Postoperative Right Bundle Branch Block: Identification of Three Levels of Block

LEONARD N. HOROWITZ, M.D., JAMES A. ALEXANDER, M.D., AND L. HENRY EDMUNDS, JR., M.D.

SUMMARY It has been postulated that postoperative right bundle branch block (RBBB) may be produced by conduction block at any of several sites. In this study the site of block and resultant pattern of ventricular activation were documented in 20 patients in whom RBBB developed during repair of congenital cardiac defects. Intraoperative epicardial and endocardial mapping and recording from the right ventricular specialized conduction system were performed before and after repair in each patient. In eight patients right bundle branch (RBB) conduction was interrupted proximally in the area of the ventricular septal defect. Right ventricular (RV) activation in these patients was delayed at all sites. In five patients RBB conduction was interrupted distally in the area of the moderator band. RV activation in these patients was delayed at most sites; however, the apical septal sites were activated normally. In seven patients, RBB conduction was interrupted terminally in the area of the terminal fascicular network. In these patients RV activation was delayed only in basilar areas. We conclude that at least three distinct types of postoperative RBBB exist and can be identified by differences in RV activation.

THE ELECTROCARDIOGRAPHIC PATTERN of right bundle branch block (RBBB) appears frequently after open heart surgery for correction of congenital malformations. This RBBB pattern often results from disruption of the right ventricular specialized conduction system during right ventriculotomy or infundibular resection, but may also occur after transatrial repair of ventricular septal defects in which neither right ventriculotomy nor infundibular resection is performed. It has therefore been postulated that the postoperative RBBB pattern may be produced by conduction block at any of several sites along the course of the bundle of His or right bundle branch (RBB). The present study was performed to identify the sites of block and consequent patterns of ventricular activation during RBBB produced during repair of congenital cardiac defects.

Materials and Methods

Twenty patients who underwent total repair of either tetralogy of Fallot (12 patients), ventricular septal defect (three patients), complete atrioventricular (AV) canal (three patients) or double outlet right ventricle (two patients) were studied during cardiac surgery after informed consent was obtained (table 1). The patients were 1–22 years old, and there were six females and 14 males. Fourteen-lead ECGs and vectorcardiograms were obtained before surgery and during the first postoperative month. RBBB was defined by prolongation of the QRS complex (usually greater than 120 msec), an S wave in leads I and V6 and a monophasic, double-peaked R wave in V1 and V2. Vectorcardiographic criteria included prolongation of the QRS loop and slowly inscribed, anterior and rightward terminal forces. No patient had RBBB preoperatively, but eight patients had postoperative RBBB with a QRS duration of less than 120 msec; however, these children were younger than 8 years of age and the typical electrocardiographic and vectorcardiographic patterns of terminal rightward delay were observed.

The sequence of epicardial activation was determined by recording bipolar electrograms at 36–78 epicardial sites on both ventricles. A bipolar plunge electrode was inserted as a reference electrode into the left ventricular myocardium. The epicardium was then mapped with a hand-held probe (bipolar platinum electrode with 1-mm interelectrode distance) at preselected sites identified on Polaroid photographs of the heart taken before the mapping procedure. Surface electrocardiographic leads I, II and III or aV1 and V6 were recorded; the band pass was 0.1–100 Hz. The electrograms were filtered at 40–500 Hz. Epicardial activation mapping was performed after cannulation but before the initiation of cardiopulmonary bypass and after termination of cardiopulmonary bypass. During cardiopulmonary bypass, bipolar electrograms were recorded from 10–15 sites on the right atrial and ventricular endocardial surfaces. Particular attention was directed toward recordings on the septum along the course of the RBBB from the membranous septum to the Purkinje fibers of the right ventricular anterior wall. Endocardial electrograms were also recorded at the apex and outflow tract. Activation mapping was performed during sinus or
performed in three patients through a vertical right ventriculotomy. Hypertrophied muscle bands were excised to leave a wall thickness of 6–10 mm. Ventricular septal defects in all patients with tetralogy of Fallot were closed using a Dacron patch and running sutures reinforced with three to five interrupted figure-of-eight sutures in all patients. The ventricle was closed using a patch of pericardium.

Transatrial repair of a membranous ventricular septal defect was performed in three patients. The ventricular septal defect was patched with either woven Dacron or Teflon felt and secured to the right ventricular side of the septum with a continuous suture and three to five interrupted figure-of-eight sutures.

Complete AV canal was repaired transatrially in three patients. The right atrium was opened through an oblique incision anterior to the crista terminalis, and complete repair of either type A or type C AV canal defect was performed using a Dacron patch secured to the right ventricular side of the ventricular septal crest with mattress sutures buttressed with pledgets. Very superficial bites were taken over the bundle of His and AV node.

Transventricular repair of double outlet right ventricle was performed in two patients. The right ventricle was opened with a vertical ventriculotomy from the pulmonary annulus to the midpoint of the right ventricle. The hypertrophied muscle mass in the right ventricular wall was excised. The ventricular septal defect was closed with an enlarged Dacron patch such that left ventricular outflow was directed through the aortic orifice. The patch was secured with several running sutures interrupted three to five times.

Results

The typical electrocardiographic and vectorcardiographic patterns of RBBB developed in all 20 patients. In patient 4 left-axis deviation compatible with left anterior hemiblock also appeared postoperatively.

In the epicardial map recorded before repair, right ventricular activation began along the anterior interventricular groove and spread concentrically toward the base in each patient. Right ventricular activation began $18 \pm 7$ msec after the onset of the QRS. Right ventricular epicardial activation was completed in $44 \pm 13$ msec (mean $\pm$ SD). In 12 patients, the earliest epicardial activation of the left ventricle occurred within 10 msec of right ventricular epicardial breakthrough. In the three patients with complete AV canal, activation of the anterior wall of the left ventricle followed activation of the posterior wall in association with left-axis deviation, as previously reported.

Before correction, electrograms could be reproducibly recorded in each patient from the His bundle, proximal RBB (along the margin of the ventricular septal defect), distal RBB (moderator band) and terminal RBB (free wall endocardial Purkinje network).

Postoperatively, three sites of block in the right ventricular specialized conduction system and three cor-

<table>
<thead>
<tr>
<th>Pt</th>
<th>Age (years)</th>
<th>Sex</th>
<th>Diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>10</td>
<td>M</td>
<td>TOF</td>
</tr>
<tr>
<td>2</td>
<td>8</td>
<td>F</td>
<td>TOF</td>
</tr>
<tr>
<td>3</td>
<td>4</td>
<td>F</td>
<td>TOF</td>
</tr>
<tr>
<td>4</td>
<td>9</td>
<td>F</td>
<td>TOF</td>
</tr>
<tr>
<td>5</td>
<td>21</td>
<td>M</td>
<td>TOF</td>
</tr>
<tr>
<td>6</td>
<td>12</td>
<td>F</td>
<td>TOF</td>
</tr>
<tr>
<td>7</td>
<td>10</td>
<td>M</td>
<td>TOF</td>
</tr>
<tr>
<td>8</td>
<td>2</td>
<td>M</td>
<td>VSD</td>
</tr>
<tr>
<td>9</td>
<td>7</td>
<td>F</td>
<td>TOF</td>
</tr>
<tr>
<td>10</td>
<td>11</td>
<td>M</td>
<td>DORV</td>
</tr>
<tr>
<td>11</td>
<td>2</td>
<td>M</td>
<td>VSD</td>
</tr>
<tr>
<td>12</td>
<td>7</td>
<td>F</td>
<td>VSD</td>
</tr>
<tr>
<td>13</td>
<td>8</td>
<td>M</td>
<td>TOF</td>
</tr>
<tr>
<td>14</td>
<td>8</td>
<td>M</td>
<td>TOF</td>
</tr>
<tr>
<td>15</td>
<td>12</td>
<td>M</td>
<td>DORV</td>
</tr>
<tr>
<td>16</td>
<td>1</td>
<td>M</td>
<td>AVC</td>
</tr>
<tr>
<td>17</td>
<td>1</td>
<td>M</td>
<td>AVC</td>
</tr>
<tr>
<td>18</td>
<td>1</td>
<td>M</td>
<td>AVC</td>
</tr>
<tr>
<td>19</td>
<td>22</td>
<td>M</td>
<td>TOF</td>
</tr>
<tr>
<td>20</td>
<td>8</td>
<td>M</td>
<td>TOF</td>
</tr>
</tbody>
</table>

Abbreviations: TOF = tetralogy of Fallot; VSD = ventricular septal defect; DORV = double outlet right ventricle; AVC = atrioventricular canal defect.
Table 2. Operative and Postoperative Data

<table>
<thead>
<tr>
<th>Pt</th>
<th>Operative approach</th>
<th>Site of block</th>
<th>Postoperative ECG</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>Operative approach</td>
</tr>
<tr>
<td>1</td>
<td>TA</td>
<td>T</td>
<td>+135</td>
</tr>
<tr>
<td>2</td>
<td>TA</td>
<td>T</td>
<td>+135</td>
</tr>
<tr>
<td>3</td>
<td>TA</td>
<td>T</td>
<td>+75</td>
</tr>
<tr>
<td>4</td>
<td>TA</td>
<td>P</td>
<td>-60</td>
</tr>
<tr>
<td>5</td>
<td>TV</td>
<td>T</td>
<td>+135</td>
</tr>
<tr>
<td>6</td>
<td>TA</td>
<td>T</td>
<td>+120</td>
</tr>
<tr>
<td>7</td>
<td>TA</td>
<td>D</td>
<td>+110</td>
</tr>
<tr>
<td>8</td>
<td>TA</td>
<td>P</td>
<td>+45</td>
</tr>
<tr>
<td>9</td>
<td>TV</td>
<td>T</td>
<td>+120</td>
</tr>
<tr>
<td>10</td>
<td>TV</td>
<td>D</td>
<td>+135</td>
</tr>
<tr>
<td>11</td>
<td>TA</td>
<td>P</td>
<td>+135</td>
</tr>
<tr>
<td>12</td>
<td>TA</td>
<td>P</td>
<td>+135</td>
</tr>
<tr>
<td>13</td>
<td>TV</td>
<td>D</td>
<td>+80</td>
</tr>
<tr>
<td>14</td>
<td>TA</td>
<td>D</td>
<td>+120</td>
</tr>
<tr>
<td>15</td>
<td>TV</td>
<td>D</td>
<td>+90</td>
</tr>
<tr>
<td>16</td>
<td>TA</td>
<td>P</td>
<td>-60</td>
</tr>
<tr>
<td>17</td>
<td>TA</td>
<td>P</td>
<td>-45</td>
</tr>
<tr>
<td>18</td>
<td>TA</td>
<td>P</td>
<td>-45</td>
</tr>
<tr>
<td>19</td>
<td>TA</td>
<td>P</td>
<td>+135</td>
</tr>
<tr>
<td>20</td>
<td>TA</td>
<td>T</td>
<td>+135</td>
</tr>
</tbody>
</table>

All patients had right bundle branch block after surgery.

*Increase from preoperative value.

Abbreviations: D = distal (moderator band); TA = transatrial; P = proximal; TV = transventricular; T = terminal.

responding patterns of right ventricular activation delay were identified.

Proximal RBBB

In eight patients, three with ventricular septal defect, three with AV canal and two with tetralogy of Fallot repaired transatrially, conduction was interrupted at the proximal RBB (fig. 1). Before repair in each of these patients, electrograms of the specialized conduction system were consistently recorded along the inferior margin of the ventricular septal defect and the moderator band; after repair, however, despite extensive mapping, none could be recorded at these or other distal sites. In these patients, the HV intervals recorded after repair were not significantly different from the values before repair. Activation maps recorded after the appearance of RBBB in these eight patients revealed delay in activation at all right ventricular sites and alteration of the activation sequence (figs. 2 and 3). Epicardial activation of the anterior wall, apex and outflow tract of the right ventricle was delayed more than 35 msec in each patient (fig. 2). Before RBBB developed, the earliest epicardial breakthrough occurred on the right ventricular anterior surface in each patient; after RBBB developed, the earliest epicardial breakthrough was located on the left ventricle, and the earliest right ventricular activation appeared along either the anterior or posterior interventricular groove 38–92 msec after the onset of the QRS complex. Right ventricular epicardial activation spread radially from the interventricular groove toward the outflow tract and the AV groove (fig. 3).

Endocardial activation of the right ventricle was also altered after creation of RBBB. Endocardial electrograms recorded on the midseptum (fig. 2) and at the apex that occurred within 30 msec of the onset of the QRS complex during recordings before repair were delayed and occurred in the middle third of the QRS complex 30–58 msec after the onset of the QRS complex.

In patient 4, who had proximal RBBB after transatrial repair of tetralogy of Fallot, left-axis deviation as well as RBBB developed. Delayed activation of the anterior left ventricular wall appeared coincident with
FIGURE 2. Selected epicardial electrograms in proximal right bundle branch block in patient 19. ECG leads 1, 2 and 3 are shown with a reference electrogram recorded in the left ventricle (LV) and mapping electrograms recorded at the right ventricular apex (RVA), anterior wall (RVAW) and outflow tract (RVOT) before and after transatrial repair of tetralogy of Fallot. The vertical lines indicate the onset of the QRS and the numbers indicate time (msec) from the vertical line to the local electrogram. Before repair, the RVAW was the earliest site in the right ventricle. After repair, the earliest right ventricular site was the apex which was activated 25 msec later than before repair. The latest ventricular epicardial activation occurred on the RVOT at 144 msec (compare with fig. 3).

FIGURE 3. Epicardial activation in proximal right bundle branch block in patient 11. ECG leads 1, 2 and 3 are shown with a schematic representation of the epicardial surface before and after transatrial repair of ventricular septal defect. Anterior and posterior projections of the heart are shown. (The lateral projection is omitted because no changes occurred in that segment of the left ventricle.) Activation times at selected epicardial sites are shown with 20-msec isochrones. Before repair, the QRS duration was 61 msec and the right ventricular activation pattern was normal. After repair, the QRS duration was 92 msec and activation at all right ventricular sites was delayed. Right ventricular activation began along the anterior interventricular groove and spread radially to the base.

FIGURE 4. Epicardial activation in proximal right bundle branch block with left anterior hemiblock in patient 4. ECG leads 1, 2 and 3 are shown with a schematic representation of the epicardial surface in the anterior, left lateral and posterior projections (from left to right). Activation times at selected epicardial sites are shown with 20-msec isochrones. Before repair of tetralogy of Fallot, areas of epicardial breakthrough were present on the anterior right and left ventricles and on the posterior left ventricle. After repair, right bundle branch block and left-axis deviation were present. Epicardial activation of the right ventricle was delayed at each site (see fig. 3). Activation of the left ventricular anterior wall was also delayed and the anterior left ventricular breakthrough site was no longer present. Posterior left ventricular activation was not significantly altered.

the right ventricular activation delay (fig. 4). There was no significant change in the HV interval measured intraoperatively after repair in this patient.

Distal RBBB

In five patients, RBB conduction was blocked distally at the level of the moderator band. In each of these patients, the moderator band was deliberately divided to relieve muscular obstruction to the right ventricular outflow. These five patients had either tetralogy of Fallot or double outlet right ventricle. After repair, electrograms of the specialized conduc-
tion system could be recorded to the level of the proximal RBB in each. RBB electrograms from the distal moderator band itself or terminal Purkinje network were not present after the appearance of RBB (fig. 5). In two patients, conduction system electrograms could be obtained at the proximal portion of the moderator band after repair (fig. 5); in the other three patients, right bundle electrograms could be recorded only at the distal margin of the ventricular septal defect.

**FIGURE 5.** Specialized conduction system map in distal right bundle branch block in patient 14. ECG lead 3 is shown with electrograms recorded from the distal His bundle (H), proximal right bundle branch (PRB), two sites in the distal right bundle branch at the septal base of the moderator band (DRB1) and at the midportion of the moderator band (DRB2) and an anterior wall Purkinje fiber (PJ). Intervals (msec) measured from the conduction system electrograms to the onset of ventricular activation are shown. Sequential recordings along the course of the right bundle branch were obtained before repair. After sectioning of the moderator band, conduction system recordings were obtained from PRB and DRB2. Electrograms recorded beyond this level showed no specialized conduction system potentials. The DRB2 electrogram recorded after repair is not shown because that area was resected.

**FIGURE 6.** Selected epicardial electrograms in distal right bundle branch block in patient 14. ECG leads 1, 2 and 3 are shown with a reference electrogram recorded in the left ventricle (LV) and mapping electrograms recorded at the right ventricular apex (RVA), anterior wall (RVAW) and outflow tract (RVOT) before and after transatrial repair of tetralogy of Fallot. The vertical lines indicate the onset of the QRS and numbers indicate time (msec) from the vertical line to the local electrograms. Before repair, the RVAW was the earliest right ventricular site. After repair, right bundle branch block (RBBB) was present and the earliest activation was at the RVA. Despite the presence of RBBB, the RVA activation time after repair was not significantly different from the value before repair. Activation of the RVAW and RVOT were delayed (compare with fig. 7).

Activation mapping after RBBB in these patients revealed delay at most right ventricular epicardial sites. In four of the five patients, activation of the right ventricular apex was delayed 3–8 msec, an insignificant change; however, activation of the anterior and inferior walls and outflow tract was significantly delayed (greater than 25 msec) compared with the values before repair (figs. 6 and 7). In the remaining patient, activation of the right ventricular apical epicardium was delayed 22 msec compared with the value before repair.

Endocardial activation of the right ventricle was delayed on the free wall and outflow tracts of the right ventricle in these patients. Activation of the midseptum and apical septum of the right ventricle, however, remained similar to patterns (fig. 5) present before repair. Although endocardial electrograms recorded at the right ventricular apex were delayed more than
10 msec in two patients, in no patient did activation of this site occur after the first one-third of the QRS complex. The extent of normally activated areas varied within this patient group.

Terminal RBBB

In seven patients, conduction in the RBB was blocked in a portion of its terminal ramifications. Each of these patients had tetralogy of Fallot; five underwent transatrial repair and two underwent repair through a right ventriculotomy. The moderator band remained intact after repair in each of these patients. In each of the patients, conduction system electrograms were recorded from the distal His bundle site, proximal and distal right bundle branch sites and the anterior wall Purkinje fibers after repair of tetralogy of Fallot and the creation of RBBB (fig. 8).

In this group, right ventricular epicardial activation was delayed only in the anterobasal segment along the AV groove and the outflow tract (figs. 9 and 10). Delays of 30–73 msec were observed at these sites. Activation of the right ventricular apex and much of the anterior and inferior walls was unchanged after appearance of RBBB.

The sequence and timing of endocardial activation of the right ventricular septum and apex were unchanged after the creation of RBBB (fig. 8). Endocardial electrograms recorded in the outflow tract, however, were fragmented and delayed and usually occurred in the last one-third of the QRS complex during the slurred S wave inscribed in electrocardiographic lead I.

Discussion

Right ventricular conduction defect after congenital cardiac surgery can result from damage to the specialized conduction system at one or more sites along its course. Identification of these sites, however, has been difficult. Pathologic studies after repair of ventricular septal defects have shown that interstitial
hemorrhage and sutures may impinge upon the bundle of His and proximal RBB, producing infarction and necrosis; these structural abnormalities may interfere with proper function of the conduction system. On the other hand, Gelband et al. showed that RBBB occurs immediately after ventriculotomy when a transventricular approach is used, suggesting that peripheral damage to the conduction system, at least in some patients, results in RBBB. Subsequently, Krongrad and co-workers presented convincing physiologic evidence that the ventriculotomy produces RBBB by interruption of a peripheral branch of the RBB at a discrete site rather than by cumulative disruption of the continuous terminal fascicular network of the RBB. This interruption of the peripheral or terminal RBB usually occurs as a result of endocardial resection of abnormal right ventricular obstructing muscular tissue without ventriculotomy as well.

In the present study we identified three distinct levels of block in the RBB that correspond to three types of right ventricular conduction abnormality. Using techniques developed by Kaiser, Kupersmith, Krongrad, Waldo and their associates, we were able to delineate the course of the RBB and document the site of block by electrophysiologic measurement in 20 patients in whom the electrocardiographic pattern of RBBB developed during repair of congenital cardiac defects.

Proximal RBBB

The site of proximal RBBB is located between the bundle of His and the RBB immediately distal to the ventricular septal defects. Anatomic studies have shown that the RBB lies along the inferoposterior margin of the ventricular septal defect in patients with isolated ventricular septal defect, tetralogy of Fallot, and AV canal defects. The anatomic location of the RBB in this position has been verified by intraoperative conduction system mapping and our prerepair data. Interruption of RBB conduction in these patients results from repair of the ventricular septal defect. As a result of the repair in some patients, the cardiac impulse from the His bundle does not reach the proximal RBB.

The nature of the RBBB in the patient in whom left anterior hemiblock also developed did not appear to be different from the RBBB in other patients with proximal RBBB. There was no evidence of conduction to the proximal RBB. It has been shown in animal ex-
Experiments that the electrocardiographic pattern of RBBB and left anterior hemiblock can result from focal lesions in the distal bundle of His.\textsuperscript{38, 39} Unfortunately, on the basis of our data, we cannot distinguish a conduction defect located in the distal bundle of His from a lesion involving both the most proximal portion of the RBB and the anterior left bundle branch fibers destined to the anterior left ventricular wall. The absence of activation in the proximal RBB would be expected in both situations, and the resultant abnormal pattern of ventricular activation would be similar. The presence of a His bundle lesion could be established if it were possible to pace the His bundle distally and produce normalization of the QRS complex. Technical problems, however, make isolated distal His bundle pacing difficult, if not impossible, in most cases.\textsuperscript{40}

The pattern of right ventricular activation in our patients with proximal RBBB is similar to that produced in animals when the proximal RBB is cut,\textsuperscript{31-35} and in human hearts with chronic complete RBBB.\textsuperscript{36, 37} In this pattern, the left ventricle is activated normally; the earliest right ventricular activation occurs by transseptal spread along the interventricular grooves and activation of the basal portions of the right ventricle completes ventricular depolarization. Activation of the right side of the interventricular septum also changed in our patients with proximal RBBB. In pre-repair records, the earliest septal activation appeared in the midanterior septum near where we recorded the RBB potentials. A similar pattern of septal activation has been shown in animals.\textsuperscript{32, 37, 38} After proximal RBBB, activation of all recorded septal sites was delayed and the earliest activated site either shifted to a superior position on the septum or became nearly simultaneous in several sites. RBBB produced by cutting the RBB in animals produced a similar result.\textsuperscript{32, 37} Further, the right ventricular apex was activated later than normal in these patients, usually in the latter half of the QRS complex.

Distal RBBB

The site of distal RBBB is located in the moderator band. Anatomic studies have shown that the RBB courses along the septum and enters the moderator band crossing to the right ventricular anterior wall.\textsuperscript{39, 40} Our data support the hypothesis advanced by Krongrad et al.\textsuperscript{2} that disruption of the moderator band is an additional mechanism by which RBBB develops after surgery. In contrast to our patients with proximal RBBB, in whom sectioning of the RBB was inadvertent, in each patient with distal RBBB the moderator band was deliberately cut. Distal RBBB was not produced in any patient in whom the moderator band was not cut. Although uncommon anatomic variants have been described in which multiple right ventricular muscle bundles and two distal branches of the RBB co-exist,\textsuperscript{41} it appears that distal RBBB occurs only when the moderator band is sectioned and thus should be apparent to the surgeon at surgery.

Right ventricular activation during distal RBBB suggests that at least some fibers of the RBB enter the right ventricular myocardium before traversing the moderator band. An anteroseptal branch of the RBB has been described in man,\textsuperscript{42} but its functional significance is uncertain.\textsuperscript{3} Sectioning of certain fibers in the canine heart that are similar to those of the moderator band only produces changes in activation of the posterior septum, and anteroseptal activation remains unaffected.\textsuperscript{57} Sectioning of the free-running false tendon in the canine heart, which is analogous to the moderator band in man, produces delayed activation of a substantial portion of the right ventricular epicardium, but apicoseptal activation remains similar to control.\textsuperscript{33, 34} In man, after sectioning of the moderator band, the majority of sites in the right ventricular epicardium are activated later than before. Some apical and paraseptal sites were activated normally.

Terminal RBBB

The site of terminal RBB is located in the peripheral ramifications of the RBB. Krongrad et al.\textsuperscript{2} suggested that this type of block resulted from disruption of a discrete segment of the right ventricular specialized conduction system rather than from sectioning of a continuous Purkinje network.\textsuperscript{2} Coggin et al.\textsuperscript{43} presented similar data collected in man and experimental animals, and anatomic evidence of such discrete conduction tissue further supports this concept.\textsuperscript{44} Our data are compatible with these findings, but do not provide additional evidence. Krongrad et al.\textsuperscript{13} demonstrated continuity of the RBB from the bundle of His to the junction of Purkinje fiber and ventricular myocardium in the presence of RBBB after ventriculotomy. Our data are consistent with their findings.

In each patient with terminal RBBB, the block resulted from a right ventriculotomy or transatrial section of hypertrophied muscle bundles. As we have shown,\textsuperscript{3} there is little difference in the effects of disrupting the terminal segments of the RBB by either ventriculotomy or myocardial resection. The pattern of right ventricular activation in terminal RBBB is notable in that activation delays are restricted to the anterobasal portion of the right ventricular outflow tract. Endocardial activation and epicardial activation of the right ventricular apex are both normal.

Significance of Different Levels of Block

The clinical importance of distinguishing among the different levels of block in the RBBB is clear. If interruption of the RBB is proximal, accompanying or subsequent damage to the LBB system could lead to complete AV block, increasing the risk of hemodynamic deterioration or sudden death.\textsuperscript{43} The functional significance of distal RBBB remains unclear. Although it appears that a variable portion of the right ventricle may be activated by branches of the RBB before the moderator band, the long-term reliability of
these fibers is unknown and their presence in all hearts has not been verified. Because of these uncertainties we agree with Kronrad et al. that sectioning of the moderator band should be avoided whenever possible. Terminal RBBB appears to be inconsequential. Delayed activation of the infundibulum should have no hemodynamic effect. Terminal RBBB should not increase the risk of subsequent complete AV block because the more proximal connections of the RBBB reach the right ventricle normally.

Clinical Identification of the Site of Block

Our data suggest a method by which proximal and terminal RBBB can be distinguished postoperatively. If right ventricular apical endocardial or epicardial activation occurs in the first third of the QRS complex in the presence of a RBBB pattern on the ECG, proximal RBBB can be excluded. This recording can usually be obtained with the electrodes placed on the right ventricle as temporary pacing electrodes postoperatively. Differentiating between distal and terminal RBBB is more difficult because of the variable amount of normal apical activation in the former. It appears, however, that distal RBBB occurs only when the moderator band is cut at surgery; otherwise, distal RBBB can be reasonably excluded.

The three types of block can be distinguished by electrophysiologic study. An electrogram from the right ventricular apex can be recorded with an electrode catheter during electrophysiologic study, a technique used by Kastor and co-workers to study ventricular activation during spontaneous RBBB. In patients who had right ventricular activation similar to that in our patients with proximal RBBB, they found delay in right ventricular apical activation. Our data verify the concept, proposed by Castellanos, Gelband and Sung and their co-workers, that measurement of the right ventricular apical activation time can identify patients with proximal RBBB. Normal activation time of the apex of the right ventricle in the presence of the ECG pattern of RBBB would identify a patient with terminal RBBB.

Acknowledgments

The authors thank Helaine Corr for her secretarial assistance, the nurses and technicians of the cardiothoracic operating rooms at Children's Hospital of Philadelphia for their invaluable assistance, and Drs. Joseph F. Spear, John A. Kastor, Chief of the Cardiovascular Section, and Mark E. Josephson, Director of the Clinical Electrophysiology Laboratory, for their enthusiastic support and editorial assistance. We particularly thank Dr. E. Neil Moore without whose pioneering work and continuing support this study would not have been possible.

References

24. Latham RA, Anderson RH: Anatomical variations in atrioven-
tricular conduction system with reference to ventricular septal defects. Br Heart J 34: 185, 1972
34. Uhley HN, Rivkin L: Electrophysiologic patterns following interruption of main and peripheral branches of the canine right bundle of His. Am J Cardiol 7: 810, 1961
Postoperative right bundle branch block: identification of three levels of block.
L N Horowitz, J A Alexander and L H Edmunds, Jr

Circulation. 1980;62:319-328
doi: 10.1161/01.CIR.62.2.319
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1980 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on
the World Wide Web at:
http://circ.ahajournals.org/content/62/2/319.citation