Auricular Fibrillation and Mitral Stenosis in Bacterial Endocarditis

By Jacques B. Wallach, M.D., Macellis Glass, B.A., Leslie Lukash, M.D., and Alfred A. Angrist, M.D.

The infrequent association of bacterial endocarditis with auricular fibrillation and with severe mitral stenosis is noted. The relevant literature is reviewed. Eighty-two cases of bacterial endocarditis superimposed on rheumatic heart disease and 47 examples of bacterial endocarditis without underlying rheumatic valvulitis are studied. Auricular fibrillation and severe mitral stenosis occurred less frequently in these groups than in patients with rheumatic heart disease without bacterial endocarditis. Various theories to account for these observations are analyzed and an alternative explanation is offered.

The RARE association of auricular fibrillation and subacute bacterial endocarditis has been stressed.1-18 This may be of diagnostic value to the clinician in differentiating rheumatic heart disease from subacute bacterial endocarditis in patients with evidence of embolization. No adequate explanation for this apparent "antagonism" has been offered. The relative infrequency of severe mitral stenosis in patients with subacute bacterial endocarditis has also been noted.5,6 At present no theory accounts for this relationship.

In reviewing the clinical features of subacute bacterial endocarditis, Libman,1 in 1918, noted that "arrhythmia is remarkably infrequent." The occurrence of auricular fibrillation in reported series of cases of bacterial endocarditis since that time are summarized in table 1.

In a review of the literature, Segal13 collected 1,351 reported cases of bacterial endocarditis; auricular fibrillation occurred in 29 (2.14 per cent). In contrast, the average incidence of auricular fibrillation in several collected series of patients with rheumatic heart disease was 48.7 per cent. However, many of the cases with alleged auricular fibrillation included in this review were inadequately reported or only casually mentioned in the original articles; the autopsy findings were usually not described.

Furthermore Segal did not distinguish between patients with established auricular fibrillation who later developed subacute bacterial endocarditis (a rather rare occurrence) and those more numerous patients with subacute bacterial endocarditis who developed transient or terminal auricular fibrillation.

McDonald18 studied seven reported series of cases of bacterial endocarditis.4, 7, 9, 10, 12, 17 Auricular fibrillation occurred in 24 (2.45 per cent) of the 980 cases reported in these seven papers but antedated the onset of the bacterial endocarditis in only two cases. In 11 instances the auricular fibrillation began after the onset of the bacterial endocarditis and in 11 other patients it was not stated which process occurred first.

These reports indicate that the development of subacute bacterial endocarditis in rheumatic patients with established auricular fibrillation is infrequent.

Material and Methods

In order to study this problem with a large series of uniformly examined cases, 8676 consecutive autopsies performed at the Queens General Hospital from 1936 to 1950 inclusive were reviewed. Five hundred and nine (5.9 per cent) of these were classified as rheumatic heart disease since they fulfilled criteria previously used12: (1) fusion and retraction of the mitral or tricuspid valves; (2) fusion and shortening of the chordae tendineae; (3) noncalcific fusion of the aortic valve; (4) any fusion, including calcific changes, of the aortic valve when it was associated with definite rheumatic

From the Department of Pathology, Queens General Hospital, Jamaica, New York.
Table 1—The Occurrence of Auricular Fibrillation in Reported Series of Bacterial Endocarditis

<table>
<thead>
<tr>
<th>Author</th>
<th>Total Cases</th>
<th>Auricular Fibrillation</th>
<th>Comment*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rothschild, Sacks, Libman</td>
<td>123</td>
<td>4</td>
<td>1 AFB terminal. 3 AFB and 1 AFL in bacteria-free stage. 1 AFB mentioned in footnote associated with Graves disease.</td>
</tr>
<tr>
<td>Cotton</td>
<td>55</td>
<td>1</td>
<td>Terminal AFB</td>
</tr>
<tr>
<td>Blumer</td>
<td>301</td>
<td>4</td>
<td>4 AFL. 1 AFL. 9 “irregularity” without classification. AFB previously ended with quinidine. Recurred at time of infection 6 months before death.</td>
</tr>
<tr>
<td>Sprague</td>
<td>20</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Thayer</td>
<td>306</td>
<td>“5%”</td>
<td></td>
</tr>
<tr>
<td>de la Chapelle, Graef</td>
<td>-</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>Fulton, Levine</td>
<td>111</td>
<td>1</td>
<td>1 established AFB. 1 transient terminal AFB (included in Fulton and Levine). 1 AFB preceding bacterial endocarditis.</td>
</tr>
<tr>
<td>Laws, Levine</td>
<td>43</td>
<td>2</td>
<td>1 established AFB. 1 AFB and 1 AFL in course of bacterial endocarditis—not autopsied. 2 AFB in course of bacterial endocarditis and 1 terminal AFB with acute bacterial endocarditis autopsied.</td>
</tr>
<tr>
<td>Segal</td>
<td>192</td>
<td>6</td>
<td>3 persistent AFB. 1 transient terminal AFB. 1 transient terminal AFB in bacteria-free stage.</td>
</tr>
<tr>
<td>Christian</td>
<td>150</td>
<td>5</td>
<td>6 AFB preceding bacterial endocarditis. 5 AFB in course of bacterial endocarditis. Both present on admission in 10 cases.</td>
</tr>
<tr>
<td>McDonald</td>
<td>196</td>
<td>21</td>
<td></td>
</tr>
</tbody>
</table>

* AFB = auricular fibrillation; AFL = auricular flutter.

mitral valve involvement; (5) active rheumatic heart disease with Aschoff bodies or characteristic verrucae; (6) bacterial endocarditis of the mitral or aortic valves with some evident previous distortion, but with the exact estimation of the severity of rheumatic damage being difficult. Cases showing the following changes were excluded from this study: (1) slight mitral changes (for example, tongue-like extensions of the valve cusps), or distortion which did not fulfill the criteria stated above (points 1 and 2); (2) slight aortic stenosis with or without slight mitral involvement; (3) calcific aortic stenosis with or without slight mitral changes; (4) calcification of the valve rings alone. Thus 103 cases of calcific aortic stenosis and 72 cases of doubtful origin were excluded from this study.

In the group of rheumatic hearts, bacterial endocarditis was present in 82 (16.1 per cent); in 5 of these the lesions were healed. These patients were compared with 47 instances of bacterial endocarditis in which the hearts did not meet the criteria for rheumatic heart disease described above. In each case the presence of auricular fibrillation and the severity of the mitral stenosis was noted. When the cardiac rhythm was abnormal, the time of onset relative to the beginning of the valvular infection was ascertained. The present study concerns those patients in whom auricular fibrillation preceded the development of bacterial endocarditis. The severity of the mitral stenosis was graded from 1 to 4 plus with 1 plus as the least severe involvement, yet sufficient to meet the criteria above, and 4 plus as the most severe involvement, representing valves described as “fish-mouth,” “button-hole,” “slit-like,” “markedly stenotic,” “could not admit one finger”; 2 and 3 plus represented the intermediate degrees of severity.

In the tables the cases of “subacute” bacterial endocarditis are also listed separately from the total cases of bacterial endocarditis although the artificial nature of this distinction is appreciated. No significant differences between these two groups are noted.

Results

There were 82 cases of bacterial endocarditis superimposed on rheumatic heart disease. Five of these were healed (all had regular rhythm) and are not included in the figures below. Of the remaining 77 cases, two patients (both with acute endocarditis) were admitted with fibrillation and the data did not indicate the length of time that this had been present; these were therefore considered with the group of established arrhythmias. Of four cases with preceding established auricular fibrillation, one patient had preceding thyrotoxicosis and two
were examples of rather acute endocarditis. Thus only six of 77 cases (7.8 per cent) could be considered to have established auricular fibrillation prior to the onset of bacterial endocarditis and this represents a maximum estimate.

Of 47 cases of bacterial endocarditis without definite underlying rheumatic heart disease the cardiac rhythm was known in 45 patients. Forty-two (93.4 per cent) had regular rhythm, one developed auricular fibrillation in the course of acute bacterial endocarditis, and two patients with terminal bacterial endocarditis had preceding auricular fibrillation.

The figures for all cases of bacterial endo-

<table>
<thead>
<tr>
<th>Grade of Mitral Stenosis</th>
<th>Rheumatic Hearts</th>
<th>Nonrheumatic Hearts</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total cases</td>
<td>82</td>
<td>47</td>
</tr>
<tr>
<td>Healed cases</td>
<td>5</td>
<td>0</td>
</tr>
<tr>
<td>Rhythm not listed</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>Residual cases</td>
<td>77</td>
<td>45</td>
</tr>
<tr>
<td>Regular rhythm</td>
<td>62</td>
<td>42</td>
</tr>
<tr>
<td>Auricular fibrillation</td>
<td></td>
<td></td>
</tr>
<tr>
<td>During infection†</td>
<td>9 (4 acute)</td>
<td>1 (acute)</td>
</tr>
<tr>
<td>Present on admission</td>
<td>2 (2 acute)</td>
<td>0</td>
</tr>
<tr>
<td>Preceded infection</td>
<td>4 (2 acute,</td>
<td>2 (2 terminal</td>
</tr>
<tr>
<td></td>
<td>1 thyrotoxic)</td>
<td>infection)</td>
</tr>
</tbody>
</table>

**TABLE 3.—Occurrence of Auricular Fibrillation in Cases of Subacute Bacterial Endocarditis Superimposed on Rheumatic and Nonrheumatic Hearts**

<table>
<thead>
<tr>
<th>Total cases of subacute bacterial endocarditis</th>
<th>Rheumatic Hearts</th>
<th>Nonrheumatic Hearts</th>
</tr>
</thead>
<tbody>
<tr>
<td>Healed cases.................................</td>
<td>4</td>
<td>7</td>
</tr>
<tr>
<td>Rhythm not listed.............................</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Residual cases...............................</td>
<td>55</td>
<td>7</td>
</tr>
<tr>
<td>Regular rhythm..............................</td>
<td>48</td>
<td>7</td>
</tr>
<tr>
<td>Auricular fibrillation Onset during infection</td>
<td>5</td>
<td>0</td>
</tr>
<tr>
<td>Present on admission Onset preceded infection</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>2 (1 thyrotoxic)</td>
<td>0</td>
</tr>
</tbody>
</table>

**TABLE 4.—Association of the Cardiac Rhythm with the Degree of Valvular Stenosis**

<table>
<thead>
<tr>
<th>Grade of Mitral Stenosis</th>
<th>Multiple Valve† All Cases of Bacterial Endocarditis 77 patients</th>
<th>Multiple Valve† Subacute Bacterial Endocarditis 55 patients</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>4+  3+  1-2+</td>
<td>4+  3+  1-2+</td>
</tr>
<tr>
<td>Total cases</td>
<td>18  26  33</td>
<td>12  19  24</td>
</tr>
<tr>
<td>Regular rhythm</td>
<td>9    25  28</td>
<td>8    18  22</td>
</tr>
<tr>
<td>Auricular fibrillation</td>
<td></td>
<td></td>
</tr>
<tr>
<td>During infection†</td>
<td>6 (2*) 1 2 (1*)</td>
<td>3    1    1</td>
</tr>
<tr>
<td>Present on admission</td>
<td>1 (1*) 0 1</td>
<td>0    0    0</td>
</tr>
<tr>
<td>Preceded infection</td>
<td>2 (1*) 0 2 (1*) (1†)</td>
<td>1    0    1</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Grade of Mitral Stenosis</th>
<th>Multiple Valve† All Cases of Bacterial Endocarditis 71 patients</th>
<th>Multiple Valve† Subacute Bacterial Endocarditis 49 patients</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>4+  3+  1-2+</td>
<td>4+  3+  1-2+</td>
</tr>
<tr>
<td>Total cases</td>
<td>14  21  36</td>
<td>8    16  25</td>
</tr>
<tr>
<td>Regular rhythm</td>
<td>7    20  31</td>
<td>6    15  23</td>
</tr>
<tr>
<td>Auricular fibrillation</td>
<td></td>
<td></td>
</tr>
<tr>
<td>During infection†</td>
<td>4    1  2</td>
<td>1    1    1</td>
</tr>
<tr>
<td>Present on admission</td>
<td>1    0  1</td>
<td>0    0    0</td>
</tr>
<tr>
<td>Preceded infection</td>
<td>2    0  2 (1†)</td>
<td>1    0    1 (1†)</td>
</tr>
</tbody>
</table>

* Acute.
† Applies to the degree of involvement of the most severely affected valve with a superimposed bacterial infection.
‡ Thyrotoxic.
carditis are summarized in table 2. No significant differences were noted when similar comparisons were made for cases of “subacute” bacterial endocarditis only (table 3).

The type of cardiac rhythm was correlated with the degree of valvular stenosis in 77 patients with active bacterial endocarditis superimposed on rheumatic heart disease. Of 18 patients with 4 plus valvular involvement, one patient with acute endocarditis had auricular fibrillation on admission and two patients (one acute, one possibly acute) had auricular fibrillation preceding the bacterial infection. Preceding auricular fibrillation did not occur in 26 cases with 3 plus valvular involvement. Of 33 patients whose hearts showed 1 to 2 plus valvular involvement, one patient was admitted to the hospital with fibrillation and two patients had pre-existing auricular fibrillation (one was thyrotoxic and one had acute endocarditis). These cases are summarized in table 4.

These figures, though too few for statistical analysis, indicate no significant variation in the incidence of auricular fibrillation in patients with different grades of valvular stenosis. No significant difference is noted when the series is divided into groups of cases including all active bacterial endocarditis or only “subacute” bacterial endocarditis, and involvement of multiple valves or only mitral valvular involvement (table 4).

In order to determine the relationship between the severity of mitral stenosis and the development of bacterial endocarditis, the percentage of cases with varying grades of mitral valvular involvement in the group with bacterial endocarditis was compared with the whole series of patients with rheumatic heart disease. The degree of mitral valve involvement was known in 458 of the 509 cases. Two hundred ten (46 per cent) of these had severe (4 plus) mitral stenosis compared with 14 of 71 cases (19.7 per cent) with bacterial endocarditis. These results appear in table 5.

These findings indicate that bacterial endocarditis does involve valves with severe mitral stenosis, but does so less often than would be expected from the over-all frequency of severe stenosis.

### Table 5.—Comparison of Severity of Rheumatic Mitral Involvement in all Rheumatic Patients and in Rheumatic Patients with Superimposed Bacterial Endocarditis

<table>
<thead>
<tr>
<th>Grade of Mitral Stenosis</th>
<th>All Patients with Mitral Stenosis (498 cases)</th>
<th>Mitral Stenosis with Superimposed Bacterial Endocarditis (71 cases)</th>
<th>Mitral Stenosis with Superimposed Subacute Bacterial Endocarditis (19 cases)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No.</td>
<td>%</td>
<td>No.</td>
</tr>
<tr>
<td>4+</td>
<td>210</td>
<td>46</td>
<td>14</td>
</tr>
<tr>
<td>3+</td>
<td>86</td>
<td>19</td>
<td>21</td>
</tr>
<tr>
<td>1-2+</td>
<td>162</td>
<td>35</td>
<td>36</td>
</tr>
</tbody>
</table>

### Comment

The remarkable infrequency of arrhythmias in patients with subacute bacterial endocarditis was pointed out first by Libman and his associates, but no explanation was offered. Levine noted that a patient with evidence of mitral stenosis is distinctly less liable to develop subsequent bacterial endocarditis. He pointed out that auricular fibrillation in rheumatic heart disease is almost always associated with mitral stenosis; and in auricular fibrillation the leaflets are apt to be dense and fibrous. He suggested that the fibrous leaflets of a valve with mitral stenosis are “less fertile soil” for streptococcus viridans. The incidence of auricular fibrillation in subacute bacterial endocarditis is about 3 per cent. The incidence of severe (4 plus) mitral stenosis in subacute bacterial endocarditis in this study is 16 per cent. There remain then 13 per cent of the cases which do not benefit from the explanation offered by Levine. Table 4 also indicates that auricular fibrillation is associated with 1 and 2 plus mitral stenosis just as frequently as with 4 plus mitral stenosis. Furthermore the figures in tables 2 and 3 show that auricular fibrillation occurs with the same frequency in bacterial endocarditis superimposed on rheumatic as on nonrheumatic hearts. Therefore, it is not the mitral stenosis, per se, or its severity, which accounts for the low incidence of auricular fibrillation in subacute bacterial endocarditis. Segal concurs in this opinion but offered no statistical evidence.

Sprague reported 20 cases of subacute bacterial endocarditis studied post mortem and
found no marked degree of mitral stenosis. He states: "It has been suggested that the rigid calcified valves of chronic mitral stenosis are less favorable ground for superimposed bacterial infection than those seen in malignant endocarditis, being subjected to less trauma since the cusps can no longer shut and so cannot strike their abnormal surfaces against one another." That some other factor in addition to less active protective antigenic agents and healing and calcification of valves is of importance is "suggested by the knowledge that this infection is much less common in childhood, at the time when rheumatic carditis is prevalent and affected mitral valves are not healed or calcified." Further it is precisely these rigid valves which Allen has indicated are subject to most trauma since

<table>
<thead>
<tr>
<th>Grade of Mitral Stenosis</th>
<th>Davis and Weiss (474 cases)</th>
<th>Wallach, Glass, Lukash and Angrist; 309 cases (458 were graded)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No.</td>
<td>%</td>
</tr>
<tr>
<td>4+</td>
<td>57</td>
<td>12</td>
</tr>
<tr>
<td>3+</td>
<td>85</td>
<td>18</td>
</tr>
<tr>
<td>1-2+</td>
<td>332</td>
<td>70</td>
</tr>
</tbody>
</table>

there is no appreciable difference in the frequency with which males and females were affected by bacterial endocarditis. These authors suggested that the progressive development of mitral stenosis meant continued "local allergy with persistent rheumatic state," and that, therefore, these patients have not developed the immunity requisite for subacute bacterial endocarditis.

Davis and Weiss graded the degree of valvular involvement from I to IV. They found that "the distribution of the degree of rheumatic valvular damage in the 47 cases of subacute bacterial endocarditis corresponded with that for 474 cases of rheumatic heart disease, indicating that the development of subacute bacterial endocarditis is independent of the degree of underlying rheumatic endocarditis." They believed that the apparent infrequency of advanced mitral stenosis with subacute bacterial endocarditis is explained by the fact that slight lesions while just as likely to develop endocarditis "are much more common than advanced ones. The same factor is held responsible for the relatively rare occurrence of auricular fibrillation in the course of subacute bacterial endocarditis." The statistics in the present study (table 6) do not support their conclusions.

The most likely explanation for the discrepancy in the frequency of the four grades of stenosis may be the use of slightly different criteria for the selection of grade I cases. It is most probable that Davis and Weiss included many cases with minimal evidence of apparent rheumatic involvement. Their grade I "included the earliest changes, consisting in definite thickening of the free margin of the valves." Many of these may have represented commonly seen fibrous or edematous non-rheumatic thickening of the valve margins. These hearts show no definite rheumatic stigmata and have not been included in our series. This diluted the number of their cases with the most severe type of valvular distortion (4 plus).

Laws and Levine also disagreed with Davis and Weiss and felt that "there is a large number of patients with persistent auricular fibrillation and valvular disease who live on
the average for several years. If the bacterial invasion of damaged valves were a mere accident, it is surprising that there are not more instances of subacute bacterial endocarditis in patients with auricular fibrillation." De Graff and Lingge14 concluded "that patients who died of subacute bacterial endocarditis have not had rheumatic heart disease long enough to develop auricular fibrillation" which they regarded as a terminal condition.

Segal12 pointed out that "the rare incidence of auricular fibrillation in active subacute bacterial endocarditis cannot be attributed entirely to the same incidence of severe mitral stenosis in this disease." The statistical evidence in the present study supports the views of Segal.15 He pointed out that "the integrity of the myocardium and the underlying physiologic and biological state would appear to be more closely related to the pathogenesis of auricular fibrillation than the grade of mitral stenosis per se." However, at present there is no known morphologic counterpart for this "state." McDonald18 concluded that "the answer to this problem seems to depend upon two factors which contribute to the development of auricular fibrillation: the integrity of the myocardium and the duration of the disease affecting the heart."

Each of the various theories summarized above have stressed individual factors such as the resistance of the fibrotic valve to infection, local trauma to the valve, local valve "immunity," the role of the patient, the role of chance in valvular infection, the integrity of the myocardium, and the duration of the rheumatic valvulitis. However, an adequate overall explanation has not been offered nor have the basic mechanisms been elucidated.

It is well to define the limitations of present day knowledge. The pathology of auricular fibrillation is not known.21 The physiologic and pharmacologic concepts of auricular fibrillation are controversial.22 The reasons for the very frequent association of auricular fibrillation with mitral stenosis (or for that matter arteriosclerotic heart disease or thyrotoxicosis) are hypothetic. Furthermore the mechanism responsible for progressive stenosis of the rheumatic valves has not been adequately demonstrated. The relationship of such factors as the age of the patient and the duration of the valvular distortion is not well recognized. Finally, the effects of stress and the role of the adrenals have not been sufficiently studied and may play a vital part.

It has been adequately demonstrated that bacterial endocarditis represents infection of nonbacterial thrombotic lesions by organisms in the bloodstream.23 Since bacterial invasion of the bloodstream occurs so frequently in all individuals,24 it cannot be the infectious factor alone which is the significant one. Therefore the presence of rather recent nonbacterial thrombotic lesions would seem more significant for the development of bacterial endocarditis, although both are required simultaneously for the initiation of bacterial endocarditis.

It appears most probable that local and general stress represents the inciting and crucial factors responsible for the occurrence of such nonbacterial thrombotic vegetations and perhaps also for the sufficient mobilization of antibacterial forces to favor localization to the affected valve. The local stress factors determined by certain physical principles (impact and contact) have been emphasized by Allen.15 The experimental production of nonbacterial thrombotic endocarditis in animals subjected to various stresses is now in progress. It is worth noting that one type of stress which has been successfully used is the repeated injection of killed bacteria.25 The relationship of this technic to the statements in the older literature concerning the high degree of bacterial immunity in patients with subacute bacterial endocarditis is of interest. The stress of injections of bacteria to give this high degree of immunity may have produced the nonbacterial thrombotic lesions, thereby reproducing the above experiment.

There is a wide range of variation in the response of affected people to stress. The heart valves of the majority of individuals do not react demonstrably to the type of stress encountered in everyday life; these do not develop rheumatic heart disease. Next are the individuals who respond to some stress with the production of rheumatic valvulitis. Once
this alteration has occurred in the ground substance of the valve, future stresses may produce further changes in this collagen more readily. These changes may be manifested pathologically as nonbacterial endocarditis in and on the rheumatic valve. This nonbacterial lesion is organized, becomes incorporated as part of the valves, and is responsible for much of the valvular distortion usually attributed to the rheumatic valvulitis.

Considerable valvular distortion (for example, "4 plus," "fish-mouth," "button-hole") of the type frequently associated with auricular fibrillation results with the healing of repeated nonbacterial thrombotic lesions. By this stage the patient has demonstrated his ability to resist bacterial infection of his nonbacterial thrombotic lesions. These then are apt to be healed and fibroized; hence the apparent immunity of patients with severe mitral stenosis of longer duration to subacute bacterial endocarditis. Occasionally the nonbacterial thrombotic vegetation becomes infected and bacterial endocarditis results. Similar events may occur when the basic alteration of the valve collagen is associated with sclerotic changes rather than rheumatic valvulitis. If bacterial infection of the nonbacterial thrombotic lesion occurred, the patient usually succumbed and therefore did not have time to develop progressive valvular distortion or auricular fibrillation. Likewise those rheumatic patients who died earlier in their disease of other complications (for example, embolization) could not go on to develop the more severe valvular distortion or bacterial infection. This aspect is in basic agreement with the conclusions of DeGraff and Linge14 and McDonald.18

Although auricular fibrillation occurs most frequently in association with such factors as increasing age of the patient, longer duration and greater severity of the valvular lesion, it is well to recognize that fibrillation is dependent on muscle rather than valve function. The myocardial changes in chronic rheumatic heart disease are only secondarily related to the valvular distortions. However, aging of the myocardium (presbycardia29) may play an additional important role similar to that which occurs in arteriosclerotic heart disease.

The exceptions to the above generalizations result from those occasional patients who continue to respond to stress with the development of rheumatic or nonbacterial thrombotic formation although the valve is markedly distorted already. Those which became infected represent the examples of bacterial endocarditis with preceding auricular fibrillation presented earlier in the paper.

SUMMARY AND CONCLUSIONS

The infrequent occurrence of auricular fibrillation and the degree of severity of rheumatic mitral valvular involvement is reported in a group of autopsied patients with bacterial endocarditis.

The unusual coincidence of bacterial endocarditis with auricular fibrillation and mitral stenosis in other series of cases is reviewed.

Previous theories for the rarity of these associations are analyzed and an alternative explanation is offered.

SUMARIO ESPAÑOL

Se informa la ocurrencia infrecuente de fibrilación auricular y el grado de severidad de envolvimiento reumático de la válvula mitral en un grupo de pacientes con endocarditis bacterial a quienes se les hizo autopsia.

La coincidencia poco usual de endocarditis bacterial con fibrilación auricular y estenosis mitral en otra serie de casos se revisa.

Teorías previas para la rareza de estas combinaciones se analizan y una explicación alternativa se ofrece.

REFERENCES


Levine, S. A.: Some unproved impressions con-


