Primary Myocardial Disease*

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During the past several years, we have observed a number of patients who developed congestive heart failure in the absence of evidence of significant systemic arterial hypertension, coronary artery disease, or valvular disease. It is the purpose of this paper to describe a group of 23 such patients in whom no satisfactory explanation of their heart disease was found at necropsy or at exploratory thoracotomy. Twenty have come to necropsy in the University of Cincinnati Hospitals, and three have had a myocardial biopsy performed during an exploratory thoracotomy. Eleven of these patients were examined during life by the senior author. These patients comprise the group referred to in this paper as idiopathic myocardial disease. Other terms for this type of heart disease have been employed. They include: primary myocardial disease,† idiopathic cardiac hypertrophy,‡ cardiomyopathy,§ and myocardosis.¶ This communication is an extension of a previous report on this subject.¶

For inclusion in this group, it was required that the patient have advanced heart disease manifested by cardiac enlargement and congestive heart failure. Clinically, the most difficult condition to exclude as a possible cause of heart failure was that of hypertension. Five of the patients we are reporting had at one time diastolic blood pressure of 100 mm. Hg or higher; however, in no instance was there sustained hypertension, and, as a rule, hypertension was present only during bouts of congestive heart failure and was usually of only a few weeks’ duration. From the pathologic studies, the exclusion of pericardial disease and valvular disease presented no problem. The most difficult decision with regard to the pathologic criteria lay in the area of coronary artery disease. Only three of the 20 necropsied patients had moderately numerous sclerotic plaques in the coronary arteries; however, significant narrowing of the lumen of the coronary arteries was absent in all patients included in this study. With regard to the myocardium itself, patients with cardiac amyloid disease and those with focal accumulations of inflammatory cells were not included. Thus myocarditis, at least in an active phase, could be reasonably well excluded from this group under discussion. No patient in this group had hemochromatosis or sarcoidosis. None had severe pulmonary emphysema or pulmonary fibrosis at necropsy; patients with glomerulonephritis, nephrosclerosis, or severe pyelonephritis were also excluded. Further, there was...
no necropsy evidence of such “collagen” diseases as lupus erythematosus, scleroderma, or dermatomyositis in these patients.

Table 1 summarizes the clinical data of the 23 patients who are reported here. It will be noted that their ages were from 18 months to 68 years at death; three are still living. Fourteen of these patients were white and nine were Negroes; 19 were males and four were females. The sex distribution means little since ten of these patients were obtained from the Cincinnati Veterans Administration Hospital where few women are admitted. It is of interest that there was a history of alcoholism in eight of these patients; in three of these, there was also nutritional cirrhosis of the liver at necropsy, and two others had fatty livers. A familial history of similar heart disease was found in only one patient. Very impressive was the long duration of congestive heart failure in many patients, 11 of whom had had symptoms for five years or more. Diastolic apical gallop rhythm was common and was described in 13 patients at sometime during the course of the illness. Systolic murmurs were also common and were described in 14 of the patients in this group. The systolic murmurs were usually apical pansystolic murmurs attributable to mitral incompetence, but were occasionally pansystolic murmurs located close to the left sternal border and increased on inspiration indicating probable relative tricuspid incompetence. As a rule, these patients tended to respond well during the first few bouts of congestive heart failure to rest, digitalization, and diuretics, and their murmurs and gallop rhythms tended to become less prominent or to disappear. In one patient, a paradoxical pulse of significant degree was observed. The ocular fundi showed

<table>
<thead>
<tr>
<th>No.</th>
<th>Name</th>
<th>Age at Death</th>
<th>Sex</th>
<th>Race</th>
<th>Blood Pressure**</th>
<th>Known Duration of Heart Disease</th>
<th>Clinical Diagnosis</th>
</tr>
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<tbody>
<tr>
<td>1</td>
<td>E.B.</td>
<td>18</td>
<td>F</td>
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<td>98/44</td>
<td>7 months</td>
<td>Idiopathic myocardial hypertrophy?</td>
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<tr>
<td>2</td>
<td>M.M.</td>
<td>15</td>
<td>F</td>
<td>N</td>
<td>90/60</td>
<td>7 years</td>
<td>Primary myocardial disease</td>
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<tr>
<td>3</td>
<td>G.P.</td>
<td>20</td>
<td>M</td>
<td>W</td>
<td>110/70</td>
<td>5 years</td>
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<tr>
<td>4</td>
<td>H.D.</td>
<td>28**</td>
<td>M</td>
<td>W</td>
<td>120/65</td>
<td>13 years</td>
<td>Aortic stenosis</td>
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<td>V.H.</td>
<td>30</td>
<td>F</td>
<td>N</td>
<td>112/90</td>
<td>1 month</td>
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<tr>
<td>6</td>
<td>W.H.</td>
<td>36**</td>
<td>M</td>
<td>N</td>
<td>110/70</td>
<td>7 months</td>
<td>Myocarditis</td>
</tr>
<tr>
<td>7</td>
<td>P.Y.</td>
<td>37</td>
<td>M</td>
<td>N</td>
<td>102/90</td>
<td>2 years</td>
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</tr>
<tr>
<td>8</td>
<td>C.F.</td>
<td>38</td>
<td>M</td>
<td>N</td>
<td>110/80</td>
<td>2 years</td>
<td>Possible mitral insufficiency</td>
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<tr>
<td>9</td>
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<td>41</td>
<td>M</td>
<td>N</td>
<td>100/90</td>
<td>6 years</td>
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<tr>
<td>10</td>
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<td>43</td>
<td>M</td>
<td>W</td>
<td>120/75</td>
<td>3 weeks</td>
<td>Congestive heart failure of unknown etiology, Hepatic cirrhosis</td>
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<tr>
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<td>F.G.</td>
<td>43</td>
<td>M</td>
<td>W</td>
<td>125/90</td>
<td>not known</td>
<td>Hepatic cirrhosis</td>
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<td>W</td>
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<td>46</td>
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<td>M</td>
<td>N</td>
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<td>Congestive heart failure of unknown etiology, Collagen disease?</td>
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<tr>
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<td>M</td>
<td>W</td>
<td>120/80</td>
<td>3 years</td>
<td>Myocardial infarction, Pneumonia</td>
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<tr>
<td>17</td>
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<td>50</td>
<td>M</td>
<td>W</td>
<td>110/80</td>
<td>7 years</td>
<td>Coronary artery disease</td>
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<td>18</td>
<td>W.M.</td>
<td>60</td>
<td>M</td>
<td>W</td>
<td>120/60</td>
<td>14 years</td>
<td>Rheumatic heart disease, Aortic stenosis and insufficiency</td>
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<tr>
<td>19</td>
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<td>60</td>
<td>F</td>
<td>W</td>
<td>100/60</td>
<td>not known</td>
<td>Hepatic cirrhosis</td>
</tr>
<tr>
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<td>E.W.</td>
<td>60</td>
<td>M</td>
<td>W</td>
<td>110/85</td>
<td>2 years</td>
<td>Arteriosclerotic heart disease</td>
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<tr>
<td>21</td>
<td>J.H.</td>
<td>64</td>
<td>M</td>
<td>W</td>
<td>95/65</td>
<td>16 years</td>
<td>Myocardial infarction</td>
</tr>
<tr>
<td>22</td>
<td>E.C.</td>
<td>66</td>
<td>M</td>
<td>N</td>
<td>120/96</td>
<td>9 years</td>
<td>Possible rheumatic heart disease</td>
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<tr>
<td>23</td>
<td>H.K.</td>
<td>68</td>
<td>M</td>
<td>W</td>
<td>110/60</td>
<td>6 years</td>
<td>Arteriosclerotic heart disease</td>
</tr>
</tbody>
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*Patient still living. Myocardial biopsy obtained at exploratory thoracotomy.

**Blood pressure recorded during final hospitalization.
no changes greater than Grade II. In no patient was there significant anemia. Cardiac fluoroscopy was performed in 13 patients, a paradoxical pulse of significant diminished pulsations of the heart.

During life, idiopathic myocardial disease may be mistaken for the more ordinary forms of heart disease. It may be seen in Table 1 that eight of these patients had a clinical diagnosis of rheumatic heart disease, and five were thought to have coronary artery disease. We have already discussed in a limited way the problem of the differential diagnosis of primary myocardial disease and hypertensive cardiovascular disease, and it must be stated that some patients who develop congestive heart failure have at this time an increase of systemic vascular resistance and may have moderate diastolic hypertension. Because of the murmurs at the cardiac apex or at the tricuspid area, patients with idiopathic myocardial disease may be thought to have rheumatic heart disease. In two patients in our group, exploratory thoracotomy was done because of a mistaken diagnosis of valvular heart disease. One patient (H.D.) had a Grade III ejection systolic murmur at the cardiac apex. This murmur was audible also at the left sternal border and in the primary aortic area, but was fainter in these locations. The murmur was considered to be consistent with aortic stenosis. His electrocardiogram showed left ventricular hypertrophy and left heart catheterization was performed. Left ventricular pressure was 190/15 mm. Hg and aortic pressure was 105/65 mm. Hg. The systolic pressure gradient of 85 mm. Hg was thought to be consistent with aortic stenosis and exploratory thoracotomy was performed. No evidence of obstruction in the aortic valve or outflow tract of the left ventricle was found. Another patient (L.S.) had congestive heart failure with atrial fibrillation. A clicking sound in early diastole was heard at the cardiac apex and the left sternal border. This was thought to be an opening snap of the mitral valve. There was left atrial enlargement on fluoroscopy and these observations were thought to be consistent with rheumatic mitral stenosis without a diastolic murmur. The possibility, however, was also considered that he might have idiopathic myocardial disease, but it was
decided that exploratory thoracotomy should be performed to exclude mitral stenosis. On exploratory thoracotomy no evidence of mitral valve disease was found. Figure 1 shows roentgenologic evidence of left atrial enlargement in an 18-month-old baby (E.B.) with idiopathic myocardial disease as evidence that mitral stenosis may be simulated radiologically as well as on physical examination. Another radiologic observation which may suggest rheumatic heart disease is that of Kerley lines. Figure 2 illustrates the chest x-ray film of a 20-year-old man (G.P.) who died of idiopathic myocardial disease after five years of congestive heart failure. Because of the Kerley lines shown here and because of the mid-diastolic apical gallop rhythm which was at times mistaken for the murmur of mitral stenosis, the patient was often thought to have rheumatic heart disease during life. The auscultatory findings in primary myocardial disease were reviewed by Harvey and Perloff.5

In addition, some of these patients may be thought to have pericardial disease. Figure 3 shows radiologic evidence of extreme cardiac dilatation in a young girl of eight years (M.M.) who had idiopathic myocardial disease. This x-ray film suggests the possibility of pericardial effusion. In addition, the feeble pulsations of the dilated heart observed during fluoroscopy also raise the question of pericardial disease. Some of these patients may have a paradoxical pulse similar to that seen in pericardial disease. Furthermore, these patients may have a very high diastolic pressure in the right ventricle exceeding one-third of the systolic pressure, also suggesting the possibility of cardiac constriction. Figure 4 illustrates an angiocardiographic study which was made in a 36-year-old Negro man who was admitted with paradoxical pulse, gallop rhythm, a very high venous pressure, and relatively clear lung fields, raising the question of pericardial disease. As illustrated in this figure, there is no evidence of increased pericardial thickness or effusion and it was concluded that he most likely had idiopathic myocardial disease, although coronary artery disease remains a possibility.

Rapid increase in the size of the cardiopericardial silhouette may also suggest pericardial effusion. Figure 5 shows the dramatic change in heart size that may occur in an eight-month period. The chest x-ray film, reproduced on the left, is that of a ten-month-old child (E.B.); and the one

Figure 2B: This x-ray film was taken during his final admission at the age of 20 years and shows generalized cardiomegaly with additional pericardial effusion.

Figure 3: Chest x-ray film of patient M.M. at the age of eight years showing gross cardiac dilatation simulating pericardial effusion.
FIGURE 4: Angiocardiogram of 36-year-old man who had paradoxical pulse and persistent elevation of venous pressure simulating pericardial disease. No increased thickening of the pericardium is shown on this study.

.on the right is from the same child eight months later, showing the marked cardiac dilatation that took place during this period of time.

Figure 6 illustrates a positive angiocardiographic study showing evidence of pericardial effusion in a patient without idiopathic myocardial disease. The wide area along the right atrial border which is not opacified by dye is seen clearly.

In older patients, the differential diagnosis of idiopathic myocardial disease and coronary artery disease may be extremely difficult. Although none of the patients whom we have studied has complained of anginal pain or has had a history of previous myocardial infarction, they often show electrocardiographic abnormalities which may be similar to those seen in coronary artery disease. However, in no instances have patterns pathognomonic of myocardial necrosis been seen. Eight of these 23 patients had atrial fibrillation; one developed atrial flutter after exploratory thoracotomy; four had P waves suggestive of right atrial enlargement; abnormal left axis deviation was found in six and abnormal right axis deviation was found in three; left ventricular hypertrophy was found on the electrocardiogram in eight and left bundle branch block was found in four; the QRS complex of the electrocardiogram was of low voltage in five subjects. In none of the patients reported here was the electrocardiogram entirely normal. Figure 7 illustrates an example of an electrocardiogram which may simulate myocardial disease.
emboli; and five, pulmonary emboli. Focal areas of myocardial fibrosis were found in 15 patients (Fig. 8) (O.M.). None had large areas of scarring, and none had extensive subendocardial fibrosis or fibroelastosis (Fig. 9). There was no evidence of amyloid disease either on routine hematoxylin and eosin stain, or on crystal violet stain. In eight patients, where the examination was made, the skeletal muscle was normal. The left ventricle was increased in thickness in 19 of the 20 necropsied patients and was dilated in the remaining one. The right ventricle was increased in thickness in 11 of the necropsied patients.

**Discussion**

Idiopathic myocardial disease is not a rare disorder. Since our original report of 18 patients submitted one year ago, we have studied five more patients who fall into this category, in two of whom the diagnosis has been confirmed at necropsy, and in the other three by exploratory thoracotomy and myocardial biopsy. In addition, we have under surveillance a number of other patients in whom idiopathic myocardial disease is the probable diagnosis, but has not yet been confirmed histologically. The recognition of the frequency of this form of heart disease is important because these patients are often thought to have more common forms of heart disease, such as coronary artery disease, pericardial disease, rheumatic or congenital valvular disease, or hypertensive cardiovascular disease; and in some instances the patients may be subjected to unnecessary thoracotomy in an attempt to correct nonexistent valvular disease, or to unnecessary needling of the heart in an attempt to remove pericardial effusion. In some instances the mature age of the patient and electrocardiographic evidence of left bundle branch block, or the presence of QS complexes in the electrocardiogram may result in the diagnosis of coronary artery disease.

Goodwin et al. have divided patients with idiopathic myocardial disease into three clinical groups: (1) those simulating
**Figure 7:** A selected electrocardiogram from patient M.B. showing abnormal left axis deviation, atrial fibrillation, and premature ventricular contractions.

**Figure 8:** Microscopic sections of myocardium of patient O.M. showing rather extensive myocardial fibrosis. This degree of fibrosis was not present throughout the myocardium of this patient, and was more extensive than that seen in the majority of patients in this series.

**Figure 9:** Gross photograph of the previously opened left ventricle of patient C.F. showing a marked degree of dilatation with no evidence of endocardial sclerosis. Portions of the normal mitral and aortic valves are included.
ischemic heart disease; (2) those simulating obstructive heart disease; (3) those simulating constrictive pericarditis. In our group of 23 patients, only one simulated aortic stenosis and only one simulated constrictive pericarditis; the others simulated ischemic or hypertensive or nonobstructive valvular disease, or pericardial effusion.

Other reports have stressed the frequency of pulmonary and systemic emboli arising from mural thrombi in idiopathic myocardial disease. Although four of our patients had systemic arterial emboli and five had pulmonary emboli, in only one patient was the embolism recognized clinically.

The etiology of this form of heart disease is uncertain. In recent years, a number of reports have emphasized the familial incidence of idiopathic cardiac hypertrophy. However, only one patient in our group had a family history of similar heart disease. Because of the wide age ranges represented and the varying history with regard to nutrition and alcoholism, it is believed likely that the category of idiopathic myocardial disease represents several etiologic forms of heart disease. Although none of the patients in our study had significant accumulation of inflammatory cells in the myocardium to suggest that myocarditis was the cause, it is known that chronic myocarditis may produce a similar clinical picture. It is possible that in some of our patients the fibrosis in the myocardium was the remnant of a previous inflammatory process and that the process may have begun as a form of myocarditis of unknown cause. In our patients, there was no evidence of neuromuscular disorders which may be associated with myocardial disease, such as Friedreich's ataxia, or progressive muscular dystrophy. None of the patients in our group had specific evidence of accepted causes of myocardial dysfunction; in particular, there was no evidence of Aschoff nodules to suggest rheumatic fever; there was no evidence of granuloma formation to suggest sarcoidosis; there was no evidence of iron deposit to suggest hemochromatosis, of glycogen deposit to suggest Von Gierke's disease, or of amyloid deposit to suggest cardiac amyloidosis. Subendocardial fibroelastosis, although present focally, principally over the papillary muscles in some of our patients, was not disproportionate to that commonly seen in long-standing congestive heart failure with cardiac dilatation, and in no instance did the hearts resemble those seen in the adult form of subendocardial fibroelastosis. There was a history of alcoholism in eight patients of our group and one must consider whether or not this could be related etiologically to the myocardial disorder. Some authors have incriminated chronic alcoholism without beriberi as a cause of myocardial degeneration. None of our patients presented a clinical picture of acute beriberi and chronic beriberi heart disease is not associated with specific histologic alterations in the myocardium which would permit its recognition without the clinical syndrome. Since three patients in our group had evidence of nutritional cirrhosis and two had fatty livers, one must also consider the relationship of nutritional cirrhosis to myocardial disease. Lunseth, Olmstead and Abboud described 12 instances of idiopathic cardiac enlargement in 108 patients dying of portal cirrhosis. These authors postulated that the cardiac enlargement was related to the increased cardiac output often found in nutritional cirrhosis of the liver. It is possible that cirrhosis of the liver could be related in some way to idiopathic myocardial disease in a few of our patients. In summary, however, it is believed that the etiology of the myocardial failure in the patients described in this group can not be explained at present.

Summary

This paper describes 23 patients who had congestive heart failure without clinical or pathologic evidence of the primary cause. Twenty of these patients were necropsied; three had myocardial biopsy. The ages of these patients were from 18 months to 68 years at death. Nineteen were males and four were females. Eight had heart
failure for five years or more; eight were alcoholic; three had nutritional cirrhosis. Transient mitral or tricuspid systolic murmurs, and apical diastolic gallop rhythms were common. Atrial fibrillation was present in eight. Right heart catheterization in five patients showed evidence of decreased cardiac output and biventricular failure. All had left ventricular enlargement at necropsy. Mural thrombi were present in 12; four had systemic emboli; five had pulmonary emboli. There was no evidence of coronary disease, myocarditis, or subendocardial fibroelastosis. Idiopathic myocardial disease is of clinical importance because it may simulate coronary artery disease, hypertensive heart disease, pericardial effusion, or rheumatic heart disease. The cause is unknown. Only one patient in this group had a family history of similar heart disease.

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Dr. Philip Cabaud, Department of Pathology, Brooklyn Hospital, Brooklyn, New York, furnished the data for patient F.S.

RESUMEN

Este trabajo describe a 23 enfermos que tuvieron insuficiencia cardíaca congestiva sin evidencia clínica de la causa primaria. Se hicieron necropsias en 20 de esos enfermos; tres tuvieron biopsia del miocardio. Las edades de esos enfermos fueron de 18 meses a 68 años. Diecinueve eran hombres y cuatro mujeres.

Ocho de esos enfermos habían tenido insuficiencia cardíaca por cinco o más años. Ocho eran alcohólicos; tres tenían cirrosis nutricional. Fueron comunes como síntomas: soplos mitrales o tricuspidos, disantímeros galopantes diastólicos apicales. La fibrilación auricular se observó en ocho enfermos.

La cateterización del corazón derecho dió evidencia de rendimiento cardíaco disminuido y de insuficiencia biventricular. Todos tenían crecimiento ventricular izquierdo en la necropsia. Se encontraron trombos murales en 12; cuatro tenían embolos de la circulación general. Cinco los tenían pulmonares. No había evidencia de enfermedad coronaria, miocarditis o de fibroelastosis subendocárdica.

La enfermedad idiopática miocárdica es de importancia clínica porque puede simular enfermedad coronaria. La causa es desconocida. Solo un enfermo de este grupo tenía antecedentes familiares de semejante enfermedad cardíaca.

RESUME

Description de 23 malades ayant une défaillance cardiaque congestive sans preuve clinique ou anatomique de la cause initiale. 20 de ces malades ont été autopsiées; 3 ont eu une biopsie musculaire. L'âge des malades varie de 18 mois à 68 ans; 19 sont du sexe masculin, 4 du sexe féminin. 8 de ces malades avaient une défaillance cardiaque durant depuis 5 ans au plus. 8 étaient alcooliques, 3 avaient un myocarde nutritionnel. Des souffles systoliques mitraux ou tricuspidiens, et un galop diastolique de la pointe étaient fréquents. La fibrillation auriculaire était présente chez huit malades. Le cathétérisme cardiaque droit chez 5 malades a montré une diminution du débit cardiaque et une défaillance bi-ventriculaire. Tous avaient une augmentation du ventricule gauche à l'autopsie; des mural thrombi étaient présent chez 12; 4 avaient des embolies périphériques et 5 des embolies pulmonaires. Il n'y avait pas de signe de maladie coronarienne, de myocardite, de fibroelastose sous-endocar- dique. La maladie myocardique primitive a une grosse importance clinique car elle peut simuler les cardiopathies dues à la sclérose coronarienne, l'hypertension, l'épanchement péricardique ou le rhumatisme cardiaque. La cause est inconnue.

Un seul des malades de ce groupe avait une histoire familiale d'affection cardiaque semblable.

ZUSAMMENFASSUNG


REFERENCES


THE UNCLASSIFIED MYCOBACTERIA

The studies which have been reviewed by the authors seem to prove that mycobacteria constitute a single group of organisms, but with gradations of progressive change. Their final and most clear-cut stage, involving loss of many of their biologic properties, but which happens to be toxic or susceptible animals, is represented by pathogenic strains. Such a change is reversible and, therefore, a saprophytic strain may, under certain conditions, turn to a virulent one, and vice versa. We have brought this about in vivo, so there is little doubt that it may also take place in vivo.

This concept of phylogenetic unity of all mycobacteria has been defended by a great many investigators since the beginning of the century and has been fully proved by recent research. Therefore, it is not surprising that the so-called atypical or unclassified mycobacteria are found to originate from pathogenic strains whether spontaneously or under the influence of antimicrobial drugs. The contrary opinion is based on theoretic considerations which are quite reasonable but which, for lack of convincing proof, cannot be taken into consideration at the moment.

The claim for placing the unclassified mycobacteria in a well defined group independent from the pathogenic one is not justified because they do not possess any fixed characteristic which was not known long ago, except the very disappointing results of the actual antimicrobial drugs on them. In fact, they are no more than attenuated and resistant bacilli, much more frequently observed today because of their progressive adaptation to antimicrobials.

Moreover, the disease caused by them remains indistinguishable from tuberculosis and, hence, must be handled similarly, also from the epidemiologic standpoint. Those who are claiming lack of contagion assume a great responsibility indeed because data offered by them show that when corresponding specific tuberculins were used, the infection index among the household contacts equals that of tuberculosis.