of help to any one interested in this subject :—

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