Aortic Valve Perforation in Bacterial Endocarditis

By Ralph Tompsett, M.D., and Glenn D. Lubash, M.D.

The modern treatment of bacterial endocarditis may be expected to result in bacteriologic cure in 90 to 95 percent of patients who complete a course of treatment. Nevertheless as many as 25 percent of patients die from emboli or cardiac failure within the first 6 months from initiation of treatment. In addition to the underlying heart disease, the additional factor of destruction of all or part of a valve may be added during the course of bacterial endocarditis and contribute to congestive heart failure, despite bacteriologic cure. The importance of the development of dynamically significant aortic insufficiency during the course of bacterial endocarditis has been described in numerous case reports and various authors have commented on its significance.

During the course of observation of approximately 150 patients with proved bacteriologic endocarditis over a 10-year period we have also recognized the importance of this complication appearing during treatment or the early convalescent period. Moreover, the clinical features have seemed to be reasonably typical in most cases and, when present, have generally been followed by progressive, often intractable heart failure. The recent interest in surgical procedures that may afford a significant chance of improvement in these patients has made it seem worthwhile to review the problem with a view to directing attention.

Table 1
Summary Data on Patients with Aortic Valve Perforation in Bacterial Endocarditis

<table>
<thead>
<tr>
<th>Case no.</th>
<th>Sex</th>
<th>Age</th>
<th>Type of heart disease</th>
<th>Cusp involved</th>
<th>Lesion of cusp</th>
<th>Relation of signs of aortic insufficiency to endocarditis</th>
<th>Duration of life after either appearance of or change in signs of aortic insufficiency (mo.)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>M</td>
<td>49</td>
<td>Syphilitic</td>
<td>Posterior and right</td>
<td>Perforation</td>
<td>Changed</td>
<td>3-5</td>
</tr>
<tr>
<td>2</td>
<td>M</td>
<td>47</td>
<td>Rheumatic</td>
<td>Posterior and left</td>
<td>Perforation</td>
<td>Appeared</td>
<td>1</td>
</tr>
<tr>
<td>3</td>
<td>M</td>
<td>48</td>
<td>Congenital (IV septal defect)</td>
<td>Posterior, right and left</td>
<td>Perforation and rupture</td>
<td>Appeared</td>
<td>33</td>
</tr>
<tr>
<td>4</td>
<td>M</td>
<td>43</td>
<td>Rheumatic</td>
<td>Right and left</td>
<td>Perforation</td>
<td>Uncertain, probably new</td>
<td>2-6</td>
</tr>
<tr>
<td>5</td>
<td>M</td>
<td>55</td>
<td>Uncertain, probably rheumatic</td>
<td>Posterior</td>
<td>Perforation</td>
<td>Perforation</td>
<td>3</td>
</tr>
<tr>
<td>6</td>
<td>F</td>
<td>53</td>
<td>Rheumatic</td>
<td>Posterior and left</td>
<td>Perforation</td>
<td>Appeared</td>
<td>½ to 6</td>
</tr>
<tr>
<td>7</td>
<td>F</td>
<td>55</td>
<td>Rheumatic</td>
<td>Right</td>
<td>Perforation</td>
<td>Changed</td>
<td>3</td>
</tr>
<tr>
<td>8</td>
<td>F</td>
<td>64</td>
<td>Rheumatic</td>
<td>Right</td>
<td>Perforation and rupture</td>
<td>Appeared</td>
<td>8</td>
</tr>
<tr>
<td>9</td>
<td>M</td>
<td>30</td>
<td>Rheumatic</td>
<td>Posterior</td>
<td>Perforation</td>
<td>Changed</td>
<td>4</td>
</tr>
<tr>
<td>10</td>
<td>M</td>
<td>71</td>
<td>Rheumatic</td>
<td>Posterior</td>
<td>Perforation</td>
<td>Appeared</td>
<td>2-8</td>
</tr>
</tbody>
</table>
to the recognition of this complication of bacterial endocarditis.

The present report summarizes observations in patients with bacterial endocarditis seen at the New York Hospital between 1944 and 1956, in whom dynamically significant aortic insufficiency appeared after initiation of therapy and in whom postmortem examination revealed perforation or destruction of one or more aortic valve cusps.

**Observations**

The postmortem records at the New York Hospital from January 1944 to January 1956 were reviewed. Forty-seven cases of bacterial endocarditis were autopsied during this period. The clinical records were reviewed and compared with the postmortem observations. Those cases were chosen that fulfilled the following criteria:

1. Absence of dynamically significant aortic insufficiency prior to the development of endocarditis. 2. The development of dynamically significant aortic insufficiency during or soon after treatment. 3. Autopsy evidence of either perforation or destruction of an aortic valve cusp due to bacterial endocarditis.

Ten suitable cases were found and are summarized in table 1. A representative illustration of the changes seen in the aortic valve is presented in figure 1.

**Discussion**

These cases illustrate what appears to be an important complication of bacterial endocarditis, namely, perforation or destruction of the aortic valve cusps. In all of the patients, bacteriologic cure of the endocarditis had been achieved or could reasonably have been anticipated had it not been for the fact that progressive cardiac failure supervened.

It is impossible to estimate the frequency of aortic valve perforation in bacterial endocarditis from the data at hand. During the 12 years covered by this study, approximately 150 patients at this hospital were recognized during life to have bacterial endocarditis and treated for it. Forty-seven cases were autopsied during this period. (It should be noted that not all of these were recognized during life and treated.) Of those cases autopsied, 15 had perforation of the aortic valve. Although only 10 cases are included here because of the criteria mentioned in the introduction, it is possible that aortic valve perforation was an important factor in other patients as well.

The very complexity of the clinical situation in which valve perforation occurs tends to obscure the fact that valve perforation has occurred and that the likelihood of progression to intractable heart failure is good. It is hoped that a greater awareness of the importance of aortic valve perforation in bacterial endocarditis coupled with improved surgical procedures for correction of aortic insufficiency may enhance the outlook for this group of patients.

**Summary**

This report describes 10 patients with bacterial endocarditis who during observation developed signs of dynamic aortic insufficiency or experienced marked worsening of the manifestations of pre-existing aortic insufficiency. The autopsy findings in these patients provide evidence that the clinical signs were associated with perforation or rupture of the aortic valve cusps. It is suggested that this sequence of events in the course of bacterial endocarditis is common and that its occurrence must be regarded as an ominous prognostic sign.
Acknowledgment

The authors are grateful to Dr. John Kidd of the Department of Pathology, Cornell University Medical College, for permission to review the autopsy records on the cases reported herein.

References


To put the discovery of the systemic circulation of the blood in its true light, we must have some notion of the history of philosophy, science and medicine. Medicine, and herein it is in contrast with Theology and Law, had its sources almost wholly in the Greeks. Not only in the doctrine of the four elements of Empedocles, a doctrine which has survived almost to our own day, and in the physical theories of Heraclitus and Leucippus, did medicine, for good or ill, first find a scheme of thought, but in the schools of Hippocrates and of Alexandria it was based also, and far more soundly, upon natural history and anatomy. The noble figure of Galen, the first experimental physiologist and the last of the great Greek physicians, stood eminent upon the brow of the abyss when, as if by some convulsion of nature, medicine was overwhelmed for fifteen centuries. To the philosophy of medicine, Galen had given more than enough; to its natural history he had contributed in the following of Hippocrates; to its discoveries he had given the greatest of all means of research, individual genius; to its methods he had given, but in vain, that indispensable method, practised first perhaps in history by Archimedes and the Alexandrians, of verification by experiment; a method, after Galen, virtually lost till the time of Gilbert, of Galileo and of Harvey.—THOMAS CLIFFORD ALLBUTT, M.A., M.D. Science and Medieval Thought. London, C. J. Clay and Sons, 1901, p. 20.
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