The Mechanism of Apparent Right Bundle Branch Block 
After Transatrial Repair of Tetralogy of Fallot

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SUMMARY The electrocardiographic pattern of right bundle branch block (RBBB) is routinely observed after transatrial repair of tetralogy of Fallot even though no ventriculotomy has been performed. The mechanism of this conduction disturbance was studied in 16 patients with tetralogy of Fallot and one patient with infundibular pulmonic stenosis. Preoperative ECGs and vectorcardiograms showed right ventricular hypertrophy and no RBBB. Epicardial activation maps were obtained before and after total surgical repair in all patients and after infundibular resection but before closure of ventricular septal defect (VSD) in four of these patients. After infundibular resection, RBBB appeared and activation was markedly delayed (> 30 msec) over the pulmonary outflow tract, but was unchanged over the body of the right ventricle. No further changes in ventricular activation occurred after closure of the VSD. This study shows that RBBB after transatrial repair of tetralogy of Fallot is usually produced by infundibular resection, but not by VSD closure, and is associated with delayed activation of the pulmonary outflow tract and base of the right ventricle which results from damage to portions of the right ventricular conduction system.

THE ELECTROCARDIOGRAPHIC PATTERN of right bundle branch block (RBBB) almost invariably appears after the total surgical repair of the tetralogy of Fallot (TOF). The production of RBBB has been attributed to occasional transection of the proximal right bundle branch during closure of the ventricular septal defect (VSD) and the almost invariable interruption of the terminal ramification of the right bundle branch by right ventriculotomy. This latter mechanism is most frequently responsible for producing RBBB during the routine transventricular repair of TOF. Previous investigators have emphasized that the electrocardiographic pattern of RBBB does not necessarily imply interruption of the proximal right bundle branch. RBBB, however, also occurs after correction of TOF performed through an atriotomy without a ventriculotomy. This study was performed to determine the etiology of RBBB after repair of TOF and to determine the significance of RBBB in TOF by studying the ventricular activation patterns.

Methods and Materials

Seventeen patients undergoing total repair of TOF (16 patients) or infundibular pulmonic stenosis (one patient) were studied during cardiac surgery after informed consent was obtained (table 1). The patients ranged in age from 4–21 years; there were nine males and eight females. Seven patients had had previous palliative operations that required repair at the time of TOF correction. We obtained 14-lead ECGs (routine 12-lead and V6R and V7) and vectorcardiograms (VCG) (Frank system) on the day before surgery and during the second postoperative week. In each patient, the preoperative ECG and VCG were compatible with right ventricular hypertrophy, and no patient had RBBB (table 1). RBBB was defined by prolongation of the QRS complex (usually 0.12 second or longer), an S wave in leads 1 and V6 and a double peaked R wave in V1 and V2. Vectorcardiographic criteria included prolongation of the QRS loop and slowly inscribed (> 30 msec) terminal forces directed anteriorly and to the right. In four patients RBBB occurred with a QRS duration < 0.12 msec; however, the typical ECG and VCG patterns of terminal rightward delay were observed.

The sequence of epicardial activation was determined by recording bipolar electrograms at 40–80 epicardial sites. A bipolar plunge electrode (teflon coated stainless steel; 0.005 inch diameter) was inserted as a reference electrode into the left ventricular myocardium by means of a 23-gauge needle. The epicardium was mapped with a hand-held probe at preselected sites marked on a Polaroid photograph of the heart which had been taken before the mapping procedure. Surface electrocardiographic leads 1, 2 and 3 were recorded; the band pass was 0.1–100 Hz. The electrograms were filtered at 40–500 Hz. All data were stored on magnetic analog tape (Honeywell 5600C)
TABLE 1. Clinical Data

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</tr>
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<td>Left Blalock-Taussig</td>
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<tr>
<td>17</td>
<td>11/M</td>
<td>RVH, RAD</td>
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Abbreviations: RAD = right-axis deviation; RVH = right ventricular hypertrophy; TOF = tetralogy of Fallot.

and were subsequently retrieved on paper at a speed of 250 mm/sec. Interval measurements were made on three to six ECG complexes for each recording site with a Hewlett-Packard desktop computer and digitizer. The reproducibility of measurements by our methods was ± 2 msec. Measurements were made at the point at which the first rapid deflection crossed the baseline. All interval measurements were made between the reference electrogram and the probe electrogram. For clarity in illustrations, the activation time was adjusted to reflect the interval between the onset of the QRS complex and the probe electrogram by adding the constant Q-reference electrogram time to each measurement.

We performed epicardial mapping in each patient during sinus rhythm before and after cardiopulmonary bypass. In four patients, an additional epicardial map was obtained after infundibular resection but before VSD repair (patients 4–6, 9). All epicardial mapping was performed when the rectal temperature was between 35.5 and 37°C.

Surgical Techniques

Transatrial repair of TOF was carried out in 13 patients. No ventriculotomy was performed; however, the hypertrophied septal and parietal obstructing bands were excised so that the remaining thickness of the right ventricular infundibulum was approximately 6–12 mm.

Transventricular repair was carried out in three patients through a vertical right ventriculotomy. Hypertrophied muscle bands were excised to leave a wall thickness of 6–10 mm. The ventricle was closed using a patch of pericardium. VSDs were closed using a dacron patch and running sutures were reinforced with three to five interrupted figure-of-eight sutures in all patients.

**Figure 1.** ECGs before and after transatrial repair of tetralogy of Fallot (patient 14). Electrocardiographic leads I, aVF, V₁, and V₆ are shown. The preoperative ECG (A) showed right-axis deviation and right ventricular hypertrophy. The postoperative ECG (B) obtained 10 days after surgery showed incomplete right bundle branch block and a minor leftward axis shift.
In one patient without a VSD, valvular and infundibular pulmonic stenosis were relieved. After valvulotomy, infundibular resection was carried out transatrially without ventriculotomy.

**Results**

The typical electrocardiographic or vectorcardiographic patterns of RBBB developed in 16 of 17 patients after transatrial or transventricular repair of TOF. In patient 14, minimal QRS prolongation was observed in association with modest terminal rightward delay in the VCG; however, criteria for RBBB were not met (figs. 1 and 2).

In the preoperative epicardial map in all patients, right ventricular activation began along the anterior interventricular groove and spread concentrically toward the base. Right ventricular epicardial activation was completed in 49 ± 8 msec (mean ± SD). In 12 of 17 patients, the earliest epicardial activation of the left ventricle occurred within 10 msec of right ventricular epicardial breakthrough. This pattern of epicardial activation is compatible with right ventricular hypertrophy. A representative example is shown in figure 3A.

**Ventricular Activation After Transatrial Repair**

RBBB or right ventricular conduction defect (one patient) and delayed activation of the right ventricular outflow tract also developed in the 12 patients with TOF in whom repair was performed through the right atrium without a ventriculotomy (fig. 4). In each patient, activation of all epicardial sites in the outflow tract was delayed 32–62 msec (fig. 5). The difference map for each patient in this group was qualitatively similar to the example shown in figure 5. In all patients, the body of the right ventricle was activated normally and delayed activation was confined to the right ventricular base and outflow tract.

Epicardial recordings were obtained after infundibular resection but before closure of the ventricular septal defect in four of the patients with transatrial repair of TOF (patients 4, 5, 6 and 9). Immediately after infundibular resection, the electrocardiographic pattern of RBBB appeared and the QRS duration in-
increased from 73 ± 8 msec to 128 ± 12 msec. Simultaneously, right ventricular activation delay occurred in the right ventricular outflow tract and base (fig. 6). The location of right ventricular epicardial breakthrough and the activation sequence of the body of the right ventricle were unchanged however. After ventricular septal defect repair, the epicardial activation sequence was unchanged from that observed after infundibular resection (figs. 6B and 6C).

RBBB developed in the patient with infundibular pulmonic stenosis after transatrial infundibular resection was performed. The right ventricular activation pattern was similar to that of all patients with TOF repaired without a ventriculotomy. Activation maps were similar to those obtained after infundibular resection in patients 4, 5, 6 and 9, discussed above.

Comparison of Ventricular Activation After Transventricular and Transatrial Approaches

The degree of prolongation of the QRS complex and right ventricular activation time after repair of TOF were comparable. The changes after transatrial and transventricular repairs were similar (table 2); however, we observed a qualitative difference in the pattern of right ventricular activation between these two groups. Although in both groups the area of delayed epicardial activation was confined to the base
of the right ventricle and pulmonary outflow tract, after transventricular repair the change from normal to prolonged activation time was abrupt; after transatrial repair, the activation delay appeared gradually across the area of infundibular resection (figs. 3 and 6). Furthermore, after transventricular repair, activation of the outflow area proceeded from areas distal to the ventriculotomy and the latest areas activated were those adjacent to the incision. After transatrial repair, the direction of ventricular activation was proximal to distal areas across the site of infundibular resection and the latest areas activated were those adjacent to the atrioventricular groove.

Relationship of Ventricular Activation to the ECG

Epicardial activation times were plotted on a time axis aligned with the QRS complex recorded in lead I to correlate activation times with the ECG (fig. 7). Before repair, the QRS duration was 75 msec and right ventricular epicardial activation occurred 31–75 msec after the onset of the QRS complex. After the creation of RBBB, all epicardial activation times which occurred during the slurred portion of the QRS corresponded to sites on the right ventricular outflow tract, while epicardial activation of the apex and body of the right ventricle remained unchanged during the midportion of the QRS complex. In fact, the entire slurred S wave was explained by delayed infundibular activation (fig. 6). In patient 14, the outflow tract activation was delayed 16 msec after repair, but no discrete area of block was defined. These delayed sites occurred during the terminal 10 msec of the QRS complex. In all patients, the prolongation of the QRS complex and slurred terminal portion were produced by delay in infundibular activation.

Discussion

In the present study we have shown that RBBB observed after the repair of TOF without a ventriculotomy is produced by delayed activation of the pulmonary outflow tract. This delay occurred at the
FIGURE 4. Right bundle branch block (RBBB) and delay in outflow tract activation after transatrial repair of tetralogy of Fallot (patient 9). Electrocardiographic leads 1, 2, and 3 are displayed with electrograms from the left ventricular (LV) anterior wall (reference) and time-aligned electrograms from the right ventricular anterior wall (RVAW), right ventricular apex (RVA) and right ventricular outflow tract (RVOT). A) Before repair, right ventricular activation was earliest in the RVAW and latest in the RVOT at 62 msec after the onset of the QRS complex (solid vertical line). B) After repair, RBBB was present and activation of the RVOT was delayed to 124 msec. Activation of the RVAW and RVA was unchanged from control.

The electrocardiographic pattern of RBBB which routinely follows the total correction of TOF may result from trauma to the right bundle branch at several different locations. Gelband et al. and Krongrad et al. have attributed postoperative RBBB in most patients to the ventriculotomy, which interrupts the peripheral conduction tissue of the right ventricle. They showed that activation of right ventricu-
ular sites distal to the ventriculotomy was delayed from control studies and this delay was correlated with the presence of RBBB. Further support of their hypothesis was furnished by the observation that RBBB usually occurs after repair of VSDs through a right ventriculotomy, but is less common after repair through an atriotomy. The observation that RBBB occurs routinely after transatrial repair of TOF therefore requires further explanation.

Before these observations were made, it was assumed that the postoperative RBBB which occurs after TOF repair was caused by right ventriculotomy alone. However, since ventriculotomy by necessity occurred before infundibular resection in the studies of Gelband and Krongrad, RBBB was present from the earliest part of the operation and the effect of disruption of the distal conduction system by infundibular resection could not be assessed. In this study, transatrial infundibular resection avoided the need for ventriculotomy and allowed us to assess the effect of infundibular resection alone. We assume that infundibular resection transects the peripheral conduction system in a location similar to that of a right ventriculotomy.

There are, of course, certain limitations in assessing the site of block within the conduction system by examining patterns of epicardial activation alone. The contribution of the septal ramifications of the right bundle branch as reflected by right septal depolarization cannot be measured without septal recordings, which were not performed in this study. Similarly, block occurring at more than one site (e.g., at the level of the moderator band as well as the endocardial Purkinje system) cannot be identified without extensive endocardial and intramural recordings. Despite these limitations, it appears that routine repair of TOF results in RBBB that is associated with activation delay confined to the outflow tract. Preliminary
FIGURE 5. Difference map after transatrial repair of tetralogy of Fallot (patient 11). A schematic anterior view of the right ventricle is shown. The data indicate the mapping sites and the magnitude of the changes between the preoperative and postoperative maps. The stippled area indicates sites at which activation was delayed more than 10 msec. The area of delayed activation was confined to the right ventricular outflow tract and base.

studies with endocardial and intramural recordings have shown that this pattern of ventricular activation does not occur after damage to the proximal right bundle branch or moderator band. Thus, although this study cannot document the interaction between the effects of peripheral Purkinje system disruption and intramyocardial delay secondary to myectomy, the site of block can probably be placed distal to the right bundle branch in the terminal ramifications of the right ventricular specialized conduction system.

Our study extends to man the results of animal experiments in which peripheral forms of RBBB have been produced by interruption of the peripheral conduction system. Moore et al. produced RBBB and delayed activation of the pulmonary outflow tract by sectioning a peripheral segment of the right ventricular Purkinje network through a right atriotomy. The pattern of right ventricular activation delay in these animals is similar to that produced by the endocardial resection in our patients.

Although RBBB commonly occurs after TOF repair, its presence is not invariable. Various series have included 1–5% of patients in whom RBBB was not observed. Krongrad et al. have published pre- and postoperative ECGs in a patient in whom RBBB was not produced by infundibular resection.
and right ventriculotomy. Our observations in patient 14 are similar. Complete RBBB did not occur and electrocardiographic and vectorcardiographic changes were unimpressive. Presumably, infundibular resection as well as ventriculotomy can occasionally avoid disrupting the terminal Purkinje network of the right ventricle such that the ECG is not markedly altered. In our case, the modest terminal delay in outflow tract activation may have resulted from disruption of a local area of the Purkinje network and produced the electrocardiographic changes which are compatible.
FIGURE 7. Epicardial activation data plotted with a lead I QRS complex before (A) and after (B) transatrial repair of tetralogy of Fallot (patient 6). Epicardial activation times of the sites on the left ventricle (solid circles), body of the right ventricle (solid circles) and outflow tract of the right ventricle (open circles) are plotted on a common time axis with a single QRS complex from lead I recorded before (A) and after (B) repair. In A, QRS duration is 62 msec and activation of the outflow tract occurred between 40-58 msec. In B, the QRS duration is 114 msec and right bundle branch block is present. Activation of the sites on the left ventricle and body of the right ventricle was unchanged; however, activation of sites on the outflow tract was delayed 35-56 msec. The sites in the outflow tract are all activated during the slurred S wave of the QRS complex.

with the pattern commonly referred to as “incomplete RBBB.”

We assume that activation of most of the right ventricle is delayed in the usual form of RBBB, a suspicion confirmed in the few studies of ventricular activation in RBBB which have been reported. However, in our patients as well as those reported by Gelband and Krongrad, typical RBBB appeared in association with delayed activation of only a portion of the right ventricle. This difference may be caused,
TABLE 2. Pre- and Postoperative Electrocardiographic Data

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*Measured at paper speed of 250 mm/sec.

The use of catheter endocardial mapping during the routine postoperative evaluation of patients with TOF may therefore identify patients in whom the block is indeed proximal and who may later develop heart block.24, 25

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References

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Retrograde Conduction in the His-Purkinje System

Analysis of the Routes of Impulse Propagation Using His and Right Bundle Branch Recordings

MASOOD AKHTAR, M.D., CAROL J. GILBERT, R.N., CCRN, FRANCIS G. WOLF, M.D.,
AND DONALD H. SCHMIDT, M.D.

SUMMARY  We analyzed routes of retrograde impulse propagation in the His-Purkinje system (HPS) in 24 patients with normal intraventricular conduction. Using intracardiac recordings from the His bundle and right bundle branch (RBB), predetermined paced ventricular cycle lengths were scanned with right ventricular extrastimuli (S2). Electrophysiologic findings suggested that retrograde activation of the HPS bundle after premature stimuli (H2) occurred via the left bundle branch (LBB) in 16 of 24 cases and via the RBB in three of 24 patients. The remaining patients demonstrated retrograde H2 activation via both RBB and LBB at different times during the scanning. In general, a linear relationship existed between retrograde conduction delays in the HPS (i.e., S2H2 intervals) and ventricular coupling intervals (S1S2) when retrograde H2 activation consistently occurred via the same bundle branch. However, we noted sudden and unexpected increases (30–90 msec) or decreases (10–55 msec) in S2H2 intervals in 11 of 24 patients; these changes occurred with and without concomitant changes in the retrograde His-right bundle (HRB) activation sequence. Retrograde H2 activation during right ventricular premature stimulation occurred exclusively through the LBB in a majority (67%) and through the RBB in a minority (12%) of cases. In some patients (21%), however, the route of retrograde H2 activation may vary and could occur via both the LBB and the RBB. Recordings from the HRB area should help to delineate the route of retrograde impulse propagation to the bundle of His.

ATRIAL EXTRASTIMULUS TECHNIQUE has been the standard laboratory method for assessment of drug effects upon the functional properties of the His-Purkinje system (HPS).1-10 Determination of the refractoriness of the HPS in the antegrade direction in man, however, is frequently precluded by longer refractoriness of the atrioventricular (AV) node, particularly after drugs like digitalis and propranolol.4, 9, 10 The ventricular extrastimulus method circumvents the above difficulties, provides a different yet practical approach to this problem, and allows determination of refractoriness of the HPS in almost all patients, albeit in the retrograde direction.11-18 While the ventricular extrastimulus method is simple, it has some inherent limitations, particularly the lack of sufficient electrophysiologic markers. The retrograde His bundle activation during basic drive beats (H2) is generally not recognizable. The His bundle depolarization in response to closely coupled ventricular premature depolarization (H2) is easily identified, but does not provide any insight into the route of retrograde impulse conduction through the HPS, i.e., via the right (RBB) or left bundle branch (LBB) systems. The manner of impulse propagation is fundamentally important for the interpretation of drug effects on the HPS when using the ventricular extrastimulus technique. The present study delineates the routes of impulse propagation to the bundle of His during right ventricular premature stimulation in 24 patients with normal intraventricular conduction.
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