

This is fully discussed in many good text-books on medicine and diagnosis.

The form of the outline of dulness in pericardial effusion is also characteristic. The pear-shape outline with the base downward; the dulness, even in the early stage of effusion, in the fifth right interspace, close to the sternum, obliterating the resonant angle formed by the lung, heart and liver; the dulness over the sternum extending to or above the second rib, together with the outline of the left border dulness, all these are easily recognized and almost pathognomonic. It is true that a greatly enlarged heart, with all its chambers dilated from myocarditis, and a weak diffusible apex-beat may present an outline of dulness which so nearly resembles that of pericarditis with effusion that it may be impossible to differentiate between them, without puncture. The limits of this paper will not permit the discussion of many of the interesting points concerning the precordial dulness of cardiac, vascular, lung and pleural diseases, and especially as compared with pericarditis.

The location of the apex-beat in pericarditis with effusion is characteristic. When it is perceptible it will always be found that the left border of dulness is relatively far removed from it, as it is not in any other cardiac disease.

In large effusions it may be obscured and at other times the right ventricle may strike the chest wall in the region of the nipple, or undulatory waves may be seen as the only evidence of the heartbeat against the chest wall. However, it matters not how the apex beat or the impulse of some other part of the heart against the chest wall be ascertained, it will be found that the point of contact of the heart against the chest wall is always relatively far removed from the left border of precordial dulness, as compared with the relations of the apex-beat to the left border dulness in all other conditions.

OTHER SIGNS.

Some of the other signs of pericarditis have been named in the reported cases. They are as follows: The relatively rapid respiration and dyspnea; the signs of compression of the left lung, evinced by the left inter-scapular and subscapular dulness and bronchial breathing; the rapid heart action, the pulsus paradoxus, and the asymmetry in size of the pulse of the radials; the irregular type of temperature; the paralysis of the left recurrent laryngeal nerve; the unequal pupils; the disturbed mental state of the patient, and still other phenomena. These are not so characteristic as the three cardinal signs first named, but are important and significant when present.

Pericarditis is an easily recognized condition. Frequent careful systematic examination of the precordium should be made in all infectious diseases, and if this is done pericarditis will not escape one. The diagnosis will then be made during life and not at the post-mortem table as is unfortunately now the case in at least 50 per cent. of the cases of pericarditis which autopsy reveals.

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Treatment of Eczema.—Spiegler recommends painting the patch with equal parts of caustic potash and distilled water for a minute at most, and then cauterizing with equal parts of nitrate of silver and distilled water. The dressing then applied can be left undisturbed until complete recovery. This method of treatment is applicable only to chronic, limited, circumscribed eczema with no signs of inflammation.—*Derm. Cbl.* from Kaposi's Festschrift.

THE PATHOLOGY AND PATHOGENESIS OF PERICARDITIS.*

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PHILADELPHIA.

The ancients supposed disease of the heart to be incompatible with life, the heart being the center of life. Galen, in the second century after Christ, observed pericarditis in the lower animals and inferred its occurrence in man. Before this knowledge had been gleaned, all peculiarities of the structure or appearance of the heart were supposed to denote attributes of character. Thus, Haller speaks of the "hairy hearts" of Leonidas. Lysander and Aristomenes, as indicative of great bravery, those "hairy hearts" undoubtedly being viscera covered with stringy fibrin.

ETIOLOGY.

The disease is now known to be of frequent occurrence. It is most common in adolescence and early adult life, between the ages of 15 and 30. It occurs in childhood and in infancy, and rarely in the fetus. It also occurs in late adult life and sometimes in senility. Knopf saw pericarditis 10 times among 459 cases of diseases of childhood, occurring between 1 and 11 years of age.

It is more frequent in men than in women, probably because they are more exposed to its causes. Concerning the relative frequency authors vary. Barthez and Rilliet observed it in 21 men and 3 women. Bamberger gives the ratio as 38 to 25, and Sibson at 35 to 28. All classes of society are liable to the affection, and it is not known that occupation or social condition has any predisposing tendency toward it.

It is customary to divide the cases into two classes: 1. Primary or idiopathic; 2, secondary or metastatic.

1. As all cases depend upon infection, and so far as we know, all the lesions depend upon the local operation of infectious agents, idiopathic pericarditis signifies nothing more than that the infectious agents having entered the circulation through some undiscoverable lesion, have produced their first visible changes in the pericardium. Inasmuch as such an accident must be much less frequent than the entrance of bacteria through recognizable lesions, primary pericarditis is extremely rare. Most writers of experience report that they have seen one or two cases of primary pericarditis, but all agree that it is very rare. Bauer in von Ziemssen's Handbook states that out of the 3000 autopsies which occur yearly in Munich, he sees only two or three cases of idiopathic pericarditis.

2. Secondary pericarditis, on the other hand, is not infrequent and is a common complication of the infectious diseases. It occurs chiefly by hematogenous metastasis, but may also result from lymphogenic metastasis from contiguous infectious processes, and from internal and external traumatism.

Traumatism as a cause of pericarditis may be disposed of in a few words, as it is of importance only as it affords an avenue of entrance for micro-organisms, or produces conditions favorable to their colonization in the tissue. External punctured and incised wounds are harmless, if performed under aseptic conditions. Ordinary wounds of the pericardium, if infected, produce a localized form of inflammation, not analog-

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ous to the usual forms. The extent and character of the lesions will, however, vary according to the nature of the infectious agent introduced.

Internal traumatic lesions are not uncommon. Foreign bodies in the esophagus and stomach may perforate the pericardium. Tuberculous vomica may rupture into it; abscesses of the spleen, liver, lung, heart wall, mediastinal glands, etc., may burrow into it. Empyemas sometimes form fistulous communications with it, and echinococcus cysts and aneurysms of the aorta sometimes rupture into it. Abscesses and other morbid conditions of the mammary gland and skin sometimes descend and invade the pericardium. In all of these conditions the morbid anatomic picture will vary according to the circumstances of the case, none of them conforming to the descriptions later to be given of the true clinical pericarditis.

Lymphogenous metastasis may occur in many of the local contiguous affections mentioned in which no actual traumatic lesion exists. In this manner, disease of the mediastinum, pleura, etc., may occasion pericarditis.

Hematogenous metastasis is seen in nearly all of the infectious diseases, but especially in the following: 1, rheumatism; 2, pyemia; 3, septicemia; 4, pneumonia; 5, chorea; 6, endocarditis; 7, other acute infectious diseases; 8, tuberculosis; 9, scurvy; 10, blood diseases, and 11, malignant disease.

Rheumatism is more liable than any other of the infectious diseases to be succeeded by pericarditis. The percentage of cases given by various writers differs widely. Bauer attributes this to the variation in the characteristics of rheumatism as it occurs in different places. The highest percentage of cases is given by Williams, who thinks it is 75 per cent. The following table gives the percentages of various authors:

Williams, 75 per cent.; Leudet, 22; Bamberger, 30; Chambers and Thompson, 20 to 30; Bauer, 16 to 20; Ball and Sibson, 20; Wunderlich, 19; Duchus, 16; Latham, 5, and Teller, 4.7 per cent. The occurrence of pericarditis is rare in subacute rheumatism and is said not to occur in chronic rheumatism. It therefore is to be looked for in the acute forms, and in gonorrheal rheumatism. The disease is more liable to occur in the polyarticular forms than in the monoarticular forms, and it is said to be most frequent in those cases in which the inflammation wanders quickly from one joint to another. Pericarditis may be the first expression of rheumatic infection, preceding the articular symptoms, or it may succeed them. The usual time for its occurrence is between the 6th and 14th days.

Pyemia is frequently accompanied by pericarditis, the relations of the two processes being so clear that no further mention of it need be made.

Septicemia is also frequently complicated by pericarditis. The statistics of Kirke and Willigk showing 5 out of 91 cases.

Pneumonia is liable to be accompanied by pericarditis in something over 7 per cent. of the cases. Leudet saw it 6 times in 83 cases.

Chorea is not infrequently complicated by pericarditis, Roger having observed it in 5 out of 71 cases, and Olivier in 1 out of 30 cases.

Endocarditis is quite frequently complicated with it. This is both because the two conditions are liable to depend upon the same cause, and because of the likelihood of metastatic infections in endocarditis.

Various other infectious diseases in which the occurrence of pericarditis does not take place with any regu-

larity must also be mentioned. Among these are scarlet fever, smallpox, relapsing fever, measles, erysipelas, gonorrhea, syphilis, dysentery, cholera, typhoid fever, vaccinia, cerebro-spinal meningitis and diphtheria.

Tuberculosis may lead to pericarditis both by metastasis, continuity of tissue, and the rupture of vomica into the cavity. The lesions occasioned may be either local or general, and the exudation is nearly always purulent. Indeed, Birsch-Hirschfeld states that the majority of cases of pericarditis with chronic purulent exudation are tuberculous.

Scurvy is usually associated with hemorrhagic extravasations into the tissue and upon the serous membranes, and is nearly always accompanied by superficial lesions of the mouth and skin, which favor the entrance of bacteria into the circulation. Both of these conditions, together with the general vital depression that exists in scurvy, favor the development of pericardial infections, so that pericarditis with sero-sanguinolent and purulent exudates are common. The effusions that can occur under these conditions may be enormous. Sometimes, in scurvy, pericarditis may be endemic and Koch is satisfied that it is identical with the *morbus cardiacus* of the ancients.

Blood diseases with diminished vital resistance, such as hemophilia, hemorrhagic diathesis, purpura, leukemia, diabetes, etc., may also be complicated with pericarditis. For the same reason the disease also occurs in alcoholism, cirrhosis of the liver and in nephritis. It is in chronic nephritis that pericarditis is most apt to occur, its development usually coinciding with the first symptoms of uremic blood poisoning. The frequency of its appearance is given by Taylor as 33 per cent.; by Bamberger, 14; by Rosenstein, 7, and by Frerichs, 4.5 per cent.

Malignant disease, as in carcinomatous invasion of the pericardium from primary disease of the mammary gland, esophagus, thyroid gland, etc., may by invasion of the pericardium lead to the diminution of its vital resistance, the occurrence of an unusual quantity of sero-sanguinolent exudation, and so predispose to the occurrence of infections, should bacteria in any way reach the tissues.

BACTERIOLOGY.

There is no specific micro-organism of pericarditis. The manifold conditions in which the disease occurs, and the varied morbid anatomic appearances which it presents, suggested this before experimental research in bacteriology proved it.

In the sero-fibrinous form of the disease, the study of numerous cases has revealed the presence of streptococci, staphylococci, pneumococci, bacillus pyocyaneus, the bacillus of Friedländer, and the bacillus tuberculosis.

In the purulent form, streptococci, staphylococci, bacillus coli communis, bacillus of Friedländer, and the tubercle bacillus have been found.

The hemorrhagic form is apt to be tuberculous, so that the tubercle bacillus may be found, together with other accidental organisms.

In the pyo-pneumo-pericarditis, and in fetid pericarditis, with communications with the lung, esophagus, etc., numerous saprophytic micro-organisms may be found.

MORBID ANATOMY.

It is customary to divide pericarditis into numerous forms according to the peculiarities of the exudate. The forms usually described being as follows:

1, Pericarditis sicca; 2, sero-fibrinosa; 3, purulenta;

4, adhesiva chronica; 5, hemorrhagica, and 6, tuberculosa.

Concerning the frequency of occurrence, the best figures obtainable seem to be those of Breitung, who collected 324 cases of pericarditis among the autopsies of the Berlin Charité, between the years 1866 and 1876, and found the following distribution:

Pericarditis sero-fibrinosa, 108; hemorrhagica, 30; purulenta, 24; tuberculosa deuteropathia, 24; tuberculosa idiopathia, 2; adhesiva partialis, 111; adhesiva totalis, 23, and ossificans, 2 cases.

It is an error to think of these names as referring to distinct forms of the disease. They are for the most part stages of the same process, and a description of the morbid process will make this clear. The first expression of the inflammatory process is found in redness and dryness of the surface of the pericardium—pericarditis sicca—upon which the vessels stand out with unusual distinctness. The natural glaze of the surface is lost, and the two surfaces, parietal and visceral, when they come in contact, rub one against the other with an unnatural degree of friction, which is painful to the patient, and which gives the well-known friction-sound to the ear of the auscultator. It may be that a few flakes of fibrin appear upon this dulled surface, and are rolled into threads by the incessant movements of the heart.

The disease may advance no further than this, and terminate in perfect recovery, but usually the excitant by which the disease is produced occasions further changes, and the inflammatory hyperemia is succeeded by a period of transudation, in which in most cases a clear serous fluid is poured out into the sac. Almost immediately a deposition of fibrin takes place from the fluid upon the surface of the membranes, chiefly, however, upon the heart itself. It forms a more or less coherent membrane which can readily be stripped off, leaving a fairly healthy surface behind. The membrane is, however, almost immediately changed in appearance by the movements of the heart, which tend to gather the sticky material into irregular forms. Upon the right ventricle, and especially upon its inferior and right surfaces, and upon the right auricle, the combined pressure and movement of the heart roll up the fibrin into threads of rounded form, which, after a time, give that part of the organ a shaggy, villous appearance—*cor villosum*. The left ventricle which has a different movement, and is freer of pressure, usually presents a reticulated membrane which is not unlike a piece of omentum upon its surface. As the accumulation of serum and precipitation of fibrin go on, the pericardial sac becomes distended, its base being broad, the precordial area tending to bulge it. The quantity of accumulated exudate usually reaches one-half to one pint, but it is not rare for it to reach a quart. Corvisart and Louis mention cases with exudates of 1000 and 1200 grams. Montague found one with 1850 grams of exudate, of which he withdrew 1000 grams by puncture. Gosselin once saw 2000 c.c. of fluid in the pericardium. The largest serous accumulation that I have seen recorded is that of Corvisart, who found 8 pounds of serum in one case.

If the bacteria upon whose activity the pericarditis depends are of markedly chemotactic influence, the exudate may be more or less purulent from the start. How often this is the case it is difficult to conclude from the number subsequently found to be purulent, and it is usually supposed that it is not uncommon for the sero-fibrinous exudate to become purulent in conse-

quence of further changes in the local conditions. In 39 cases studied by Louis, the exudates were: Serous, 9; purulent, 7; sero-sanguinolent, 10, and sero-purulent, 13.

The purulent exudates are apt to be larger than the sero-fibrinous exudates. Martin has seen 3¼ pounds of purulent matter, and Alonzo Clark, 1 gallon of sero-pus in the pericardium. In cases of purulent exudation the pericardium is still found to be covered by the fibrin as previously described.

Should general conditions, such as the existence of scorbutus, or of local conditions, such as extremely active inflammation, exist, blood may be added to the exudate, whether previously serous or purulent. It is chiefly in scorbutus that bloody effusions occur, and it is almost inconceivable to what extent the pericardium may be distended. Andrea has seen 2 pounds of blood in the pericardial sac, and in scorbutic cases Kyber has found as much as 10 liters of bloody fluid.

The terminations of the process will depend upon its severity and the extent of damage done. When the disease does not advance beyond the stage of congestion, the return to the normal is a very simple matter. So soon as the membrane becomes covered with fibrin, however, new conditions present themselves and it is well-nigh impossible for recovery to take place without leaving marked changes behind.

The deposition of fibrin upon the already diseased surface is shortly succeeded by the degeneration and destruction of its covering endothelial cells. The connective tissue of the sub-endothelial layer of the pericardial membrane now begins to grow, and into the spaces of the fibrinous deposit a delicate, well-vascularized granulation tissue grows, extending as the fibrin melts away. Transmigrated leucocytes penetrate the tissues and aid in the removal of the fibrin.

The result of the presence of this growing tissue is that contiguous surfaces of the membrane are apt to grow together by firm fibrous unions, and that upon surfaces not in contact, more or less regular connective tissue indurations, and callous formations form. The length of time required for the formation of these adhesions is probably considerable. Their duration is permanent, though in the course of time the influence of traction may modify them.

While the absorption of the serum from sero-fibrinous exudates presents no great difficulties, the case is different when the exudate is purulent, for the burrowing tendency of the pus must be considered. Purulent exudations may be absorbed. They may, however, burrow in various directions and discharge externally. The usual seat of pointing is the precordium. Fabricius saw a purulent pericarditis point in the 2d left intercostal space. Wyss saw the pus penetrate a rib and be discharged externally. Rich reports a case that bored through the sternum and presented as a subcutaneous enlargement the size of an apple on the anterior chest wall. Sometimes the pus burrows down the back simulating the descending abscesses of Pott's disease; rarely it points sub-phrenically. One of the interesting terminations is into the lung, the pus being discharged through the bronchial tubes. This occasions what is known as a pneumo-pericarditis or a pro-pneumo-pericarditis. In these cases, not only is the pus discharged into the bronchial tubes, but there is an opportunity for the purulent effusion in the pericardium to receive saprophytic bacteria from the lung and become fetid.

The purulent inflammation also leads to great thickening

ing of the membrane. Horn has reported an interesting case of extraordinarily thick pericardium and Nunier has seen the membrane thickened and callous, with imbricated scales like a pineapple.

After evacuation of the pus there is usually some residual exudate, and in cases which terminate by reabsorption, the residuum is prone to undergo calcification. A most interesting case is reported by Drummond in which there was an extensive bone-like deposit between the layers of the muscular tissues of the heart—"a thick bone-like mass ran across the whole left ventricle, penetrating the entire wall of the heart like a wedge and reaching into the cavity of the left ventricle." Variot observed a case in which the left ventricle was covered by a solid hard plate of chalky matter on its whole anterior surface. Other cases of calcification have been observed by Tiessier and Richards.

PROGNOSIS.

The effect of pericarditis upon the heart is of great importance. No considerable disease of the epicardium is possible without involvement of the heart. The superficial layers of muscular fibers usually show cloudy swelling, later hyaline or fatty degeneration. In cases with purulent exudate the pus may also gradually work its way between the muscular bundles.

The changes that thus take place during the height of the disease predispose to acute dilatation of the heart, and a fatal termination of the disease may thus be brought about. If this does not occur, and if the patient recovers from the pericarditis, the regenerative cicatricial processes that go on lead to fibroid interstitial changes in the wall of the heart. If many adhesions form and interfere with the action of the heart, a very marked hypertrophy of the organ may occur to compensate for the embarrassment.

The tuberculous disease of the pericardium differs from the form described in that it is essentially chronic and purulent from the beginning. A proper description of the various pictures presented by this disease is made impossible by the time allotted to this paper.

Syphilitic pericarditis has been observed by Orth and by Lancereux, who observed gummatous formations in the pericardia of syphilitic children.

ETIOLOGY OF PERICARDITIS.*

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The etiology of pericarditis has been discussed so often and by so many that it is indeed difficult to present a discussion of it to a body of this character in such a manner as to arouse interest. For this reason it seemed better to disregard the extensive literature upon this subject and utilize the reports of three hundred cases as a text, dwelling upon some points of interest presented by them and drawing attention to some things in which they differ from generally accepted statements. The cases in hand consist of 80 cases ending in recovery and 220 ending in death, of which 190 were examined postmortem; 244 of these cases are acute pericarditis and 56 are of the chronic form with partial or complete obliteration of the pericardial sac. The cases are derived in part from the records of the Cook County Hospital, where

many of the cases were seen personally and in part from private records.

The immediate exciting cause of the pericardial inflammation is usually unknown and apparently undiscoverable by our present methods. We know that there are certain diseases in which pericarditis frequently occurs as a complication, but whether the irritant causing the inflammation is a bacterium or a toxin is still uncertain. For example, the pericarditis accompanying the chronic diffuse nephritis may be of toxic origin; some authors, indeed, speak of it as a uremic pericarditis, or it may be of bacterial origin as others believe. So also the pericarditis of rheumatism may be due to a bacterium, or may be as some suspect the joint inflammations to be, of toxic origin. In a limited number of cases bacteria have been found in the exudate, pus cocci, pneumococci, tubercle bacilli, bacillus pyocyaneus and others, but in many cases the exudate is sterile. In many cases the irritant body reaches the pericardium through the lymph channels, or by direct extension, but doubtless there are numerous cases, apparently of this sort, which are really hematogenous.

It has been customary to divide the cases of pericarditis into two groups, primary and secondary, meaning by the former cases in which there is no manifest disease, to which the pericarditis appears as a complication. Most authors state that careful attention to etiology has reduced the primary cases to a vanishingly small group. This is doubtless true, but there is no apparent reason why the pericardium should so differ from other serous membranes that it does not become the seat of a primary inflammation. Of the cases which went to autopsy there is but one which could in any way be regarded as a primary pericarditis, and even in this case there was an old adhesive pleuritis, which might easily have been tubercular or the remains of a former pneumonia. This fact might be regarded as strong evidence against the existence of a primary pericarditis. There are 12 cases with 3 deaths in which the diagnosis of pericarditis alone is made. It is impossible to say how many of these cases should be regarded as examples of primary pericarditis, but in all the pericarditis was the prominent clinical feature and in several careful search for some cause failed to reveal any. Although there are no examples of pericarditis following injury of the pericardium in this series, such cases occur and in them the relation of cause and effect is too manifest to require any discussion.

Let us first take up the 244 cases of acute pericarditis or rather the 232 cases left after deducting the 12 cases already mentioned, grouped according to the primary disease and in the order of relative frequency.

First come 79 cases of pericarditis with acute pneumonia of which 73, i. e., 92.4 per cent. ended fatally. In 55 of them it is stated what lobes were involved in the pneumonia. In 23 cases the pneumonia was in the right lung, 21 in the left, and 11 times both lungs were involved. The percentages do not differ very greatly from those given by Aufrecht, Grisolle and Huss as to the relative frequency of the right, left and bilateral pneumonia, as shown in the following table:

AUFRECHT, GRISOLLE, HUSS.	PERSONAL.
Per cent.	Per cent.
Right-sided pneumonia . . . 52.0	23 cases, i. e., 42
Left-sided pneumonia . . . 35.2	21 cases, i. e., 38
Bilateral pneumonia . . . 12.8	11 cases, i. e., 20

* Read in a Symposium on Pericarditis at the Fifty-second Annual Meeting of the American Medical Association, in the Section on Practice of Medicine, and approved for publication by the Executive Committee of the Section.