Further Observations on the Etiology of the Right Bundle Branch Block Pattern Following Right Ventriculotomy

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SUMMARY

Fifteen patients with various congenital heart defects were studied during open heart surgery in order to establish the precise mechanism by which a right ventriculotomy causes a right bundle branch block (RBBB) pattern on the scalar electrocardiogram (ECG). All required a right ventriculotomy for the correction of their defects. In each, the right ventriculotomy was carried out in steps, with incisions (3 to 7) of approximately 1 cm in length. Six simultaneous scalar ECG leads were recorded prior to the first incision and following each incision. The QRS duration was then measured and related to the length of the ventriculotomy.

Following the right ventriculotomy, 12 of the 15 patients developed an RBBB pattern; the remaining three did not. In all cases, the total increase in QRS duration occurred during one specific incision of the right ventricular free wall and was not related to the total length of the ventriculotomy or to the sequence of the incisions. The site at which incision of the right ventricle caused the RBBB pattern was located at between 40 to 75% of the distance between the pulmonary artery annulus and the inferior border of the heart in nine patients; in three patients the site was higher on the right ventricular free wall.

Our results explain why some patients who had a right ventriculotomy during open heart surgery do not develop an RBBB pattern on the scalar ECG. Also, since no relation was found between the length of the incision and the QRS duration, our results suggest that the ventriculotomy-induced RBBB pattern is unlikely to be due to disruption of a continuous Purkinje network but is probably due to disruption of a distal branch or branches of the right bundle.

Additional Indexing Words:
Specialized atrioventricular conduction system
Electrocardiogram
Tetralogy of Fallot
Congenital heart disease

The electrocardiographic (ECG) pattern of right bundle branch block (RBBB) observed following surgical correction of various forms of congenital heart disease has been attributed to surgical injury of various parts of the right intraventricular conduction system. Although injury to the proximal part of the right bundle branch can occur during open-heart surgery, the electrocardiographic pattern of RBBB has been shown recently by Gelband and associates to occur immediately following the right ventricular incision, and prior to closure of the ventricular septal defect or pulmonary infundibular resection. This finding suggests that the postoperative RBBB pattern in these patients is related primarily to the right ventriculotomy. The precise mechanism, however, by which a right ventriculotomy causes this electrocardiographic pattern is unknown. It is not known whether alteration in the right ventricular activation sequence is dependent on the length of the incision, and thus on progressive interruption of the right ventricle Purkinje network, or whether this electrocardiographic pattern is caused by severing a distal branch or branches of the right bundle at a certain location. Further, if the latter is the case, this specific location has not been identified.

This study was undertaken to identify the precise mechanism by which a right ventriculotomy causes distortion of right ventricular activation sequence, QRS prolongation, and an RBBB pattern and to identify the precise anatomical location of the surgical incision which causes the prolongation of the QRS.

Materials and Methods

Fifteen patients with various forms of congenital heart disease requiring a right ventriculotomy were studied dur-
ing open-heart surgery. A summary of the pertinent clinical data is provided in table 1. The patients ranged in age from 15 months to 50 years. There were seven males and eight females. None had a preoperative ECG with an RBBB pattern. All studies were performed during the period of cardiopulmonary bypass and prior to aortic cross-clamping. During periods of data collection, body temperature was measured from the retrocardiac portion of the esophagus or from the rectum. The temperature varied from patient to patient, as determined by the requirements of the surgical procedure, but in each patient, all recordings were made at the same temperature.

Seven patients were studied during atrial rhythm produced by pacing the atria through a bipolar electrode

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<tr>
<th>Name</th>
<th>Diagnosis</th>
<th>Age</th>
<th>Sex</th>
<th>Temperature (°C)</th>
<th>Preanesthetic medication (mg)</th>
<th>Anesthesia</th>
<th>Type of ventriculotomy</th>
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<tr>
<td>1) T.C.</td>
<td>T/F</td>
<td>15 mo</td>
<td>M</td>
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<td>SEC − 30</td>
<td>SC − 70</td>
<td>MS − 8</td>
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<td>M</td>
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<td>H, N₂O</td>
</tr>
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<td>3) J.M.</td>
<td>T/F</td>
<td>31 mo</td>
<td>M</td>
<td>32</td>
<td>MS − 1</td>
<td>C, H, N₂O</td>
<td>H, N₂O</td>
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<td>4) T.D.</td>
<td>VSD</td>
<td>5½ y</td>
<td>F</td>
<td>32</td>
<td>MS − 1</td>
<td>C, H, N₂O</td>
<td>H, N₂O</td>
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<td>5) C.E.</td>
<td>T/F</td>
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<td>TH − 250</td>
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<td>30</td>
<td>PH − 15</td>
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<td>H, N₂O</td>
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<td>5 y</td>
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<td>PH − 12</td>
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<td>H, N₂O</td>
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<td>VSD, PS</td>
<td>5 y</td>
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<td>28</td>
<td>SEC − 50</td>
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<td>PS</td>
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<td>SC − 100</td>
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<td>VSD</td>
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<td>13) G.B.</td>
<td>VSD</td>
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<td>32</td>
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<td>C, H</td>
<td>H, N₂O</td>
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<td>15) L.J.</td>
<td>VSD, STRAD, TV</td>
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<td>28</td>
<td>SEC − 30</td>
<td>C, N₂O</td>
<td>H, N₂O</td>
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Table 1
Clinical Data for 15 Patients Studied

Abbreviations: M = male; F = female; T/F = tetralogy of Fallot; VSD = ventricular septal defect; IHSS = idiopathic hypertrophic subaortic stenosis; PS = pulmonary stenosis; STRAD. TV = straddling tricuspid valve; mo = months; y = years; L = longitudinal; T = transverse; SEC = seconal; MS = morphine sulfate; SCOP = scopolamine; SC = succinyl choline; C = cyclopropane; H = Halothane; A = Atropine; TH = Thiopental; PH = Phenergan, P = Pancurare; DEM = Demerol; V = Valium.
placed at the region of the sinoatrial node. The atria were paced at a rate slightly in excess of the spontaneous rate. Stimuli were provided by a special digital threshold stimulator (Medtronics 1187). All leads in contact with the heart were isolated both from the ground and from the recording instruments by isolation transformers. Four patients were studied during normal sinus rhythm and four patients were studied during a junctional rhythm in which the morphology and the frontal orientation of the QRS complex were identical to the QRS morphology and orientation prior to surgery.

In each patient, six frontal ECG leads (I, II, III, aV_{l}, aV_{r}, aV_{F}) were monitored simultaneously on a switched-beam oscilloscope (Electronics for Medicine, Model DR-12) and recorded on a magnetic tape recorder (Honeywell 5600) at 1\% ips. ECGs were recorded at a sensitivity of 1 to 2 cm/mV. Following institution of cardiopulmonary bypass, six frontal ECG leads were recorded to provide baseline data on QRS duration and orientation prior to the right ventricular incision. Then, the ventriculotomy was carried out in steps, with 3 to 7 incisions of approximately 1 cm in length performed for each ventriculotomy. After each incision, the recording of the six frontal scalar ECG leads was repeated. In some patients, the same recordings were repeated after closure of the ventricular septal defect and/or infundibular resection.

At the end of the surgical procedure and following the closure of the ventriculotomy, several measurements were obtained in order to identify precisely the location of the ventriculotomy on the right ventricular free wall, and its relation to the various structures on the cardiac surface. These measurements were as follows: the distance from the 1) pulmonary artery annulus to the diaphragmatic surface of the heart (length of the heart) 2) pulmonary artery to the upper margin of the ventriculotomy; 3–5) left anterior descending coronary artery to the upper, middle, and lower parts of the ventriculotomy; and 6) total length of the ventriculotomy. The distance from the lower end of the ventriculotomy to the diaphragmatic border of the heart was obtained by subtracting the length of the ventriculotomy plus the distance from the pulmonary artery to the upper part of the incision from the total length of the heart. Also, photographs of the ventriculotomy and its relation to the right ventricular free wall were obtained during surgery.

Following completion of data collection, the tracings were replayed at 15/16 ips, thereby expanding the electrocardiograms by a factor of two for greater accuracy in measurement of the QRS complex. The QRS duration was determined from the onset of the QRS inscription in any of the six simultaneous leads to the latest inscription seen at any of these six simultaneously recorded ECG leads. All measurements were made in duplicate or triplicate using a vernier measuring device having an accuracy of ±1 msec. The frontal plane QRS axis was estimated from the six frontal ECG leads.

Results

Fourteen patients had a vertical and one patient had an oblique ventriculotomy. The QRS duration increased in 12 patients at the time of ventriculotomy, and in three patients the post-ventriculotomy QRS duration remained identical to the control value. The preoperative QRS duration for those patients who developed an RBBB pattern following the ventriculotomy ranged from 60 to 88 msec (mean 71 msec), and the postoperative QRS duration ranged from 85 to 125 msec (mean 107 msec). The mean increase in QRS duration was 36 msec, a value similar to that reported by Gelband and associates.

Figure 1 shows five groups of six simultaneous frontal ECG leads obtained during open-heart surgery, from a 2-year-old girl, operated on for a ventricular septal defect. An increase in QRS duration from 65 to 99 msec, a marked widening of the S wave in leads I, II, aV_{F}, and a widening of the R' in aV_{r} was observed following the third and last incision. No further changes were seen following the VSD closure. These changes persisted in the patient's postoperative electrocardiogram (fig. 2).

Patients were separated into four groups on the basis of the pattern of development of QRS prolongation (table 2). The pattern of an increase in QRS duration with the last incision illustrated in figure 1 occurred in three of the 15 patients studied (group A). In four patients, there was no change in QRS duration following the first several incisions. An increase in QRS duration then occurred with an additional 1 cm incision. Further enlargement of the ventriculotomy did not result in a further increase in QRS duration (group B). In five patients, the QRS complex was maximally prolonged immediately following the first 1 cm incision, with no further increase on subsequent incisions.

Table 2

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<th>Incision number</th>
<th>B.L.</th>
<th>1</th>
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<td>65</td>
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<td>77</td>
<td>77</td>
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<td>65</td>
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<td>113</td>
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<td>B.W.</td>
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B.L. = Baseline; QRS Duration.
Five groups of six simultaneous frontal electrocardiograms obtained during open-heart surgery on a 2-year-old patient with a ventricular septal defect. The first group (base line) was obtained immediately prior to the ventriculotomy. The following three groups were obtained during the ventriculotomy (incision) with each group taken following an incision of approximately 1 cm (marked with an arrow). During the incision there is a progressive rightward shift in the cardiac frontal vector (FAQRS) from 80° to 120°. There is also an inconsequential increase in P-R interval during the recording period from 118 to 170 msec. Paper speed = 100 mm/sec; S = stimulus.

Figure 1

In four of the seven patients in whom the QRS increased, slight changes in QRS waveform were noted prior to the increase in QRS duration. The changes include deepening of the S wave in lead I and an increase in r' in aV_R similar to that commonly interpreted as "incomplete RBBB" (fig. 1).

Localization of the Site Causing QRS Prolongation

Figure 5 is a close-up photograph of the free right ventricular wall (D. L.), taken during operation and a diagram reconstructed from the photograph and the multiple measurements obtained at surgery. The horizontal strips show the border between incisions 1, 2, and 3. In this patient the increase in QRS duration occurred following the third incision of about 1 cm (figures 1 and 2), and occurred at 64% of the distance from the pulmonary artery to the inferior border of the heart. In this patient, the last incision was carried
through the hypertrophied and anteriorly displaced moderator band of the right ventricle, proximal to its insertion into the anterior papillary muscle.

Figure 6 shows the location of the right ventriculotomy on each of the 15 patients studied, the site of each incision in relation to the right ventricular surface, the length of the ventriculotomy, and the order in which each small incision was carried out for each ventriculotomy.

In nine of the 12 patients who developed RBBB following the ventriculotomy, the middle of the incision which caused the QRS prolongation was located at between 40% to 73% of the length of the heart (the
distance from the pulmonary artery annulus to the inferior border of the heart). In three patients, this site was located at 25, 30, and 31% of this distance. The total lengths of the heart in these three patients, two with tetralogy of Fallot and one patient with a ventricular septal defect, were the smallest in our study, measuring 6, 6.5, and 5 cm, respectively.

In the seven patients in whom the increase in QRS duration did not occur with the first incision (groups A and B), the site at which this increase occurred was at 40 to 73% (mean 61) of the distance from the pulmonary artery annulus to the inferior border of the heart. In the five patients (group C), in whom the QRS duration increase occurred with the first incision, this site was higher on the right ventricular free wall, and ranged from 25 to 47% (mean 35%) of this distance.

In the 11 patients with a longitudinal ventriculotomy who developed an RBBB pattern, the distance of the ventriculotomy from the anterior descending coronary artery ranged from 2 to 3 cm at the top of the incision, and from 2 to 5.5 cm at the bottom. In the three patients who did not develop an RBBB pattern, the distance of the ventriculotomy from the anterior descending coronary artery was (top to bottom) 1.25–1 cm; 2–2 cm; and 3–3.5 cm in each.

Discussion
A right ventricular conduction delay following open-heart surgery may be caused by any or a combination of several mechanisms.1–4, 6, 7, 13 Titus et al. in 1963 have shown that many patients operated on for repair of their ventricular septal defect had hemorrhage, suture placement, and infarction of their right bundle branch following open-heart surgery. These lesions were usually associated with similar lesions in other parts of the specialized A-V conduction system. In only one of 12 specimens studied in detail with histologic techniques were the authors able to show an isolated hemorrhage into the right bundle branch which did not involve the A-V node, the His bundle, or the left bundle branch. Latham and Anderson6 more recently suggested that edema and pericellular exudate might also interfere with the proper function of the His bundle and bundle branches following open-heart surgery.

However, several reports have shown that the RBBB pattern might result primarily from a right ventriculotomy and the peripheral disruption of the right ventricular conduction system.8–13 This has been shown to be equally true for canines.11, 12 The finding that this conduction disturbance can be explained by a similar cause in humans also,8–10, 13 has been recently questioned, however, because some patients do not develop an RBBB pattern following a right ventriculotomy.17 Our results show that in 12 of 15 patients studied in whom an RBBB developed following surgery the development of this pattern was associated with the right ventriculotomy. In three patients, including one patient in whom a ventricular

Figure 5
Close-up photograph and schematic drawing of the right ventricular free wall and right ventriculotomy on the patient D. L. (fig. 1) indicating the precise location of the incision resulting in prolongation of QRS duration (shaded areas on the drawing denote areas not seen on photograph).
A RBBB following open-heart surgery recently characterized by a septal defect was closed, and pulmonary infundibular resection done, an RBBB did not develop immediately following the right ventriculotomy, and in all three, the postoperative electrocardiogram did not show an RBBB. These results show that in most patients operated recently in our institution, the RBBB results from peripheral disruption of the right ventricular conduction system during the ventriculotomy rather than injury to the proximal right bundle branch caused during the VSD closure or infundibular resection.

The results also show that in all patients studied by us in whom an RBBB occurred during the ventriculotomy, the pattern appeared with an incision of about 1 cm or less and that no direct relationship exists between the length of the ventriculotomy and QRS duration. This observation, therefore, suggests that the delay of right ventricular activation sequence is due to disruption of a peripheral branch or branches of the right bundle branch or its arborization located at a specific site, rather than a continuous disruption of a right ventricular Purkinje network. Our observation also explains why some patients who had a right ventriculotomy such as those described by Anderson et al., and most patients in whom a small incision is made on the free wall of the right ventricle, do not develop an RBBB.

In ten patients, the ventriculotomy was carried out in sequence from its upper part toward the inferior border of the heart. In the other five patients, however, the ventriculotomy was not carried out in such a sequence. Also, the QRS prolongation was not related to the order in which the incisions were made in the various groups of patients. It is, therefore, clear that the increase in QRS duration and the site of its occurrence are independent of the order in which the separate incisions were performed, and that the QRS prolongation which occurs with a specific incision is not the result of a previous and cumulative injury to the right ventricular conduction system manifested only with the last incision.

In most of our patients, the right longitudinal ventriculotomy was carried out lateral to the insertion of the moderator band into the base of the anterior papillary muscle, which suggests that in these patients the disruption occurs at the very distal part of the

Figure 6

Precise location of the right ventriculotomy and its relation to the right ventricular free wall obtained from multiple measurements in each of the patients studied. The number next to each incision represents the order in which the incisions were made. The cross-hatched areas represent the incision which caused the QRS prolongation. The diagrammatic presentations are: Group A: 3 patients in whom the QRS prolongation occurred on the last incision; Group B: 4 patients in whom QRS prolongation occurred following several 1 cm incisions in each, with no additional increase in QRS duration on subsequent enlargement of the ventriculotomy; Group C: 5 patients in whom QRS prolongation occurred with the first incision; Group D: 3 patients in whom QRS prolongation did not occur.
right bundle branch and most probably following the insertion of some of its terminals into the right ventricular myocardium.11-22 This location is identical to the location of the incision which caused the distortion of right ventricular activation sequence previously described by Genender and associates in canine hearts.11 The likelihood that right bundle branch terminals had already penetrated into the right ventricular myocardium in spite of the development of an RBBB pattern on the ECG support the conclusion of Gelband et al.18 that, in these cases, this electrocardiographic pattern may be of little clinical prognostic significance.

In other patients, however, the RBBB occurred with the incision of the moderator band proximal to its insertion into the base of the anterior papillary muscle (fig. 6). Such an injury to the moderator band is likely to occur in patients with an anterior displacement of the moderator band and the anterior papillary muscle and also with a longitudinal ventriculotomy used in all but one patient in our study. Disruption of the right bundle branch at the level of the moderator band is also likely to occur in patients with severe right ventricular outflow obstruction who require pulmonary infundibular resection and excision of the markedly hypertrophied moderator band. Our observation that in some patients disruption of the moderator band, and therefore of the right bundle branch, occurs during surgery therefore demonstrates an additional mechanism by which an RBBB can develop following surgery.

In their study Gelband et al.18 demonstrated that, on completion of the ventriculotomy, right ventricular activation was delayed at sites lateral to the ventriculotomy. Since activation time did not change medial to the ventriculotomy following infundibular resection and VSD closure, they assumed “that no further injury occurred with further procedures.”

If and when such injury to the proximal part of the right bundle branch occurs, it might not always be detected with the epicardial recording technique previously used,18 or following a ventriculotomy which causes an RBBB pattern on the electrocardiogram. Sodi-Pallares et al.,21 Scher et al.,22 and Burchell et al.23 have shown that, in the canine, left ventricular septal activation precedes right ventricular septal activation, and that parts of the interventricular septum are excited through a left ventricular excitation wave. Similar findings were demonstrated by Durrer for isolated human hearts.20 It is therefore likely that parts of right ventricular epicardial surface close to the interventricular septum might be activated through an excitation wave originating in the left ventricle. If epicardial recordings are therefore obtained medial to the ventriculotomy and close enough to the interventricular septum, in the presence of an intact left bundle branch, no delay, or a very minimal delay in activation sequence will be detected in the presence of a proximal disruption of the right bundle branch.

The clinical and prognostic significance of right bundle branch disruption is related to whether right bundle branch terminals have already been inserted into the right ventricular myocardium. Since the anatomic importance of the anterior-septal fascicle of the right bundle branch as recently defined24 is not yet clear,17,25 we feel that the disruption of the right bundle branch at the level of the moderator band is of clinical importance. This is so mainly because it is a major fascicle of the right bundle branch and disruption occurs prior to its insertion into right ventricular myocardium. Further, in the absence of precise knowledge of a more proximal insertion of right bundle branch terminals into right ventricular myocardium, a lesion to the moderator band should be considered to be of similar prognostic significance to a lesion involving the proximal part of the right bundle branch which might occur during a VSD closure. Current electrocardiographic criteria do not differentiate these various sites of injury and might, in many cases, be of little aid in establishing the patient’s prognosis.

In four patients, changes in QRS waveform occurred prior to QRS prolongation (fig. 1). These changes included deepening of the terminal forces in leads I and aV1, and development of a more prominent r’ in aV4R, and are similar to the ECG pattern commonly interpreted as “incomplete right bundle branch block.”26 These changes resulted from peripheral alteration in right ventricular activation sequence following the incision of the right ventricular free wall rather than delayed conduction (“incomplete block”) in the right bundle branch. The difficulties, overlap, arbitrariness, and inadequacy of the current electrocardiographic interpretation and criteria for “incomplete” and “complete” RBBB as a true representative of cardiac electrophysiological phenomena have been commented upon previously.27-29

In our institution, the RBBB pattern following open heart surgery is associated with the right ventriculotomy in most patients. No electrocardiographic criteria, however, are currently available to identify additional injury to the proximal part(s) of the right bundle branch after disruption of its peripheral ramifications. It is suggested therefore that current electrocardiographic analysis of the postoperative RBBB pattern (or even spontaneously occurring RBBB) might carry different prognostic significance in various groups of patients. Groups in whom the RBBB
pattern is due to peripheral disruption of the right bundle branch after its insertion into the right ventricular myocardium (right ventricular conduction delay) and groups in whom a proximal lesion (a true RBBB) or combined peripheral and proximal lesions occurred may therefore carry different prognoses. The improved understanding of the various sites of possible injury to the specialized A-V conduction system, and in particular to the right bundle branch, during open-heart surgery, improved surgical techniques, and the possibility of intraoperative identification of the right bundle branch* will hopefully serve in the future to further reduce the occurrence of injury to the right ventricular conduction system during open-heart surgery.

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Further Observations on the Etiology of the Right Bundle Branch Block Pattern Following Right Ventriculotomy

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