Myocarditis

By CLARENCE E. de la Chapelle, M.D., and CHARLES E. Kossmann, M.D.

In THE PAST, many practicing physicians in this country as well as abroad designated almost every disease of the heart muscle as "myocarditis." In the majority, the disease was probably replacement fibrosis of the myocardium. The latter was undoubtedly the result of coronary arteriosclerosis or of an inadequate coronary blood supply to a myocardium hypertrophied by arterial hypertension.

In time the pendulum swung to the other extreme. Few clinicians made a diagnosis of myocarditis except in the presence of rheumatic fever even though many of them were aware of the present-day meaning of the term, namely, inflammation of the myocardium.

Data assembled in the past 10 to 15 years show that myocarditis is not uncommon at postmortem examination. Therefore, the lesion cannot be a rarity in clinical medicine. The one individual who has made physicians aware of this fact by means of his splendid, meticulous pathologic studies and reviews is Saphir.1, 2, 9, 14 Renewed interest in the clinical recognition of myocarditis has also been stimulated by the innumerable reports of electrocardiographic abnormalities, particularly in the presence of acute infectious diseases, from military hospitals during and following World War II. The rather liberal use of the electrocardiograph in the clinical work-up of patients in the hospitals of the Armed Forces undoubtedly contributed to the frequency of these reports. Two diseases which occurred among our troops in two entirely different parts of the globe, namely, scrub typhus fever in the South Pacific and diphtheria in Central Europe, are examples in which electrocardiographic studies elicited much valuable information and stimulated more frequent clinical recognition of myocardial involvement in these two diseases.

Pathologic Incidence

As seen in the accompanying table (table 1) from Saphir and Gore,1, 2 based on a review of 1402 cases of myocarditis verified by pathologic examination, myocarditis occurs in practically every type of acute disease and with a wide variety of etiologic agents. These cases were studied between 1942 and 1946 when more than 40,000 autopsies were recorded at the Armed Forces Institute of Pathology.3

The incidence of myocarditis in 1250 consecutive necropsies, exclusive of fetal and neonatal deaths and medical examiner's cases, performed at Bellevue Hospital between January 1951 and December 1952 was only 3.3 per cent. The number of sections for microscopic examination taken from each heart ranged from 3 to 15 in the 42 cases analyzed.

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<table>
<thead>
<tr>
<th>No. of Cases</th>
<th>Age</th>
<th>Sex</th>
<th>Race</th>
</tr>
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<tbody>
<tr>
<td></td>
<td></td>
<td>Male</td>
<td>Female</td>
</tr>
<tr>
<td>2</td>
<td>11-20</td>
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<td>0</td>
</tr>
<tr>
<td>4</td>
<td>21-30</td>
<td>0</td>
<td>4</td>
</tr>
<tr>
<td>3</td>
<td>31-40</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>8</td>
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<tr>
<td>9</td>
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<td>61-70</td>
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<td>4</td>
</tr>
<tr>
<td>4</td>
<td>71-80</td>
<td>3</td>
<td>1</td>
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<tr>
<td>Total 42</td>
<td></td>
<td>26</td>
<td>16</td>
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</tbody>
</table>
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In 13 of the 42 cases the myocarditis was associated with acute or subacute bacterial
### Table 1.—Diseases Associated with Myocarditis* (From Gore and Saphir, 1948)

<table>
<thead>
<tr>
<th>Disease</th>
<th>Column 1</th>
<th>Column 2</th>
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<tr>
<td>Rickettsial diseases</td>
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<tr>
<td>Scrub typhus</td>
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<td>227</td>
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<tr>
<td>Epidemic typhus</td>
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<td>48</td>
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<tr>
<td>Rocky Mountain spotted fever</td>
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<td>19</td>
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<tr>
<td>Diphtheria</td>
<td>144</td>
<td>221</td>
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<tr>
<td>Subacute bacterial endocarditis</td>
<td>208</td>
<td>208</td>
</tr>
<tr>
<td>Rheumatic heart disease</td>
<td>130</td>
<td>130</td>
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<tr>
<td>Meningococcemia</td>
<td>111</td>
<td>256</td>
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<tr>
<td>Scarlet fever</td>
<td>24</td>
<td>44</td>
</tr>
<tr>
<td>Weil's disease</td>
<td>7</td>
<td>8</td>
</tr>
<tr>
<td>Relapsing fever</td>
<td>6</td>
<td>11</td>
</tr>
<tr>
<td>Syphilis (gummatous)</td>
<td>2</td>
<td>66</td>
</tr>
<tr>
<td>Chagas' disease</td>
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<td>1</td>
</tr>
<tr>
<td>Schistosomiasis</td>
<td>5</td>
<td>41</td>
</tr>
<tr>
<td>Malaria</td>
<td>5</td>
<td>135</td>
</tr>
<tr>
<td>Trichinosis</td>
<td>2</td>
<td>2</td>
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<tr>
<td>Acute encephalitis</td>
<td>13</td>
<td>144</td>
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<tr>
<td>Poliomyelitis</td>
<td>13</td>
<td>94</td>
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<tr>
<td>Infectious mononucleosis</td>
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<td>9</td>
</tr>
<tr>
<td>Measles</td>
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<td></td>
</tr>
<tr>
<td>Guillain-Barré syndrome</td>
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<td>30</td>
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<tr>
<td>Mumps</td>
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<td>8</td>
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<tr>
<td>Epidemic hepatitis</td>
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<td>400</td>
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<tr>
<td>Smallpox</td>
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<td>9</td>
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<tr>
<td>Virus pneumonia</td>
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<td>222</td>
</tr>
<tr>
<td>Tuberculosis</td>
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<td>581</td>
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<tr>
<td>Boeck's sarcoid</td>
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<tr>
<td>Cocciidiodermatitis</td>
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<tr>
<td>Blastomycosis</td>
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<tr>
<td>Actinomycosis</td>
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<td>9</td>
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<tr>
<td>Torulosis</td>
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<td>6</td>
</tr>
<tr>
<td>Septicemia</td>
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<td>Streptococcus</td>
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<td>Staphylococcus</td>
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<td>107</td>
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<tr>
<td>Pneumococcus</td>
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<td>18</td>
</tr>
<tr>
<td>Other acute bacteremias</td>
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<tr>
<td>Acute glomerulonephritis</td>
<td>14</td>
<td>160</td>
</tr>
<tr>
<td>Acute tonsillitis</td>
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<td>Unknown</td>
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<tr>
<td>Acute nasopharyngitis</td>
<td>41</td>
<td>Unknown</td>
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<tr>
<td>Cellulitis, lymphangitis, and wound infections</td>
<td>13</td>
<td>Unknown</td>
</tr>
<tr>
<td>Tularemia</td>
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<td>16</td>
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<tr>
<td>Brucellosis</td>
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<td>4</td>
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<tr>
<td>Miscellaneous (postinfectious)</td>
<td>13</td>
<td>Unknown</td>
</tr>
<tr>
<td>Exfoliative dermatitis</td>
<td>7</td>
<td>44</td>
</tr>
<tr>
<td>Arsenical reaction</td>
<td>1</td>
<td>18</td>
</tr>
<tr>
<td>Sulfonamide hypersensitivity</td>
<td>105</td>
<td>Unknown</td>
</tr>
<tr>
<td>Disease unknown (so-called &quot;idiopathic&quot;)</td>
<td>43</td>
<td></td>
</tr>
<tr>
<td>Starvation</td>
<td>33</td>
<td>50</td>
</tr>
<tr>
<td>Heat stroke</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Surviving less than 24 hours</td>
<td>16</td>
<td>45</td>
</tr>
<tr>
<td>Surviving more than 24 hours</td>
<td>13</td>
<td>26</td>
</tr>
<tr>
<td>Carbon monoxide poisoning (limited to patients who survived for an appreciable interval after the lethal exposure)</td>
<td>1</td>
<td>30</td>
</tr>
<tr>
<td>Emetine</td>
<td>1</td>
<td>70</td>
</tr>
<tr>
<td>Burns</td>
<td>11</td>
<td>45</td>
</tr>
</tbody>
</table>

* The figures in the first column represent the number of times myocarditis was encountered. Wherever possible the number of cases of each disease, screened to ascertain the first figure, is given in column 2. The ratio of the two thus provides a crude index of the frequency of myocarditis in each disease.

endocarditis, and in nine it was part of a rheumatic carditis. In eight cases myocarditis was found in association with sepsis or pyemia. The remaining 12 cases presented myocardial inflammatory changes in a variety of diseases including tuberculous, glomerulonephritic, granulomatous infections, disseminated lupus erythematosus, fungus infection (aspergillosis), bronchiectasis, and lobular pneumonia. In two instances the question of alkalosis and electrolyte imbalance and hypersensitivity to sulfonamide therapy arose as possible causes of the myocarditis. No instances of so-called Fiedler's (isolated) myocarditis were met with in this group.

Of the 42 cases, 16 were in the age group from 61 to 80 years, and 17 were in the period from 41 to 60 years. Therefore, 33, or the majority, were above the age of 40 years. Although Bellevue Hospital has a pediatric service, no children came to necropsy with any evidence of myocarditis during this period of two years when the 1250 postmortem examinations were
performed. The youngest adult with myocarditis was 19 years old.

There were 26 males and 16 females in the group. The usual ratio of males to females in Bellevue Hospital necropsies is 2:1. Only six Negroes were represented as compared to 36 white; among the latter were two white Puerto Ricans and one Chinese.

Some four of the 42 were considered to have died unexpectedly although they were seriously ill at the time of death.

Myocardial involvement was not recognized clinically in the majority of the 42 cases. In the 13 cases of acute and subacute bacterial endocarditis the correct diagnosis of the "endocarditis" was made in only six. In none of the 13 was mention made of myocarditis in the final clinical diagnosis. In contrast, one-half of the rheumatic cardiac patients were considered to have "active myocarditis." Two who were submitted to mitral commissurotomy were found to have rather widespread rheumatic myocarditis involving not only the auricular appendages, so commonly seen in biopsy specimens taken at the time of operation, but also the myocardium of the ventricles. Clinically both patients were considered to be free of rheumatic infection.

Practically no patients with communicable diseases are admitted to Bellevue Hospital. We, therefore, reviewed 1000 necropsies performed at Willard Parker Hospital, a municipal institution for the care of communicable diseases in New York, in the period from 1932 to 1952. Seventy-eight cases of myocarditis were disclosed, or an incidence of 7.8 per cent. This is readily accounted for by the known high incidence of myocarditis in the type of infectious diseases admitted to this hospital.

Analysis of these 78 autopsies reveals the largest number of cases with myocarditis, as determined histologically, namely 18, in individuals with diphtheria. Fourteen occurred in poliomyelitis, and 14 also were found in patients presenting various forms of tuberculosis. Nine were associated with measles, seven with scarlet fever, six with meningococccemia or meningococcus meningitis, and only two in pertussis. Six of the remaining eight cases of myocarditis occurred in a variety of diseases including pneumonia, otitis media, influenza meningitis, aplastic anemia, croup with tetany, and erythema multiforme bullosum. There were two instances of isolated myocarditis.

The incidence of myocarditis is known to be unusually high in poliomyelitis and diphtheria. In the former an incidence of 100 per cent has been reported in some epidemics (1949). Dolgopol, while pathologist to Willard Parker Hospital, published a review of cases of poliomyelitis coming to postmortem examination and found an incidence of 26.6 per cent. The experience with diphtheritic myocarditis in this hospital was reported in considerable detail by Burkhardt and his associates.

These data from a large general hospital and a hospital for contagious diseases compare with an incidence of 4.3 per cent among 5626 autopsies performed at the Michael Reese Hospital, reported by Saphir, and is in contrast to a 9 per cent incidence found by the same observer at the same hospital in a study of 1000 other consecutive autopsies when more (about 25) than the usual number of blocks were taken from each heart for microscopic examination.

In summary, the pathologic data indicate the likelihood that the over-all incidence of myocarditis is approximately 10 per cent. This takes for granted, however, that all individuals, including children, dying of infections of bacterial, viral, protozoal, rickettsial, helminthic and fungal origin are included. In addition, this figure will obtain only if multiple sections of the heart, more particularly of the myocardium, are examined histologically.

CLINICAL INCIDENCE

The incidence of myocarditis as based on clinical recognition is distinctly lower than the pathologic incidence. Having failed to find any recent figures on this problem, particularly analyses made since the advent of multiple electrocardiographic records, including precordial and extremity leads, we turned to the clinical records of Bellevue Hospital to seek an answer. This large municipal hospital is affiliated with four medical schools whose faculties are responsible for the diagnostic work-up and care of all the patients. Here we found that a
diagnosis of myocarditis was made only 16 times among 68,000 discharged patients in 1952, an incidence of 0.02 per cent. This includes 11 patients diagnosed as "rheumatic myocarditis, active," two patients with a diagnosis of "acute isolated myocarditis, unknown cause," one patient with "idiopathic myocarditis," and finally, one case of "acute bacterial myocarditis" and one of "subacute bacterial myocarditis."

Since, as already mentioned, Bellevue Hospital admits practically no communicable diseases, we obtained some figures from the record files of Willard Parker Hospital which is mainly for the care of such diseases. Among 4946 discharges during the year 1951, no diagnosis of myocarditis was noted, but during 1952 among 6452 discharges one diagnosis of "acute myocarditis complicating meningococcemia" was made and proved to be correct at necropsy.

From these figures, it is quite obvious that pathologists recognize myocarditis at necropsy far more frequently than physicians diagnose it during life. The discrepancy suggests that the signs and symptoms of myocarditis are frequently overshadowed by those of the primary disease, or indeed, that they are completely lacking in many instances. It is also probable that myocarditis is common in many diseases but is of such a mild degree that it is not recognizable. In most of these instances, recovery is the rule. How many of those who recover have residual myocardial changes is a moot question. Some of the areas of myocardial fibrosis seen at necropsy without sufficient reason for their presence may represent the residual scars of previous inflammatory reactions.

DEFINITION

The term "myocarditis" as used pathologically should be limited to hearts which demonstrate evidence of acute, subacute or chronic inflammation of the myocardium, either focal or diffuse. If no active inflammatory process is found in the myocardium the term should not be employed. An important exception is diphtheritic myocarditis in which, during the early course of the disease, no inflammatory cellular infiltrate is seen. The lesion, during that period, is mainly a parenchymatous one.

According to Karsner¹⁰ "acute myocarditis" to the pathologist "indicates a condition in which there is, associated with muscle degeneration or necrosis, an infiltration into the interstitial tissues of cells usually found either in acute or subacute exudative processes." He does not believe that a differentiation between acute parenchymatous and acute interstitial myocarditis is justifiable.

In those instances in which fibrous tissue is present, especially if it is interstitial in distribution and also if there is scarring of muscle bundles, "myocardial fibrosis" is apt to be the proper designation. If, on the other hand, the scarring is of perivascular distribution, then the lesion is most likely postinflammatory in nature, and commonly the end result of rheumatic myocarditis. In such instances collection of residual inflammatory cells may be seen. Sections of this description usually represent "healed myocarditis."

PATHOLOGIC FEATURES

The diagnosis of myocarditis usually requires examination of multiple sections from various parts of the myocardium. Gross recognition of myocarditis is usually difficult, often impossible. Sometimes the heart may be flabby or pallid or yellowish with dilated chambers. Mural thrombi may be present. However, in many instances the heart appears normal.

Microscopically, there may be found interstitial infiltrations of histiocytes, lymphocytes and eosinophils or neutrophilic polymorphonuclear leukocytes between the muscle fibers and in the perivascular connective tissue. In some sections the muscle fibers may exhibit fatty, granular, or hyaline degeneration. Focal embolic lesions or focal intrafascicular infiltration of inflammatory cells may be seen in the myocardium in the presence of acute and subacute bacterial endocarditis.

In bacteremia, especially when the Staphylococcus is the etiologic organism, abscesses, usually microscopic in extent, are not uncommonly encountered. These lesions are usually found in relation to terminal branches of the coronary arteries.
In idiopathic myocarditis, also known as Fiedler's or interstitial myocarditis, the inflammatory changes are limited to the myocardium, hence the frequent designation "isolated" myocarditis. In the early stages, the heart may present softening, areas of fatty change and zones of hyperemia. Mural thrombi, usually intraventricular, are commonly found.

Histologically there may be seen diffuse bands of lymphocytes, histiocytes, plasma cells, and occasionally eosinophils. Sometimes nucleated giant cells are recognized similar to those found in the granulomata of syphilis and tuberculosis. In conjunction with this cellular response there may be disseminated areas of necrosis as well as areas of replacement fibrosis.

A similar microscopic picture is said to occur in the myocardium as the result of hypersensitivity to sulfonamide drugs or as an allergic response to the administration of penicillin, serum, or other sensitizing agents.

It is now generally accepted that a so-called virus myocarditis occurs not infrequently in such viral or suspected viral diseases as anterior poliomyelitis, infectious mononucleosis, varicella, infectious hepatitis, measles, mumps, atypical primary pneumonia and several others. Histologically, the lesion includes a focal or diffuse invasion of the interstitial tissue mainly by lymphocytes, and less numerous as a rule are the polymorphonuclear leukocytes. In some instances only lymphocytes and monocytes are seen. Plasma cells are rarely seen in this type of myocarditis; likewise necrosis of muscle fibers does not seem to be a prominent feature.

The virus of poliomyelitis has been isolated from the hearts of patients dying of poliomyelitis. Although this has not been confirmed by other observers, there are authentic reports of a virus having been isolated from anthropoid apes who apparently died from myocarditis. It was possible to produce practically identical myocardial lesions with this virus in mice and guinea pigs. This virus has come to be known as the "encephalomyocarditis (EMC)" virus. Three instances of this disease in man have been reviewed.

Among the infectious granulomata of the heart are included syphilis and tuberculosis, but they rarely involve the myocardium. The latter is most frequently seen as microscopic miliary tubercles occurring in the presence of generalized miliary tuberculosis, or as an extension from tuberculous mediastinal lymph nodes. Rarely, nodular tuberculomata may be found in the myocardium. Other granulomata which may be seen in the myocardium include actinomycosis which usually extends from the mediastinum or lung, and in which lesion the ray fungus is readily demonstrated; and sarcoidosis, which may produce rather typical interstitial or intrafascicular lesions simulating tuberculous granulomata.

Trichinosis may cause myocarditis in the form of a diffuse infiltration of polymorphonuclear leukocytes, plasma cells, and sometimes of eosinophils. The parasites, although usually not detectable, may be recovered following digestion of a fresh specimen.

In Chagas' disease, seen principally in Brazil but also in other Latin American countries, the protozoans (Trypanosoma cruzi) may lodge in the myocardium and cause necrosis and inflammation of the interstitial tissue.

In malaria the plasmodium, usually Falciparum, may produce thrombi in the capillaries of the myocardium with resultant ischemic changes in the muscle fibers. The parasites may be seen in these capillaries together with agglutinated erythrocytes.

**Classification**

Myocarditis may be acute, subacute, or chronic, the differentiation being made arbitrarily on the basis of duration. Chronic myocarditis, when employed in the true sense of the term, implies continued activity, or at least frequent recurrences, of the inflammatory process over an appreciable period of time. The most common cause of the latter type of myocarditis is rheumatic fever.

Saphir has proposed a useful working classification of myocarditis which includes four main types: (a) myocarditis following infectious diseases, with or without endocarditis; (b) the specific type with characteristic anatomic structure, or identifiable pathogenic organism (rheumatic fever, tuberculosis, blastomycosis,
Chagas' disease, and other conditions); (c) those due to chemical poisons, physical agents, or hypersensitive states; and (d) the isolated type, unassociated with any known illness. In another classification suggested by Friedberg myocarditis is divided into: (1) infectious and toxic myocarditis; (2) suppurative myocarditis; and (3) idiopathic or isolated (Fiedler's) myocarditis.

CLINICAL Diagnosis

The diagnosis of myocarditis is made mainly by inference in the presence of one of the many types of diseases previously enumerated. According to the fifth edition of the "Nomenclature and Criteria for Diagnosis of Diseases of the Heart and Blood Vessels" the diagnostic signs of myocarditis include the following:

Sinus tachycardia. Abnormal rhythms. Enlargement of the heart. Systolic murmur at apex due to mitral incompetency. Faintness or sharp quality of the first heart sound. Evidence of cardiac insufficiency. Electrocardiographic changes such as defective atrioventricular or intraventricular conduction, or S-T and T-wave changes. Fever. Leukocytosis. Increased sedimentation rate.

Symptoms of myocarditis are by no means specific. In fact they are not infrequently very slight or completely absent. Frequently the symptoms are caused by the primary disease rather than by the myocarditis. Occasionally patients complain of vague aches and pains over the precordial area which have no relation to physical effort, appearing even at rest in bed, and of variable duration, ranging from minutes to hours. No particular type of radiation has been described.

Some patients complain of palpitation both with and without any appreciable increase in heart rate or arrhythmia. Weakness and fatigue seem to be relatively common complaints; less often anorexia or headaches are mentioned.

When heart failure intervenes as it does in those cases of myocarditis with rather severe myocardial inflammation, then the symptoms are the same as those met with in any instance of heart failure. It occurs quite often in the presence of rheumatic myocarditis but is also common in "isolated" myocarditis and not infrequently in that associated with diphtheria and with bacterial endocarditis. In the absence of valvular disease, it definitely indicates the presence of myocarditis, taking for granted, of course, that the patient is in the midst of an infection or convalescing from one.

The usual complaints when heart failure intervenes include dyspnea and orthopnea. Occasionally, however, the picture is confused by the presence of shock, the result of peripheral vascular collapse, which is not a rare occurrence in the presence of the severe and toxic types of acute infectious disease. It may occur with little or no impairment of the myocardium and, therefore, cannot be considered a symptom indicative of myocarditis except possibly in the case of diphtheritic myocarditis.

Sinus Tachycardia in the cases we have reviewed was not common and could readily be due in many instances to the primary disease rather than to the myocarditis, per se. However, in patients with acute rheumatic fever associated with myocarditis, it is rather common to have a tachycardia, and particularly one which is out of proportion to the height of the fever. On the other hand bradycardia may occur in the presence of myocarditis, including that caused by rheumatic fever. It has been noted quite frequently in children and young adults (fig. 2) after the first week or two of the illness and has been ascribed by some to overactivity of the vagus. Bradycardia is also said to be observed often in primary atypical pneumonia.

All patients with bradycardia in whom myocarditis is suspected should be checked by electrocardiogram in order to rule out heart block, which is not uncommon in several types of myocarditis, including that occurring in diphtheria, rheumatic fever and Chagas' disease.

Abnormal Rhythms, including premature ventricular systoles, paroxysmal supraventricular tachycardia, atrial fibrillation, atrial flutter and idioventricular rhythm, have been described as occurring in the presence of myocarditis. Rheumatic myocarditis seems to have the greatest incidence of rhythmic disturbances,
but diphtheritic myocarditis is also known to be complicated by a number.26, 27

On occasion the appearance of an arrhythmia in the presence of an acute infections disease will arouse the first suspicion of an underlying myocarditis or lead to the taking of an electrocardiogram which in turn may furnish information suggestive of myocardial disease.

Enlargement of the Heart. In many instances of myocarditis the heart is of normal size. When obvious enlargement is found at the bedside it is likely that the patient has a rather severe and diffuse myocarditis. In such instances it is often transient and may recede rather rapidly, although in Fiedler's (isolated) myocarditis it usually persists and progresses.

The material which has been reviewed would indicate that enlargement of the heart in myocarditis is clinically not common. The heart is most apt to enlarge in the presence of rheumatic, diphtheritic and chagasic myocarditis among the specific myocarditides, and in Fiedler's (isolated) myocarditis among those of unknown origin.

Systolic Murmur at Apex Due to Mitral Incompetency. An apical systolic murmur is a rather common finding in myocarditis but is by no means conclusive evidence of an inflamed myocardium. Sometimes it appears only when heart failure intervenes, at other times it seems to accompany the elevation of temperature, and occasionally it may parallel the presence of anemia. In those cases of acute and subacute bacterial endocarditis with complicating myocarditis or with focal abscesses of the myocardium, a systolic murmur at the apex may be the result of underlying pre-existing valvular deformity, or of destruction of the valve cusp by the vegetative process, or finally as a sign of myocardial failure.

Diastolic murmurs, either at the aortic or mitral areas, are rare in uncomplicated cases of myocarditis. They are most apt to be heard in children with rheumatic carditis associated with aortic or mitral valvulitis or both. Even in these circumstances they may be transient, disappearing after the carditis has subsided.

Faintness or Sharp Quality of the First Heart Sound. Normal heart sounds have been described in many instances of proven myocardi
ditis, possibly in the majority. However, among the fairly common signs elicited on physical examination has been a faint, muffled or impure first sound, usually described as being heard at the apex. At times it assumes the valvular quality of the second sound. A split first heart sound, usually apical in site, has also been described. Occasionally it will be the second sound which shows this change. None of these altered sounds is characteristic of myocarditis, being readily heard in the presence of fever, anemia, altered metabolic states and in many other types of cardiac abnormalities.

Gallop rhythm may be elicited in the presence of myocarditis even in the absence of clinical heart failure. It is most frequently heard in children who are having a severe bout of rheumatic carditis, but it has also been reported in other types of myocarditis, particularly in patients with Fiedler's (isolated) myocarditis.

Evidence of Cardiac Insufficiency. Occasionally a correct diagnosis of myocarditis has been made in the presence of an acute infection because of the appearance of heart failure in a patient without pre-existing heart disease. This combination of events, especially in children or in young adults, makes the presence of myocarditis likely. Cardiac insufficiency in the presence of recurrent rheumatic carditis is a rather common finding particularly in patients with established valvular deformities, although it is also known to occur during the initial bout of rheumatic fever with carditis.

Progressive myocardial insufficiency, either rapid or gradual, is one of the outstanding clinical features of Fiedler's (isolated) myocarditis.28 It is common among the late manifestations of the myocarditis associated with subacute bacterial endocarditis. Since the advent of antibiotic therapy it is also encountered in the healing and healed stage of this type of endocarditis, although one author's29 experience has led him to believe that this form of therapy has decreased the occurrence of heart failure considerably.

It has been said that pulsus alternans probably occurs more often during cardiac insufficiency complicating myocarditis than other myocardial disease. Although an example is
shown in rheumatic myocarditis in figure 1, it is uncommon in our experience. We have seen it most often in the degenerative types of heart disease.

Circulatory collapse or shock is, on occasion, seen in patients with myocarditis and is probably more often peripheral than central in origin. It seems to occur most often in patients with diphtheria. This mechanism may account for the sudden death in these patients. Nevertheless it is well known that unexpected and sudden death is fairly frequent in other types of myocarditis. Among 117 cases of sudden death Lisa found 59 cases of myocarditis, 20 of "infectious" type and 39 of "toxic" type. On the basis of the evidence presented he concluded that infection is a more frequent cause of sudden death than arteriosclerosis. In our 42 cases of myocarditis coming to necropsy at Bellevue Hospital at least four were regarded as having died unexpectedly.

Intracardiac thrombosis is met with in some cases of myocarditis, especially in the Fiedler's type of lesion where heart failure and also embolic phenomena are fairly common. Emboli may be another cause of sudden death even when there is no clinical evidence of active infection.

Fever is a nonspecific finding in many examples of myocarditis and one which may follow any type of pattern. However, there is an appreciable number of cases which show no fever during the course of the disease. This holds true for some of the cases of recurrent or chronic rheumatic carditis which at postmortem are found to have rather extensive signs of inflammation of the myocardium. Fever, therefore, is most apt to be a reflection of the primary disease rather than of the myocarditis, per se.

Leukocytosis. What has been said of fever might be said of leukocytosis, namely, that it is not specific, merely being a response to the primary disease. Many cases of myocarditis, especially the viral and rickettsial types, show no alteration in the total or differential count of white blood cells.

Increased Sedimentation Rate. This laboratory aid seems to be a bit more helpful in establishing a diagnosis of myocarditis than fever and the white blood cell count. There are a significant number of proven examples of myocarditis in which the erythrocytic sedimentation
rate is reported as normal. This is particularly true in the presence of chronic or recurrent rheumatic myocarditis and despite evidence of rather widespread inflammation in the tissues of the heart. The two patients previously described in the Bellevue Hospital series who died following mitral commissurotomy and who at necropsy were found to have rather widespread rheumatic myocarditis had normal sedimentation rates, no fever, and normal white blood cell counts. This negative laboratory information contributed to the erroneous opinion that they were free of active rheumatic infection. It is known that newer nonspecific tests of inflammation, such as the determination of the C-reactive protein, display similar clinical limitations.21

If initially elevated, the sedimentation rate may be a helpful guide in determining when a patient with myocarditis may be permitted out of bed.

Although not mentioned among the signs listed in the “Criteria,”21 hypotension and a small pulse volume have been noted by a number of observers in the course of myocarditis. Hypotension may be seen in those patients even in the absence of heart failure or shock. Possibly it is caused by hyperthermia.

**Electrocardiogram**

The manifestations of acute myocarditis are often so minor just preceding a serious clinical episode that the electrocardiogram assumes considerable importance as the laboratory aid which may first display suspicious evidence of the lesion. Paradoxically, this aid may also be deceiving if inadequate attention is paid to the many extracardiac variables which can modify it from time to time22 particularly in the course of a febrile illness. An inspection of many sporadic reports on the electrocardiogram in myocarditis suggests that the changes noted may have been due in some instances to extracardiac causes, or to cardiac causes other than the myocarditis itself.

The “extracardiac causes” which may be encountered in many of the diseases with which myocarditis is associated may be listed as follows: (a) chemical, including alkalosis,33 acidosis,33 abnormalities of serum electrolytes (particularly potassium and calcium),34,35 recent ingestion of a meal with its probable effect on the serum potassium,36 and hypoxia37; (b) mechanical, including hypotension as often seen with fever and its consequent effect on the gradient of pressure in the walls of the cardiac chambers, on cardiac position, and on coronary flow especially when shock levels are reached; displacement such as may be caused by pneumonia and effusion; different positions of the patient when recording serial records; (c) pharmacologic, such as drugs which may modify the record; and (d) thermal, namely hyperpyrexia. A sustained induced temperature of 105 to 107 F. may occasionally cause distinct abnormalities of the T wave in the extremity and precordial leads.38 Lower temperatures, as produced by intravenous typhoid vaccine, will invariably cause the ventricular gradient, regarded as a vector, to shorten and rotate in a counterclockwise manner both in the frontal plane and in the sagittal plane viewed from the left.39,40 This occurs even if the fever is prevented with a simultaneously administered antipyretic.40 In the electrocardiogram, the change in the magnitude and direction of the gradient manifests itself usually as a lowering of the T wave in lead I and lowering or inversion in leads II, III, and aVF.

The “cardiac causes” other than myocarditis which may produce electrocardiographic abnormalities include previously existing or progressive valvular deformities or myocardial necrosis. This complicates the interpretation of the record when, for example, inflammation is superimposed on rheumatic valvular, hypertensive, arteriosclerotic, or other types of cardiac disease. Further, there are often diffuse pathologic changes over and above the usual focal inflammatory lesions such as cloudy swelling and fatty infiltration usually regarded as degenerations but considered by some pathologists to be part of the inflammation.10 The role of these in producing aberrations of the electrical behavior of the heart is uncertain.

The dominant effect of most of these variables is on the labile recovery process represented in the finished record by the S-T segment and the T wave; myocarditis dominantly affects the same process. The consequent need
acute infectious diseases which can cause myocarditis is probably close to 33.3 per cent.41

Myocarditis itself may affect rhythmicity, conductivity, and the basic processes of excitation and recovery in both the atria and the ventricles. These effects will manifest themselves in the clinical electrocardiogram as abnormal rhythms and extrasystoles, atrioventricular and intraventricular block, and modifications in the form and duration of the deflections comprising the atrial and ventricular complexes.

Disturbances of rhythm include sinus tachycardia, occasionally atrioventricular nodal rhythm or tachycardia, and premature systoles of various origins. Of fairly common occurrence is a sinus bradycardia in the course of rheumatic carditis (fig. 2). Except in rheumatic myocarditis and an infrequent case of diphtheria,26 circus rhythm is rare. Ventricular tachycardia has been encountered principally in terminal instances of diphtheritic myocarditis. An example has been described in specific (tuberculous) myocarditis,41 and we have observed an example of ventricular and bidirectional ventricular tachycardia in the course of acute focal myocarditis of unknown cause (figs. 3 and 4).

for careful differentiation in interpretation of the records is apparent. A final conclusion on the significance of the electrocardiographic findings will depend, as it always should, on evaluation of them in the light of the total clinical picture.

The incidence of electrocardiographic abnormalities in diseases which can cause myocarditis varies from the rare example of specific myocardial involvement in tuberculosis41 to almost a 100 per cent incidence in South American trypanosomiasis.25, 42. 43 Naturally the figure varies with the severity of the primary disease, possibly with the same primary disease caused at different times by pathogens with different degrees of viscerotropism as in poliomyelitis,6 and with other factors difficult to define or still unknown. The over-all incidence of abnormal electrocardiograms in the course of

Fig. 2. R. J., white male, 17, with acute rheumatic myocarditis and polyarthritis with sinus bradycardia and prolonged Q-T interval. The bipolar limb leads (I, II, III), the augmented unipolar limb leads \((aV_R, aV_L, aV_F)\), and the precordial leads \((V_1, V_2, V_3)\) were recorded two weeks after the onset of articular pains, and after two days of 120 mg. of corticotropin (ACTH) intramuscularly daily. The heart rate was 43 per minute. Bazett's index was 0.464; the body temperature was normal. Two days before therapy the temperature was 103 F., but the heart displayed a relative bradycardia with a rate of 82 per minute. Time lines occur every 0.04 second. The precordial leads were recorded with the string tension adjusted so that 1 mv. = 0.5 cm. Unless otherwise stated the symbols and technical data are the same in all subsequent electrocardiographic illustrations.

Fig. 3. A. L., Puerto Rican female, 42, with acute focal interstitial myocarditis of unknown cause with some hemorrhage into the epicardial fat. Heart weight 420 Gm. The electrocardiogram was made on March 4, 1952. To be noted are the low voltage of QRS and the inverted T waves in leads \(V_2\) and \(V_4\). The precordial leads were recorded at normal sensitivity (1 mv. = 1 cm.) of the string.
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Fig. 4. Electrocardiogram of same patient presented in figure 2 recorded on March 6, 1952, shortly after the onset of a shock-like state. Death occurred two days later. In leads I, II, and III there is a bi-directional ventricular tachycardia; in leads V₁ and V₅ it is an ordinary ventricular tachycardia; in lead V₃ there appears to be a type transitional between the two.

Atrioventricular block occurs characteristically in diphtheritic and in chagasic myocarditis, but may be encountered in lesser grades in other infections and diseases, possibly more frequently in those diseases of actual or suspected viral or rickettsial origin such as poliomyelitis, typhus fever, infectious mononucleosis, and Fiedler’s myocarditis. Sporadic examples are reported in other diseases. Intraventricular block also occurs most frequently in diphtheria and in Chagas’ disease, and in the latter the block almost without exception is of the right bundle branch. Bundle-branch block may be encountered with any diffuse, extensive inflammation such as occurs in Fiedler’s myocarditis. The first electrocardiogram published in a case of Fiedler’s myocarditis revealed a left bundle-branch block and, at times, incomplete atrioventricular block with ventricular premature systoles (fig. 5). Another example with left bundle-branch block is shown in figure 6. In both of these the block was characterized by a rather wide QRS interval (0.18 and 0.16 second, respectively).

Abnormalities of the P wave have been noted principally when the atrial pacemaker was altered. However, in some instances attention...
has been called to high or peaked P waves. No consideration appears to have been given at all to the atrial T wave ($T_a$). In the electrocardiogram of a 52 year old man with an abscess of the left atrial wall there was distinctive downward displacement of $T_a$ (the P-R segment) seen best in leads II and aVF with reciprocal elevation in lead aVR (fig. 7). At necropsy a few days later the heart, which weighed 680 Gm., revealed inflammatory areas in the atrial appendages as well as in the ventricles. There were in addition a gross abscess of the left atrial wall, an acute bacterial (Streptococcus hemolyticus) endocarditis of the aortic valve with a mycotic aneurysm of one leaflet and an infarct measuring 1 cm. in diameter in the posterior wall of the left ventricle probably embolic in origin. The abnormal $T_a$ could be ascribed to atrial suppuration, atrial hypertrophy, or both.

Since the lesions in myocarditis are usually focal and microscopic, modifications of QRS are not usually significant unless there is also intraventricular block, or an extensive destruction of fibers. Unusual notching may occasionally be seen (fig. 7), but to ascribe this to the myocarditis is difficult even when serial records are obtained. Theoretically QRS may be modified much as with infarction if the myocarditis includes a localized abscess in the ventricular muscle. In our experience such a lesion caused by the Streptococcus viridans produced no abnormalities of QRS, and the abscess was not suspected until it ruptured through the visceral pericardium causing a purulent pericarditis. Subsequently it spread to include the junctional tissues and caused atrioventricular block.

In a third abscess of the myocardium with two mycotic aneurysms of the interventricular septum, there were no distinctive abnormalities until just before death when the suppurative process ruptured into the myocardium and so doing severed the right bundle-branch (figs. 8 and 9). The precordial leads displayed deep Q waves in leads V1 and V2 (fig. 8) such as are often seen with anteroseptal infarction complicated by right bundle-branch block.

![Fig. 7. R. D., white male, 49, with acute bacterial endocarditis (Streptococcus hemolyticus) of the aortic valve, abscess of the left atrial wall, and generalized monocytic infiltration of the myocardium. Heart weight, 680 Gm. The electrocardiogram shows a sinus tachycardia with notching of the R wave in leads II, III, and aVF, a diphasic P wave in lead V1, and more than usual displacement of the P-R segment ($T_a$ wave) downward in leads II and aVF, and upward in lead aVR (arrows).](image1)

![Fig. 8. E. G., white male, 39, with calcific aortic stenosis with acute bacterial endocarditis (Staphylococcus albus). The electrocardiogram shows abnormalities of the S-T segment and T wave such as might be caused by left ventricular hypertrophy. The heart weight was 860 Gm. with hypertrophy dominantly of the left ventricle.](image2)
FIG. 9. Electrocardiograms of the same patient presented in figure 8 recorded four days later and six hours after the onset of epigastric and lower substernal pain. There is a right bundle-branch block with deep Q waves in leads V₁ and V₅, and considerable displacement of the S-T junctions in all leads. The patient died six hours later.

In addition to the calcific aortic stenosis, ventricular hypertrophy and acute staphylococcal endocarditis of the aortic valve, there were two mycotic aneurysms approximately 1.5 cm. in diameter at the base of the interventricular septum. One of these extended anterolaterally and actually involved the anterior descending branch of the left coronary artery which, on microscopic section, showed periarteritis but no occlusion. The other aneurysm extended medially through the septum into the anterior right ventricular wall and caused a bulge of the surface of this chamber near the atrioventricular groove. It involved the right coronary artery which also showed inflammatory changes in its walls. Both of the aneurysms were surrounded by necrotic myocardium infiltrated with acute and chronic inflammatory cells, small abscesses and hemorrhage. This acute inflammation of the anterior part of the heart apparently caused the terminal electrocardiographic picture simulating myocardial infarction.

The voltage of the QRS deflections may sometimes be lowered in the type of carditis which progresses to heart failure, or when there is an associated pericardial effusion.

The S-T segment has often been reported to be abnormally elevated or depressed at its origin, or to show an abnormal form in the course of myocarditis. However, neither of these, particularly the displacement, is ever very outstanding with a few notable exceptions.²⁷, ⁵⁶ Although not all the causes of change in the form or location of this segment are known, in the light of existing theory displacement of the origin of the segment (S-T junction or J) should not be great in a diffuse myocarditis provided that there is no intraventricular block or unusual hypertrophy. Examination of a good number of the published records as well as our own bears out the theory. Most of them show little or no displacement if measurement is made with the P-R segment as the level of reference. This is the case despite the fact that pathologic correlation will often disclose partially destroyed myocardial fibers where a current of injury might be expected to exist. Even in localized inflammation such as a myocardial abscess or tuberculoma⁴¹ the displacement is not great although it was present in an example of the latter just after a paroxysm of ventricular tachycardia, and in an example of the former just described, when the abscess ruptured into the myocardium.

Nevertheless, serial records in a variety of myocarditides will at times show some change in the position of the S-T junction and perhaps more often a modification of form. The latter is always associated with a change in the T wave, the most common electrocardiographic abnormality encountered in myocarditis. The change which occurs indicates that the average direction of recovery is altered from normal in different ways. If the new direction of the T wave, regarded as a vector, has a direction backward, upward, and to the left, the clinical record will display lowering of the T wave in lead I, lowering or inversion in leads II and III, and inversion in several or all of the precordial leads, usually on the right half or more of the precordium. This change suggests that the normal ventricular gradient is altered so that recovery is prolonged on the epicardial surface as compared with the endocardial, or at the apex as compared with the base. As noted earlier, fever alone favors this change. If the new direction is forward and to the right and either upward or downward, the T wave characteristically will be inverted in lead I and in left precordial leads (fig. 11).

The duration of abnormality of the S-T segment and T wave varies in different diseases and with different severities of the same disease,
but usually evolves over a period of a few days to six weeks if the patient recovers. Naturally this interval may be modified by therapy.

No important changes in the U wave have been described though it is likely that they occur.

Except in rheumatic myocarditis, the Q-T interval has been largely neglected. No measurements seem to have been made of the Q-U interval.

The electrocardiogram may at times be within normal limits when necropsy soon after displays extensive anatomic disease. An example is shown in figure 10. At necropsy a few days later, there was found an acute bacterial endocarditis of unknown cause with diffuse myocarditis. Even though various deflections

Fig. 10. J. R., white male, 33, with acute bacterial endocarditis of the mitral and tricuspid valves of undetermined cause with acute myocarditis and necrosis. The heart weighed 450 Gm. The record illustrates no definite deviations from normal even though the S-T junction is slightly elevated in leads II and aVl. The sensitivity of the string when recording the chest leads was 1 mv. = 0.7 cm.

Fig. 11. R. O., white male, 7½, with acute rheumatic carditis and polyarthritis. At the time the electrocardiogram was made the patient had been sick for eight weeks and the day before had received 300 mg. of cortisone by mouth. To be noted is the inversion of the T wave in leads I, aVl, and Vs with a diphasic T wave and prominent U wave in lead V1.

Fig. 12. Electrocardiograms on the same patient presented in figure 11 recorded 26 days later after a course of cortisone of gradually diminishing dosage which at the time of the record was 50 mg. daily. The serum potassium at the time was 3.4 mEq. per liter. All clinical manifestations of rheumatic activity had subsided. Compared with figure 11, the changes in the direction and form of the T waves are to be noted, particularly the high, pointed T wave in lead II, which is not an uncommon finding usually in the convalescent stage of rheumatic myocarditis.
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are not beyond normal quantitatively, qualitative changes may often be suggestive. Of fairly frequent occurrence in this regard is the development of high, peaked T waves in the healing stage of rheumatic carditis (figs. 11 and 12).

Prognosis

There is very little that can be said regarding the prognosis of myocarditis since so few cases are recognized at the bedside, excluding those of specific type such as the rheumatic. It seems improbable, therefore, to consider the prognosis of myocarditis in relation to the disease of which it is a complication or a manifestation. There is a group, however, usually classified as Fiedler’s (isolated) myocarditis which does not seem related to any particular disease.

In the latter group, that is, Fiedler’s myocarditis, the prognosis, both immediate and ultimate, is poor. These patients are prone to many complications such as embolic phenomena, serious conduction defects and, most important, progressive and rather rapid heart failure. Occasionally death is rather sudden or unexpected. The usual cardiac therapy does not seem to alter the prognosis of these cases even in those instances where the correct diagnosis was made early in the course of the disease. However, the new hormones such as corticotropin (ACTH) and cortisone may be found to be effective in these cases. One case report is already at hand indicating that corticotropin therapy proved beneficial in curing a 7 year old boy with myocarditis of undetermined etiology simulating Fiedler’s type.58

It is conceivable that some of the myocarditides which occur in the presence of acute and subacute bacterial endocarditis may react favorably to the antibiotic therapy now being used routinely in such cases. Certainly the prognosis of these bacterial forms of endocarditis has become distinctly favorable since the use of the antibiotics. Unfortunately two adverse features of antibiotic therapy have made their appearance in recent years: (1) an increase in penicillin-resistant bacteria,59 especially among strains of Staphylococcus and slightly less so among the Streptococci which cause endocarditis; (2) the rather frequent occurrence of heart failure, commonly progressing to death, in patients in whom the endocarditis has been “cured” by antibiotic therapy.60 Most of the latter patients have a complicating myocarditis of rather extensive nature and which does not respond as well to the antibiotics as the endocardial lesions.

The prognosis of rheumatic myocarditis is dependent on the prognosis of the primary disease, namely rheumatic fever. Its mortality from the acute attack varies between 1 and 4 per cent and is practically always due to acute carditis.61 However, the highest mortality rate in children with rheumatic heart disease occurs during a period of three to five years after the initial attack of rheumatic fever.62 During this interval, carditis is an important, if not the most important, factor in causing death. In about 80 per cent of all cases, the cause of death in children with rheumatic heart disease is rheumatic fever and carditis, the latter including myocarditis. As seen in the nine instances of rheumatic heart disease coming to necropsy in the Bellevue Hospital group myocarditis may also occur in adult life. All nine cases were adults ranging from 19 to 64 years. The majority died of congestive heart failure, and it seems likely that the underlying myocarditis was at least a contributory factor to the heart failure as well as to death.

Possibly the prognosis of rheumatic myocarditis is already being influenced by the increasing use of hormone therapy including cortisone and corticotropin. The preliminary report issued in June 1952 by the Cooperative Rheumatic Fever Study63 stated: “In the type of cases admitted to the trial and with the regime of treatment laid down, it appears that individual symptoms, signs or laboratory observations may have been affected more favorably by one or another of these three drugs, but no consistent pattern is evident. In short, no firm conclusions can at present be drawn concerning the drug most effective in the control of the acute illness. The cases have not been under observation sufficiently long to provide data on the prevention of rheumatic heart disease.” The three drugs used in the study were cortisone, corticotropin and acetylsalicylic acid. Other reports have been appearing more recently suggesting rather striking
results by the use of the hormones but with larger doses than those employed in the cooperative study. Wilson and her co-workers imply that the short-term administration of corticotropin modifies the natural course of active rheumatic carditis with respect to severity, duration and absence of overt clinical evidence of residual cardiac damage.

Diphtheritic myocarditis must always be given a poor prognosis even though a number of patients survive this very serious complication of diphtheria, regardless of whether the site of initial infection is in the nose or throat or in the skin. The fatality rate from myocarditis seems to be highest in the later phase of the disease including the convalescent period, but unexpected circulatory or cardiac failure as well as sudden death may occur during any part of the clinical course. In one-third of a large series of cases reviewed by Gore the manifestations of myocarditis appeared at a time when the patient seemed to be well on his way to convalescence. He states that the interval between diphtheria and the onset of cardiac symptoms has been designated as the “deceptive interval of apparent improvement.” Early clinical diagnosis of the primary disease and early administration of adequate amounts of antitoxin undoubtedly help in preventing myocarditis and thereby lessen the mortality. Long periods of convalescence sufficient to heal the myocardial process may also reduce the mortality rate even though the residual damage of the myocardium which some cases may show will not necessarily be prevented.

It would seem the part of wisdom to give all patients with myocarditis occurring in various infectious diseases sufficient rest in bed to permit the myocardial inflammatory process to pass into a healed stage as determined clinically and electrocardiographically.

**TREATMENT**

Among the many diseases which may cause myocarditis there are some which can be prevented. Among these are rheumatic fever and diphtheria. The latter can be prevented by proper immunization measures. Rheumatic fever can be prevented to a great extent by routinely treating all infections of the upper respiratory tract, especially tonsillitis and pharyngitis, with appropriate and intensive antibiotic therapy. In children and young adults who already have rheumatic heart disease, the prophylactic use of sulfadiazine will reduce the rate of recurrent rheumatic fever and carditis. Experience with the use of long-acting antibiotics over the past few years is also very encouraging in the prevention of recurrent rheumatic fever.

Bacterial endocarditis can be prevented by the prophylactic use of chemotherapy in any patient known to have congenital or acquired heart disease for one day prior to, and for 48 hours after, operative procedures such as extraction of a tooth, tonsillectomy, and after childbirth. Correction of sepsis or pyemia by intensive antibacterial therapy in conjunction with appropriate surgical procedures or by the use of therapeutic enzymes (streptodornase-streptokinase) will prevent the occurrence of bacterial endocarditis and probably myocarditis too.

In the management of diphtheritic myocarditis, the importance of early recognition of the primary disease as well as the early administration of adequate amounts of antitoxin should be given major emphasis. It is well to keep in mind the so-called “deceptive interval of improvement” which has been observed so often prior to the onset of cardiac complications. Since the manifestations of myocarditis may occur late in the course of the disease, convalescence should be prolonged in order to allow healing of the myocardial damage with subsequent reduction in the number of sudden or unexpected deaths.

The management of rheumatic myocarditis will usually include the use of salicylates, cortisone, or corticotropin. As previously quoted, the Cooperative Rheumatic Fever Study in its preliminary report concerning the relative merits of these three drugs was unable to draw any firm conclusions as to which was most effective in the control of rheumatic fever. Needless to say these conclusions do not permit one to make a definitive choice among these three therapeutic agents. However, reports are beginning to appear intimating the superiority of the hormones and their effectiveness in
controlling the acute manifestations of rheumatic fever including carditis and in reducing the incidence or severity of sequelae. Perhaps with increasing experience and modification of dosage, the hormones will assume an important role in the treatment of rheumatic carditis and possibly in the prevention of structural deformities.

Regardless of which medicines are used, bed rest and supportive measures are indicated until clinical manifestations and electrocardiographic records indicate that the myocardial inflammatory process has become quiescent. If heart failure, arrhythmia, or other circulatory complications should intervene, they must be handled in the same manner as in other types of heart disease.

As an important preventive of myocarditis, the therapy of acute and subacute bacterial endocarditis should not be delayed. Antibiotic therapy, preferably beginning with penicillin, should be started within 48 hours after the diagnosis of bacterial endocarditis has been made, only withholding the drug long enough to obtain blood for culture. Subsequently, depending on the in vitro characteristics of the microorganism and the clinical response, the most effective antibiotic or combination of antibiotics should be employed. If signs of heart failure intervene, the therapy should include the measures usually outlined, such as restricted sodium intake, digitalization, diuretics if needed, continued bed rest and correction of anemia if present. Any arrhythmia which may arise must also be treated by the necessary therapeutic agents to prevent heart failure or embolic complications.

Therapy of isolated (Fiedler’s) myocarditis consists mainly of the treatment of the congestive heart failure which these patients so frequently present. In view of the rather high incidence of thromboembolic complications, anticoagulant therapy should be seriously considered. Cortisone and corticotropin may lead to recovery, at least temporarily, of some of these cases as suggested earlier. Since there is always a possibility that some instances of Fiedler’s myocarditis may have a bacterial or viral origin, the use of antibiotics should also be considered.

The treatment of that form of myocarditis considered to be “allergic” or associated with hypersensitivity, for example, to sulfonamides, antibiotics or biologic sera, should include the use of antihistamines, and possibly the corticosteroids. In the management of patients with myocarditis secondary to parasitic infestation, the parasiticide indicated for the specific causative agent should be employed. As manifestations of myocardial involvement appear, the necessary measures to control them must be instituted. Similar considerations apply to the management of the myocarditides occurring in the presence of rickettsial and viral diseases.

SUMMARY

A review of selected papers on myocarditis published during the past decade and an analysis of some original data together with personal experiences have been presented. All of these indicate that the clinical diagnosis of myocarditis is made far too infrequently as contrasted with the high incidence of this disease as recognized by pathologists. It is estimated that approximately 10 per cent of all patients coming to necropsy will demonstrate some evidence of myocarditis.

The discrepancy between the clinical and the pathologic incidences can possibly be ascribed to two factors, namely, (a) the relatively innocent nature of the clinical and laboratory findings in many cases, and (b) the apparent reluctance of the clinician to make the diagnosis.

It is likely that a correct diagnosis of myocarditis will be made more often and cardiac catastrophies avoided if the clinician will consider the possibility of the diagnosis, particularly in the course of all types of infectious diseases.

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