Complete Heart Block Complicating Bacterial Endocarditis

By Kyuhyun Wang, M.D., Fredrick Gobel, M.D., Donald F. Gleason, M.D., and Jesse E. Edwards, M.D.

SUMMARY
Among 142 cases of bacterial endocarditis (BE), complete heart block (CHB) was found in six cases (4%) and first-degree (1°) or second-degree (2°) A-V block in 14 cases (10%).

The aortic valve was involved in 18 of 20 cases with atrioventricular (A-V) conduction disturbance, including all six cases of CHB. Anatomic observations (four autopsy, one operative) were made in five of the six cases of CHB. In these cases, a common finding, in addition to involvement of the aortic valve, was extension of the infection to adjacent structures resulting in cardio-aortic fistulae. CHB likely resulted from extension of infection to the major conduction tissues.

Five of the six patients with CHB died suddenly while in the hospital. One patient was treated with electric pacing while the infection was being controlled and, 38 days later, underwent successful replacement of the aortic valve. Conduction abnormalities are important possible complications of aortic valvular BE. Prompt pacing may be a lifesaving procedure, allowing eradication of infection as a Prelude to surgical therapy.

Additional Indexing Words:
Aortic valvular endocarditis
Cardioaortic fistula
Atrioventricular conduction disturbance

Patients with bacterial endocarditis (BE) continue to have a high risk of immediate mortality (30%)1 despite the advent of antibiotic therapy. On occasion, complete heart block (CHB) may complicate BE and contribute to poor prognosis. The purpose of this study was to determine the frequency and the pathologic features of CHB in patients with BE.

Methods and Materials
Records were reviewed of all adult patients who had diagnostic coding of BE between 1961 and 1971 at the University of Minnesota Hospitals (UHM) and the Minneapolis Veterans Administration Hospital (MVAH). One hundred forty-two cases were found acceptable for inclusion in this study (table 1).

Criteria used for including cases as examples of BE were as follows. (1) Acquired valvular or congenital heart disease and positive blood cultures (66 cases). (2) Direct examination of interior of heart, either at operation or autopsy (59 cases). (3) Typical clinical profile of BE and response to antibiotic therapy in spite of negative blood cultures (17 cases).

Negative blood cultures were obtained in 14 patients (25%) with pathologically proven BE and in 17 patients who fulfilled the third criterion. Of the patients with negative blood
cultures, many had received some form of antibiotic therapy prior to transfer to our hospitals.

In each case, the electrocardiograms were reviewed for the nature of A-V conduction. When conduction abnormalities were demonstrated to have been present before the onset of BE, the cases were not considered to be examples of A-V conduction disturbance caused by BE.

There were 57 deaths; 51 autopsies were performed. Special consideration was given to the six cases in which CHB was discovered. Among these, five deaths occurred. Autopsies had been done on four. Records of each of the latter four autopsies were reviewed. In three of these cases, the specimens of heart also were available for review. In the one living patient with CHB, the aortic valve was visualized at operation.

Results

Among the 142 cases, the most common sites of evident valvular disease in order of decreasing frequency were the aortic valve alone, 55 cases; the mitral valve alone, 39 cases; and aortic and mitral valves together, 28 cases. Of the 142 patients with BE reviewed, CHB was documented in six (4%) and 14 others (10%) showed 1\textdegree\ or 2\textdegree\ A-V block (table 2).

In each of the six cases with CHB, the aortic valve was the site of preexisting disease and by direct examination was also shown to be the site of BE. In the 14 cases of incomplete heart block, the sites of background disease were considered to be the aortic valve alone in eight cases, the aortic and mitral valves together in four cases, and the mitral valve alone in two cases. Five of the 14 patients with incomplete A-V block died. Autopsy in each revealed the aortic valve to be involved.

Thus, of the 55 patients with a background of aortic valvular disease alone, six (11%) showed CHB and eight (15%) showed 1\textdegree\ or 2\textdegree\ A-V block. Among the 28 cases with background disease both in the aortic and mitral valves, there were no cases of CHB, but four (14\%) with incomplete A-V block. In patients who clinically had evidence of only mitral valve disease, there were two cases of incomplete A-V block. In none of the other categories of background disease were there any cases of A-V conduction disturbance.

The cases with CHB showed a strong tendency for the infection not only to involve the aortic valve extensively but also to extend to and beyond the aortic wall, as evidenced by the fact that a mycotic aneurysm and a cardioaortic fistula were demonstrated in five of the six cases (one at operation and four at autopsy (figs. 1–3; table 3).

The sites of termination of the fistulae beginning in the aorta were as follows: right atrium (case 3), right atrium and left ventricle (case 2), and both ventricles (case

<table>
<thead>
<tr>
<th>Basis for diagnosis</th>
<th>Cases (no.)</th>
<th>Positive No.</th>
<th>Positive %</th>
<th>Negative No.</th>
<th>Negative %</th>
<th>No culture taken (no.)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Autopsy</td>
<td>51</td>
<td>42</td>
<td>75</td>
<td>14</td>
<td>25</td>
<td>3</td>
</tr>
<tr>
<td>Operation</td>
<td>8</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Clinical features only</td>
<td>83</td>
<td>66</td>
<td>80</td>
<td>17</td>
<td>20</td>
<td>0</td>
</tr>
<tr>
<td>Total</td>
<td>142</td>
<td>108</td>
<td>78</td>
<td>31</td>
<td>22</td>
<td>3</td>
</tr>
</tbody>
</table>

Table 2

<table>
<thead>
<tr>
<th>Site of underlying valvular disease</th>
<th>All</th>
<th>CHB</th>
<th>1\textdegree/ or 2\textdegree\ HB</th>
</tr>
</thead>
<tbody>
<tr>
<td>AV only</td>
<td>55</td>
<td>6</td>
<td>8</td>
</tr>
<tr>
<td>AV &amp; MV</td>
<td>28</td>
<td>0</td>
<td>4</td>
</tr>
<tr>
<td>MV only</td>
<td>39</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>MV &amp; TV</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>None</td>
<td>10</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Right side</td>
<td>9</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Total</td>
<td>142</td>
<td>6</td>
<td>14</td>
</tr>
</tbody>
</table>

Abbreviations: AV = aortic valve; MV = mitral valve; TV = tricuspid valve.
Case 3. (a) A portion of aortic valve. The posterior cusp is perforated (point of arrow). Involvement of the aortic sinus by infectious process is obscured by the cusp. (b) Left atrium. The bilobed protrusion (between arrows) above the mitral valve represents extension of infection from the posterior aortic sinus to the atrial septum. This lesion has not perforated. (c) Right atrium (R.A.). A mycotic aneurysm (between arrows) of the posterior aortic sinus has presented against the septal wall of the right atrium. The aneurysm has perforated (probe).

4), one case each and left ventricle (tract bypassing the aortic valve), two cases (cases 1 and 6).

In two of three cases (cases 2 and 3) of CHB in which autopsy was done and in which there was demonstrated a fistula originating in the posterior aortic sinus, the infectious process extended to the atrial septal wall and involved the A-V node or bundle of His (fig. 3).

In the one case (case 4, fig. 4) where the fistula began in the right aspect of the anterior sinus of a bicuspid aortic valve, there did not appear to be direct involvement of the conduction system by the infection. In this case, extensive coronary atherosclerosis and multiple acute myocardial infarcts were present. While some of the latter may have been of embolic origin, no coronary emboli were demonstrated. The multiplicity of infarcts suggests that some part of the major conduction system had been so involved, but this was not demonstrated.

Organisms were identified in five cases with CHB and were not particularly unusual. Alpha streptococcus was recovered from three patients, coagulase-positive staphylococcus from one patient, and E. coli from the fifth.

Signs of aortic insufficiency were present in five cases, in four of which these signs preceded the appearance of CHB.
There were individual variations in the development of the conduction disturbance in all six cases. In case 1, the patient exhibited normal conduction at the time of hospital admission and then developed $1^\circ$ A-V block with left-axis deviation (fig. 5a). Two days later, CHB appeared and the patient died on that day from ventricular fibrillation. In case 2, normal A-V conduction on admission was followed by CHB noted on a subsequent electrocardiogram taken on the tenth hospital day. A transvenous endocardial pacemaker
Case 2. (a) Interior right atrium (R.A.). Anterior to the ostium of the coronary sinus (C.S.) is a zone of discoloration at the lowermost portion of the atrial septum. This corresponds to the lesion seen in figure 2b. It lies at the anticipated location of the A-V node (rectangle represents a section taken prior to the illustration and not intended to show the major conduction tissue; section of the latter obtained following the preparation of this illustration). (b) Low-power photomicrograph of atrial septum, A-V node (A.V.N.) and central fibrous body. The atrial septum shows hemorrhage, as well as leukocytic infiltration. The latter feature is shown more clearly in c and d. Elastic tissue stain; 20 ×. (c) A-V node (A.V.N.) and atrial septum. The latter shows hemorrhage and leukocytic infiltration. A-V node is involved by inflammatory process. H & E; 45 ×. (d) High-power view of A-V node showing muscle fibers separated by infiltrating leukocytes, edema, and hemorrhage. H & E; × 150.

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Table 3

Essential Data in Six Cases of Complete Heart Block Complicating Bacterial Endocarditis

<table>
<thead>
<tr>
<th>Case</th>
<th>Age (yr)</th>
<th>Sex</th>
<th>Background state of aortic valve</th>
<th>Cardioaortic fistula</th>
<th>Bacteriology</th>
<th>Sequence of conduction disturbance</th>
<th>Administration of digitalis</th>
<th>Serum potassium (mEq/liter)</th>
<th>Cause of death</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>64</td>
<td>M</td>
<td>No. 10 Starr-Edwards prosthetic</td>
<td>Posterior aortic sinus to LV</td>
<td>Coagulase-positive staphylococcus</td>
<td>Normal → 1° → CHB</td>
<td>No</td>
<td>6.5</td>
<td>Ventricular arrhythmia</td>
</tr>
<tr>
<td>2</td>
<td>77</td>
<td>M</td>
<td>Senile calcific</td>
<td>Posterior aortic sinus to LV and RA</td>
<td>α-Hemolytic streptococcus</td>
<td>Normal → CHB</td>
<td>Yes</td>
<td>4.4</td>
<td>Sudden death while being electrically paced</td>
</tr>
<tr>
<td>3*</td>
<td>77</td>
<td>M</td>
<td>Congenital bicuspid</td>
<td>Posterior aortic sinus to RA</td>
<td>α-Hemolytic streptococcus</td>
<td>AF → CHB</td>
<td>Yes</td>
<td>4.7</td>
<td>Congestive heart failure</td>
</tr>
<tr>
<td>4†</td>
<td>56</td>
<td>F</td>
<td>Congenital bicuspid</td>
<td>“Right” αortic sinus to LV and RV</td>
<td>Multiple negative blood cultures</td>
<td>Normal → CHB → Normal</td>
<td>Yes</td>
<td>4.5</td>
<td>Following aortic valve replacement (3 days)</td>
</tr>
<tr>
<td>5*</td>
<td>74</td>
<td>M</td>
<td>No. 10 Starr-Edwards prosthetic</td>
<td>No autopsy</td>
<td>E. coli</td>
<td>AF → CHB → AF</td>
<td>Yes</td>
<td>5.7</td>
<td>Congestive heart failure</td>
</tr>
<tr>
<td>6</td>
<td>49</td>
<td>M</td>
<td>Congenital bicuspid</td>
<td>Posterior aortic sinus to LV</td>
<td>α-Hemolytic streptococcus</td>
<td>CHB → 2° → 1°</td>
<td>Yes</td>
<td>4.5</td>
<td>Aortic valve replaced, living</td>
</tr>
</tbody>
</table>

*Cases 3 and 5 each had atrial fibrillation with variable A-V conduction, developed CHB, and later returned to variable A-V conduction.
†In addition to the fistula, case 4 showed multiple small myocardial infarcts.
‡At the time when CHB developed.
§Right aspect of anterior sinus in congenital bicuspid aortic valve.
Abbreviations: LV = left ventricle; RA = right atrium; RV = right ventricle; CHB = complete heart block; AF = atrial fibrillation; 1° = first-degree A-V block; 2° = second-degree A-V block.
was inserted into the right ventricle. The patient died suddenly 3 days later while pacing was in effect.

Two patients (cases 3 and 5) showed atrial fibrillation and transient CHB. The ventricular rates were regular at 25 and 30 beats/min, respectively. In each, following pacing and withholding digitalis, CHB disappeared while atrial fibrillation continued. Death resulted from congestive heart failure and sepsis. In case 4, transient CHB was noted preoperatively. The patient died of ventricular fibrillation 3 days following emergency aortic valve replacement. Case 6 presented with CHB. Following medical treatment and transvenous pacing, conduction improved initially to $2^\circ$ A-V block, then to $1^\circ$ A-V block (fig. 5b). The patient remains alive and well following aortic valve replacement.

Discussion

CHB complicating BE has only uncommonly been reported. Approximately 14 such cases are described in the English literature. Rabinovich and associates, in a clinical report, described one case of CHB among 141 cases of BE. Pearce and Guze found second- or third-degree heart block in three cases among 85 cases of BE. In each patient, myocardial abscesses were found but the anatomic details were not further described. Shinebourne and associates found no case of

Figure 4

Case 4. (a) Perforation of aneurysm involving right aspect of anterior sinus of bicuspid aortic valve into septal wall of right ventricular infundibulum. (b) Low-power photomicrograph of perforation of aortic wall (A.) and of the sinus tract leading to the right ventricle (R.V.). Elastic tissue stain; $\times 5$. 

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A-V block in 93 cases of BE. Conversely, when Penton and co-workers reviewed 224 cases of CHB, one was in a case of BE. In our series, there was a 4% incidence (six cases) of CHB complicating BE. This incidence increases to 11% if only patients with isolated aortic valvular disease are included.

Our study emphasizes the common involvement of the aortic valve when heart block complicates BE. In each of the six patients with CHB and in 12 of the 14 with 1° or 2° A-V block, aortic valvular disease was associated with BE. In only two cases without clinical evidence of aortic valvular disease was BE associated with any degree of heart block.

It will be recalled that, in four of five cases with CHB in which anatomic observation was available, a fistula beginning in the posterior (noncoronary) aortic sinus was present. In the fifth case, the fistula began in the right aspect of the anterior sinus of a bicuspid aortic valve. CHB likely resulted from direct extension of the infectious process to the A-V node or bundle of His. The proximity of the A-V node and the bundle of His to the posterior aortic sinus makes CHB understandable under these circumstances.

In our material, the common finding of involvement of the posterior aortic sinus in association with mycotic aneurysm and cardio-aortic fistulae conforms to the observations of Roberts and Somerville who reported on A-V conduction disturbances in BE.

The pathologic details of one of our patients with only 1° A-V block were reported by Wang and associates. Infection of the aortic valve included the posterior cusp. This was associated with mycotic aneurysms of the ascending aorta and posterior aortic sinus. The aneurysms bulged into the right atrial aspect of the atrial septum. Histologic examination revealed an inflammatory process encroaching upon but not invading the A-V node.

Our material showed that CHB in association with BE may be transient (four of six patients). It is possible that digitalis therapy played an additive role in causing CHB in cases 3 and 5, since conduction improved when digitalis was discontinued. In case 3,
However, necropsy later revealed heavy infiltration by neutrophils of the A-V node. No necropsy was done in case 5. In Stenström's report on a patient with BE, CHB reverted to 2° A-V block, then to 1° A-V block before death. Histologic examination revealed healing inflammation in the A-V node and in relation to the bundle of His. In addition to the pattern of CHB reverting to lesser degrees of conduction disturbance, the reverse may also be seen. In one of our patients with CHB, the latter disturbance was preceded by 1° A-V block (case 1).

That the development of CHB is a serious manifestation in BE is supported by our observations. It will be recalled that our six cases of CHB were among 142 cases of BE. Death occurred in five of the six cases with CHB, while the overall mortality of patients with BE in this study was 40%.

The high rate of death among patients with CHB is probably related to complexity of the disease which includes compounding of the effects of CHB, aortic valvular dysfunction, the high chance of runoff through a fistula, and infection. Care of the patient should include management of the CHB with pacing, control of serum potassium levels, and avoidance of digitalis intoxication.

The value of maintaining an adequate cardiac rate is supported by our case 6. In this case, after increasing the ventricular rate by pacing, circumstances allowed the infection to be overcome. These procedures, in turn, set the stage for an orderly approach to the surgical management of the destructive aortic valvular disease.

References

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