Some Aspects of the Pathogenesis of Cardiac Tuberculosis

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Tuberculosis of the heart is not a common condition. It is very often an autopsy finding, which clinically was of little or no importance, being but a part of a generalised tuberculous infection. Sixteen examples of cardiac tuberculosis were found among the records of 3,500 cases post-mortem in the Institute of Pathology in the last ten years, an incidence of only 4.6 per 1,000. Of these sixteen cases there was direct evidence for regarding the condition as tuberculous in origin in ten only, but it is possible to bring forward strong circumstantial evidence that the cause of the pathological changes found in the other six cases was originated by infection with the tubercle bacillus.

The following is a brief summary of the pathological features of these sixteen cases:

Case 1: Male, aged 23 years; generalised miliary tuberculosis with tubercles in the myocardium; endocardium and pericardium not affected; tuberculosis of the right epididymis with surgical removal two years previously; acute fibro-caseous tuberculosis of the right seminal vesicle; ante-mortem thrombi in the periprostatic, right internal and common iliac veins; infarction of the lung.

Case 2: Female, aged 50 years; generalised miliary tuberculosis with tubercles in the myocardium; endocardium and pericardium not affected; fibro-caseous tuberculosis of the upper lobe of the left lung; early tuberculous meningitis; also acute non-specific right sided mastoiditis, ethmoidal and sphenoidal sinusitis.

Case 3: Female, aged 15 years; generalised miliary tuberculosis with myocardial and subendocardial tubercles; pericardium not affected; fibro-caseous tuberculosis of the lower lobe of the right lung with tuberculous hilar adenitis; tuberculous meningitis; localised tuberculous peritonitis.

Case 4: Male, aged 22 years; generalised miliary tuberculosis with myocardial and subendocardial tubercles; pericardium healthy; right sided renal phthisis with tuberculosis of ureter, bladder, prostate and seminal vesicle; non-bacterial thrombotic endocarditis of the mitral valve superimposed on healed rheumatic endocarditis.

Case 5: Female, aged 45 years; solitary tuberculoma in the myocardium; endocardium and pericardium healthy; no other tuberculous lesions found in any part of the body; diabetes and hyperglycaemic coma. (Partial post-mortem examination only performed.)

Case 6: Female, aged 6 years; generalised miliary tuberculosis with subendocardial and myocardial tubercles; endocardial tuberculosis; large caseating areas in myocardium, extending through the endocardium and discharging caseous material into the heart cavity, and in direct communication with caseating areas.
in the pericardium; acute sero-fibrinous pericarditis; large active tuberculous focus
in apex of left lung; tuberculosis of the mediastinal glands; tuberculous ascites
with enlarged peritoneal glands; chronic venous congestion of liver secondary to
cardiac tamponade. (See plate 1.)

CASE 7: Female, aged 50 years; generalised miliary tuberculosis; early localised
fibrinous pericarditis in relationship to a caseous hilar lymph node; normal endo-
cardium and myocardium; extensive tuberculous lesion right upper lobe with
slight fibrosis but no cavitation; tuberculous broncho-pneumonia; both calcified
and caseous hilar lymph nodes, with direct extension to the pericardium.

CASE 8: Female, aged 63 years; acute fibrinous and organising tuberculous
pericarditis; normal endocardium; focal areas of myocardial fibrosis but otherwise
no lesion demonstrated in the heart muscle; caseous lymph node at the root of the
aorta; auricular fibrillation with bundle branch block and chronic venous conge-
tion; generalised arterio-sclerosis; old scarring in lungs. (See plate 2.)

CASE 9: Male, aged 60 years; fibrinous and organising pericarditis; normal
endocardium and myocardium; bilateral apical fibrous scars in lungs; caseous
tracheo-bronchial lymph glands; chronic venous congestion; thrombosis of pro-
static veins; infarction of lung.

CASE 10: Male, aged 20 years; acute sero-fibrinous pericarditis; normal endo-
cardium and myocardium; tuberculous abscess in lung; bilateral pleurisy; chronic
peritonitis; cardiac cirrhosis.

CASE 11: Female, aged 22 years; generalised miliary tuberculosis; chronic
adhesive pericarditis; normal endocardium and myocardium; primary tuberculous
complex in lower lobe of right lung; tuberculosis of right inferior group of tracheo-
bronchial lymph glands; bilateral pleural effusions; extensive fibro-caseous tuberc-
ulosus of the retro-pancreatic lymph glands; tuberculous peritonitis.

CASE 12: Male, aged 29 years; generalised miliary tuberculosis; chronic tuberc-
ulous pericarditis with adherent pericardium; normal endocardium and myo-
cardium; primary tuberculosis of right upper lobe with involvement of the bronchi
and extension to the left lung; tuberculous pneumonia; tuberculous ulceration of
left main bronchus; tuberculous pleurisy with effusion; tuberculous peritonitis with
ascites.

CASE 13: Male, aged 33 years; chronic adhesive pericarditis; endocardium and
myocardium not affected by tuberculosis but both show evidence of previous rheu-
matic infection; tuberculosis with caseation in left upper lobe of lung; tuberculous
pleurisy; calcified hilar lymph glands; tuberculosis with destruction of both adrenals
and development of Addison’s disease.

CASE 14: Male, age not known (carried in dead); chronic adhesive pericarditis;
normal endocardium and myocardium; chronic fibroid phthisis with cavitation of
both lungs; terminal haemorrhage from ruptured blood vessel in cavity of left lung.

CASE 15: Female, aged 38 years; adhesive and obliterative pericarditis with
patches of calcification; normal endocardium and myocardium; old left sided
pleurisy.

CASE 16: Female, aged 30 years; calcified pericarditis; normal endocardium and
myocardium; old standing tuberculosis with caseation in hilar lymph glands; Pick's disease; thickened and fibrosed pleura with patchy calcification; non-specific inflammatory reaction in lung, possibly, but not definitely, tuberculous; death followed post-operative mediastinal haemorrhage.

Of the sixteen cases the pericardium was affected in eleven, and these eleven cases include all six cases in which direct proof of cardiac tuberculosis was not obtained. Even assuming that all eleven cases were, in fact, tuberculous, this gives an incidence of only 3.1 per 1,000 in the records of this department for tuberculous pericarditis.

This incidence is very much smaller than that recorded in the literature. Osler in 1893 was able to review seventeen cases which he had seen personally, and wrote: “Tuberculosis follows hard upon rheumatic fever as a cause of pericarditis—in 1,000 autopsies there were 275 cases with tuberculous lesions, in 7 of which the pericardium was involved.” Norris (1911) found 1,780 cases of tuberculosis in 7,219 autopsies, and 82 of them had tuberculous pericarditis; and Kornblum, Bellet, and Ostrum (1933) state that the incidence in general autopsies of tuberculous pericarditis is approximately 1 per cent., and that tuberculous involvement of the pericardium occurs in about 4 per cent. of autopsies on patients with pulmonary tuberculosis.

Tuberculosis of the Endocardium

Tuberculosis of the endocardium must be regarded as a condition of little practical importance, since when it does occur it is invariably but part of a very serious and often generalised form of tuberculosis such as that of the miliary type. Here tubercles may be found within the musculature usually of the ventricle, or just underneath the endocardium, tubercle bacilli having been carried to these sites by the blood of the coronary arteries. Occasionally a subendocardial tubercle may rupture into the cavity of the heart, and this serves to hasten the inevitable end-result. Rarely a fulminating tuberculous pericarditis may spread by direct extension into the myocardium and rupture through the endocardium into the heart cavity. Tuberculosis of the endocardium, or of the valves, akin to acute or subacute bacterial endocarditis, is an extraordinarily rare condition.

Baker (1935), in an exhaustive survey of the literature, found about thirty cases reported, but concluded that only four of them could be regarded as having established their claim to be tuberculous endocarditis, and he regards but one of these thirty cases as being the only fully established case of tuberculous endocarditis analogous to rheumatic or bacterial endocarditis. Since then Davie (1936) has reported two cases of what appear to be true tuberculous endocarditis, and Bevans and Wilkins (1942) reported a further case. This rare condition, interesting especially because of its rarity, should alone be termed “tuberculous endocarditis,” the other more frequent finding being designated “endocardial tuberculosis.” Davie put forward a new hypothesis to account for the rarity of tuberculous endocarditis. His theory assumed the existence of a tuberculous allergic endocarditis similar in type to rheumatic endocarditis, and he postulated the rare coincidence of a tuberculous bacillæmia during the phase of the allergic endocarditis to explain the
accepted form of tuberculous bacterial endocarditis. So he brought the latter condition "into line ætiologically with subacute bacterial endocarditis."

One still wonders, however, why a tuberculous bacteræmia should spare the valve which has been affected by rheumatism when the relatively non-pathogenic streptococcus viridans can so often multiply there and cause disaster.

To complete this brief discussion of tuberculous endocarditis, it should also be pointed out "that there is no evidence that chronic valvular disease can originate either directly from tuberculous inflammation or indirectly from the toxic effect of tuberculosis elsewhere in the body" (White 1946).

Cases 3, 4, and 6 are examples of subendocardial tubercle formation occurring as part of a miliary tuberculosis. Case 6 also shows the spread of myocardial tuberculosis to the endocardium and the rupture of a caseating focus into the heart cavity. In this case tubercle bacilli were demonstrated by Ziehl-Neelson staining in the material about to be discharged into the blood stream.

**Tuberculosis of the Myocardium**

Tuberculosis of the myocardium is usually unsuspected during life. Occasionally, however, a small tubercle may involve the auriculo-ventricular conduction system and lead to heart block; or an aneurysm of the heart wall may occur, rupture, and lead to sudden death (White 1946). This condition is also rare. Raviart (1906) in a series of 7,683 cases of tuberculosis found myocardial tuberculosis only 49 times (or 0.63 per cent.). In our series there were 6 cases among the 3,500 post-mortem reports examined, which is an incidence in general pathology of only 1.7 per 1,000 cases.

Cases 1, 2, 3, 4, and 6 all showed tuberculous infection of the myocardium and all had associated generalised miliary tuberculosis. Case 5 is an example of solitary myocardial tuberculoma found at post-mortem. This case showed no other active tuberculous lesions, although old pleural adhesions were found; unfortunately, however, only a partial post-mortem examination was carried out. In this particular case the tuberculoma had not interfered with the patient's condition and was quite unsuspected. Death occurred in diabetic coma. Case 6, which, incidentally, illustrates almost all the findings of acute cardiac tuberculosis, was also an example of how a very active pericardial tuberculosis can spread directly into the heart muscle: in this case there were undoubtedly many extensions of caseous material into the blood stream and some at least, if not all, occurred after the onset of the pericarditis.

**Tuberculosis of the Pericardium**

Tuberculosis of the pericardium, though not a common disease, is nevertheless an important one. The pericardium may be infected in one of three ways:

1. Via the blood stream.
2. Via the lymphatics.
3. By direct extension.

Osler (1893) wrote: "Tuberculous pericarditis is due in the majority of cases to infection of the membrane by caseous mediastinal lymph nodes. The disease may
be confined to the glands and to the pericardium." This view is widely accepted by all authorities on the subject, but it is only within recent years that the real reason for the close association between pericarditis and hilar lymph adenitis has been appreciated. This followed the work of Rich and others. True, in miliary tuberculosis, small tubercles may form on any serous membrane, including the pericardium, but a tuberculous effusion or a tuberculous pericarditis, in the usual conception of the term, does not usually develop. The pathogenesis of tuberculosis of all serous cavities is similar. Effusion will only develop in the presence of either a massive dose of organisms, hypersensitivity, or both. In the discussion on this subject, Rich (1944) states: "As in the case of the meninges, tubercle bacilli do not escape freely from the blood stream into the serous cavities. The writer has not encountered in the literature an instance of tuberculous effusion occurring as a prompt and direct result of the intravascular injection of tubercle bacilli into experimental animals, nor in his own experience has he ever seen that event happen in any of the many normal and hypersensitive animals, that have been subjected to intravenous or intra-arterial injections of large numbers of tubercle bacilli." And again: "In miliary tuberculosis in human beings, likewise, miliary tubercles develop on serous surfaces, but abundant effusions into the cavities do not occur."

Experimentally it has been found by many workers, that an abundant effusion will occur if tubercle bacilli are injected in large numbers directly into the pleural or peritoneal cavities of hypersensitive animals, but hypersensitivity is necessary, or effusion will not occur. It can be assumed that the same reasoning holds true for pericardial effusions, and this argument agrees well with the already established fact that tuberculous hilar lymph glands, healed or active, are invariably found at autopsy and that direct extension or lymphatic spread from these glands had occurred. It must be pointed out, however, that although large numbers of tubercle bacilli require to be injected into the serous cavities of even hypersensitive animals to produce effusions, "this does not mean that a proportionally large number must be discharged into the cavity in the human being, for the degree of inflammatory exudation depends upon the balance between the number of bacilli and the degree of hypersensitivity, and the human being can develop a decidedly higher degree of hypersensitivity than laboratory animals" (Rich, 1944). Indeed in the human it is very probable that a tuberculous effusion can develop following the discharge into a serous cavity of tuberculoprotein and disintegrated dead bacilli, no actual living organisms being required.

Actually a tuberculous hilar lymph node could affect the pericardium by any one of three different methods. Firstly, the gland could rupture through the pericardium and discharge caseating material into the sac. Such cases have been described. Secondly, there could be a lymphatic spread from the gland to the parietal pericardium and Hannesson (1941) states that this is the most likely method of spread in most instances, but to assume this is to postulate that a retrograde extension occurs and this is never a very attractive hypothesis. Thirdly, the tuberculous material could pass directly from the gland to the pericardium. Harvey and Whitehill (1937) were able to demonstrate this mode of extension in three cases and this
would seem to be the usual way by which the condition is brought about. In Case 7 the pericarditis was localised to that part of the parietal layer in contact with a caseating lymph gland.

It should be pointed out that the demonstration of a tuberculous gland, especially when it is not in contact with the pericardium, does not necessarily mean that this gland infected the sac, since it may be one of the group of glands draining the pericardium and so infected by the serositis rather than being the cause of the serositis. In this way, many so-called retrograde lymphatic spreads may conceivably be explained.

From this general discussion of the pathogenesis of tuberculous pericarditis, the various types met with in practice will be described briefly.

These can be divided into four groups:

1. That associated with generalised miliary tuberculosis.
2. That associated with generalised serous membrane tuberculosis.
3. That due to extension from neighbouring structures.
4. That developing independently.

That associated with Generalised Miliary Tuberculosis:

The first group has already been dealt with. In this series there were no true examples of this particular type. Case 6, although showing pericarditis and miliary tuberculosis, must be regarded as being of the third type, and, as has been mentioned previously, in this case it is very probable that the miliary tuberculosis followed the pericarditis and not vice versa.

That associated with Generalised Serous Membrane Tuberculosis:

The second group includes many cases of so-called polyserositis or Concato's disease. Under this generic term is included a well defined group of cases with chronic inflammatory thickening of the serous membranes, often associated with recurrent serous effusions. Burrell, Hare, and Ross (1929) believe that chronic tuberculosis of the great serous sacs progressing to the production of polyserositis is a condition associated with a high degree of immunity to the infection, and that in more chronic cases the infective element is slowly submerged as the clinical picture becomes one of mechanical obstruction to the heart and circulation. It is in this way that Pick's disease may be produced.

That this condition is usually due to tuberculosis is not accepted by all. But it is not difficult to imagine how this could be produced by infection with the tubercle bacillus and it does seem that tuberculosis is, at any rate, a common cause for the condition. Case 11, for example, showed chronic adhesive pericarditis, bilateral pleural effusions and peritonitis, in other words, inflammation of the three serous membranes. If in this case the tuberculous foci in the lung and glands had been healed and not evident, the patient would have presented the picture of a polyserositis. The serous membranes may be extensively affected with minimal tuberculosis elsewhere. Case 9 is an example with healed pulmonary tuberculosis, caseous tracheo-bronchial glands, and a fibrinous but organising pericarditis, this patient dying from a pulmonary infarction associated with thrombosis of the pro-

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static veins; and it is easy to see how the allergic reaction, which occurs in any serositis, may be maintained or heal with gross fibrosis and thickening, when the focus, which was the cause of that serositis, becomes quiescent and at autopsy is found only as a small area of scar tissue, the aetiology no longer obvious. It is easy to see, too, how the pleura and pericardium could be implicated by the same group of caseous lymphatic glands which later might heal. The end result of this pathology is the syndrome called polyserositis.

That other disease processes apart from tuberculosis may cause this syndrome cannot, of course, be denied.

That due to Extension from Neighbouring Structures:

The third group, where extension occurs from neighbouring structures, is the largest group and includes most cases of acute fibrinous and sero-fibrinous pericarditis. Cases 6, 7, 8, 9, and possibly 10 are examples of this group. In Case 7 a direct extension was demonstrated histologically between the caseating gland and the pericardium.

In the early stages of this process the membranes are only a little thickened and often tubercles can be seen just underneath the endothelial layer. In other instances the contiguous surfaces of the pericardium are covered with yellow caseous material and in certain cases collections of thick cheesy pus may be found between the two layers. It is obvious how in these cases the tuberculous process might involve the myocardium directly as has been mentioned previously.

With the advent of hypersensitivity, effusion occurs. Osler (1893) describes this effusion as being of four main types:

1. A simple plastic exudate is formed, similar to that of rheumatic pericarditis, with little serous effusion or thickening of the membrane. Histologically, however, one finds tubercle formation in the disorganised pericardium.

2. An extensive sero-fibrinous exudate is found and here the membranes may be greatly thickened.

3. Hæmorrhagic effusions may develop. The colour of these may be bright red but more commonly is of a reddish-brown or chocolate colour.

4. The effusion may be purulent. And, to quote Osler, "This, too, apparently from the onset and not following paracentesis. The exudation may be enormous and has been diagnosed as left sided empyema."

These effusions may be of very great size indeed, but since the effusion is slow in development and so unlike rheumatic pericarditic effusion, it is relatively well tolerated by the patient (White 1946). Incidentally, White mentions that "A pericardial friction rub may be heard over the præcordium even in the presence of a large effusion" and Harvey and Whitehill (1937) found a friction rub in 30 per cent. of twenty cases with large effusions (over 300 c.c.). They state: "Consequently a large effusion does not prevent the appearance of a friction rub." In Case 6 a præcordial friction rub was recorded as having been heard. It is very difficult indeed to imagine how this rub could possibly be produced.

The patient may die in this stage of the disease, but in many cases, probably the
majority, the condition settles and healing takes place. The end result of healed acute tuberculous pericarditis is the important clinical variety.

With healing, the pericardial cavity may be partially or completely obliterated, so-called adhesive pericarditis, and occasionally the adhesions may extend to and involve other structures producing the condition known as chronic adhesive mediastinitis or mediastino-pericarditis. A similar condition can be produced by rheumatic fever, which is, in fact, the commonest cause of this particular pathological change. Unless these adhesions hamper the heart’s contractions, the condition is usually unsuspected. Blalock and Burwell (1941) state: “Even generalised union of the epicardium and the pericardium, if non-constricting, may produce no significant disturbances of cardiac function. It is believed that most cases (of mediastino-pericarditis) follow rheumatic fever.”

The more important complication following acute tuberculous pericarditis is the condition known as chronic constrictive pericarditis. In this condition the thickened fibrotic pericardial surfaces are firmly adherent and enclose the heart in a sac of scar tissue, which, like all other scar tissue, contracts with age. It is a slowly progressive condition, taking many years to develop and so often met with in elderly patients. It has been recorded in octogenarians (Osler 1893), and Blalock and Levy (1938) gave as the average age of 42 cases, 40.3 years; they had one example in a patient over 70 years of age, three in patients over 60, and nine in patients over 50. In this connection, Graham, Singer, and Ballou (1935) state that in the aged, tuberculosis is the most common cause of pericarditis.

Constrictive pericarditis produces a characteristic clinical picture of which there are two varieties, one in which the superior vena cava is obstructed (“superior mediastinal pressure syndrome”) and the other and more common condition where the inferior vena cava and often the hepatic veins are constricted (“inferior mediastinal pressure syndrome”). The patho-physiological aspects of the condition are well dealt with by Blalock and Burwell (1941). According to these authors most of the symptoms and signs are dependent on two changes. (1) Diminution in the amount of blood pumped by the heart per minute and an inability to increase the output per beat. This diminution leads to weakness, fatigue, tachycardia, low pulse pressure and a limited tolerance for exercise. (2) Increase in the venous pressure. This increase is also apparent in the pulmonary circulation but “various findings indicate that the congestion is more severe in the systemic than in the pulmonary area.” The increase in venous pressure leads to venous distension, to engorgement of the liver, to the formation of peripheral oedema, to epistaxis, and to the transudation of fluid into the pleural and peritoneal cavities.

It is thus apparent that the alterations in constrictive pericarditis include changes in the venous pressure which are similar to those of congestive heart failure and changes in the cardiac output and blood pressure reminiscent of those changes which occur in patients suffering from surgical shock. To quote Blalock and Burwell (1941), “However, in the latter condition, the veins are usually collapsed in sharp contrast to the distended veins seen in constrictive pericarditis.”

White (1946) writes that it is the hepatic vein obstruction secondary to the con-
striction of the heart itself, with or without an additional factor of local blocking, that leads to hepatic congestion, enlargement, and eventually cirrhosis in the inferior mediastinal pressure syndrome. It is this particular end-result that is called Pick's disease, and at post-mortem examination the so-called "sugar icing of the liver" is found due to the chronic peritonitis which is produced. Pick's disease may or may not be associated with polyserositis, although, of course, it is possible that Pick's disease may follow polyserositis.

The striking condition of pericarditis calciosa is sometimes seen when calcium is deposited in the greatly thickened and fibrotic pericardium of a chronic pericarditis. Occasionally the plaques of calcium are small and of no real importance except that they show up well on radiograms of the chest. At other times, however, the heart may be completely encased in a thick deposit of calcium, the condition picturesquely called "armoured heart" or "marble heart."

This condition has always aroused a considerable amount of interest or curiosity. One notes that marble heart disease appears to have been recognised by the American author Hawthorne as long ago as 1818. The condition is described in a story by this author entitled "Ethan Brand." Ethan Brand is related to have committed suicide by plunging into the burning lime kiln which in former days he had attended. Hawthorne concluded his story as follows: "The marble was all burnt into perfect snow-white lime. But on its surface, in the midst of a circle, snow-white too and thoroughly converted into lime, lay a human skeleton in the attitude of a person who, after long toil, lies down to long repose. Within the ribs, strange to say, was the shape of a human heart." This story in more detail is recorded by Hewitt (1932).

In the present series there were two cases of pericarditis calciosa. Case 15 had small plaques of calcium in the pericardium which was associated with an adhesive obliterative pericarditis and an old unilateral pleurisy. No active tuberculous infection was found. The condition of the pericardium in Case 16 was more striking and the whole heart was encased in calcium. Here there was an old caseous hilar lymph gland and healed pleurisy with some non-specific but probably tuberculous chronic inflammatory changes in the lung. No evidence of rheumatic infection was found in either case and the evidence, admittedly circumstantial, was that the cause of both cases was tuberculosis.

The aetiology of most cases of constrictive pericarditis is, in fact, still in debate. Since at post-mortem one finds only scar tissue, and the damage done by this scar tissue, it is exceedingly doubtful if positive proof of an aetiological factor can ever be forthcoming in every case. One has to rely entirely on indirect evidence. In our sixteen cases of tuberculosis of the heart there are included six cases of chronic pericarditis. Two of these cases with calcification of the pericardium have just been discussed. All the other cases had associated active tuberculous infection in some other part of the body. Case 11 had tuberculosis of the lung, tracheo-bronchial glands, both pleural membranes, peritoneal lymph glands, and peritoneum. Case 12 had tuberculosis of the lungs, bronchi, pleura, and peritoneum. Case 13 had tuberculosis of the lungs, pleural cavities, and both adrenal glands with resultant Addison's disease. It should be pointed out that this case also showed evidence of
previous rheumatic infection which may well have been the cause of the pericarditis, but the evidence seems to indicate that it actually was a tuberculous pericarditis. And Case 14 had chronic fibroid phthisis with cavitation of both lungs.

No one denies that tuberculosis can and does produce constrictive pericarditis in many cases. But in just what percentage of cases of this syndrome can the tubercle bacilli be implicated? That is the question which cannot be satisfactorily answered.

This problem is discussed at length by Blalock and Levy (1938). To put the problem in its proper perspective, the following authors are quoted:

Wells (1902): "Many cases of adherent pericardium are probably of tuberculuous origin although not showing any anatomic characteristics of tuberculosis. However, it is to be understood that this is not the only cause."

Lilienthal (1925): "Few, if any, of the calcifications of the pericardium are of tuberculuous origin."

Sprague, Birch, and White (1932): "Tuberculosis of the insidious type is the most probable cause of the constrictive pericarditis of Pick."

White (1935) reported fifteen cases of constrictive pericarditis and wrote the following: "The etiology of chronic pericardial disease can be assigned as follows: Tuberculosis in two (questionable in two others), pneumonia with polyserositis (including both pleuritis and pericarditis) in two, sepsis in one, rheumatism in none, uncertain or unknown in ten (in five of which there was a definite history of acute pericarditis)."

And finally, Blalock and Levy (1938) write: "Of the nineteen patients with undoubted constrictive pericarditis which have been studied—eleven were found to be tuberculous in origin and five others were believed to be" and "It is likely that this disease (i.e. tuberculosis) accounts for the majority of cases of chronic constrictive pericarditis." Regarding the possibility of the condition being of rheumatic origin, they state: "Our experience and particularly that of White in Boston, makes one wonder seriously if rheumatic infection ever results in marked constrictive pericarditis." The strongest evidence that rheumatism can not be regarded as a cause of constrictive pericarditis would appear to come from White (1935), who states: "A series of 1,000 children with rheumatic infection studied—and followed over a period of ten years, has shown in not a single instance any evidence of chronic constrictive pericarditis, even though the heart was often seriously involved in other respects, and even though acute pericarditis had been noted in many cases during their acute rheumatic infection."

Thus it would appear that at any rate rheumatic fever cannot be incriminated as a cause of this syndrome.

*That Developing Independently:*

There remains one other variety of tuberculosis of the heart, namely, that condition which is said to develop independently. Briefly this is a condition which "... presents the bread and butter appearance. No other active foci of tuberculosis are found in the body" (Thompson, 1933). In this rather rare variety the peri-
carditis is supposed to be either the only or the oldest tuberculous lesion and the condition is called “primary tuberculosis of the pericardium.” It occurs in elderly patients and the tuberculous nature of the condition is usually unsuspected clinically. Thompson notes the following points and gives as his opinion, that it should be possible to recognise clinically that it is, in fact, a form of tuberculosis.

1. The average age is over 60 years.
2. The onset is insidious in patients without previous history of cardiac symptoms or evidence of previous rheumatic disease, syphilis or arterio-sclerotic heart disease.
3. There is unexplained fever in all cases.
4. There is a rapidly progressing increase in symptoms of cardiac insufficiency, proceeding relentlessly to a fatal termination within a very few months of the appearance of the first symptom. “It is precisely this rapidly fatal evolution and this striking failure to respond to treatment that identified this disease.”

Some authors consider that in this condition tuberculous infection occurs only in the pericardium and not in other tissues, unless it so happens that spread from pericardium to other parts of the body takes place. This view seems most unreasonable and it would appear much more likely that the true primary tuberculous focus, for example in lungs and mediastinal glands, had healed and was no longer demonstrable clinically or at post-mortem examination. The term “anatomically primary cases,” which is often applied to this group is therefore not a good one. The fact remains, however, that the pericarditis may be the only active tuberculous focus found and when this is so, the condition may be called primary tuberculosis of the pericardium. In Cases 8 and 9, the pericarditis was the dominating feature and although both patients had caseous lymph nodes they could probably be included in this group. One patient was aged 60 and the other 63 years.

**Summary**

1. Cardiac tuberculosis is quite a rare condition. Only sixteen cases were found among the records of 3,500 cases post-mortem in the last ten years in the Institute of Pathology.
2. Tuberculous endocarditis is extremely rare and it would appear that only four cases have ever been recorded. Endocardial tuberculosis, however, is quite frequently found as part of a general miliary tuberculosis or as an extension inwards of a tuberculous pericarditis.
3. Tuberculosis of the myocardium may be part of a general miliary tuberculosis or it may be the result of spread from a very active tuberculous pericarditis.
4. The usually accepted picture of tuberculous pericarditis with effusion will only be found if hypersensitivity to the tubercle bacillus is associated.
5. It would appear that:
   - (a) Chronic adhesive pericarditis, where the two layers of pericardium are partially or completely fused together, is often due to tuberculosis.
   - (b) Chronic mediastino-pericarditis, where the two layers of pericardium as well as being glued together are strongly attached to other mediastinal structures with consequent hampering of the heart’s action as it attempts to contract, is seldom due to tuberculosis.
(c) Polyserositis, which may be followed by constrictive pericarditis, is very often due to tuberculosis.

(d) Constrictive pericarditis may present as either a superior or an inferior mediastinal pressure syndrome and is most commonly due to tuberculosis.

(e) Calcified pericardium is a subvariety of constrictive pericarditis and so is also most commonly due to tuberculosis.

(f) Rheumatic fever seldom, if ever, causes constrictive pericarditis.

6. The important literature on the subject is briefly reviewed.

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Review


Perhaps the best features of this book are its arresting title and the foreword by Professor Sir Cyril Burt. Dr. von Andics has analysed the case-histories of one hundred persons who have been seen in the Potzel Clinic in Vienna or its branches, most of whom came from the poorer classes of society. All had failed—obviously—in their attempts to commit suicide, and in some cases these do not seem to have been very determined attempts. As might be expected, they had become weary of life for such causes as maladjustment to their environments. Forty-three per cent. were between the ages of 20 and 40, and forty-three per cent. were males. Only eleven of seventy-eight cases were regarded as of normal or supra-normal sexuality. The author does not give any very clear idea of "the meaning of life," thus leaving the impression that her book title is barmecidal.
Some Aspects of the Pathogenesis of Cardiac Tuberculosis

Plate 1

(Case 6, female child aged 6 years)
Acute sero-fibrinous tuberculous pericarditis with extension into the myocardium.

Plate 2 (x50)

(Case 8, female subject aged 63 years)
Section through pericardium showing tuberculous pericarditis.