Late Postoperative Conduction Disturbances
After Repair of Ventricular Septal Defect
and Tetralogy of Fallot

Analysis by His Bundle Recordings

By M. J. GODMAN, M.B., M.R.C.P., N. K. ROBERTS, M.B.,
AND T. IZUKAWA, M.D., F.R.C.P. (C)

SUMMARY
His bundle electrograms were recorded in five children who developed complete heart block (CHB) five months to five years after cardiac surgery. In four children who had an electrocardiographic pattern of right bundle branch block and left axis deviation (RBBB and LAD) preceding the appearance of CHB, the site of block was distal to the area generating the His potential. The fifth child, who had an electrocardiographic pattern of RBBB with normal axis prior to the development of CHB, had block occurring proximal to the area generating His potential. All five children had had transient CHB in the immediate postoperative period.

In an additional 17 postoperative patients, His bundle recordings were obtained during sinus rhythm. Five out of ten patients with RBBB and LAD on the electrocardiogram and one patient with LBBB had H-V interval prolongation, a recording suggesting incomplete bilateral bundle branch block or trifascicular block. Six patients with RBBB only on the electrocardiogram had normal H-V intervals. All the patients with H-V interval prolongation had had CHB transiently in the immediate postoperative period.

This study suggests that patients with an electrocardiographic pattern of RBBB and LAD who have had transient CHB postoperatively may have, more extensive residual damage to the conduction system than suspected on the basis of the surface electrocardiogram alone and because of the risk of late recurrence of CHB should be given a guarded prognosis.

Additional Indexing Words:
Tetralogy of Fallot Intraventricular conduction Ventricular septal defect
Open heart surgery

A variety of conduction disorders may complicate repair of ventricular septal defects or tetralogy of Fallot. Permanent complete heart block (CHB) is the most serious of these disturbances and is associated with a poor prognosis.1 Transient complete heart block occurring in the immediate postoperative period, on the other hand, has been associated with a good prognosis.2 More recently the electrocardiographic pattern of right bundle branch block (RBBB) and left axis deviation (LAD) has been reported to complicate repair of ventricular septal defects in 7–11% of cases.3–5 In one series, it was associated with a high incidence of late postoperative CHB, particularly when there had been a history of CHB transiently in the immediate postoperative period.6

At the Hospital for Sick Children, Toronto, late CHB developed at intervals of five months to seven years postoperatively in 11 children who had repair of a ventricular septal defect or the tetralogy of Fallot. All 11 children had previously had CHB transiently in the immediate postoperative period. Seven of the 11 also had electrocardiographic features of RBBB and LAD preceding the development of late heart block. This suggests that the complete heart block results from bilateral bundle branch block or trifascicular block.7

Complete heart block postoperatively can result from a lesion at one of several levels and the site of the pacemaker controlling the ventricles influences...
prognosis. Therefore all patients with the pattern of RBBB and LAD postoperatively are not necessarily at the same risks of late CHB. It has been our experience and that of others that the surface electrocardiogram alone is of limited value in identifying the level of the block.

The present study was therefore undertaken to assess the value of His bundle electrograms in patients with late postoperative conduction disturbances. In particular, we have attempted to determine the usefulness of these recordings in defining the site of conduction disturbances, and in identifying patients potentially at risk of developing late postoperative CHB.

**Methods**

**Patients**

His bundle electrograms were recorded in two groups of patients.

**Group I**

This group consisted of five patients with late postoperative CHB. The clinical and electrocardiographic data of these patients are summarized in table 1. All five patients had had transventricular CHB documented electrocardiographically in the immediate postoperative period. They subsequently returned to sinus rhythm but CHB recurred at intervals from five months to 5.5 years postoperatively. Four of the five had Stokes-Adams episodes requiring insertion of ventricular demand pacemakers which were operating in the continuous mode at the time of study. These four patients had an electrocardiographic pattern of RBBB and LAD preceding the development of late postoperative CHB. Since the patients in this study belonged in the pediatric age group, left axis deviation was considered to be present if the frontal plane axis was less than 0 degrees. The fifth patient had an electrocardiographic pattern of RBBB with a normal axis. (A normal axis was arbitrarily defined as being between 0 degrees and 120 degrees. We are not aware of any comprehensive data on the "normal axis" in RBBB in the pediatric age group.) Complete heart block returned five months later, alternating with periods of second degree heart block of the Wenckebach type; at the time of study second degree block was present. Only the fifth patient had P-R interval prolongation when in sinus rhythm. All five patients had an electrocardiographic pattern with a broad QRS morphology when in CHB.

**Group II**

The second group consisted of an additional 17 postoperative patients, all of whom were in sinus rhythm at the time of study. The clinical, electrocardiographic and electrophysiological data of these patients are summarized in table 2. None of the patients were receiving any medication at the time of study. Ten of the 17 had an electrocardiographic pattern of RBBB and LAD; six, RBBB with normal axis; and one, LBBB with normal axis. Five of the ten patients with RBBB and LAD; three of the six patients with RBBB alone, and one patient with LBBB had had CHB transiently in the immediate postoperative period. None, however, had had any recurrence of CHB during the interval between the immediate postoperative period and the time of study, which ranged from six weeks to 11 years.

**Electrophysiological Studies**

His bundle electrograms were recorded with either a unipolar or bipolar electrode catheter (USCI No. 5 or No. 6). Recordings were made on photographic paper using a rapid writer from a DR8 Electronics for Medicine Recorder at a paper speed of 100 mm/sec. A low level filter of 12 Hz and a high level of 2000 Hz was employed. A simultaneous standard lead II electrocardiogram was recorded.

Atrial pacing was performed in 13 of the 17 patients from Group II. The stimulus frequency was increased until second degree atrioventricular block developed or

**Table 1**

<table>
<thead>
<tr>
<th>Case no.</th>
<th>Diagnosis</th>
<th>Age at surgery</th>
<th>Interval between surgery and late CHB</th>
<th>ECG pattern preceding CHB</th>
<th>Site of A-V block</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Ventricular septal defect</td>
<td>10 months</td>
<td>5.5 years</td>
<td>RBBB + LAD</td>
<td>Distal to site of His potential</td>
</tr>
<tr>
<td>2</td>
<td>Ventricular septal defect</td>
<td>18 months</td>
<td>22 months</td>
<td>RBBB + LAD</td>
<td>Distal to site of His potential</td>
</tr>
<tr>
<td>3</td>
<td>Ventricular septal defect</td>
<td>2 years</td>
<td>3 years</td>
<td>RBBB + LAD</td>
<td>Distal to site of His potential</td>
</tr>
<tr>
<td>4</td>
<td>Tetralogy of Fallot</td>
<td>10 years</td>
<td>7 months</td>
<td>RBBB + LAD</td>
<td>Distal to site of His potential</td>
</tr>
<tr>
<td>5</td>
<td>Tetralogy of Fallot</td>
<td>8 years</td>
<td>5 months</td>
<td>RBBB</td>
<td>Proximal to site of His potential</td>
</tr>
</tbody>
</table>

Abbreviations: RBBB = right bundle branch block; LAD = left axis deviation.

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### Table 2

Clinical Electrocardiographic and Electrophysiological Findings in Postoperative Patients with Bundle Branch Block

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age (yrs)</th>
<th>Sex</th>
<th>Cardiac lesion</th>
<th>Interval between surgery &amp; study</th>
<th>CHB postoperatively</th>
<th>Rhythm</th>
<th>QR8 (msec)</th>
<th>P-P (msec)</th>
<th>P-R (msec)</th>
<th>A-H (msec)</th>
<th>H-V (msec)</th>
<th>Atrial pacing (beats/min)</th>
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<tbody>
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<td>1</td>
<td>13½</td>
<td>F</td>
<td>VSD</td>
<td>11 yr</td>
<td>Transient</td>
<td>Sinus</td>
<td>145</td>
<td>900</td>
<td>150</td>
<td>85</td>
<td>40</td>
<td>200</td>
</tr>
<tr>
<td>2</td>
<td>8</td>
<td>M</td>
<td>Tet of Fallot</td>
<td>3 mo</td>
<td>Transient</td>
<td>Sinus</td>
<td>130</td>
<td>480</td>
<td>140</td>
<td>85</td>
<td>35</td>
<td>160*</td>
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<tr>
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<td>10</td>
<td>M</td>
<td>Tet of Fallot</td>
<td>2 yr</td>
<td>Transient</td>
<td>Sinus</td>
<td>130</td>
<td>650</td>
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<td>80</td>
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<tr>
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<td>9</td>
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<td>VSD</td>
<td>8 yr</td>
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<td>Sinus</td>
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<td>490</td>
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<td>180*</td>
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<td>—</td>
<td>Sinus</td>
<td>135</td>
<td>640</td>
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<td>85</td>
<td>40</td>
<td>—</td>
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<tr>
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<td>10</td>
<td>M</td>
<td>Tet of Fallot</td>
<td>2 mo</td>
<td>Transient</td>
<td>Sinus</td>
<td>140</td>
<td>580</td>
<td>130</td>
<td>75</td>
<td>40</td>
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**RBBB + Normal Axis**

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age (yrs)</th>
<th>Sex</th>
<th>Cardiac lesion</th>
<th>Interval between surgery &amp; study</th>
<th>CHB postoperatively</th>
<th>Rhythm</th>
<th>QR8 (msec)</th>
<th>P-P (msec)</th>
<th>P-R (msec)</th>
<th>A-H (msec)</th>
<th>H-V (msec)</th>
<th>Atrial pacing (beats/min)</th>
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<td>VSD</td>
<td>5 yr</td>
<td>Transient</td>
<td>Sinus</td>
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<td>450</td>
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<td>70</td>
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**RBBB + LAD**

<table>
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<th>Case No.</th>
<th>Age (yrs)</th>
<th>Sex</th>
<th>Cardiac lesion</th>
<th>Interval between surgery &amp; study</th>
<th>CHB postoperatively</th>
<th>Rhythm</th>
<th>QR8 (msec)</th>
<th>P-P (msec)</th>
<th>P-R (msec)</th>
<th>A-H (msec)</th>
<th>H-V (msec)</th>
<th>Atrial pacing (beats/min)</th>
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<td>8</td>
<td>12½</td>
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<td>VSD</td>
<td>8 yr</td>
<td>—</td>
<td>Sinus</td>
<td>140</td>
<td>830</td>
<td>140</td>
<td>80</td>
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<tr>
<td>9</td>
<td>13½</td>
<td>M</td>
<td>VSD + PS</td>
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<td>Sinus</td>
<td>155</td>
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<td>M</td>
<td>VSD</td>
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<td>—</td>
<td>Sinus</td>
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<td>Sinus</td>
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<td>660</td>
<td>125</td>
<td>60</td>
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<td>F</td>
<td>VSD</td>
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<td>Sinus</td>
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<td>Transient</td>
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<tr>
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<td>125</td>
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<tr>
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<td>F</td>
<td>Tet of Fallot</td>
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<td>Transient</td>
<td>Sinus</td>
<td>130</td>
<td>580</td>
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<td>F</td>
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<td>Transient</td>
<td>Sinus</td>
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<td>610</td>
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<td>Sinus</td>
<td>135</td>
<td>760</td>
<td>155</td>
<td>65</td>
<td>70</td>
<td>200</td>
</tr>
</tbody>
</table>

*Rate at which second degree A-V block of Wenckebach type developed. In all 4 cases this response was interpreted as being within the normal range.
an atrial rate of 200/min with 1:1 A-V conduction was produced.

The following intervals, in milliseconds, were measured from the His bundle electrogram. All intervals were measured in five separate complexes and the mean of these five measurements calculated.

1) The A-H interval was measured from the onset of atrial activation A to the first rapid deflection of the His bundle potential.

2) The H-V interval was measured from the first rapid deflection of the His bundle potential to the earliest inscription of ventricular activation, whether this was the V wave in the His bundle electrogram or the onset of the QRS complex in the simultaneously recorded ECG.

Right bundle potentials were not recorded in any patient. The A-H interval was taken to represent the conduction time through the A-V node to activation at the His bundle recording site and the H-V interval to represent the conduction time from activation at the His bundle recording site to the onset of ventricular activation. A prolonged H-V interval in the presence of true bundle branch block suggests associated impairment of conduction in the bundle of His or the contralateral bundle branch system.9-10

On the basis of previous studies in our laboratory in children, the range of the normal A-H interval is 54-118 msec and of the normal H-V interval, 20-42 msec.11 The H-V interval increases with age and the H-V intervals in this study have therefore been compared with the expected H-V interval for a given age.11, 12

**Results**

**Group I**

In each of the four children who had an electrocardiographic pattern of RBBB and LAD preceding late CHB, the His bundle electrogram suggested that the site of block was distal to the site from which the His bundle electrogram was recorded (table 1). The recordings in these cases showed that each A wave was accompanied by a His potential (fig. 1). Late postoperative recurrence of CHB in these four children, therefore, appears to have been the result of bilateral bundle branch block or trifascicular block, although the possibility of block in the His bundle itself, distal to the recording site, cannot be excluded.

In the fifth patient in this group, who was in second degree heart block of Wenckebach type at the time of study, the His bundle electrogram suggested that the site of block was proximal to the site from which His bundle activity was being recorded (fig. 2).

**Group II**

The electrophysiological findings in the 17 postoperative patients, who were in sinus rhythm at the time of study, are summarized in table 2, together with the relevant clinical and electrocardiographic data. The H-V interval in these cases was related to the H-V interval expected for the patient's age. Figure 3 shows the regression line for the H-V interval with age, the outer limits representing two standard errors of the estimate. This graph is based on His bundle recordings in 100 children investigated in our laboratory for congenital heart disease or suspected congenital heart.11

All six patients who had an electrocardiographic pattern of RBBB with a normal axis had H-V intervals within the expected range, although three of the six had had CHB transiently in the immediate postoperative period. Five of the ten patients with an electrocardiographic pattern of RBBB and LAD had H-V intervals that were prolonged for the patient's age groups. All of these five had had transient CHB postoperatively. The remaining five patients with the electrocardiographic pattern of RBBB and LAD had H-V intervals that were within normal limits; only one of them had had transient CHB postoperatively.

One patient with LBBB, who also had had CHB transiently in the immediate postoperative period, had a long H-V interval for age.

**Discussion**

Full assessment of the prognosis and management of CHB depends on the precise identification of the site of the conduction delay. The location of block within the conduction system mainly determines the significance of the block; a low idioventricular pacemaker is more likely to be associated with major symptoms or Stokes-Adams episodes than a pacemaker situated above the bifurcation of the bundle of His.10 This is supported by the His bundle recordings in the five children with CHB in this report. Four of the five were symptomatic and required cardiac pacing; all had block distal to the site from which the His potential was recorded. The fifth patient, who like the other four had a broad QRS complex when in CHB, was shown to have block occurring proximal to the site of origin of the His potential. This patient was asymptomatic, and although he developed CHB five months postoperatively, it was again only transient and disappeared eight days after detection. Following return to sinus rhythm he has remained well during a follow-up period of one year after the His bundle recordings.

As indicated by these findings, the width of the QRS complex in postoperative CHB may not
always be a reliable indicator of the location of the ventricular pacemaker. A wide QRS complex usually indicates a low idioventricular pacemaker. An electrocardiographic pattern of RBBB, however, is common after ventriculotomy or repair of a ventricular septal defect, and this decreases the significance that can be attached to a broad QRS complex in surgically induced complete heart block.14, 15 Anderson et al., in a recent report of two cases of early postoperative CHB, have also

Figure 1

Complete A-V block distal to the His potential. The top tracing is a standard lead II and the bottom is the His bundle electrogram. The atrial electrogram is labeled A and the His potential H. Paper speed 100 mm/sec. The patient had a permanent demand ventricular pacemaker operating here in the continuous mode at a rate of 75 beat/min. Each atrial depolarization complex is followed by a His potential. In the first complex the A potential is buried in the QRS complex but a His potential is identified immediately following the QRS complex. These data indicate that the A-V block is distal to the recording site and lies between the bundle of His and the Purkinje myocardial junction.

Figure 2

In this recording each QRS complex is preceded by a His potential. The H-V interval does not vary and is constant at 40 msec. There is gradual prolongation of the A-H interval, until finally atrial conduction is blocked, and no His potential is recorded, suggesting that the site of block and impaired conduction is proximal at the A-V node.
**CONDUCTION AFTER SURGERY FOR VSD & TF**

![Graph](image)

**Figure 3**

The H-V interval in the postoperative cases is related to the H-V interval expected for the age. This figure shows the regression line (middle line) for the H-V interval with age, with the outer limits (upper and lower lines) representing two standard errors of the estimate. This regression line was derived from measurements of the H-V interval in 100 children studied in our laboratory. Symbols: Open circle = Right bundle branch block; open square = Left bundle branch block; open triangle = Right bundle branch block and left axis deviation; opaque symbols indicate those patients with bundle branch block who had had transient CHB in the immediate postoperative period.

Dreifus et al. believes that the criteria of Mobitz based on the behavior of the P-R interval are not as reliable in identifying the site of A-V block as is a classification based on the QRS duration. Our experience and Anderson's would suggest that neither the P-R interval nor the breadth of the QRS complex is reliable in identifying the site of A-V heart block in postoperative conduction disorders.

While our findings would suggest that late postoperative CHB is usually the result of bilateral bundle branch block or trifascicular block, this is clearly not invariably the case, as was demonstrated by the fifth patient we studied. Accurate identification of the level of conduction failure can only be made by utilization of His bundle recordings.

Although permanent CHB is the most serious of the conduction disturbances following repair of ventricular septal defects, it is now less common as an immediate postoperative complication. On the other hand, the majority of patients have an ECG pattern of RBBB following operation. A smaller number of patients develop the pattern of RBBB combined with LAD postoperatively.

Two possible mechanisms of production of postoperative RBBB have been proposed. Gelband et al. have convincingly demonstrated that a vertical ventriculotomy alone may be responsible for appearance of a right bundle branch block pattern. They also showed in their cases that during the remainder of the surgical procedure no further injury occurred to the right ventricular conduction system. However, pathological studies have demonstrated traumatic lesions can occur in the bundle of His and both right and left bundle branches. Direct trauma to the right bundle proximally and more peripheral damage to the right conduction system both may be present in some patients, although this was not the case in series studied by Gelband et al.

Gelband et al. proposed that if the ECG pattern of RBBB seen postoperatively is due to the ventriculotomy there would be no risk of complete heart block in patients who later developed left bundle branch block since the main right bundle is intact. Downing et al. also claimed that RBBB and LAD did not carry a bad prognosis in childhood although they believed the ultimate prognosis was undermined. In our experience children with this

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pattern have a high risk of development of late postoperative CHB. Our finding is supported by Wolff et al. who found that 20% of children with RBBB and LAD developed late postoperative CHB. However, one patient in our series with this pattern postoperatively followed up to 11 years has remained free of any complication.

It is important to attempt to identify the individual patient with RBBB and LAD who is particularly at risk of developing late postoperative CHB. The ability to recognize disorders in conduction involving both bundle branches at an early stage may help to identify patients at high risk. The surface electrocardiogram does not identify damage to the conduction system as accurately and completely as the His bundle electrogram. A number of studies in nonsurgical patients have demonstrated the usefulness of His bundle recordings in detecting bilateral bundle branch block or trifascicular block, by allowing for a more precise measurement of the conduction time and delay in the bundle of His and the bundle branches. In particular, His bundle recordings may detect conduction defects in the contralateral bundle branch system which are not defined in the surface ECG.

In this report all the patients with RBBB alone had H-V intervals which were in the same range as those of children of the same age with normal QRS complexes. This finding suggests that in these cases there is no associated damage in the bundle of His or the left bundle branch system, even though three of the six cases with RBBB had CHB transiently postoperatively. Indeed in some, as suggested by Gelband et al., the right main bundle may also have been intact and the RBBB pattern purely a result of the ventriculotomy.

In contrast, five out of the ten patients with RBBB and LAD and the one patient with LBBB had prolonged H-V intervals for their age. This suggests that there were more extensive lesions than suspected from the surface ECG, producing bilateral bundle branch block, trifascicular block, or impaired conduction in the distal portion of the His bundle. The six patients with prolonged H-V intervals had all had CHB transiently in the postoperative period. Transient CHB postoperatively, in association with RBBB and LAD on return to sinus rhythm, therefore, seems to indentify patients most likely to have extensive quantitative damage to the conduction system as defined by His bundle recordings.

We suggest that the H-V interval prolongation in some patients with RBBB and LAD is more probably due to impaired conduction in the left posterior division of the left bundle and that this is the result of residual damage in patients who have had CHB transiently in the immediate postoperative period. If this is so then this information may help in identifying a subgroup of postoperative patients with RBBB and LAD who are at a particularly high risk of developing late CHB. At present the division of the P-R interval into its component parts has not altered the management of the patient in this report since we believe that until further information is available all patients with RBBB and LAD should be carefully observed. If the occurrence of CHB or Stokes-Adams attacks could be accurately predicted, a prophylactic pacemaker could be employed. However, the exact order of risk of development of CHB is not demonstrated in this study and is not known. It may well be that the risk when plotted against time for patients with RBBB and LAD and a prolonged H-V interval will be very high.

Ideally, damage to the conduction system should be avoided at the time of surgery. This requires clear delineation of the anatomic course of the His bundle and the bundle branches. Kaiser et al. have described such an approach using an electrode probe to localize the specialized A-V conduction pathways. Wider application of this type of technique may abolish the serious risks incurred by patients in whom patterns of RBBB and LAD developed at the time of surgery.

References


Circulation, Volume XLIX, February 1974
7. IZUKAWA T, CLARKE M, TRUSLER GA: Late complete trifascicular heart block resulting from cardiac surgery. Circulation 44 (suppl II): II-181, 1971


16. SPEAR JF, MOORE EN: Electrophysiologic studies on Mobitz Type II second degree heart block. Circulation 44: 1087, 1971


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