Diagnosis of Trifascicular Damage Following Tetralogy of Fallot and Ventricular Septal Defect Repair

STEVEN M. YABEK, M.D., JAY M. JARMAKANI, M.D., AND NIGEL K. ROBERTS, M.D.

SUMMARY We evaluated the usefulness of the surface electrocardiogram to predict the presence of postoperative trifascicular damage. We used the pre and postoperative electrocardiograms and postoperative His bundle electrograms from 50 patients an average of 3.8 years following tetralogy of Fallot (TF) and ventricular septal defect (VSD) repair. Right bundle branch block (RBBB) and RBBB with left anterior hemiblock (LAH) developed in 88% and 18%, respectively, and 22% had transient postoperative complete heart block (CHB). Mean P-A and A-H conduction times were 16.6 and 84.9 msec and were not different from 37 age-matched normal patients and 61 patients with unoperated TF and VSD. The mean H-V conduction time of 48.5 msec was significantly increased compared to the other two groups. Ten postoperative patients had H-V times of 55 msec or greater, indicating trifascicular damage. Of these, five had electrocardiographic bifascicular block (RBBB with LAH) with or without additional 1° A-V block. The remaining five patients’ electrocardiograms did not suggest trifascicular damage. Since postsurgical trifascicular damage may progress to late onset CHB, conduction studies are indicated to identify patients at risk, despite surface electrocardiographic findings.

SYNCOPAL EPISODES AND SUDDEN DEATH have been known to follow tetralogy of Fallot (TF) and ventricular septal defect (VSD) repair. Various reports have associated these events with complete heart block (CHB) due to surgically-induced trifascicular damage. Certain postoperative electrocardiographic patterns, particularly right bundle branch block (RBBB) with left anterior hemiblock (LAH), have been associated with trifascicular damage. Many recent reports have attempted to assess the prognosis of patients with postoperative bifascicular block (RBBB with LAH) as seen on the standard electrocardiogram. Although the clinical significance and prognostic value of this electrocardiographic finding is not agreed upon, the ability to diagnose postoperative trifascicular damage accurately and therefore to assess the potential risk for late onset CHB from the standard surface electrocardiogram, has not been critically tested.

It was the purpose of this study to determine whether the postoperative surface electrocardiogram can be used to predict the presence of occult but potentially serious conduction disturbances. This was accomplished by evaluating the pre- and postoperative electrocardiograms and postoperative His bundle electrograms from 50 patients following TF and VSD repair.

Material and Methods

The study group was composed of 50 patients undergoing postoperative cardiac catheterization from 10 days to 20 years following complete repair of TF (40) and VSD (10). Their ages ranged from 4.8 to 22.5 years, with a mean of 10.9 years (table 1). Preoperatively, all patients were in sinus rhythm and had no electrocardiographic evidence of arrhythmias or conduction disturbances. Many were selected for postoperative re-evaluation because of clinically apparent hemodynamic abnormalities or the presence of historical or electrocardiographic evidence of surgically-induced conduction disturbances, such as the occurrence of transient postoperative CHB. They did not, therefore, represent an unselected, postoperative group of patients. In all patients, operation consisted of a vertical right ventriculotomy and patch repair of the septal defect. Infundibular resection and outflow reconstruction were performed when required in patients with TF.

All pre- and postoperative electrocardiograms were reviewed with careful attention paid to detect transient conduction disturbances occurring during the early postoperative period. Operative and subsequent physicians’ notes were also carefully reviewed for the same purpose. Patients with bundle branch block, left axis deviation or first degree (1°) A-V block preoperatively were specifically excluded. Postoperative RBBB was diagnosed when the QRS duration increased by at least 40 msec compared to the preoperative tracing, and terminally prolonged R waves were present in the right precordial leads. Left anterior hemiblock was diagnosed when the mean frontal plane QRS axis was less than −30° and the initial 20 msec vector was directed inferiorly and to the right. The electrocardiogram used for analysis was obtained within 48 hours of catheterization.

All studies were performed in the postabsorptive state under sedation with meperidine, 1 mg per kg, chlorpromazine, 0.5 mg per kg, and promethazine, 0.5 mg per kg. Informed consent for electrophysiological investigation was obtained for each patient. Following the hemodynamic investigations, bipolar (1 cm interelectrode distance) His bundle electrograms were obtained, and along with three simultaneously obtained surface electrocardiographic leads, were recorded on photographic paper running at 100 mm/sec. Low and high level filters of 12 Hz and 2000 Hz were employed for the His bundle electrograms. P-A, A-H, and H-V intervals were measured as previously described.10-12

His bundle electrograms on an age-matched group of 37 hemodynamically and electrocardiographically normal children served as controls. Findings from the first 27 of these children, studied by one of us at another institution, have been previously reported.12 These children were studied to rule out significant congenital heart disease and were found
to have either normal hearts or minimal, hemodynamically insignificant lesions (e.g., pulmonary gradients < 15 mm Hg; straight back syndrome; vascular ring; etc.) Additionally, conducting intervals were obtained from 61 patients with unoperated TF (30) and VSD (31) and also served as controls. No patient included in this study was taking digitals or other anti-arrhythmic drugs.

Results

All patients except two were in sinus rhythm with 1:1 atrioventricular (A-V) conduction at the time of postoperative catheterization. Two patients had junctional rhythms. Immediate pre- and postoperative electrocardiographic comparison revealed significantly increased P-R intervals (mean increase = 18.6 msec; \( P < 0.05 \)) and QRS durations (mean increase = 45.7 msec; \( P < 0.001 \)) following surgery (table 1).

Table 2 lists the specific electrocardiographic conduction disturbances found at the time of study. Ten patients (20%) had first degree A-V block. Complete RBBB was present in 44 cases (88%) of which nine (18%) also had LAH. Another six of the patients (12%) with RBBB had transient LAH for varying periods postoperatively, but had only RBBB at the time of study. Transient postoperative CHB, lasting from a few minutes to two weeks, occurred in 11 patients (22%).

Mean values for the P-A, A-H, and H-V conducting times in the group of 37 normal children were 21.5 msec, 87.8 msec, and 31.6 msec, respectively (table 3). The conduction times for the 50 postoperative patients are shown in table 1, and are compared to the normal group and the group with unoperated TF and VSD in figure 1. The mean P-A and A-H conduction times for the postoperative patients were 16.6 msec and 84.9 msec, respectively, and were not statistically different from normal, although five postoperative patients had minimally increased A-H times, ranging from 130 to 150 msec. His-Purkinje conduction, as represented by the H-V interval, was significantly delayed in the postoperative group, with a mean value of 48.5 msec. Ten patients had H-V times of 55 msec or greater, indicating trifascicular damage. The mean P-A, A-H, and H-V intervals (± SD) for the group of 61 patients with unoperated TF and VSD were 19.1 ± 40 msec, 85.0 ± 20.4 msec, and 33.9 ± 6.6 msec, respectively, and were not statistically different from normal (fig. 1).

The conducting intervals and the electrocardiographic findings for each of the ten children with postoperative trifascicular block are listed in table 4. Four patients had findings suggesting the presence of trifascicular damage, namely RBBB together with LAH and first degree A-V block, three of whom had a history of transient CHB in the early postoperative period. One patient had RBBB, LAH, a normal P-R interval and transient postoperative CHB. Of the remaining patients, one had RBBB, 1° A-V block and transient postoperative CHB. Three patients had only RBBB, although one of these had transient postoperative LAH. Figure 2 shows the electrocardiogram and His bundle electrogram from one of these patients. The last patient demonstrated only a borderline P-R interval prolongation at the time of study, although her H-V interval measured 65 msec (fig. 3). This patient had complete RBBB and LAH postoperatively, but both disappeared on the third postoperative day.

Conducting intervals in the ten patients developing postoperative 1° A-V block showed that two had isolated A-H prolongation (135 and 150 msec), four had isolated H-V prolongation (160, 85, 65, and 60 msec) and two had prolongation of both intervals. In two patients, all conducting intervals were within the normal range.

Five of the 11 patients developing transient postoperative complete heart block had residual trifascicular damage (table 4).

Discussion

The occurrence of CHB following TF and VSD repair constitutes the most significant surgically-induced conduction system abnormality. Refinements in the surgical technique have lowered the incidence of permanent CHB occurring immediately following surgery to about 1%, 10, 11 but late onset CHB, which may occur many years following the surgical correction and present with syncopal episodes or sudden, unexpected death, remains a problem. 12, 13

The postoperative electrocardiographic pattern of complete RBBB with LAH develops in 7 to 25% of cases following TF and VSD repair, 2, 4, 10, 11 and has been associated

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**Table 1. Data on 50 Postoperative Patients Following Tetralogy of Fallot and Ventricular Septal Defect Repair**

<table>
<thead>
<tr>
<th>Age (yr)</th>
<th>Preop P-R (msec)</th>
<th>Preop QRS (msec)</th>
<th>Postop P-R (msec)</th>
<th>Postop QRS (msec)</th>
<th>Postop A-H (msec)</th>
<th>Postop H-V (msec)</th>
</tr>
</thead>
<tbody>
<tr>
<td>4.8-22.5</td>
<td>10-200 50-110</td>
<td>110-240 60-160</td>
<td>40-30* 50-150</td>
<td>30-160</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean</td>
<td>10.9 46.1</td>
<td>144.4 71.9</td>
<td>163.0 117.6</td>
<td>16.6 84.9</td>
<td>48.5</td>
<td></td>
</tr>
<tr>
<td>SD</td>
<td>21.7 14.4</td>
<td>36.2 23.7</td>
<td>13.7 26.1</td>
<td>20.1</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Two patients with junctional rhythm had P-A intervals less than 0 (—30 and —40 msec).

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**Table 2. Electrocardiographic Data in 60 Children Following Tetralogy of Fallot and Ventricular Septal Defect Repair**

<table>
<thead>
<tr>
<th>No.</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>1° A-V block</td>
<td>10 20</td>
</tr>
<tr>
<td>RBBB</td>
<td>44 88</td>
</tr>
<tr>
<td>with LAH</td>
<td>9 18</td>
</tr>
<tr>
<td>with transient LAH</td>
<td>6 12</td>
</tr>
<tr>
<td>Transient postop CHB</td>
<td>11 22</td>
</tr>
</tbody>
</table>

**Table 3. Conduction Times in 37 Normal Children***

<table>
<thead>
<tr>
<th>No.</th>
<th>P-R</th>
<th>P-A</th>
<th>A-H</th>
<th>H-V</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>139</td>
<td>21.5</td>
<td>87.8</td>
<td>31.6</td>
</tr>
<tr>
<td>SD</td>
<td>18.2</td>
<td>7.6</td>
<td>18.9</td>
<td>6.4</td>
</tr>
<tr>
<td>Mean + 2 SD</td>
<td>37</td>
<td>126</td>
<td>45</td>
<td></td>
</tr>
</tbody>
</table>

*Abbreviations: A-V = atrioventricular; RBBB = right bundle branch block; LAH = left anterior hemiblock; CHB = complete heart block.

**Table 3. Conduction Times in 37 Normal Children***

*Mean age: 8.3 years. SD = standard deviation.
with the occurrence of late onset CHB.\(^1,2\) In one series, five of 24 patients with postoperative RBBB and LAH developed late CHB.\(^3\) Four of those patients had a transient episode of CHB in the early postoperative period. In another report, RBBB, LAH, and early transient CHB were present in four of five patients developing late onset CHB.\(^4\) Other studies have found that surgical bifascicular block is not associated with a poor prognosis and reported no patients who died suddenly or developed late onset CHB.\(^5,6\) Few of the reported patients in these latter studies, however, suffered from transient postoperative CHB. The apparent difference in clinical outcome in patients with postoperative bifascicular block may be explained by differences in patient populations that grouping based on standard electrocardiograms does not make apparent. The ability to detect the presence of surgically-induced trifascicular damage may be of more prognostic importance than is the finding of a particular electrocardiographic pattern such as RBBB with LAH.

In our group of 50 postoperative patients, ten were found to have trifascricular damage, on the basis of prolonged His-Purkinje conduction times. This probably represents a higher incidence than would be expected in a nonselected group of postoperative patients. Five of the ten patients with trifascicular damage had the electrocardiographic pattern of RBBB with LAH. Four of these also had transient postoperative CHB. These results are similar to those reported by Godman et al.\(^1\) In another invasive study of 18 patients with postoperative bifascicular block by Pahlajani et al., four were found to have prolonged H-V conduction times,
although none progressed to CHB during the follow-up period.

Bifascicular block, together with the history of transient CHB in the early postoperative period, may suggest trifascicular damage. This does not mean, however, that the presence of RBBB with LAH will include all patients with trifascicular damage. Four of our patients with H-V prolongation had RBBB alone and one patient had a normal postoperative QRS duration. The latter patient, and one with RBBB and right axis deviation, did develop postoperative first degree A-V block. These latter five patients had postoperative electrocardiograms at the time of study which did not suggest the presence of surgically-induced trifascicular damage, yet each patient had an H-V conduction time of 55 msec or greater. Any patient may be subject to late onset CHB due to trifascicular damage, and only invasive studies can accurately identify those patients at highest risk. In the cooperative study by Moss et al., describing 20 documented cases of late onset CHB following ventricular defect repair,18 complete heart block occurred up to 14 years after surgical correction. The ECG immediately preceding the onset of CHB was available for study in 15 cases and only four children had the electrocardiographic combination of RBBB with LAH immediately prior to CHB. Eight had only RBBB and three had normal QRS durations. Eleven of the 20 children had transient CHB during the immediate postoperative period.

There has been some question as to whether trifascicular
damage is in fact a precursor of CHB or sudden death. Narula and co-workers,16 in a long-term series, reported an 80% mortality in patients with RBBB, LAH, and markedly prolonged H-V intervals. Scheinman et al.17 found that CHB occurred in 18% of a group of similar patients with trifascicular damage who were followed for a mean period of only 15 months. Since additional fibrosis of the conduction system occurs with age, CHB after trifascicular damage would not be a surprising development.

The prolonged His-Purkinje conduction times observed in our postoperative patients were probably caused by the surgical intervention. Conduction times obtained from 61 nonoperated patients with TF and VSD were not different from normal (fig. 1). No unoperated patient had an H-V time greater than 50 msec and only one had a value greater than 45 msec. Even if we exclude the ten patients with grossly abnormal H-V times (> 55 msec), the mean ± SD H-V for the remaining 40 postoperative patients was 41.4 ± 6.8 msec and was still significantly greater (P < 0.001) than the mean value for the normal group (31.6 ± 6.4 msec) and the group of patients with unoperated TF and VSD (33.9 ± 6.6 msec). Surgical correction of TF and VSD seems to cause damage mainly to the distal portion of the conducting system. The His bundle and bundle branches are particularly vulnerable because they are so close to the inferior and distal margins of the septal defect.18 The A-V node may not be so vulnerable at the time of surgical repair. Although five of our patients had prolonged A-V nodal conduction times postoperatively, A-H values for these patients were only minimally prolonged (range = 130–150 msec) and the mean value for the entire group was normal.

Surgically-induced trifascicular damage may be a precursor of late onset CHB following TF and VSD repair. Trifascicular disease can only be firmly diagnosed by demonstrating a prolonged H-V interval. This prolongation can occur in the absence of electrocardiographic bifascicular block and some of our patients with bifascicular block did not have trifascicular involvement. The occurrence of transient CHB postoperatively may suggest postoperative trifascicular damage and possible persistent disease to the trifascicular system.

The prognosis of patients with certain electrocardiographic patterns such as RBBB with LAH cannot be made in the absence of information regarding conduction along the remaining functioning bundle branch. Unfortunately, this information can be obtained only by invasive means. We agree with Steeg et al.,4 that patients presenting with postsurgical RBBB and LAH may fall into two different populations defined by either peripheral damage to the conduction system or damage to the His bundle or its proximal branches. The former has not been associated with transient postoperative CHB5,4 and these patients have been found to have a generally benign course. The latter population, patients with trifascicular damage, may have a serious prognosis.

In addition to His bundle electrocardiography, atrial pacing and measurement of refractory periods utilizing the atrial extrastimulus technique can provide additional information on the integrity of the conducting system.19 Prolonged A-V nodal and His-Purkinje refractory periods have been found in patients with postsurgical bifascicular block and normal His bundle electrograms.7
We recommend that His bundle electrograms be obtained for patients whose history portends of Stokes-Adams attacks and that intracardiac electrophysiological study should be seriously considered in any patient with transient postoperative CHB or electrocardiograms demonstrating bifascicular block or progressively lengthening P-R intervals. Our results and previous reports of late postoperative CHB in patients whose electrocardiograms did not show bifascicular block present some rationale for follow-up electrophysiological studies in all children following ventricular defect repair. At the present time, we cannot recommend the prophylactic use of permanent pacemakers in asymptomatic patients with evidence of trifascicular damage, but this group certainly deserves very careful follow-up and continued study.

References
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Temporary Inhibition of Permanently Implanted Demand Pacemakers

PEVRAIZE LATIF, M.D., AND GORDON A. Ewy, M.D.

SUMMARY Temporary inhibition of permanently implanted demand pacemakers has been previously described. Demand pacemakers may be inhibited by waving a magnet over the region of the pacemaker generator or by chest wall stimulation. The former may not inhibit most of the bipolar pacemakers, whereas the latter may be time consuming and may cause patient discomfort.

Another method is described which utilized a commercially available Cordis Omnicon Programmer, Model 166-B, to temporarily inhibit bipolar and unipolar pacemakers. By placing the programmer over the skin where the pacemaker generator is implanted and/or over the area of the subcutaneous perivascular lead and activating the programmer multiple times at a rate faster than the pacing rate, the demand pacemakers are inhibited. After testing the efficacy in vitro, the method was successfully tried on 45 patients. Fifteen of these patients had unipolar pacemakers. Pacemakers marketed by Medtronic, Cordis, Starr-Edward, C.P.I., and Arco were tested.

Temporary inhibition of permanent demand pacemakers is desirable under various clinical situations. The method herein described has the advantages of being simple, quick, painless, and is effective for both unipolar and bipolar pacemakers.

TEMPORARY INHIBITION OF PERMANENTLY IMPLANTED DEMAND PACEMAKERS, so that one can record the patient's intrinsic nonpaced electrocardiogram, is desirable under a variety of clinical situations. Three basic methods have been described.1-4 Chest wall stimulation with an external pacemaker or waving a magnet over the pacemaker generator are the two methods most commonly used. The third utilized electromagnetic impulses generated by the Medtronic Model 5855 external rate control transmitter.5, 6 Chest wall stimulation is time consuming since various areas of the chest may have to be paced to determine the precise position for inhibition.1, 2 In addition, chest wall stimulation causes some discomfort to the patient...
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