Echocardiographic Assessment of Right Bundle Branch Injury After Repair of Tetralogy of Fallot

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SUMMARY Nineteen patients, ages 3½–18 years, with electrocardiographic evidence of right bundle branch injury after intracardiac repair of tetralogy of Fallot, underwent invasive intracardiac electrophysiologic evaluation 1–13 years (mean 4.4 years) postoperatively. Categorization of the site of right bundle branch injury as proximal or distal was made by determining the V-RVA interval. In 11 of the patients, the V-RVA interval was prolonged (> 35 msec), indicating proximal right bundle branch injury and in the other eight it was normal (< 35 msec), indicating distal bundle branch injury. Within 24 hours of the study, all patients were studied by M-mode echocardiography. Measurements were made of the tricuspid valve closure, mitral valve closure and the difference between the two, or the delta value. All but one patient with distal bundle branch injury had delta values of less than 40 msec (range 8–38 msec), while 10 of 11 patients with proximal bundle branch injury had delta values greater than 40 msec (range 41–116 msec). There was a significant positive correlation (r = 0.74, p < 0.001) between V-RVA and the delta value. We conclude that the delta value is an indicator of relative activation delay of the right ventricle, and therefore, in most cases, proximal vs distal bundle branch injury can be diagnosed noninvasively.

RIGHT BUNDLE BRANCH BLOCK is the most common electrocardiographic feature in children who have undergone complete surgical repair of tetralogy of Fallot.1 However, the surface electrocardiographic pattern of right bundle branch block may result from injury to the proximal right bundle branch, as a result of ventricular septal defect closure,2,3 or might be secondary to interruption of the distal Purkinje fiber network at the time of the ventriculotomy4 or infundibulec- tomy,6 leaving the proximal portion of the right bundle intact. Because a lesion of the proximal main right bundle branch would theoretically leave the patient vulnerable to develop complete atrioven- tricular (AV) block should compromise of the left bundle branch occur, the differential diagnosis of proximal vs distal bundle branch injury is prognosti- cally important.

Sung et al.7 suggested that the most sensitive method of defining the site of right bundle branch injury is to measure the V-RVA interval. This interval, which is a measurement of the activation time of the apex of the right ventricle (normally the earliest site of right ventricular activation) relative to the activation of the left ventricle,8,9 would be expected to be prolonged in proximal right bundle branch injury.7 The present study was undertaken to determine whether relative activation delay of the right ventricle could also be assessed noninvasively by the echocardiographically determined relationship between the tricuspid and mitral valve closures (Tc and Mc).

Material and Methods

Study Group (table 1)

Nineteen patients, ages 3½–18 years (mean 7.9 years), were studied 1–13 years (mean 4.4 years) after complete surgical repair of tetralogy of Fallot performed via a right ventriculotomy. Preoperative ECGs revealed a left anterior hemiblock in one patient (patient 9) and the absence of right ventricular conduction defects in all patients. Postoperatively, all patients had electrocardiographic evidence of right bundle branch injury on the surface ECG as evidenced by either a complete right bundle branch pattern (18 patients) or by right ventricular activation delay (delayed S wave in lead I and V6 without prolongation of the QRS beyond normal values) in one patient. Additional conduction defects included electrocardiographic evidence of a left anterior hemiblock pattern in seven patients.

All patients underwent cardiac catheterization after sedation with meperidine HCl, promethazine HCl and chlorpromazine HCl after obtaining appropriate informed consent. Residual hemodynamic lesions and right ventricular pressures are summarized in table I. After the hemodynamic portion of the study, all patients underwent intracardiac electrophysiologic investigation. Surface electrophysiographic leads I, II and III were recorded simultaneously with bipolar recordings of the high right atrial electrogram, His bundle electrogram and right ventricular apex electrogram. Fluoroscopic guidance of the electrode tip was used to record the right ventricular apex electrograms. The intracardiac electrophysiologic data was displayed on an oscilloscopic recorder (Electronics for Medicine DR-8) filtered at 40–500 Hz, and permanently recorded at a paper speed of 100 mm/sec. Basic electrophysiologic intervals (PA, AH, HV) were measured as previously described.11 The V-RVA interval was measured as the interval between

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the earliest deflection of the QRS complex on the surface ECG to the first fast deflection of the bipolar right ventricular apex electrogram (fig. 1A). Normal values for these intervals in pediatric patients in our laboratory are PA 3–54 msec, AH 51–107 msec, HV 25–53 msec and V-RVA 5–35 msec.

While the upper limits of normal of V-RVA in small infants with very fast heart rates have not been established, investigations in our laboratory indicate that between ages 6 months and 18 years (encompassing all of the patients in the study group), the V-RVA increases only slightly (mean 4 msec). The designated upper limit of normal for V-RVA in this study (35 msec) represents more than 2 standard deviations above the mean throughout childhood.

Within 24 hours of the invasive electrophysiologic investigation, all patients underwent M-mode echocardiographic examination. Recordings were taken of the mitral and tricuspid valves at a paper speed of 100 mm/sec using a Unirad ultrasonoscope and a pediatric ultrasound transducer. Lead II of the surface ECG was simultaneously recorded. Care was taken to obtain the best possible definition of the closure point of the mitral and tricuspid valve leaflets, by defining both anterior and posterior leaflets of the AV valve. Recordings at a paper speed of 50 mm/sec were also obtained, which sometimes aided in identifying the precise point of AV valve closure. Caliper measurements, to the nearest 5 msec, were made of the point of Tc, defined as the interval between the Q wave on the surface ECG to the point of the tricuspid valve closure, Mc, defined as the interval between the Q wave on the surface ECG to the point of mitral valve closure, and the difference between the two (Tc−Mc), referred to as the delta value (fig. 1B). At least three measurements were performed and averaged for each determination to correct for possible respiratory variation in the measurements. For 75% of the determinations, the maximum beat-to-beat variation in the AV valve closure was < 10 msec, however, variations as large as 25 msec were encountered. In cases with variations greater than 10 msec, at least six closures were measured to obtain an average value. Standard regression analysis and two-tailed t tests were used for statistical purposes. Before performing a t test, a variance ratio (F) was calculated using Snedecor’s table for the variance ratio.14 For data pairs with significantly different (p < 0.05) standard deviations, t tests were performed after a standard computer program (Tektronix 4051) for adjusting the degrees of freedom was applied.

**Echocardiographic Control Group**

To establish normal values for the echocardiographic measurements in our laboratory, we studied 19 children, ages 9 months to 18 years,
referred for evaluation of “innocent murmurs.” All
patients had normal ECGs without evidence of con-
duction disturbances. Determinations were made of
Tc, Mc and delta values. The mean Tc was 69 ± 11
msec and the mean Mc was 49 ± 9 msec. The
observed mean delta value for the control group was
21 ± 12 msec, which is in close agreement with
previously published values in normal children.
While the smallest delta values (< 15 msec) were
observed in six patients 3–6 years of age, the remain-
ing 13 patients (including two infants) had delta values
greater than 15 msec, which had no apparent ten-
dency to increase with age. This finding has been pre-
viously reported.

Results

Intracardiac Electrophysiologic Data (table 2)

Complete evaluation of the basic electrophysiologic
intervals could be obtained in 16 of the patients. In
patient 17, only the V-RVA interval could be obtained
(the His bundle was not identified), and in two other
patients the low amplitude of the P wave on the sur-
face ECG precluded measurement of the PA interval.
For the study group, the PA interval ranged from
0–40 msec and the AH interval ranged from 40–100
msec. All of these values are within normal limits.
Prolongation of the HV interval (> 53 msec), indi-
cative of abnormal conduction within the His-
Purkinje system, was detected in four of the patients
(patients 3, 4, 15 and 16).

The V-RVA intervals for the group ranged from
3–84 msec. In 11 of the patients the interval was
prolonged, indicating the presence of proximal right
bundle branch injury. Eight patients had a normal V-
RVA interval, indicating the presence of injury to the
distal ramifications of the right bundle branch with
preservation of a normal activation time of the apex of
the right ventricle.

Relationship between V-RVA Interval,∗
Hemodynamic and Electrocardiographic Findings

A prolonged V-RVA interval was found in three
patients with a right ventricular systolic pressure of
less than 40 mm Hg and in eight patients with a right
ventricular systolic pressure greater than 40 mm Hg.
Although a prolonged V-RVA interval occurred more
frequently (73%) in the patients with a high right ven-
tricular pressure, the longest V-RVA interval in the
study group (84 msec) was in patient 16, whose right
ventricular pressure was only 26/8 mm Hg. Further-
more, normal V-RVA intervals were recorded in
patients with strikingly elevated right ventricular
pressures (patients 2 and 3). Therefore, in our
patients, right ventricular pressure alone was a poor
indicator of the functional status of the right bundle
branch system.

The documentation of injury to the His bundle or
distal conduction system (manifest as a prolonged HV
interval) was not predictive of a prolonged V-RVA in-
terval. Of the four patients with prolonged HV inter-

Figure 1. (A) Surface electrocardiographic leads I, II, and III are
simultaneously recorded with the bipolar right ventricular electrogram (RVA) from
patient 18. The V-RVA interval is measured as the interval from the initial deflection of
QRS complex (V) on the surface ECG to the initial fast deflection of the right ventricular
electrogram (arrow). Normal values are less than 35 msec. The V-RVA interval in this
patient is 60 msec, which is prolonged and indicates proximal right bundle branch
injury. (B) Echocardiographic recording of the tricuspid and mitral valves from the same
patient. Electrocardiographic lead II is simultaneously recorded. The tricuspid
valve closure (Tc) and mitral valve closure (Mc) are measured from the onset of the
QRS complex on the surface ECG to the point of respective valve closure. The delta
value (defined as Tc–Mc) is 70 msec.
TABLE 2. Electrophysiologic Data

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Range 0-30 40-100 35-85 3-84

Mean ± sd 22 ± 10 69 ± 18 47 ± 13 40 ± 22

*Normal V-RVA.

Abbreviations: PA = internal from P wave on surface electrocardiogram to atrial electrogram of His bundle recording catheter; AH = interval from atrial electrogram to His bundle electrogram; HV = interval from His bundle electrogram to onset of QRS; V-RVA = interval from onset of QRS in surface electrocardiograms to right ventricular apex electrogram.

vals, two had normal V-RVA intervals and two had prolonged intervals.

The total QRS duration measured by the surface ECG was not related to the degree of prolongation of the V-RVA interval (fig. 2). Hence, this parameter was of little value in determining whether proximal or distal right bundle branch injury was present.

Echocardiographic Data (table 3)

The mean timing of Mc occurred somewhat later in the control echocardiographic group compared with the postoperative group (49 ± 9 msec vs 38 ± 20 msec, p < 0.05). The Tc was delayed in the study group compared with the control group (84 ± 23 msec vs 69 ± 11 msec, p < 0.02) with a wider variance encountered in the study group (p < 0.004). The tendency toward a more delayed point of Tc relative to Mc in the postoperative patients resulted in a prolongation of the delta value to 46 ± 23 msec in the postoperative group, compared with the control value of 21 ± 12 msec (p < 0.001). The range of delta values in the postoperative group was wide, 8-117 msec.

![Figure 2](http://circ.ahajournals.org/)

**Figure 2.** Plot of the V-RVA interval as a function of the total QRS duration as determined by the surface ECG in 19 patients with right bundle branch injury after surgical repair of tetralogy of Fallot. There is a poor correlation (r = 0.06) between the QRS duration and the V-RVA interval.

**Relationship between Tc, Mc, Delta Value and V-RVA Interval**

Within the wide range of delta values, two groups of patients were identified (fig. 3). All but one patient (no. 8) with V-RVA intervals of less than 35 msec had delta values less than 40 msec, while all but one patient (no. 19) with prolongation of the V-RVA interval had delta values greater than 40 msec. Hence, the delta value appeared to discriminate between those with prolonged and normal V-RVA intervals (sensitivity 91%; specificity 88%).

To determine whether the absolute value of Tc alone was predictive of a prolonged V-RVA interval, this variable was plotted graphically as a function of the V-RVA interval (fig. 4). While the patients with prolongation of the V-RVA interval tended to have greater Tc values, the correlation was not sufficient to discriminate in many instances between the patients with normal and prolonged V-RVA intervals.

When the delta value was plotted as a function of the V-RVA interval (fig. 5), a significant positive correlation was observed (r = 0.74, p < 0.001) between these two variables, with the delta value more closely reflecting the V-RVA interval and, therefore, a better indicator of proximal vs distal bundle branch injury.

As in the case of V-RVA interval, the delta value could not be predicted by the postoperative right ven-
TABLE 3. Echocardiographic Data

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Range 43-145  (−) 13-68  8-116

Mean ± sd 84 ± 23  38 ± 20  46 ± 23

*Patients with normal V-RVA.

Abbreviations: Tc = tricuspid valve closure; Mc = mitral valve closure.

tricuscular pressure. While patients with prolonged delta values were more likely to have a right ventricular systolic pressure > 40 mm Hg, we did observe two patients with normal delta values and markedly elevated right ventricular pressures (patients 2 and 3).

Discussion

Experimental animal work14, 15 and observations in human patients2-6 show that the electrocardiographic pattern of right bundle branch block may result from injury to the right bundle branch system from the differentiated portion of the His bundle, to the distal ramifications of the right ventricular Purkinje fiber network. This might explain why published accounts of the prognosis of patients with various electrocardiographic conduction defects after repair of tetralogy of Fallot have been seemingly disparate.1, 16, 17 Patients now believed to be at risk to develop late postoperative complete AV block are those who have sustained injury to the proximal portions of the specialized conduction system. Although the appearance of left-axis deviation postoperatively on the surface ECG implies proximal injury to the anterior division of the left bundle branch, an associated right bundle branch block pattern need not implicate associated injury to the proximal portion of

FIGURE 3. Delta values of 19 postoperative patients grouped according to whether the V-RVA interval is normal (<35 msec) or prolonged (>35 msec). The delta value is significantly prolonged (p < 0.009) in patients with prolonged V-RVA intervals.

FIGURE 4. Plot of the V-RVA interval as a function of the tricuspid valve closure (Tc) in 19 postoperative patients. The correlation coefficient (r = 0.58, p < 0.05) suggests patients with prolonged V-RVA intervals tend to have delayed Tc.
difficulties exist with the electrocardiographic method.

Electrocardiographic analysis in children of the relationship between the tricuspid valve closure and mitral valve closure in a variety of conditions, including right bundle branch block, has been reported. Milner et al. reported data in 25 patients with right bundle branch block (13 patients postoperatively) and found that all but three patients had delta values less than 50 msec. These authors did not offer an explanation for the observed delay in the three patients. They concluded that right bundle branch block has little effect upon Tc and, therefore, the delta value. However, delta values greater than 40 msec were observed more often in the right bundle branch block group than in their control group (46% vs 13%). This would suggest that right bundle branch block does result, in some patients, in prolongation of the delta value. More recently, Brooks et al. studied 20 patients with right bundle branch block with echocardiographic and phonocardiographic techniques. These authors described two groups of patients with right bundle branch block: those with increased delta values and wide splitting of the first heart sound (eight patients) and those with normal delta values, and single first heart sounds by phonocardiography (10 patients). These investigators suggested that the delayed tricuspid component of the first heart sound and prolonged delta value in the first group might result from global activation delay of the right ventricle, and hence, represent proximal right bundle branch injury, while the finding of a single first heart sound and normal delta value would suggest distal bundle branch injury.

Our observations confirm this hypothesis by demonstrating a significant correlation between the delta value and the invasively determined measurement of relative right ventricular activation delay, the V-RVA interval. In the presence of sinus rhythm, and in the absence of extreme prolongation of the PR interval, ventricular excitation is believed to be primarily responsible for completing AV valve closure. Hence, while hemodynamic influences such as ventricular compliance and diastolic flow characteristics certainly influence AV valve closure, the relationship of AV valve closure is, in part, a mechanical expression of the electrical sequence of ventricular depolarization. Our findings indicate that in proximal right bundle injury delayed activation of the major portion of the right ventricle results in a detectable delay in Tc, while in distal bundle branch injury, preservation of normal conduction to the apical region, papillary muscles and comparatively larger portions of right ventricular free wall and septal myocardium results in a more normal Tc relative to Mc. We suggest, therefore, that the site of right ventricular conduction delay can be accurately and noninvasively determined by the echocardiographic measurement of the delta value. Further investigation is required to discern the effects of severely elevated end diastolic ventricular pressures and ventricular dysfunction upon the delta value, but
this approach affords a relatively simple, safe and widely available method of identifying patients with proximal right bundle branch injury, who may be at higher risk in the future for developing more advanced degrees of disturbance of AV conduction.

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References

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