Reversible Complete Heart Block following Cardiac Surgery

Analysis of His Bundle Electrograms

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SUMMARY
His bundle electrograms were recorded before and throughout the postoperative course in two children who developed temporary complete heart block after surgical correction for tetralogy of Fallot. In one the complete heart block was associated with prolonged duration of the QRS complex, and in the other there was no prolongation of the QRS complex. His bundle electrograms in the patient with widened QRS identified the site of block to be between the low atrium and the His bundle, and in the patient with a QRS of normal duration the block was distal to the His bundle. Complete heart block disappeared within 3 weeks in both patients. Subsequent His bundle studies showed normal A-V conduction in the first patient except for the presence of persistent complete right bundle-branch block. In the other, prolongation of the P-R interval was persistent, and atrial pacing at high rates produced a Wenckebach type of block distal to the His bundle. These studies indicate that electrophysiologically the site of complete heart block following definitive intracardiac surgery can occur at multiple sites. Furthermore, in complete heart block the site of the pacemaker may not be accurately predicted from routine electrocardiograms alone, and His bundle recordings may be necessary to clarify this site.

Additional Indexing Words:
Wenckebach Tetralogy of Fallot Interventricular conduction

STUDIES which identify the sites of electrophysiologic delay and/or complete A-V block following open-heart surgery remain scanty. Recently Rosen et al.1 demonstrated in two postoperative patients, who developed complete heart block with widened QRS complexes, that the block was distal to the His bundle. We would like to report two patients who, following surgery for tetralogy of Fallot, developed complete heart block which reverted to 1:1 A-V conduction after 1–3 weeks duration. His bundle electrograms were recorded preoperatively and throughout their postoperative courses. The analysis of these electrograms demonstrated that the site of complete block in one patient was between the low atrium and the His bundle, and in the other it was between the His bundle and the peripheral ventricular conduction system. In the first patient the QRS was prolonged with a configuration of right bundle-branch block; in the other patient with the complete block distal to the His bundle, the QRS was normal. Following the return to normal A-V conduction, the first patient had a normal response to pacing the atrium at high rates, while the second patient had an abnormal response with

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the development of a Wenckebach type of block distal to the His bundle in the ventricular conduction system. It was evident that the His bundle electrograms were necessary to accurately localize the site of complete heart block and, in addition, provided evidence of electrophysiologic abnormalities following return to 1:1 A-V conduction.

**Methods**

Two 8-year-old children with tetralogy of Fallot had cardiac catheterization under Demerol sedation (2 mg/kg im). The method of Scherlag et al.\(^2\) was used to record from the A-V conduction system. His bundle and right bundle bipolar electrograms were recorded between two electrodes 1 cm apart with a no. 6 Elecath Damato hexapolar catheter and an A-C Princeton preamplifier model 113. The output of the preamplifier went to a Sanborn Polybeam oscillographic and tape-recorder system. The frequency response of the entire system was flat between 30 and 1000 Hz. The electrograms were recorded simultaneously with lead II. Postoperatively, right atrial pacing was conducted at rates up to 180 beats/min using a Medtronic battery-powered pacemaker.

One assumes in humans that rapid deflections recorded from the anatomic region of the His bundle which occur at a time not coincident with either atrial or ventricular muscle excitation are generated by currents from excitation waves in the distal A-V node, His bundle, or proximal bundles. This assumption seems reasonable on the basis of the work of Alanis, Gonzalez, and Lopez\(^3\) who originally defined A-H and H-V intervals from electrograms recorded in isolated dog hearts, and on the additional information provided by a comparison of the intracellular and extracellular waveforms of the A-V node and specialized ventricular conduction system.\(^4\) \(^5\) Therefore, with this technic the waveforms ascribed to the His bundle are those which could be produced by currents from the distal A-V node, His bundle, or proximal bundle branches.

The interval from the time of the initial sharp downward deflection of the atrial waveform to that of the rapid deflection between the maximum and minimum of the His bundle waveform (A-H interval) was considered to estimate the A-V nodal conduction time.\(^6\) The time from His bundle excitation to the onset of ventricular activation (H-V interval) was measured from the rapid deflection of the His bundle waveform to the onset of the ventricular deflection. Our experience in children has shown that a wide variety of atrial waveforms are recorded in the vicinity of the His bundle; particularly, shape changes may occur during a single run without apparent shifts in the catheter position. Such shape changes make it difficult to select a precise point on the atrial electrogram which represents the same instant during atrial activation on a beat-to-beat basis. This variability in the atrial waveform results in a timing variation for the A-H interval of ±10 msec. In contrast, the accuracy in the timing of the His bundle deflection to the onset of ventricular excitation (H-V interval) and the right bundle-branch excitation time to ventricular activation onset was estimated to be within ±2 msec.

**Results**

**Case 1**

This 8-year-old boy was followed from infancy for tetralogy of Fallot at Duke University Hospital. The preoperative electrocardiogram (fig. 1A) revealed no apparent abnormalities of the P waves; the P-R interval was 0.12 sec, and the QRS indicated right ventricular hypertrophy. At cardiac catheterization the His bundle electrograms were normal (fig. 1B). The A-H interval was 60 msec, and the H-V interval was 41 msec.

At definitive surgical repair a vertical right ventriculotomy was performed, an infundibullectomy was done, and the ventricular defect

![Figure 1](http://circ.ahajournals.org/)

**Figure 1**

Preoperative electrocardiogram and His bundle electrogram in patient 1. The electrocardiogram (A) showed right ventricular hypertrophy, normal P waves, and a normal P-R interval of 0.12 sec. The QRS duration was 0.65 sec. In (B) the His bundle electrogram (HBE) along with the lead II electrocardiogram indicated normal A-V conduction as evidenced by an A-H interval of 60 msec and an H-V interval of 41 msec. The heart rate was 110 beats/min. A = atrial muscle excitation; H = His bundle deflection; V = onset of ventricular activation.

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was closed with a Teflon patch by continuous suture. After the defect was closed, the patient was noted to be in complete heart block. Pacing wires were implanted in the epicardium of the right ventricle, and the patient was returned to the recovery room in good condition. He continued to do well and maintained a heart rate between 70 and 96 beats/min; however, during sleep, there were transient episodes of bradycardia (heart rate 50 beats/min), and these were easily controlled with a demand pacemaker.

The electrocardiogram (fig. 2A) indicated complete heart block with an atrial rate of 125 beats/min and a ventricular rate of 75 beats/min. The QRS complex was widened, and its configuration was that of complete right bundle-branch block.

Cardiac catheterization was performed 1 week postoperatively. Hydrogen electrode curves revealed no intracardiac shunts, and the right ventricular systolic peak pressure was 32 mm Hg. His bundle electrograms (fig. 2B) revealed A-V block with dissociation between the atrial and His bundle waveforms; each ventricular beat was preceded by a His bundle potential with a constant H-V interval of 30 msec. Despite considerable attempts at repositioning the electrode catheter, neither a right bundle potential nor a double deflection from the His bundle, e.g. "split BH" waveform, could be detected.

Two weeks after the postoperative cardiac catheterization complete heart block disappeared, and the rate increased to 120 beats/min. The electrocardiogram (fig. 3A) then demonstrated a P-R interval of 0.16 sec with complete right bundle-branch block.

**Figure 2**

Electrocardiogram and His bundle electrogram during postoperative complete heart block in patient 1. The electrocardiogram (A) indicated A-V dissociation with widening of QRS. The QRS pattern was that of complete right bundle-branch block (QRS duration 0.12 sec). In (B) lead II and the His bundle electrogram showed that there was complete block of conduction between the atrium and the His bundle. Each QRS complex was preceded by His bundle depolarization with an H-V interval of 30 msec. The sawtooth marks indicate that the tracing was continuous. The second His bundle deflection is followed by an atrial complex superimposed on the initial portion of ventricular excitation. Atrial rate was 125 beats/min; ventricular rate was 75 beats/min.
Figure 3

Electrocardiogram and His bundle electrogram after return to normal sinus rhythm in patient 1. The electrocardiogram (A) indicated a normal P-R interval (0.16 sec) and complete right bundle-branch block (QRS duration 0.12 sec). In (B) the His bundle electrogram (HBE) indicated normal A-V conduction with an A-H interval of 65 msec and a H-V interval of 31 msec. Right atrial pacing (C) at a rate of 180 beats/min demonstrated a normal A-V conduction response with prolongation of the A-H interval to 150 msec with no change in the H-V interval of 30 msec. S = pacemaker stimulus artifact; additional abbreviations same as in figure 1.

(QRS duration 0.12 sec). Two months later His bundle electrograms (fig. 3B) demonstrated an A-H interval of 65 msec (60 msec preoperatively) and H-V interval of 31 msec (41 msec preoperatively) during normal sinus rhythm with a rate of 110 beats/min. The high right atrial wall was paced at a rate up to 180 beats/min (fig. 3C) with maintenance of 1:1 A-V conduction. At a rate of 180 beats/min there was prolongation of the A-H interval from 65 to 150 msec while there was no change in the H-V interval (30 msec). Prolongation of the A-H interval of this amount in response to atrial pacing at a rate of 180 beats/min is within normal limits for our laboratory.

Case 2

The second patient was 8 years old and had tetralogy of Fallot. The electrocardiogram demonstrated right ventricular hypertrophy, normal P waves, and a P-R interval of 0.16 sec.

At cardiac catheterization the His bundle electrogram (fig. 4B) revealed normal A-V conduction with an A-H interval of 82 msec and an H-V interval of 49 msec. At corrective surgery a vertical right ventriculotomy was performed through which the ventricular septal defect was closed with a Teflon patch by a continuous suture. The infundibulum was resected, and a Dacron patch was used to
form a roof for final closure of the ventriculotomy. The patient did well in the operating room and maintained a heart rate of 96 beats/min; however, on arrival in the recovery room he was noted to have complete heart block with an atrial rate of 155 beats/min and a ventricular rate of 85 beats/min. The patient’s condition remained stable, the ventricular rate remained unchanged, and the QRS duration was 0.07 sec. Cardiac catheterization on the sixth postoperative day revealed a right ventricular peak systolic pressure of 28 mm Hg, and hydrogen electrode curves showed no shunt.

The electrocardiogram (fig. 5A) at that time showed complete heart block with an atrial rate of 134 beats/min and a ventricular rate of 85 beats/min. The QRS duration was normal (0.07 sec) with an incomplete right bundle-branch-block pattern (rsR’ in lead V1). The His bundle electrogram (fig. 5B) showed complete block of conduction between the His bundle and peripheral ventricular conduction system. The A-H interval was prolonged (240 msec) with a His bundle deflection after every atrial waveform. Although there was complete dissociation between the His bundle waveforms and the ventricular complexes, there was a right bundle potential preceding each QRS with a normal RB-V interval of 14 msec (fig. 5C). Atropine (0.4 mg i.v.) produced an increase in atrial rate from 130 to 150 beats/min with a shortening of the A-H interval from 240 to 210 msec. There was no effect on the ventricular rate of 82 beats/min.

Two days following the cardiac catheterization, complete heart block disappeared. The electrocardiogram now demonstrated a heart rate of 97 beats/min with a P-R interval of 0.30 sec and a QRS duration of 0.07 sec. The first-degree heart block persisted although the P-R interval was noted to gradually decrease to 0.25 sec (fig. 6A). Two months later His bundle electrograms (fig. 6B) revealed an A-H interval of 120 msec, an interval shorter than that measured during complete heart block. The H-V interval was 58 msec, a value greater than the abnormally long preoperative H-V interval of 49 msec. The high right atrial wall was paced at increasing rates, and varying A-V block occurred when the pacing rate reached 160 beats/min (fig. 6C). Although there was varying A-V block, the A-H interval remained rather constant. The varying block was due to a Wenckebach type phenomenon distal to the His bundle. After each dropped QRS complex, the H-V interval achieved its shortest value of 58 msec; this value was the same as that measured during normal sinus rhythm and was associated with a QRS configuration in lead II similar to that recorded during the control state. Thereafter, each subsequent beat was associated with the development of an aberrant QRS complex and with an increasingly longer H-V interval until A-V conduction failed distal to the His bundle.

Thus far, both of our patients have maintained 1:1 A-V conduction for 1 year since the disappearance of the complete heart block.

Discussion

Perhaps one of the most intriguing findings of the study was that the duration of the QRS during complete heart block was of no value.
Postoperative electrocardiogram and His-bundle electrograms during complete heart block in patient 2. The electrocardiogram (A) indicated complete heart block with a QRS of normal duration (0.07 sec). In (B) lead II indicated complete heart block with a ventricular rate of 82 beats/min and an atrial rate of 130 beats/min. The accompanying His bundle electrogram revealed a His bundle deflection after each atrial depolarization complex; however, the A-H interval was prolonged (240 msec). There was complete dissociation between the His bundle complex and the ventricular complex, indicating that the site of block was between the His bundle and distal ventricular conduction system. The recording shown in (C) was made after the catheter was positioned in the right ventricular cavity. This showed a normal time relationship between excitation of the right bundle branch and the onset of ventricular activation. These data, along with those presented in the next figure (fig. 6), indicated that the right bundle branch was not interrupted at surgery, although a large part of the infundibulum was resected.

Gelband et al.8 state that right bundle-branch block (RBBB) invariably follows total surgical correction of tetralogy of Fallot, and, in their patients, an average increase of 39 msec in QRS duration occurred in all patients. They interpreted the RBBB to be due to the vertical right ventriculotomy rather than due to surgical damage to the main right bundle branch. Our second patient, who had a vertical right ventriculotomy, showed no postoperative RBBB as they define it, since there was no increase in duration of the QRS.
Figure 6

Electrocardiogram and His bundle electrogram after recovery from postoperative complete heart block in patient 2. The electrocardiogram (A) shows a normal QRS duration (0.07 sec), and rs' pattern in lead V₁, and first-degree heart block (P-R interval 0.25 sec). The His bundle electrogram in (B) indicates an A-H interval of 120 msec and a prolonged H-V interval of 58 msec. Atrial pacing produced variable A-V block (C) when the rate reached a value of 160 beats/min. During this period of variable A-V conduction, note that the A-H interval was prolonged but remained rather constant while the H-V interval showed cyclic changes of increasing H-V-interval duration until conduction failed distal to the His bundle. Associated with this variable block were changes in the shape of the QRS complex (lead II). Following each dropped beat, the shortest H-V interval (58 msec) was associated with a QRS complex in lead II, similar to that of the baseline state shown in (B). The subsequent beats have an increasing prolongation of the H-V interval with an accompanying aberrant QRS complex in lead II.

Postoperatively. In our opinion, this patient indicates that the genesis of the postoperative RBBB can be uncertain since a vertical right ventriculotomy does not always produce RBBB in tetralogy of Fallot.

In the second patient, the site of complete block was distal to the bundle of His, yet the QRS duration was normal with a normal RB-V interval. Two separate His bundle deflections were recorded by Rosen et al. in a patient with complete heart block; one followed each atrial complex and the other preceded each ventricular complex. We found no such deflections in this patient. Therefore, whether the ventricular pacemaker was located in the branching portion of the His bundle or high in one of the bundle branches remains unknown.
HEART BLOCK FOLLOWING SURGERY

The ability of excitation waves to propagate from one bundle branch to the other via the interconnections at the bundle of His has been demonstrated recently in an in vitro dog preparation.\(^1\) In order to demonstrate in patients the activation sequence in the bundle branches and their interconnections at the branching portion of the bundle of His, simultaneous recordings from right and left bundle branches are necessary. Such simultaneous multiple recordings will be necessary to pinpoint pacemaker sites if they are located in the proximal portion of the bundle branches.

Lev et al.\(^1\) have shown that, in patients undergoing surgery for ventricular septal defect, injuries can occur at multiple sites, i.e., the SA node, the A-V-node area, His bundle, and bundle branch system. Our two patients illustrate an analogous electrophysiologic finding in that the site of complete block can occur either between the atria and His bundle or between the proximal His bundle and the peripheral conduction system. The specific type of damage incurred in our patients remains speculative; however, return to 1:1 A-V conduction indicates that the site of block was not the determining factor in the reversibility of the conduction. However, since his return to normal sinus rhythm the patient with the more distal A-V conduction lesion has persisted with prolongation in both the time of transmission from atrium to His bundle and the time from His bundle to the ventricle. In addition, atrial pacing produced an unusual type of block distal to the His bundle (Wenckebach phenomenon in the specialized ventricular conduction system).

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**References**


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