Chronic Pericardial Effusion Complicating Endomyocardial Fibrosis


Pericardial effusion is relatively common in tropical Africa, and because of the prevalence of tuberculosis in such developing countries most large sterile pericardial effusions are frequently regarded as tuberculous unless proof to the contrary is forthcoming. With the recent introduction of chemotherapy such a diagnosis demands the long-term exhibition of antituberculous drugs. It has been known for some years, however, that endomyocardial fibrosis may be accompanied by a pericardial effusion. Since then there have been reports of several short series of patients suffering from endomyocardial fibrosis some of whom had pericardial effusions. Abrahams confirmed that pericardial effusions were common when the right ventricle was severely affected by endomyocardial fibrosis, and pointed out some of the difficulties in diagnosis. Nevertheless, the association of endomyocardial fibrosis and pericardial effusion does not seem to be widely recognized, and certainly the frequency of this combination is not appreciated. Accordingly, we have selected from our files six recent examples of this "syndrome" for detailed report.

Clinical and Pathologic Data

The Patients

Of the six patients four were male and two were female; their ages ranged from 10 to 40 years. All were seen between September 1960 and November 1961, so that the condition cannot be considered to be rare. The majority had had symptoms for from 1 to 5 years before they first attended hospital, but in two (cases 5 and 6) the process seemed more acute, for they were first examined soon after the onset of the illness. The commonest presenting symptom was abdominal swelling, and five of the six patients complained of this. Dyspnea and cough were also frequent complaints, but chest pain was absent. The commonest provisional diagnosis made by the referring physician was tuberculosis in one form or another; the correct diagnosis was suspected in only one instance (case 6). Two patients died (cases 3 and 5), while the remainder have been followed for periods of from 10 to 23 months.

Clinical Features

All cases presented a remarkably similar clinical picture. The relevant facts are tabulated in table 1. Thus the arterial pulse was usually small and sometimes paradoxical. Atrial fibrillation was established in four patients, and in a fifth there was an atrial arrhythmia due to varying degrees of AV block with haphazard ventricular excitation (case 6). The central venous pressure, judged in the jugular veins, was always grossly raised, and systolic expansion was the rule—telling of tricuspid incompetence. In one patient (case 3) the greatly distended neck veins showed no visible pulsation. The most constant auscultatory finding was triple rhythm from the addition of a third heart sound; best heard at the cardiac apex, this third heart sound was widely propagated, and sometimes dominated the cardiac cycle. An apical systolic murmur was heard in only two patients (cases 3 and 4) and in only one instance was
### Physical Findings

<table>
<thead>
<tr>
<th>Case no.</th>
<th>Rhythm</th>
<th>Arterial</th>
<th>Systolic expansion</th>
<th>Venous</th>
<th>Mitral area</th>
<th>Tricuspid area</th>
<th>Base</th>
<th>Asites</th>
<th>Dependent edema</th>
<th>Liver</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. N.T.</td>
<td>Atrial fibrillation</td>
<td>B.P. (100/75)</td>
<td>Systolic</td>
<td>Triple rhythm, 3rd sound dominant, no murmurs</td>
<td>Systolic murmur, triple rhythm</td>
<td>Pulse</td>
<td>Gross</td>
<td>Minimal</td>
<td>2 inches below right costal margin, firm</td>
<td></td>
</tr>
<tr>
<td>2. B.N.</td>
<td>Regular</td>
<td>B.P. (120/75)</td>
<td>Systolic</td>
<td>Triple rhythm, no murmurs</td>
<td>Triple rhythm</td>
<td>Base</td>
<td>Systolic murmur, triple rhythm</td>
<td>Severe</td>
<td>Minimal</td>
<td>1 inch below right costal margin, firm</td>
</tr>
<tr>
<td>3. F.O.</td>
<td>Rapid atrial fibrillation, ectopic beats</td>
<td>B.P. (105/75)</td>
<td>Systolic</td>
<td>Very high venous pressure with no pulsation visible</td>
<td>Short systolic murmur, triple rhythm</td>
<td>Pulse</td>
<td>Severe</td>
<td>Minimal</td>
<td>2½ inches below right costal margin</td>
<td></td>
</tr>
<tr>
<td>4. K.M.</td>
<td>Atrial fibrillation</td>
<td>B.P. (100/80)</td>
<td>Systolic</td>
<td>Systolic murmur, triple rhythm</td>
<td>Triple rhythm</td>
<td>Base</td>
<td>Dual rhythm</td>
<td>Absent</td>
<td>2½ inches below right costal margin, firm</td>
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<tr>
<td>5. M.T.</td>
<td>Atrial fibrillation</td>
<td>B.P. (120/90)</td>
<td>Systolic</td>
<td>Triple rhythm, no murmurs</td>
<td>Dual rhythm</td>
<td>Base</td>
<td>Slight</td>
<td>Absent</td>
<td>3 inches below right costal margin, firm</td>
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<tr>
<td>6. F.F.</td>
<td>Irregular atrial arrhythmia</td>
<td>B.P. (100/60)</td>
<td>Systolic</td>
<td>Triple rhythm, no murmurs</td>
<td>Dual rhythm</td>
<td>Base</td>
<td>Absent</td>
<td>Absent</td>
<td>1½ inches below right costal margin</td>
<td></td>
</tr>
</tbody>
</table>

### Electrocardiography

Electrocardiograms were recorded in all patients. Atrial fibrillation was confirmed in four patients (cases 1, 3, 4, and 5) and atrial arrhythmia in a fifth (case 6). The presence of a large pericardial effusion does not modify nor add to the clinical picture.

### Radiology

The heart shadow was huge and globular, and immediately suggested the presence of a pericardial effusion. Pulmonary congestion was not a feature, and the peripheral fields were remarkably clear. Nevertheless, the orthodiastolic radiograph by itself was not diagnostic of pericarditis without effusion. A very similar shadow was seen in patients (cases 1, 3, 4, and 5) and atrial arrhythmia in a fifth (case 6). The presence of a large pericardial effusion does not modify nor add to the clinical picture.

### Endocarditis

The murmur of tricuspid incompetence elicited the auscultatory findings in each case, and mitral murmur was absent.
picture. Very occasionally, disease of the heart muscle of unknown etiology may be similar radiologically, although in this condition pulmonary venous congestion is usually present, and left ventricular enlargement is frequently distinguishable. Representative radiographs are illustrated in figures 1 and 2.

**Pathology**

It is convenient to discuss the morbid anatomy of the condition at this juncture, for the results of the physiologic studies and also some of the factors in the differential diagnosis are thereby more readily appreciated.

The gross pathologic features have been described thoroughly by Davies and Ball in detail. Two of the present series of patients died and were examined post mortem with findings that were in every way typical. In both instances the right ventricle alone was the seat of significant disease, the left ventricle being almost completely spared macroscopically. The fibrosis of the endocardium extended as a dense, white membrane from the apex of the ventricle, which was obliterated, up to the tricuspid valve. The septal papillary muscle of the tricuspid valve was incorporated in the fibrotic process, as was the valve cusp which it served, the latter being plastered to the ventricular wall. The tricuspid valve was thus rendered grossly incompetent and, in fact, appeared as a crescentic membrane covering about half of the atrioventricular communication. The outflow path of the ventricle was also heavily involved, but the process seemed to halt just short of the pulmonary valve, which was not affected. Apart from the ventricle another most conspicuous finding was the aneurysmal enlargement of the right atrium, the wall of which was of paper thinness in places. In both patients who died, the right atrium was also the site of extensive antemortem thrombus formation. Another striking feature was the deep groove on the surface of the heart marking the obliterated apex of the right ventricle. This external depression was noted by Davies and Ball, and we concur with them that it is caused by an indrawing of the anterolateral wall of the ventricle as the walls of the inflow tract of that chamber approximate. These points are well illustrated in figure 3.

**Physiologic Studies**

Cardiac catheterization was done in four patients (table 2).

Owing to the gross dimensions of the right atrium and the peculiar distortion of the tricuspid valve, it was difficult to catheterize the right ventricle, and on one occasion it proved

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**Figure 1**

*Case 1. Posteroanterior chest radiographs. Note persistence of effusion over 14-month period. (Checked by aspiration.)*

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impossible. Even when the right ventricle was successfully catheterized, it was possible to advance the catheter into the pulmonary artery in only one patient. The rise in central venous pressure was confirmed, for the right atrial pressure was very high. A "dip and plateau" or "square-root" form of right ventricular pressure tracing was the rule, the plateau frequently reaching 50 per cent of the systolic peak. The mean pressures were approximately the same throughout the right side of the heart. The hemodynamic adjustments in this disease will be reported in detail elsewhere. Here it is enough to stress that these data suggest restriction of diastolic filling by endocardial rigidity, and that ventricular filling—and hence right ventricular forward output—is maintained principally by the very high central venous pressure, for right ventricular systole must be relatively unimportant in this respect.

Pressure tracings from two patients (cases 1 and 5) are illustrated in figure 4, including those from one of the patients who died (case 5). The right atrial tracings show gross tricuspid incompetence and the diastolic plateau is obvious in the right ventricular tracings; the similarity of the atrial and ventricular pressures in both patients is also evident.

The figures for arterial oxygen saturation

Table 2

Results of Physiologic Studies

<table>
<thead>
<tr>
<th>Case no.</th>
<th>R.A. Absolute Mean</th>
<th>R.V. Pressures (mm. Hg)</th>
<th>P.A. Absolute Mean</th>
<th>% Oxygen saturation</th>
<th>Oxygen consumption (ml./min.)</th>
<th>Cardiac output (L./min.)</th>
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<tr>
<td>1. N.T.</td>
<td>30/18 24</td>
<td>36/12 27</td>
<td>36/17 27</td>
<td>90.5 49</td>
<td>350</td>
<td>6.1</td>
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<tr>
<td>4. M.K.</td>
<td>22/12 18</td>
<td>.  .</td>
<td>.  .</td>
<td>90 46</td>
<td>230</td>
<td>2.9</td>
</tr>
<tr>
<td>5. M.T.</td>
<td>24/19 23</td>
<td>23/12 18</td>
<td>.  .</td>
<td>. .</td>
<td>.</td>
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</tr>
<tr>
<td>6. F.F.</td>
<td>16/13 14</td>
<td>15/10 13</td>
<td>.  .</td>
<td>92 57</td>
<td>.</td>
<td>.</td>
</tr>
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</table>

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Figure 3

Case 5. Four views of the heart. A, left upper. The external anterior view showing the massive right atrium (A), and the groove on the anterolateral surface (B). B, right upper. View of the tricuspid valve (C) from the opened right atrium (A). The large antemortem clot (G) is well shown. C, left lower. Horizontal section through both ventricles about 1.5 inches above the apex and at right angles to the septum (F). The septal papillary muscle of the tricuspid valve (H) is incorporated in the fibrotic thickened endocardium (J) as is the valve cusp which it serves (I). D, right lower. The apices of the left (E) and right (D) ventricular cavities. A, right atrium; B, anterolateral groove; C, tricuspid valve; D, right ventricular cavity; E, left ventricular cavity; F, interventricular septum; G, clot in right atrium; H, septal papillary muscle of tricuspid valve incorporated in the fibrotic process; I, septal chordae and cusp of tricuspid valve incorporated in the fibrotic process; J, thickened endocardium.

in table 2 should also be noted; it will be seen that some degree of desaturation occurred in the three patients in whom it was measured. Abrahams\(^6\) first reported this feature of right ventricular endomyocardial fibrosis, and it was studied in greater detail by Abrahams and Parry.\(^9\) Although absolute proof is lacking, it seems very possible that this arterial oxygen desaturation is caused by a shunting of blood from the azygos system to the pulmonary veins, which thus bypasses the right heart. This shunt is initiated and maintained by the extreme rise in central venous pressure. The arguments concerning the genesis of the arterial oxygen saturation have been advanced elsewhere.\(^9\)

Clinical Course

It must be pointed out that all these patients were African and came from the lowest strata of society. Being illiterate, they did not appreciate temporal relations clearly, so
that no great reliance should be placed on historical facts in the individual case. Nevertheless, the onset of the illness was usually insidious, and symptoms may have been present for months or years before orthodox medical aid was sought; most of this period was spent sampling the various therapeutic skills provided by the “traditional practitioners.” Once they attended hospital regularly, the natural history could be studied factually, for no specific therapy appeared to influence the course of the disease. The course was usually protracted, because, contrary to expectation, the immediate prognosis regarding life is good. Indeed, one patient (case 1), who stated that his symptoms had been present for 5 years, has been followed for 15 months with no apparent deterioration. A striking feature is the persistence of the pericardial effusion, for in none of the four surviving patients has the effusion resolved (fig. 1). The ascites, once it has formed, follows an identical refractory course. The pattern of disease is thus no different from right ventricular endomyocardial fibrosis without effusion. In both of the fatal cases extensive ante-mortem thrombosis in the right atrium was found at necropsy. Atrial fibrillation was established in both of these patients and presumably favored the development of thrombosis. It seems likely that progressive enlargement of this clot eventually led to partial occlusion of the right atrial cavity and of the atrioventricular orifice, and brought the flow of blood through the right heart to a level incompatible with life. At the same time there would have been an extreme rise in venous pressure. Support is lent to this hypothesis by the finding in one patient (case 5) of a ruptured pericardial vein which had led to sudden hemorrhage into the pericardium and relative tamponade. We presume that this rupture was caused by the exceptionally high venous pressure consequent on thrombotic occlusion of the right atrium. Possibly the blood staining of the pericardial fluid, which is so

Figure 4

Pressure tracings from cases 1 and 5. Note “dip-and-plateau” form of right ventricular tracings, and large V wave in right atrial tracings. Note also the similar level of pressure in all parts of the right heart.
common in endomyocardial fibrosis, is produced by minor venous hemorrhages of a like genesis.

**Diagnosis**

There are two main diagnostic problems—firstly, the confirmation that a pericardial effusion is present and, secondly, the definition of the underlying heart lesion.

There are several ways in which the suspected presence of a pericardial effusion may be confirmed, but the simplest method is pericardial aspiration. With care, and use of the subxiphisternal approach, this is a simple, safe, and rapid procedure; all our patients were so treated. The analyses of the pericardial fluid and of the ascitic fluid, when this was obtained, are shown in table 3, together with the values for the serum proteins in the individual cases. The peripheral fluid was frequently blood stained and often contained an excess of lymphocytes. In addition, the protein content was high, ranging from 2.9 to 5.9 Gm. per cent and averaging 4.6 Gm. per cent. The appearance and composition of the ascitic fluid was very similar. The relative proportions of the various protein fractions in both serum and pericardial fluid were also alike, although the absolute values were always higher in the patient’s serum. Finally, culture of the fluid for organisms, including *Mycobacterium tuberculosis*, was always negative.

The presence of a pericardial effusion having been established, two clinical features should immediately suggest that the heart itself is also affected: firstly, a third heart sound, and secondly the raised jugular venous pressure, and more particularly the systolic expansion of the neck veins signifying tricuspid incompetence. In our experience in Nigeria the combination of tricuspid incompetence and pericardial effusion is pathognomonic of right ventricular endomyocardial fibrosis.

In the morbid anatomy, the gross enlargement of the right atrium in this condition was stressed. The proportions of this chamber in the individual case may be demonstrated by angiocardiography, although with experience the diagnosis may be made clinically without it. Figure 2A shows the posteroanterior radiograph of the chest in case 6; the conventional venous angio- cardiogram is shown in figure 2B. The gross dimensions of the right atrium are well shown, and the shadow of the pericardial effusion is clearly visible well outside the right atrial border.

The size of the right atrium can also be demonstrated at cardiac catheterization. Figure 5A shows the posteroanterior chest radio-

**Figure 5**

*Case 5. A, left. Posteroanterior chest radiograph. B, center. Posteroanterior chest radiograph after introduction of air into the pericardial cavity, taken with the patient lying on his left side. Note the thin pericardial membrane above the air bubble. C, right. Radiograph taken during cardiac catheterization. The cardiac catheter lies entirely within the right atrium, demonstrating the size of that chamber.*

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graph in case 5. The radiograph in figure 5C was taken during cardiac catheterization, and the cardiac catheter lay entirely within the grossly dilated right atrium. Again, though not strictly necessary for diagnostic purposes, cardiac catheterization, by virtue of the pressure valves and tracings, confirms the presence of endomyocardial fibrosis.

Discussion

Any proper consideration of endomyocardial fibrosis must take into account the soil in which the disease thrives. As has been stated, it is virtually limited to the impoverished, illiterate classes, a population exposed from infancy to the multiple ravages of protein malnutrition, bacterial infections, particularly the dysenteries, falciparum malaria, hookworm anemia, and numerous other helminthic and protozoal infestations. For social and economic reasons, and because of the shortage of medical manpower, these afflictions are all too often incurable as well as uncontrollable. Conditions are also ideal for the widespread dissemination of tuberculosis, which may now be said to have assumed epidemic proportions and to be the most important disease in the tropics, if not the most important "tropical disease." Its importance in this respect has, of course, been heavily underlined by the recent introduction of successful therapeutic agents, so that tuberculosis is now a curable disease. In the wake of these drugs have come great "propaganda" drives, re-emphasizing the prevalence, and devoted to the control and elimination of, tuberculosis from the tropical underdeveloped countries, sponsored and financed by bodies such as the World Health Organization. At times almost an emotional atmosphere exists; an attitude of mind is created whereby the doctor frequently succumbs to the temptation to treat a patient for tuberculosis for long periods of time purely on assumption and without definitive proof. Nor is he to be blamed for this, continually exposed as he is to such an environment. Laboratory facilities for diagnosis are scanty, time-consuming, and costly; the mental attitude of the doctor frequently is "This patient could well have tuberculosis, which I can at least treat, so why not go ahead?"

It will thus be seen that there is a prejudice in favor firstly of diagnosing, and secondly of treating for tuberculosis, on purely clinical grounds, without laboratory confirmation. In the particular problem under discussion there are other features that support such a presumptive diagnosis. The onset of the illness

<table>
<thead>
<tr>
<th>Case no.</th>
<th>Total</th>
<th>Albumen</th>
<th>α1</th>
<th>α2</th>
<th>β</th>
<th>γ</th>
<th>Total</th>
<th>Albumen</th>
<th>α1</th>
<th>α2</th>
<th>β</th>
<th>γ</th>
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<td>3.0</td>
<td>0.5</td>
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<td></td>
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<tr>
<td>2. B.N.</td>
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<td>1.85</td>
<td>0.58</td>
<td>0.96</td>
<td>1.44</td>
<td>3.67</td>
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<td>3. F.O.</td>
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<td></td>
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<td>1.94</td>
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<td>2.84</td>
<td>0.54</td>
<td>1.02</td>
<td>1.38</td>
<td>2.42</td>
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<td>0.5</td>
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<td>0.43</td>
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</table>
is insidious with slight fever and malaise. The chest radiograph suggests a pericardial effusion; the fluid, when aspirated, is either straw colored or blood stained, frequently contains an excess of lymphocytes, and always has a high protein content. The fact that culture of the fluid, when undertaken, is negative for Myco. tuberculosis, is of no diagnostic value, for the tubercle bacillus unfortunately seems to be very difficult to grow in the tropics, even when proper facilities are available. The high venous pressure, if noted, suggests cardiac tamponade, a recognized complication of tuberculous pericarditis, and the added presence of ascites merely enlarges the diagnosis to one of tuberculous "polyserositis." At our patients' age virtually the whole of this population has been exposed to the tubercle bacillus, so that a positive tuberculin test is of no diagnostic help. The absence of parenchymal lung lesions or of an enlarged gland for biopsy purposes is likewise of no significance.

So far we have dealt exclusively with the similarities between the two conditions; it is now time to deal with the factors helpful in the differential diagnosis. Of these, the first and most important is to be aware that endomyocardial fibrosis may present clinically as a pericardial effusion. Further than this, in our experience in Western Nigeria, it is a common method of presentation, and is indeed much commoner than tuberculous pericarditis, which is relatively rare in hospital practice.

When the clinical course of the two diseases is considered, certain difficulties arise. Endomyocardial fibrosis is relatively benign, and patients usually survive for years without visible deterioration. The course of tuberculous pericarditis, however, varies according to whether tubercle bacilli are found in the fluid—that is assuming that antituberculous chemotherapy is not given. Harvey and Whitehill reported on 20 patients who had bacteriologically proved tuberculous pericarditis with large effusions. The average duration of life from the onset of symptoms was 3.4 months. With the introduction of specific therapy this gloomy prognosis has been drastically improved.

Since we are concerned here with patients in whom tubercle bacilli were not found in the pericardial fluid, and in whom no other tuberculous lesion was demonstrated, these types of cases need not concern us further. Harvey and Whitehill showed that the prognosis of 34 cases, unproved bacteriologically, but clinically certain, was much better, for
only two died while in the hospital, although 21 had tuberculous lesions elsewhere in the body. Perhaps of more significance is the length of time for which a bacteriologically unproved tuberculous pericardial effusion may persist without specific therapy. Peel\textsuperscript{11} gave data on five such cases, and in these the effusion had absorbed in from 5 to 12 weeks, an average of 8 to 9 weeks. This relatively rapid absorption of the fluid is in contrast to the complete failure to absorb, which is a constant feature of endomyocardial fibrosis.

Turning to the clinical features, we find that the most important point in differential diagnosis is the presence of systolic expansion of the neck veins. As has already been stated, we consider this pathognomonic of endomyocardial fibrosis with effusion; the statement bears repetition.

Atrial fibrillation is uncommon in tuberculous pericarditis and when present is almost always transient and related to digitalis therapy.\textsuperscript{10} By contrast, permanent atrial fibrillation, unrelated to digitalis therapy, occurs in the majority of cases of endomyocardial fibrosis with effusion.

If the differential diagnosis is still in doubt, pericardial aspiration with air replacement may be of help. With this technic the thickness of the pericardium may be demonstrated. This procedure was carried out in one of our patients (case 5) and the resulting radiograph is shown in figure 5B. It will be seen that the pericardium is thin and certainly not of the thickness to be anticipated in tuberculous pericarditis of this duration. If sufficient fluid is replaced with air, some estimate of heart size may be made and the enlarged heart of endomyocardial fibrosis is in contrast to the normal size of the heart underlying a tuberculous effusion.

As has been stated, angiocardiography and cardiac catheterization may be useful in diagnosis, although with clinical experience they are by no means essential. Their main value lies in demonstrating the severity of the cardiac lesion. Similarly, pericardial biopsy should be completely unnecessary, although we had to submit one patient to thoracotomy for this purpose in 1960 (case 2).

It remains to exclude other forms of pericarditis—benign, nonspecific, and rheumatic, and that associated with malignant disease; these should raise no difficulties in differential diagnosis.

We have no firm views as to how the pericardial effusion in endomyocardial fibrosis is produced. It has been suggested\textsuperscript{4} that the disease starts as a pericarditis so that initially the effusion may be inflammatory in origin. Certainly the sustained high central venous pressure would favor the persistence of an effusion once formed, possibly by obstructing lymphatic drainage. It is perhaps pertinent here to point out that we have never seen a pericardial effusion when the left side of the heart is solely or predominantly the seat of the fibrotic process; it seems that the presence of severe right ventricular disease is essential, with the consequent gross rise in central venous pressure.

It may be thought that we have belabored unduly the question of distinguishing endomyocardial fibrosis from tuberculosis with effusion. We would reply that accuracy in clinical diagnosis is not an academic exercise, even in the modern laboratory age, and is particularly valuable where laboratory aids, in the widest sense, are scanty or nonexistent. Finally, long continued antituberculous therapy is not without complications and is very expensive, as well as irksome to the patient and doctor; we believe that with a more critical clinical approach the exhibition of these drugs to certain patients without tuberculosis can be prevented.

Summary

Six patients are described in whom right ventricular endomyocardial fibrosis was accompanied by a large pericardial effusion. This effusion did not appear to modify either the course of the disease or the physical signs, which included gross elevation of the venous pressure, tricuspid incompetence, and massive ascites with complete absence of dependent
edema. Atrial fibrillation was a very common finding.

Cardiac catheterization confirmed the presence of tricuspid incompetence and showed a "dip-and-plateau" contour in the right ventricular tracing. In addition the mean pressures recorded from the pulmonary artery and all parts of the right heart were virtually the same.

Two patients died and were examined post mortem. The findings included gross enlargement of the right atrium, complete disorganization of the tricuspid valve, and progressive obliteration of the right ventricular cavity. It is suggested that death may be due to gradual occlusion of the right atrium by antemortem thrombosis.

The condition must be distinguished from tuberculous pericarditis. Important factors in the differential diagnosis are the presence of tricuspid incompetence with systolic expansion of the neck veins, atrial fibrillation, and the demonstration of cardiac enlargement, due to aneurysmal dilatation of the right atrium.

Acknowledgment

The pressure tracings were recorded with apparatus obtained through a generous grant from the Nuffield Foundation, to whom we express our thanks. We are also grateful to the Medical Illustration Department, University College, Ibadan, for the illustrations, and, finally, we wish to thank those of our colleagues who kindly referred patients to us.

References


The Effects of Atmospheric Electricity on Muscular Motion

Now, indeed, the effects of storm electricity, as they say, having been investigated, not of thunder and lightning alone, it occurred to us also to test what sheet lightning and northern lights would produce in animals prepared in the customary way. Therefore we adapted our animals to an atmospheric conductor not only during lightning but during northern lights. But no contractions were ever then produced, perhaps because either such coruscations do not depend on electricity or, if they do, either in too remote a place, or they occur for some very different reason than thunderbolts. But these are questions for the physicists.—Luigi Galvani. Commentary on the Effect of Electricity on Muscular Motion. Translated by Robert Montraville Green, M.D. Cambridge, Massachusetts, Elizabeth Licht, Publisher, 1953, p. 39.
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