CHAPTER XII.

DISEASES OF THE PERICARDIUM.

I. Pericarditis.

Three forms are recognized clinically:

(1) Plastic, dry, or fibrinous pericarditis.

(2) Pericarditis with effusion (serous or purulent).

(3) Pericardial adhesions or adherent pericardium.

Fibrinous pericarditis may be fully developed without giving rise to any physical signs that can be appreciated during life. In several cases of pneumonia in which I suspected that pericarditis might be present, I have listened most carefully for evidences of the disease and been unable to discover any; yet at autopsy it was found fully developed—the typical shaggy heart. We have every reason to believe, therefore, that pericarditis is frequently present but unrecognized, especially in pneumonia and in the rheumatic attacks of children. On the other hand, it may give rise to very marked signs which are the result of—

(*a*) The rubbing of the roughened pericardial surfaces against one another when set in motion by the cardiac contractions.

(*b*) The presence of fluid in the pericardial sac.

(*c*) The interference with cardiac contractions brought about by obliteration of the pericardial sac together with the results of adhesions between the pericardium and the surrounding structures.

(1) Dry or Fibrinous Pericarditis.

The diagnosis rests upon a single physical sign—"*pericardial friction*"—which is usually to be appreciated by auscultation alone, but may occasionally be felt as well. Characteristic pericardial friction is a rough, irregular, grating or shuffling sound which occurs irregularly and interruptedly during the larger part of each cardiac cycle. It is almost never accurately synchronous either with systole or diastole, but *overlaps* the cardiac sounds, and encroaches upon the pauses in the heart cycle. It is seldom exactly the same in any two successive cardiac cycles and differs thereby from sounds produced within the heart itself. Pericardial friction seems very near to the ear and may often be increased by pressure

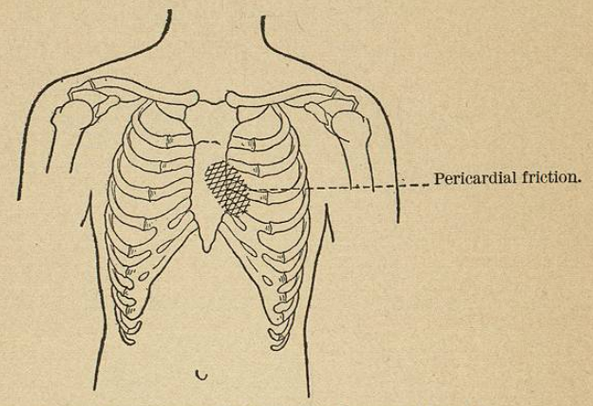

Fig. 147.—Showing Most Frequent Site of Audible Pericardial Friction.

with the stethoscope; it is not materially influenced by the respiratory movements.

It is best heard in the majority of cases in the position shown in Fig. 147; that is, over that portion of the heart which lies nearest to the chest wall and is not covered by the margins of the lungs; but not infrequently it may be heard at the base of the heart or over the whole precordial region. The sounds are fainter if the patient lies on the right side, and sometimes intensified if, while sitting or standing, he leans forward and toward the left, so as to bring the heart into closer apposition with the chest wall.

Pericardial friction sounds often change rapidly from hour to hour, and may disappear and reappear in the course of a day.

In rare cases the friction may occur only during systole or only during diastole. In such cases the diagnosis between pericardial and intracardial sounds may be very difficult.

Differential Diagnosis.

(*a*) *Pleuro-Pericardial Friction.*

Fibrinous inflammation affecting that part of the pleura which overlaps the heart may give rise to sounds altogether indistinguishable from those of true pericardial friction when the inflamed pleural surfaces are made to grate against one another by the movements of the heart. Such sounds are sometimes increased in intensity during forced respiration and disappear at the end of expiration, while true pericardial friction is usually best heard if the breath is held at the end of expiration. If a friction sound heard in the pericardial region ceases altogether when the breath is held, we may be sure that it is produced in the pleura and not in the pericardium, but in many cases the diagnosis cannot be made correctly.

(*b*) *Intracardiac Murmurs.*

From murmurs due to valvular disease of the heart, pericardial friction can usually be distinguished by the fact that the sounds to which it gives rise do not accurately correspond either with systole or diastole, and do not occupy constantly any one portion of either of these periods. Cardiac murmurs are more regular, seem less superficial, and vary less with position and from hour to hour. Pressure with the stethoscope does not increase so considerably the intensity of intracardiac murmurs. When endocarditis and pericarditis occur simultaneously, it may be very difficult to distinguish the two sets of sounds thus produced. The pericardial friction is usually recognized with comparatively little difficulty, but it is hard to make sure whether in addition we hear endocardial murmurs as well.

(2) Pericardial Effusion.

Following the fibrinous exudation, which roughens the pericardial surface and produces the friction sounds just described, serum may accumulate in the pericardial sac. Its quantity may exceed but slightly the amount of fluid normally present in the pericardium, or may be so great as to embarrass the cardiac movements and finally to arrest them altogether. In chronic (usually tuberculous) cases, the pericardium may become stretched so as to hold a quart or more without seriously interfering with the heart's action, while a much smaller quantity, if effused so rapidly that the pericardium has no time to accommodate itself by stretching, will prove rapidly fatal.

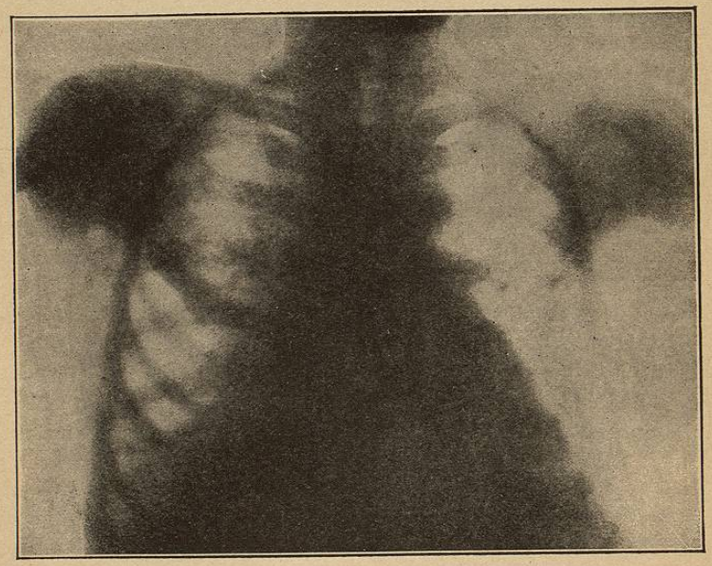

Fig. 148.—Pericardial Effusion, Cardio-hepatic Angle obtuse. (From v. Ziemssen's Atlas.)

Hydropericardium denotes a dropsy of the pericardium occurring by transudation as part of a general dropsy in cases of renal disease or cardial weakness. The physical signs to which it gives rise do not differ from those of an inflammatory effusion, and, accordingly, all that is said of the latter in the following section may be taken as equally an account of the signs of hydropericardium.

Hæmopericardium, or blood in the pericardial sac, due to stabs or to ruptures of the heart, is usually so rapidly fatal that no physical signs are recognizable.

Physical Signs of Pericardial Effusion.

In most cases a pericardial friction rub has been observed prior to the time of the fluid accumulation. The presence of fluid in the pericardial sac is shown chiefly in three ways:

(1) By *percussion*, which demonstrates an area of dulness more or less characteristic (see below).

(2) By *auscultation*, which may reveal an unexpected feebleness in the heart sounds when compared with the power shown in the radial pulse.

(3) By the signs and symptoms of *pressure* exerted by the pericardial effusion upon surrounding structures.

Bulging of the precordia is occasionally to be seen in children; in adults we sometimes observe a flattening of the interspaces just to the right of the sternum between the third and sixth ribs.

(1) *The Area of Percussion Dulness.*—The extent of the dull area depends not only on the size of the effusion and the position of the patient, but also on the amount of "give" in the pericardium and in the lungs as well as on the size of the lingula pulmonalis. Allowing for these uncertain factors, we may say: (*a*) One of the most characteristic points is the unusual[1] extension of the percussion dulness a considerable distance to the left and beyond the cardiac impulse. (*b*) Next to this, it is important to notice a change in the angle made by the junction of the horizontal line corresponding to the upper limit of hepatic dulness and the nearly perpendicular line corresponding to the right border of the heart. In health this cardio-hepatic angle is approximately a right angle; in pericardial effusion it is much more obtuse (see Fig. 149). Rotch has called attention to the importance of dulness in the fifth right intercostal space as a sign of pericardial effusion, but a similar dulness may be produced by enlargement of the liver.

[1] In health the cardiac dulness extends about three-fourths of an inch beyond the cardiac impulse, but in pericardial effusion the difference is greater.

Except for the two points mentioned above (the unusual extension of the dulness to the left of the cardiac impulse and the blunting of the cardio-hepatic angle), there seems to me to be nothing characteristic about the area of dulness produced by pericardial effusion.

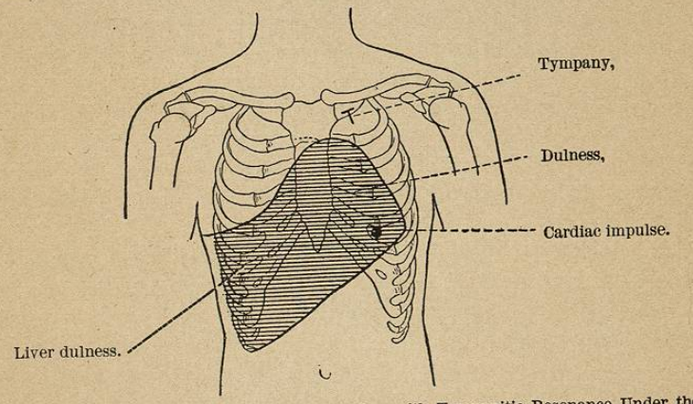

FIG. 149.—Percussion Dulness in Pericardial Effusion, with Tympanitic Resonance Under the Left Clavicle.

The "pear-shaped" or triangular area of percussion dulness mentioned by many writers has not been present in cases which have come under my observation. In large effusions percussion resonance may be diminished in the left back, and under the left clavicle the percussion note may be tympanitic from relaxation of the lung. Traube's semilunar space may be obliterated, but this occurs also in pleuritic effusions.

In some cases the area of dulness may be modified by change in the patient's position. After marking out the area of percussion

dulness with the patient in the upright position, let him lie upon his right side. The right border of the area of dulness will sometimes move considerably farther to the right. A dilated heart can be made to shift in a similar way, but to a lesser extent. Comparatively little change takes place if the patient lies on his left side, and no important information is elicited by placing him flat on his back or by getting him to lean forward.

Unfortunately, it is only with moderate-sized effusions occurring in a pericardial sac free from adhesions to the surrounding parts that this shifting can be made out. Large effusions may not shift appreciably, and less than 150 c.c. of fluid probably cannot be recognized by this or by any other method. But with large effusions the lateral extension of the area of dulness may be so great as to be almost distinctive in itself, *i.e.*, from the middle of the left axilla nearly to the right nipple.

(2) Feebleness of the heart sounds and of the apex impulse is of diagnostic importance only when it gradually takes the place of the normal phenomena as one watches the heart from day to day. Under these conditions they have some confirmatory value in the diagnosis of pericardial effusion.

Broncho-vesicular breathing with increased voice sounds may be heard over the tympanitic area below the left clavicle and occasionally between the scapulæ behind. This is a result of compression of the lung.

(3) Pressure exerted by the pericardial exudation upon surrounding structures may give rise to dyspnœa, especially of a paroxysmal type, to dysphagia, to aphonia, and to an irritating cough. The "paradoxical pulse," small and feeble during inspiration, is occasionally to be seen, but is by no means peculiar to this condition and has no considerable diagnostic importance.

(4) *Inspection and palpation* usually help us very little, but two points are occasionally demonstrable by these methods:

(*a*) A smoothing out of the intercostal depression in the precordial region, especially near the right border of the sternum between the third and the sixth ribs.

(*b*) A progressive diminution of the intensity of the apex impulse until it may be altogether lost. If this change occurs while

the patient is under observation, and especially if the apex impulse *reappears* or becomes more distinct when the patient lies on the right side, it is of considerable diagnostic value. In conditions other than pericardial effusion, the apex impulse becomes *less* visible in the right-sided decubitus.

Differential Diagnosis.

(1) Our chief difficulty is to distinguish the disease from hypertrophy and dilatation of the heart. In the latter, which often complicates acute articular rheumatism with or without plastic pericarditis, the apex impulse is often very indistinct to sight and touch as in pericardial effusion. But the area of dulness is less likely to extend beyond the apex impulse to the left or to modify the cardio-hepatic angle, or to shift when the patient lies on the right side. Pressure symptoms are absent, and there are no areas of broncho-vesicular breathing with tympanitic resonance under the left clavicle or in the axilla. Yet not infrequently these differentiæ do not serve us, and the diagnosis can be made only by puncture.

(2) I have twice known cases of encapsulated empyema mistaken for pericardial effusion. In one case a needle introduced in the fifth intercostal space below the nipple drew pus from what turned out later to be a localized purulent pleurisy, but the diagnosis was not made until a rib had been removed and the region thoroughly explored. It is not rare for pleuritic effusions to gather first in this situation, viz., just outside the apex impulse in the left axilla.

Such effusions may gravitate very slowly to the bottom of the pleural cavity or may become encapsulated and remain in their original and very deceptive position. In such cases the signs of compression of the left lung are similar to those produced by a pericardial effusion, and the results of punctures may be equivocal as in the case just mentioned. If there is *any* dulness, even a very narrow zone, in the left axilla between the fifth and eighth ribs, though there be none in the back, the likelihood of pleurisy should be suggested.

As between pleuritic and pericardial effusion the presence of a

good pulse and the absence of marked dyspnœa favors the former. In the two cases above referred to in which encapsulated pleurisy was mistaken for pericarditis, the general condition of the patient struck me at the time as surprisingly good for pericarditis.

If *both* pleurisy and pericarditis are present, the area of pericardial dulness is not characteristic until the pleuritic fluid has been drawn off. The persistence of dulness in the cardio-hepatic angle and beyond the apex beat after a left pleurisy has been emptied by tapping, and after the heart has had time to return to its normal position, should make us suspect a pericardial effusion.

Despite the utmost care and thoroughness in physical examination, many cases of pericardial effusion go unrecognized, especially in infants, in elderly persons, or when the lung borders are adherent to the pericardium or to the chest wall.

In the rheumatic attacks of children, it should be remembered that pericarditis is even more common than endocarditis.

Adherent Pericardium.

In the majority of cases the diagnosis cannot be made during life, unless the pericardium is adherent, not only to the heart, but to the walls of the chest as well. When this combination of pericarditis with chronic mediastinitis is present, the diagnosis may be suggested by

(*a*) A systolic retraction of the chest wall in the region of the apex impulse, at the base of the left axilla and in the region of the eleventh and twelfth ribs in the left back (Broadbent's sign). Such retraction is more marked during a deep inspiration. (It should be remembered that systolic retraction of the interspaces in the vicinity of the apex is very commonly seen in cases of cardiac hypertrophy from any cause, owing to the negative pressure produced within the chest by the contraction of a powerful heart.) A quick rebound of the cardiac apex at the time of diastole (the diastolic shock) is said to be characteristic of pericardial adhesions, but is often absent.

(*b*) Collapse of the cervical veins during diastole has been noticed by Friedreich, and the paradoxical pulse, above described, is

said to be more marked in adherent pericardium than in any other known condition. Most recent writers, however, place no reliance upon it.

(*c*) When the lungs are adherent to the pericardium or to the chest wall, as is not uncommonly the case, the absence of the phrenic phenomenon (Litten's signs) and of any respiratory excursion of the pulmonary margins may be demonstrated. Since pericardial adhesions are most often due to tuberculosis, the discovery of tuberculosis in the lung or elsewhere may be of aid in diagnosis.

(*d*) Broadbent considers that the absence of any shift in the position of the apex beat, with respiration or change of patient's position, is an important point in favor of mediastino-pericarditis. In health and in valvular or parietal disease of the heart, the apex beat will swing from one to two inches to the left when the patient lies on his left side, and the descent of the diaphragm during full inspiration lowers the position of the cardiac impulse considerably.

(*e*) The presence of hypertrophy or dilatation affecting especially the right side of the heart, and not accounted for by the existence of any disease of the cardiac valves, of the lung, or of the kidney, should make us suspect pericardial and mediastinal adhesions. Such adhesions embarrass especially the right ventricle, because it is the right ventricle far more than the left which becomes attached to the chest wall. The left ventricle is more nearly free.

(*f*) Since the space enclosed by the divergent costal cartilage just below the ensiform is but loosely associated with the central tendon of the diaphragm, Broadbent looks especially at this point for evidence of mediastinal or pericardial adhesions, the effect of which is to arrest completely the slight respiratory movements of this part of the abdominal wall.

(*g*) Adherent pericardium, occurring as a part of a widespread chain of fibrous processes involving the pleura, the mediastinum, and the peritoneum, may give rise in young persons to a train of symptoms and signs suggesting cirrhosis of the liver. Ascites collects, the liver is enlarged, yet there are no signs in the heart, kidneys, or blood sufficient to explain the condition. In any such case adherent pericardium should be considered. Fig. 150

show the appearance in cases of this kind in which the diagnosis was verified by autopsy.

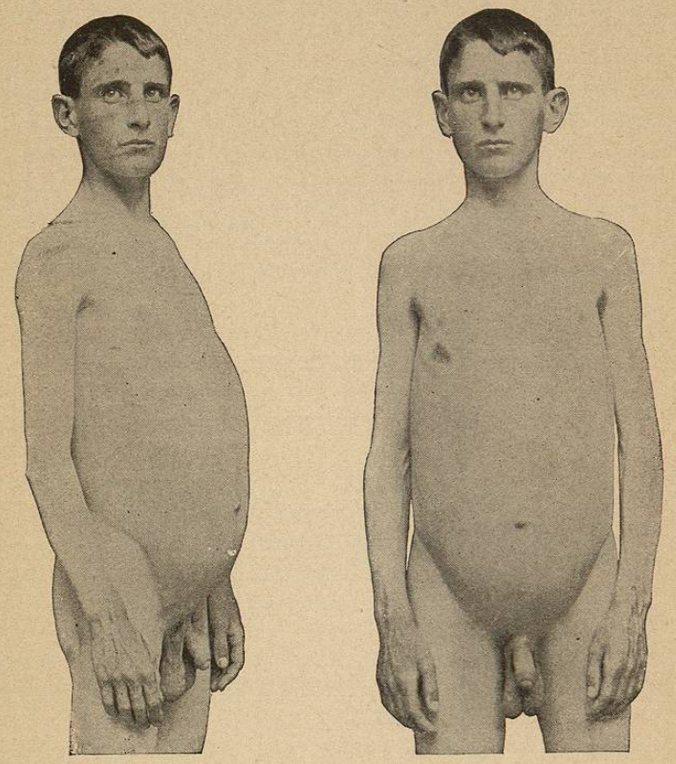

FIG. 150.—Adherent Pericardium, Ascites.

Summary.

The diagnosis of adherent pericardium with chronic mediastinitis is suggested by

(*a*) Systolic retraction of the lower intercostal spaces in the left axilla and in the left back, followed by a diastolic rebound.

(*b*) The absence of any change in the position of the apex impulse with respiration or change of position.

(*c*) The presence of hypertrophy and dilatation of the right ventricle without obvious cause.

(*d*) The absence of any respiratory excursion of the lung borders near the heart and of the abdominal wall at the costal angle.

(*e*) The presence of signs like those of hepatic cirrhosis in a young person and without any obvious cause.