TUBERCULOUS PERICARDITIS.

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Tuberculosis follows hard upon rheumatic fever as a cause of pericarditis. The affection is usually overlooked clinically, and possibly in some cases anatomically. In 1000 autopsies, the majority of which were made at the Montreal General Hospital, there were 275 cases with carditis. The affection is usually overlooked clinically, and possibly in relative frequency may be gathered from the following figures, furnished by Dr. Welch's records at the Pathological Laboratory of the Johns Hopkins Hospital. Of instances of pericarditis, 6 were tuberculous. Of late years attention has been called to the frequency of its occurrence, and yet the records in the literature are not very numerous; thus, the Index-Catalogue contains only thirty references, and the Index Medicus to July, 1892, only twenty-seven. The Transactions of the Pathological Society of London include but five cases to date.

The following is a brief summary of the cases which have come under my observation in Montreal, Philadelphia, and Baltimore:

CASE I.—Female, aged forty-three years; sixteen ounces of dark exudate in pericardium; general miliary tuberculosis.

CASE II.—Male, aged five years; caseous masses and fresh villous exudate; miliary tubercles on the peritoneum and in lungs; bronchial and mediastinal glands caseous.

CASE III.—Female, aged thirty-eight years; chronic pulmonary tuberculosis; fresh tuberculous pericarditis.

CASE IV.—Male, aged sixty-four years; acute tuberculous pericarditis; acute tuberculous pleurisy; chronic pulmonary tuberculosis.

CASE V.—Male, aged fifty years; chronic pulmonary tuberculosis; recent tuberculosis pericarditis.

CASE VI.—Male, aged forty-five years; chronic pulmonary tuberculosis; chronic tuberculous pericarditis.

CASE VII.—Male, aged twenty-eight years; chronic pulmonary tuberculosis; tuberculosis of pleura; acute tuberculous pericarditis.

CASE VIII.—Mary B., aged thirty-six years; tuberculous pyelitis; fibro-caseous changes at the apices; sixteen ounces of turbid hemorrhagic serum in pericardium; thickening of the layers.

CASE IX.—John G., aged fifty years; tuberculous mediastinal abscess; tuberculous infiltration of parietal layer of pericardium; acute pericarditis.

CASE X.—Max S., aged forty-five years; primary tuberculosis of bowels; chronic tuberculous pericarditis, the membranes from three to four lines in thickness; bronchial glands caseous.

CASE XI.—Male, aged seventy-two years; death from pneumonia; heart hypertrophied; pericardium adherent, greatly thickened, and tuberculous; calcification of bronchial glands; no other tubercles in body.

CASE XII.—John T., aged sixteen years; tuberculosis cerebro-spinal meningitis; tuberculosis of mediastinal glands; old tuberculous pericarditis.

CASE XIII.—Moses B., aged twenty-four years; tuberculosis of mediastinal glands; chronic tuberculous pericarditis, dilatation of heart; a few tubercles in lung.

CASE XIV.—Wm. H. T., aged fifty-two years; dropsy; hypertrophy and dilatation of heart; chronic tuberculous pericarditis; fibroid tubercles in lungs; miliary tubercles in viscera.

CASE XV.—John P., aged thirty-eight years; dropsy and dyspnea; tuberculous pericarditis with effusion; tuberculous pleurisy; old foot in lungs.

CASE XVI.—Emma B., colored, aged thirty-nine years; dyspnea, cough, and anasarca; tuberculous pericarditis, with effusion; tuberculosis of mediastinal and bronchial glands; scattered tubercles in lungs, liver, and spleen.

CASE XVII.—John C., aged sixty-five years, admitted with cough, fever, and signs of consolidation at left base. Death on second day after admission. Chronic pulmonary tuberculosis; fresh tuberculous pneumonia; arterio-sclerosis; patch of recent tuberculous pericarditis.

ETIOLOGY.—Tuberculous pericarditis is not limited to any age. The youngest of my cases was a child of five years; the oldest a man of seventy-two years. Parrot, Duckworth, Rolleston and Letulle have reported cases in infants under a year. In Brackmann's Göttingen Thesis, of 66 cases collected from the literature 19 were in children. It does not seem to be at all uncommon in old men, and there are two cases on record in octogenarians. Males seem more prone to the disease than females; there were only four women on my list.

Tuberculous pericarditis is due in a majority of instances to infection of the membrane from caseous mediastinal lymph glands. The disease may be confined to these glands and to the pericardium; thus, in Case XI. of my series, the patient, an old man, aged seventy-two years, died of pneumonia after a short illness. There was no tuberculosis of the lungs or other viscera; the pericardium was thickened, both layers adherent, and presented cheesy masses and gray nodules. The heart was enlarged, weighing seventeen ounces. The mediastinal glands were calcified, particularly the bronchial group. Case IX. is of special interest, showing the mode of extension from the anterior mediastinum to the pericardium. The patient, a man aged fifty years, had pulmonary...
tuberculosis, and died of acute tuberculous pleurisy with sero-purulent-exudate. Upon the external layer of the pericardium, three and a half inches in extent, was a flattened tuberculous abscess with cheesy and purulent contents. The subjacent pericardium was much thickened, and was itself cheesy, and inflammation had extended through and produced an acute fibrinous inflammation of both layers with very little fluid exudation. Tubercles could not be seen. This association of disease of the mediastinal lymph glands with pericarditis has long been recognized. Zeiker laid special stress on it in his paper on the traction diverticula of the oesophagus, caused by diseased lymph glands, and of his 54 cases there were 9 instances of total and 1 of partial synechia of the pericardial layers. Of the 101 instances of disease of the lymph glands of the mediastinum, collected by Berényi, the glands were caseous in 18 cases in which the pericardium was involved. Kast, who quotes these figures, reports an instance (Virchow's Archiv, Bd. xvi.) of acute tuberculous pericarditis due to perforation of a purulent mediastinal lymph gland into the pericardial sac. In children the affection is in all probability most commonly transmitted in this way from mediastinal and bronchial glands. In Case II., a child aged five years, the bronchial glands were greatly enlarged, projected deeply into the lung tissue, and presented on section a uniform caseous appearance. Both layers of the pericardium were greatly thickened; their adjacent surfaces roughened and irregular; not covered with the usual false exudation, but presenting strands of firm tissue, and flat, slightly elevated caseous masses, which were yellow-white in color, and equally distributed on both layers. There were scattered miliary tubercles in the lung, on the peritoneum, in the kidneys, and in the liver, and these were in all probability entirely secondary to the advanced bronchial and pericardial disease.

A second, less common, mode of extension is from the pleura or from the lung. A very good illustration of this was recently met with in Case XVII., a man aged sixty-five years, who was admitted to ward F of the Johns Hopkins Hospital with acute tuberculous pneumonia, and died on the following day. There was old disease of the lungs, and both acute and chronic tuberculous pleurisy. The left lung was strongly adherent to the pericardium, the cavity of which contained a small amount of clear fluid. The inner surface of the parietal layer was smooth, and presented a number of oechymoses. At the superior reflection, where it was adherent to the underlying thickened and infiltrated pleura, there was an eruption of fresh gray miliary tubercles.

And, lastly, there are instances in which the pericardium appears to be involved with the pleura and peritoneum in a general tuberculosis of the serous membranes. In some of these cases the extension can be shown to have been directly from the pleura and pericardium into the peritoneum, while in others it would appear that the extension was from the peritoneum into the serous membranes of the thorax.

MORBID ANATOMY.—The picture is extremely varied. Practically there are two groups of cases: those with firm adhesions between the pericardial layers, usually with great thickening; and those with recent exudation, fibrinous, sero-fibrinous, hemorrhagic, or purulent. The cases with adhesions are the most numerous. Of the 17 cases in my series, 2 cases, Nos. IX. and XVII., may be excluded, as in the first there was only tuberculous infiltration of the parietal layer, and in the other an eruption of fresh miliary tubercles alone. Of the remaining 15 cases, in 8 only was there thickening of the layers without exudation. The statistics, however, of Rousseau (Paris Thesis, 1882) and Lancereaux show that the chronic adhesive form is most common. Of the 35 observations analyzed by the former, in 21 there was adherent pericardium; while in 12 of Lancereaux's 14 cases there were adhesions.

Both layers are, as a rule, uniformly thickened, and in the extreme instances it is impossible to separate them at any point. In other cases the process is more local, and the synechia may be limited to the front of the heart, leaving large portions of the base and of the left auricle free. The reflection of the pericardium at the great vessels and the adjacent mediastinal tissues may be uniformly infiltrated and the vessels surrounded by a solid mass. In Case X., "the layers of the pericardium were adherent and measured six to ten mm. in thickness, and showed groups of tubercles, many of which had fused together in uniform cheesy masses. The tubercles and caseous masses can be readily distinguished in each layer, since between them there is an infiltrated connective tissue which is free from tubercules. At the base of the heart the thickened pericardial layers formed a solid cheesy mass surrounding the aorta."

The membranes may reach a thickness of from three to ten millimeters, the increase being due to the growth in the leaves of tubercles, the development of caseous masses, and to the new growth of connective tissue. Frequently it can be seen that the two enormously thickened layers are united by a clear, infiltrated tissue, which may itself not present any tubercles. In the earlier stages of this process the membranes are little, if at all, thickened, the tubercles are seen just beneath the endothelial layer, and there may or may not be a fresh exudate of yellowish fibrin. In other instances the contiguous surfaces of the thickened layers are covered with flat, yellowish caseous masses, as noted in Case II. Collections of thick cheesy pus are occasionally found between the layers.

The condition of the heart in this chronic adhesive form is most interesting. As is usual in adherent pericardium, particularly when the layers are very thick, there is enlargement of the organ, which may
reach an extreme grade. In Case XIV, the heart with the thickened pericardial membranes weighed thirty-six ounces. At the time of death the cardiac muscle is more or less degenerated, and may show fatty or fibroid changes. The tuberculous process rarely invades the ventricular muscle, though the thin auricle may be much infiltrated, as in Case XV., in which the appendix was converted in great part into cheesy material, only a thin film of muscle substance remaining.

In the cases with effusion there may be—(a) a simple plastic exudate similar to that of ordinary rheumatic pericarditis, with little or no serous effusion, and with scarcely any thickening of the membrane, the eruption of villiary tubercles alone indicating the nature of the process. More commonly there is (b) extensive aero-fibrinous exudate, consisting of flakes of lymph and a turbid serum. When the process lasts for any length of time, the membranes, as in Cases XV. and XVI., may be very greatly thickened and the anatomical picture may resemble very closely that of a fatal case of rheumatic pericarditis. The tubercles, however, can be seen with distinctness in the membranes, and there are usually flat areas of cheesy infiltration beneath the fibrinous layers. (c) In some cases the exudate is hemorrhagic, as in Cases I. and VIII. of my series. The membranes here may be deeply engorged, and hemorrhagic foci may be seen in them. The color of the effusion may be bright red, but is more commonly a reddish-brown or chocolate color. The amount of the effusion may be large, ranging from 500 c.c. to 2000 or 3000 c.c. An instance of most extensive effusion has been reported by Musser, with whom, when his colleague at the Philadelphia Hospital, I had an opportunity of seeing the case. The patient, a colored man, aged twenty, was admitted to the Philadelphia Hospital on the fifth week of an illness characterized by cough, dyspnea, and irregular chills. There was extreme orthopnea. There was absolute dulness over the greater part of the left chest from the second rib in front and from the middle of the scapula behind. There was an area of modified tympany along the vertebral column in the infra-scapular region. Thirty-seven ounces of a bloody fluid were aspirated, presumably from the pleural cavity. A distinct friction sound was heard after the aspiration, and it was thought that he had both pleurisy and pericarditis. At the post-mortem the pericardial sac contained sixty-four ounces of bloody serum. There were tubercles in both layers of the pericardium, also in the left pleura. The bronchial glands were enlarged and caseous. And, lastly, (d) the effusion may be purulent, and this, too, apparently from the outset and not following paracenteris. The exudation may be enormous, and the cases have been diagnosticated as left-sided empyema. In Kast's case, already referred to, the inflammation was due to perforation of a softened mediastinal lymph gland into the pericardium. No instance of purulent exudate has come under my direct care.

Clinical history.—We may recognize four groups of cases.

First group: Latent tuberculous pericarditis. A considerable number of all the cases on record belong here. The disease is discovered accidentally in individuals who have died of other affections, or of chronic pulmonary tuberculosis. An interesting illustration of this was Case XI., a well-nourished old man of seventy-two years, who was admitted to my wards in the Philadelphia Hospital with pneumonia, of which he died. There was no suspicion whatever that the pericardium was involved. As already mentioned there was found an adherent, greatly thickened, tuberculous pericardium; calcification of the bronchial glands, but no tubercles in other parts of the body.

In Cases VI., XII., XIII., and XIV. the disease was also latent, and there was no suspicion of pericarditis during life.

Second group: With symptoms of cardiac insufficiency following the dilatation and hypertrophy consequent upon chronic adhesive pericarditis. The clinical features are really those of cardiac dropsy.

Case XIV.—W. H. T., aged fifty-two years, admitted to ward F of the Johns Hopkins Hospital, June 27th, 1889, complaining of shortness of breath, swelling of the legs, and incontinence of urine.

Family history good. Father died of acute pleurisy, mother of old age, two brothers died when young.

The patient had had scarlet fever, measles, and malaria; denies syphilis. Has had rheumatic pains, but has never been in bed with acute rheumatism. Has used tobacco freely, alcohol in moderation. He was well and strong until two years ago, when he had a "bilious attack," and was in bed three weeks. He was well last winter until February, when he began to be short of breath and had a cough, which has lasted until the present time. About four weeks ago the shortness of breath increased, and his feet became swollen.

Present condition: An emaciated man, with dry, harsh skin; legs and scrotum oedematous; abdomen not swollen. Pulse, 104, tension increased; temperature, 102°.

Heart: Apex beat faintly visible in the fourth interspace; palpable in the nipple line; feeble. Cardiac dulness begins as high as second left interspace, near sternum. To the right it extends 2.5 cm. beyond the sternum; there is no thrill. The sounds are feeble; the second louder, more marked than the first. At the apex the diastolic pause is shortened—the sounds succeed each other at equal intervals of time; the second, at the left margin of sternum, is reduplicated. Careful examinations of the heart on the 1st and 2d of July showed as a special feature the flatness extending into the second left interspace; the impulse was extremely feeble; the sounds were clear, and in the third and fourth interspaces quite loud; the second was accentuated. At the auricular the second was feeble. Lungs: Clear anteriorly; resonance defective at right base. There were numerous rales at base and cracking rales over the left mammary region.

Abdomen: Soft; liver and spleen normal.

Urine clear; no albumin, no casts; sp. gr., 1010.
The case was regarded as one of cardiac hypertrophy and dilatation without valve disease. He was given tincture of digitalis every four hours, and saline purges.

He failed rapidly; the heart became very feeble; Cheyne-Stokes breathing developed, and he died on the morning of the 5th.

On admission the temperature was 102°, but after this did not rise above 99°, except on the evening of the 30th.

**Abstract of autopsy (by Dr. Welch).** Peritoneum smooth. In the thorax there were extensive adhesions on both sides. The pericardial sac was obliterated. The heart occupied an unusually large area. The surface was everywhere covered by a yellowish-white opaque layer, consisting of firm connective tissue and caseous matter, containing, especially on the surface, numerous gray and cheesy milliary tubercles. These exudations averaged 1 cm. in thickness over the whole surface, the thickest being over the outer surface of the ventricle. The heart, including the thickened pericardium and the base of the aorta, weighed thirty-six ounces. The myocardium was pale yellowish-brown, soft, with mottled fatty degenerations beneath the endocardium. In the apex of the left ventricle there were a few parietal thrombi. The thickness of the wall of the left ventricle was 18 mm.; of the wall of the right, 6 mm. The cavities were dilated; the aortic valves slightly thickened, but competent. The mitral orifice admitted three fingers. The valvular segments were a little thickened. There were small gray tubercles on the endocardium of the right auricle about the orifice of the superior cava.

The lungs were edematous and deeply pigmented; they contained masses of gray, fibroid, not cheesy, tubercles.

There were abundant miliary tubercles on the omentum. The spleen contained a few milliary tubercles. The kidneys presented a number of opaque caseous masses, many in streaks. The liver also presented a few tubercles. In the intestines there were a few scattered milliary tubercles in mucous and submucous layers, but no ulcers. There were no tubercles in the brain.

In the chronic adhesive form of tuberculous, as of simple, pericarditis, the clinical features may be those of cardiac dropsy, and a diagnosis is made either of simple hypertrophy and dilatation of the heart when, as in the case just given, there are no special auscultatory signs, or of mitral insufficiency when there is at the apex a loud blowing murmur. The diagnosis of adherent pericardium, always uncertain, is doubly so in cases admitted with dyspnea, dropsy, and the signs of cardiac dilatation, since under these circumstances it is almost impossible to make a satisfactory physical examination.

**Third group:** Acute tuberculosis. The clinical picture may be that of an acute tuberculosis, either general or with cerebro-spinal manifestations. The following is a good illustration of an acute miliary tuberculosis, the primary disease being in all probability in the mediastinal glands and pericardium:

**CASE XIII.**—Moses B., aged twenty-four years, admitted to ward F of Johns Hopkins Hospital, January 25, 1890, complaining of cough and loss of strength. Owing to mental dulness and apathy it was difficult to get any satisfactory information from the patient as to his family or personal history. He states that his present illness began only three weeks ago, though he has had a slight cough all winter. Since the onset of the illness he has been in bed and has had fever, cough, and night-sweats. There has been loss of appetite and great weakness.

**Present condition:** Temperature, 100.8°. Patient looks very heavy and dull; there are sordes on the lips, and the tongue is covered with a brownish fur. The skin shows traces of a squamous erythematous and there are well-marked nodes on the skin. The respiration is 37. The expectoration is muco-purulent and is at times blood-tinged. The physical examination of the lungs shows clear percussion resonance, the note perhaps a little higher in pitch at both bases behind. Anteriorly there are numerous piping and moist rales on the left side below the third rib; behind, the rales are universal.

Heart: Apex beat in fifth interspace below the nipple. The area of dulness does not appear to be increased. The first sound is very feeble and muffled at the apex, and possibly accompanied with a soft murmur. The second sound is well heard at the base, and both sounds are unusually loud to the right of the sternum in the sixth intercostal space. The examination of the abdomen is negative.

During the three days the patient was in the hospital there was irregular fever (from 101° to 103°), increasing debility, with hurried respirations, tremor of the extremities, and gradual failure of the heart. The blood-cells were so numerous with inspiration and expiration that it was impossible to hear the heart sounds. The examination of the sputum was negative. The urine was dark-yellow in color, acid, trace of albumin, and there were several casts seen. The diagnosis of acute tuberculosis was made.

**Autopsy (by Dr. Welch).** Abstract: Body of a large, strongly built, well-nourished, muscular man; macular eruption present on the skin; large node on the left tibia.

No special changes in the brain.

The deep cervical glands slightly enlarged, containing numerous small tubercles and caseous areas. Extensive pleural adhesions on both sides. Both layers of the pericardium were adherent over the entire heart, greatly thickened, and contained in the membranes numerous milliary tubercles. The anterior mediastinal lymph glands were enlarged and presented numerous milliary tubercles and caseous masses. The heart was enlarged and dilated. The length of the left ventricle from apex to edge of aortic valve, 15 cm.; thickness of wall of left ventricle, 16 mm.; the columns carneae in the left ventricle were extremely prominent.

The lungs were congested throughout, edematous, and contained scattered tubercles surrounded by areas of pneumonia. No caseation except in the tubercles themselves. The mesenteric glands were enlarged and those near the spleen were caseous. The kidneys showed much fatty degeneration in the convoluted tubules.

In the following case the tuberculosis was chiefly manifested in the cerebro-spinal meninges, and the clinical picture was that of ordinary tuberculous meningitis:

**CASE XIV.**—John T., aged sixteen years, admitted to ward F of Johns Hopkins Hospital, November 5, 1890, complaining of pain in back and head. Patient is well-nourished, not emaciated.
Osler: Tuberculous Pericarditis.

His mother is dead; father, five brothers, and six sisters living and well; does not know of what his mother died; says he has been ill for three weeks with headaches, tired feelings, pains over the body. He has been obstinately constipated for ten days; has been in bed for two weeks; says he had a chill a week ago. When admitted he looked very ill; temperature, 95°. After having been in bed for two hours with hot bottles to his feet, temperature rose to 98°. He passed a quiet night; temperature rose to 101° at 8 P.M.; pulse, 72.

On the 6th, the examination revealed the following: Tongue heavily coated, white. Patient is rational, answers questions well, but shows a great dislike to be moved in bed; lies on the left side with the head a little drawn back, and when an attempt is made to move him he resists, saying that it is painful. There is no paralysis; pupils are of medium size and active. Abdomen not swollen, not tender. Spleen not enlarged. Heart sounds are clear at apex and base. Examination of lungs negative.

On the 7th and 8th he was much worse; became apathetic; rambled; the temperature ranged from 97° to 102°. Heart was examined again on the 8th, and the note reads: "Sounds at apex and base are quite clear."

On the 9th and 10th the meningeal symptoms were more pronounced; he became unconscious and the discharges were passed involuntarily. At times the arms and legs seemed a little stiff, resisting flexion. Pupils moderately dilated; convergent squint in left eye; the retinal veins slightly distended and engorged; no swelling of the disks, but the physiological cupping was absent.

On the 11th there was a good deal of rigidity of the arms and of neck. Abdomen retracted. Death took place on the morning of the 12th.

The case was regarded as one of meningitis, probably tuberculous. There were no features calling special attention to the heart.

Autopsy (by Dr. Welch). Abstract: In thorax, glands at root of neck and in mediastinum enlarged and caseous. A very large caseous gland lay just beneath the esophagus cartilage. One was closely adherent to the pericardium. Some of these glands were full of miliary tubercles. The external surface of the pericardium was thickly studded with small tubercles. The two layers of pericardium were adherent, and between them was a mass of partly caseous, partly gelatinous-looking tissue from two to fourteen millimetres in thickness. Over the left ventricle was a small cavity filled with cheesy pus. The heart weighed 542 grammes. The muscle substance was firm. Wall of left ventricle, 14 mm.; of right, 3 mm. Valves were normal.

The lungs presented numerous irregular areas of hemorrhage, chiefly beneath the pleura. There were no tubercles. On the pleural surface of the diaphragm, extending from the pericardium on the right side, there were a few miliary tubercles. No special changes in liver, spleen, and kidneys. The brain and spinal cord presented a typical picture of tuberculous meningitis.

Fourth group: Cases with symptoms of acute pericarditis. This group, the most important in many respects, includes cases in which the pericarditis is acute and accompanied with more or less exudation of a sero-fibrinous, hemorrhagic, or purulent character. Here, too, the process may be latent, as in Case VII., a young man aged twenty-eight years, who died under my care, of hemoptysis. There was chronic tuberculosis of both lungs with extensive pleural adhesions.

"The layers of the pericardium were united by soft adhesions, which could be readily torn through. On each membrane were innumerable small granulations, and here and there a nodular tubercle from one to two millimetres in diameter." This case is of special interest, as it shows the first stages of the process which ultimately causes enormous thickening of the pericardial membranes with universal adhesions. The tubercles, when small, may be readily overlooked. Acute plastic pericarditis in chronic tuberculosis is not, however, always due to the eruption of miliary tubercles. In two recent autopsies on patients with chronic pulmonary tuberculosis dying in my wards there was simple pericarditis without a trace of tubercles, and with little or no exudation.

The following case, which was admitted to ward E under the care of Dr. Thayer in my absence, illustrates the mode of onset and the clinical features of a tuberculous pericarditis which came on in a strong, well-developed, muscular man, and proved fatal within three weeks:

Case XV.—John P., aged thirty-eight years, admits August 18th, with swelling of the legs and dyspnea. The family history is good. He has been, as a rule, healthy, though in his childhood and youth he had many of the infectious diseases. He has been a moderate drinker. He denies syphilis. The present illness began about two weeks ago with pain in the left shoulder and about the heart. Feet began to swell ten days ago, and he has had cough and shortness of breath for about the same length of time. He has had no nausea; his appetite has been fairly good. Within the past few days he has become much worse.

On admission the patient had intense orthopneea; pulse 130, but moderately full. He had a distressing cough, with clear watery expectoration. There was great edema of the lower extremities and of the scrotum. The finger-tips and mucous membranes were bluish in color. In the examination of the heart at the time no murmur could be detected, but the second sound was accentuated at the pulmonary carilage.

On the following morning the patient was quiet; respirations 28 to the minute; pulse 84, the beats irregular both in rhythm and force, the volume fair, and tension not diminished.

Thorax apparently symmetrical, but the manubrium very prominent, expansion equal. In front, resonance on both sides good, though on the left side flatness begins at the fifth rib midway between the nipple and axillary line, and the dulness seems here to be somewhat movable. There are numerous sibilant and sonorous rales to be heard in front. Passing down the left side and into the axilla, the breath-sounds become more feeble and fine moist rales are heard. Pitch of resonance at the extreme left base is higher than at the right, and the vocal resonance is somewhat diminished.

On auscultation there are numerous coarse and medium fine rales to be heard at both bases. At the extreme left base the respiratory murmur is almost absent.
Heart: Point of maximum impulse difficult to localize; slight general heaving over the whole precordial area; relative dulness begins above at second rib and extends well to the right of the sternum. The prominence above noted is just at the junction of the manubrium and gladiolus, and just at this point there is dulness over a very limited area. Relative dulness extends outward to a point nearly 5 cm. outside the nipple. (Chart I.) The heart-sounds are heard with the greatest intensity at the fifth space a little inside of the nipple line. There the first sound is full and booming; the second not so loud. Passing inward toward the sternum the sounds become associated with a superficial, soft, squeaking sound, diastolic in time, heard loudest over the mid sternum, and pericardial in character. This is heard faintly all inward toward the sternum the sounds become associated with a superficial, soft, squeaking sound, diastolic in time, heard loudest over the mid sternum, and pericardial in character. This is heard faintly all.

The examination of the abdominal organs is negative. The urine is yellow-colored, acid, sp. gr. 1024, distinct trace of albumin; several hyaline casts were found. Throughout the 20th and 21st the patient remained in much the same condition. On the 22d there was a slight rise in temperature to 100.5°; the pulse varied greatly in rate and character; at noon was slow, regular, and full, from 70 to 90 per minute, and again was as rapid as 140. The patient, in many respects, was better. The cedema of the legs had disappeared. The urine had increased in quantity. On the 19th only 180 c.c., and on the 20th 350 c.c. had been passed. On the 21st and 22d the amounts were 700 and 1100 c.c.

23d. The temperature has been between 97° and 98°; at the morning visit the pulse was 148, regular in force and rhythm; the respirations 32. The patient was lying quietly on left side. The physical signs practically those noted above with the exception that there is an extension of the cedema at the bases of the lung. The patient died suddenly at 4.30 P.M. to-day.

Autopsy (by Dr. Flexner). Large, muscular man; moderate cedema of the legs and of the subcutaneous tissue of trunk. Fat well retained, both beneath skin and in omentum and mesentery.

Pericardium is adherent to the left pleura; the sac thickened and contains a considerable amount of clear serum. Both layers are covered with a thick fibrous deposit, looking like a hairy coat. The thickness of the pericardium over the heart is 5 mm. When incised the thickened layer is grayish in color, with many opaque or yellowish points scattered here and there, often continuous, and having the well-recognized characteristics of tubercles of this membrane. The heart was greatly enlarged. The valves were normal. The thickness of the left ventricle was 17 mm.; length of ventricle, 84 cm.; mitral orifice, 104 cm. in circumference. Thickness of right ventricle, 6 mm.; tricuspid orifice, 12 cm. in circumference. The walls of the auricular appendix are almost completely converted into a grayish-white material with only a thin internal film which appears like muscle. In the endocardium of the left ventricle are numerous exchymoses. There are also a few small ones on the right ventricle and in the auricles, and on the endocardium of the auricles are a few small, round, whitish miliary tubercles.

The left pleura is much thickened; parietal and costal layers adherent in places, but where not in actual contact there is clear serum between them. The costal pleura strips up with difficulty, and is very hard and cuts like cartilage. The diaphragmatic pleura is especially thickened, and on section it is seen to be composed of a dense, almost cartilaginous, grayish tissue, containing yellow, opaque, caseous masses. The apex of the left lung is retracted, hard to the touch, and on section it is seen to be composed of a dense, deeply pigmented connective tissue, and old areas of caseation; no calcification. There are a few small foci of miliary tubercles, and scattered fibrous tubercles elsewhere in the lung.

The right lung is voluminous, and in the greater part of its extent free from adhesions, but the pleural surfaces present numerous grayish-white elevated masses, single and conglomerate, which can be scraped off with difficulty. The costal pleura presents similar tubercles. About these there are, here and there, fresh fibrin. In the apex the upper lobe presents a few foci of fibrous miliary tubercles.

The spleen contains a few scattered tubercles. Nothing of note in the abdominal viscera; the intestines did not present tubercles.

Here there was no suspicion before death that the process was tuberculous, nor in reality was there any clew to indicate that this was the possible nature of the trouble.

The following instance is the only one which has come under my
admission, the note made by Dr. Thayer was as follows: Rather sparely nourished woman; dorsal decubitus; lips and mucous membranes somewhat pale; tongue slightly coated; respirations 30 per minute; pulse 104, regular in force and rhythm, tension not increased; temperature on admission 98.2°.

Expansion equal on both sides of the thorax. Percussion everywhere clear. On auscultation the only abnormal signs are fine moist rales during inspiration at both apexes.

Heart: There is slight heaving in the cardiac area; the point of maximum impulse is hard to determine. The area of cardiac dulness, as indicated on Chart II, begins about the second rib and extends far to the right of the sternum. Flatness begins at the third. The general outline of the area as indicated is triangular, and it extends to the left, far down into the seventh interspace. This flatness in the cardiac region is distinctly movable.

On auscultation the sounds are feeble at the apex, become louder in the fourth and fifth interspaces, and are still more distinct at the base, where the second aortic sound is accentuated.

Abdomen: The abdomen is full, generally tympanitic, and the walls are a little tense. The liver flatness begins at the seventh rib in the nipple line, and extends five fingers' breadth below the costal margin. The edge is not distinctly palpable.

The spleen is not palpable.

There is now no swelling of the legs, but she states that they were swollen four days ago.

The urine was cloudy, acid, sp. gr. 1012; slight trace of albumin, no sugar, numerous leukocytes.

During the first ten days in hospital the patient improved. The temperature chart shows a great irregularity. The morning register was at 98° or 98.6°, and in the afternoon, between four and six o'clock, rising to 101°, sometimes to 103°. An ice-bag was placed over the heart and she was given stimulants. For the second ten days, from July 29th to August 8th, there was distinct improvement; the temperature was lower and only once reached 102°. On August 1st it was noted that the area of dulness was diminished, and there was a friction rub heard in the mid-sternal line—synchronous, however, with the respiratory, not with the cardiac movements. On the 3d it was noted that there was a well-marked rubbing friction heard in the second and third left interspaces, and heard as far out as the nipple line and over the sternum adjacent to these spaces. This was the first time since her admission that there was a well-marked friction sound. The pulse has been very variable, ranging from 98 to 112. The patient is brighter and seems to be improving.

Between August 8th and 20th the irregular fever persisted, and on several occasions rose in the afternoon to 103°. She complained at times of pain in the precordial region and of general abdominal pain. The pulse has ranged from 100 to 125. The note on the 17th was as follows: "There is moderate heaving over the lower sternum and the epigastrium; the point of maximum impulse is rather difficult to determine. Relative dulness begins at the second rib and extends well to the right of the sternum. The absolute flatness, however, scarcely exceeds the normal limit. The heart-sounds heard at the outer limit of the dulness are very feeble, and the first is followed by a short systolic puff. Passing inward the murmur becomes louder, and it disappears above the fourth rib. In
the fever has persisted, she has, curiously enough, steadily gained in
weight; thus on August 8th she weighed 104 pounds; on the 16th the
same; on the 29th, 109 pounds; September 5th, 110; and September
13th, 114 pounds.

September 28th. The abdomen has become more swollen. On exa-
nimation there was distinctly movable dulness, though fluctuation could
not be readily obtained. There was tenderness in the region of the liver,
but no nodular masses could be felt. The friction murmur up and down
the sternum was more marked, and there was also a friction sound.
On inspiration a creaking, leathery, friction sound. The physical signs in the lungs
show increase in the rales which are heard over the whole of the right
back. The tactile fremitus is a little increased, and resonance is defe-
cutive at the right base. There is no pleural friction.

On the 29th it was noted particularly that the pitch of the percussion
toward the outer side between the scapula and the axilla was higher, but
without any trace of tympanitic quality. The pericardial murmurs are
loud. During the past week the patient has been worse. There is more pain in the epigastic region, the cough is very worrying at night, and
there is increasing edema about the legs and the skin of the back.

At her own desire the patient was taken to her home on September
27th.

Up to September 1st the patient had been under the care of my
first assistant, Dr. Thayer, and the case was regarded as one of pericar-
ditis with effusion, and when he left for his vacation she seemed to be
improving. During the time she was under my care I made her the
subject of several very careful examinations, and repeatedly demon-
strated the condition to the class of graduate students, and discussed
frequently the probable nature of the trouble. The persistence of the
fever and the marked involvement of the lungs suggested to my mind
the possibility of the existence of tuberculosis. We could not, however,
for some time obtain any sputa for examination, but after September
1st, on several occasions she brought up a muco-purulent expectoration, which was very thoroughly examined
with negative results. Notwithstanding, it seemed to me that the case was
unlike any ordinary rheumatic or septic pericarditis, and I felt
justified in dictating, the last morning I saw her, "that the protracted
course of the illness, the signs of pulmonary trouble, and swelling of the
abdomen, make it probable that the patient has tuberculosis." Three days after her return home she died, and we were fortunate
enough to secure an examination, which was kindly made by Dr. Flex-
ner. The following is an abstract of his report:

Autopsy. There was a considerable quantity of clear serum in the
peritoneum, both layers of which were smooth. There were no
pleural adhesions, no tubercles on either layer. The parietal pericardial
space uncovered by lung unusually large, measuring 15 by 20 cm. The
papetal pericardium is everywhere free. The outer surface shows
numerous white elevations, many of which are confluent and more
massed in some places than in others. On incision the layer is greatly
thickened, in places as much as 6 mm. About 300 c.c. of turbid
serum escaped. The visceral layer was everywhere covered with
flaky, yellowish-white fibrin. On section this layer is greatly thick-
ened and contains numerous yellowish, caseous masses; thus the
thickness of the wall of the left ventricle is 3 cm., nearly one-half
of which is the thickened pericardium. At the root of the aorta are
masses of caseous glands, adherent to the pericardium. The weight of the
heart with the thickened sac was 1110 grammes. The chambers
were dilated. There was no valvular disease.

The lungs were voluminous, contained many scattered and conglom-
erate tubercles, many of which were just beneath the pleura. There
was diffuse bronchitis, but there were no cavitities. The bronchial glands
were caseous. Liver, spleen, and kidney contained tubercles; those in
the spleen were large and caseous. There were a few small tuberculosis
ulcers in the small intestines. The mesenteric glands and the glands
about the pancreas were caseous.

Diagnosis.—The diagnosis of tuberculous pericarditis is extremely
uncertain. In the large group of cases in which the membranes are
thickened and united, the difficulties are those which pertain to the
recognition of adherent pericardium, difficulties which are enormously
enhanced by the state of cardiac insufficiency with which these cases
usually come under observation for the first time. In children with a
history of repeated attacks of rheumatism, the bulging precordium, sys-
tolic retraction at the apex, the fixation of the upper limit of cardiac
dulness, and the diastolic rebound, speak for adherent pericardium; and
if in a case of this sort there has been no history of rheumatism, and
if, on the other hand, there are indications elsewhere of tuberculosis, a
probable diagnosis may be made. In the cases which set in as acute
pericarditis, unless there are evidences of tuberculosis in other parts, as,
for instance, in the left pleura or in the peritoneum, or there are signs
of local disease in the lung and tubercle bacilli have been found in the
expectoration, the diagnosis can rarely be made. The effusion may be
equally as great in tuberculous as in rheumatic pericarditis. If para-
centesis be performed, the presence of a bloody exudate is decidedly in
favor of tuberculosis; once, at least, tubercle bacilli have been found
(Kast). The clinical features themselves offer no criteria, though
it would seem probable that in the acute cases with sero-fibrinous
exudation the course is more protracted and the fever more irregular
than in the ordinary forms of pericarditis; and in such a case, as in
XVI. in my series, the development of diffuse signs in the lungs may
lead to a strong suspicion that the process is tuberculous.

TREATMENT.—It is not improbable that tuberculosis of the peri-
cardium may, as a similar process in the peritoneum, recover com-
pletely. Possibly some of the cases of simple adherent pericardium
are instances of healed tuberculosis. The chronic adhesive form persists
in all likelihood for years, producing few if any symptoms until the
compensation fails in the hypertrophied and dilated heart.

It is highly probable that a majority of cases which terminate in
general synechia of the membranes present no clinical features; the
process is slow, insidious, essentially chronic, and not associated with
definite symptoms. A case which has set in acutely must be dealt with
as any other form of pericarditis, the indications being, first, to limit, if
possible, the intensity of the inflammation; and, secondly, to prevent the
evil consequences of the presence of a large amount of fluid in the sac.

We have no medicinal agents at our command which have any positive
influence in controlling the ordinary inflammation of serous membranes.
In Guy's Hospital Reports of a year or two ago, ...ropos of the treatment
of pericarditis, there is a story told of Sir William Gull which is worth
quoting in this connection. "He once met a practitioner on a case of
rheumatism, in which he detected a pericardial rub. He said nothing
of this to the patient's friends, but approved the general treatment, and
they came away together. 'Oh, Dr. Gull, it was very good of you not
to let them see I had made that dreadful oversight. I cannot think
how I can possibly have failed to detect the pericarditis.' 'Never
mind,' said Gull, 'it is just as well; for if you had detected it, perhaps
you might have treated it.'" There is one measure in the utility of
which we may have great confidence, namely, the ice-bag applied con-
tinuously over the precordium. It allays the pain when present, and
appears to check the tendency to effusion, while under its use an exudate
may be absorbed with rapidity. It is very much to be preferred to
blister or the thermo-cautery. In some instances the patients com-
plain very much of the intensity of the cold of the ice-bag, and in such
I was in the habit, in Philadelphia, of using Leiter's coil, through which
the water flowed continuously, and it could be arranged to have any
temperature thought necessary.

A second indication holds good in tuberculous as in other forms of