Sinus Node Function and Conduction System after Complete Repair of Tetralogy of Fallot

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SUMMARY
Twenty-three children underwent electrophysiological studies during routine postoperative catheterization two months to five years after complete correction of tetralogy of Fallot. The aim of the study was to investigate the whole conduction system, including sinus node function, using His bundle recordings and atrial pacing.

H-V intervals were normal at rest and with pacing in twenty-two patients, including four patients with evidence of bifascicular block on the surface ECG. One patient with cardiomegaly and evidence of diffuse myocardial damage had a prolonged H-V interval but did not develop a block at this level during pacing.

A-H interval was slightly prolonged in four patients and normal in all others.

The threshold of pacing-induced atrioventricular block ranged from 75 to 240/min and was somewhat age dependent $(r = -0.35)$. Two patients fell below the 95% confidence limit of this regression and are considered abnormal. One returned to normal after intravenous injection of atropine.

Corrected sinus node recovery time ranged from 60 to 2000 msec. Three patients had values above 500 msec which are considered abnormal. These patients had other minor signs of sinus node dysfunction, i.e., episodes of sino-atrial block at rest or intermittent sinus bradycardia.

Thus, while the His Purkinje system performed satisfactorily in all patients, sinus node dysfunction and A-V node dysfunction were demonstrated in a few patients after correction of tetralogy of Fallot.

OPEN HEART SURGERY in congenital heart disease carries a risk of damage to the conduction system at various sites, impairment of the His-Purkinje system being the most widely recognized hazard. While the incidence of early postoperative permanent complete heart block (CHB) has been reduced to approximately 1%, late CHB has been observed increasingly in recent years. It has been suggested that an electrocardiographic pattern of right bundle branch block (RBBB) with left axis deviation (LAD), especially if associated with a history of transient CHB in the immediate postoperative period, increases the risk of late CHB. However, the His-Purkinje system is not the only part of the conduction system at risk, and clinical and electrophysiological evidence of sick sinus syndrome after intracardiac surgery has been reported.

This study has therefore been undertaken to assess the whole conduction system as well as the sinus node function in an unselected group of children after complete repair of tetralogy of Fallot, using His bundle recordings and atrial pacing.

Material and Methods

Patients
The study group included 23 unselected children who underwent catheterization 2 months to 5 years (mean 8 months) after complete repair of tetralogy of Fallot (21 patients) or double outlet right ventricle with pulmonary stenosis (2 patients). An atrial septal defect (ASD) was associated in seven. Their ages ranged from 2 to 22 yr (mean 9.6 yr). Before operation all children were in sinus rhythm and presented no atrioventricular (A-V) conduction defect. The clinical and electrocardiographic data are summarized in table 1.

A control group of eight children with tetralogy underwent electrophysiological studies preoperatively, most without pacing because of the risk of hypoxic spells. Three of these children had an associated ASD. Their ages ranged from 3½ to 13½ yr (mean 9.1 yr). The clinical and electrocardiographic data are summarized in table 2.

Technique
All studies were done under light sedation with a mixture of pethidine, promethazine and chlorpromazine, and before any angiograms were taken.

The technique of Scherlag et al. was used to obtain recordings of the A-V conduction system. Bipolar and tripolar electrode catheters (USCI) were used, and recordings were made on a standard 3-channel electrocardiographic writer (Mingograf 34, Elema-Schönder) equipped with a preamplifier. Paper speed was 100 mm/sec.

A standard lead I electrocardiogram and a high atrial electrogram from the stimulating intracardiac electrode, were recorded simultaneously with the His bundle electrogram (HBE).

Atrial pacing was performed with a second electrode.
catheter. Care was taken to place the tip at the junction of the right atrium and superior vena cava, close to the sinoatrial node (SA node), in order to avoid bias for P-A and A-H intervals to and make sure of SA node depolarization, even in the presence of intraatrial conduction defects.

Three modes of pacing were used in every patient: first, pacing with continuously increasing stimulus frequency until Mobitz type I second degree A-V block developed, or until an atrial rate of 240/min with 1:1 conduction was achieved. Secondly, the pacing rate was increased stepwise by increments of 10 beats/min, with short interruptions, over the same range. Finally continuous pacing was performed for 2 min at 130/min. The two latter modes were used to determine sinus node recovery time.

Measurements

The following intervals were measured at rest and during atrial pacing.

P-A: (intraatrial conduction time). From the earliest onset of the P wave on the standard ECG, or the A wave on the high atrial electrogram, to the onset of the A wave on the HBE.

A-H: (conduction time through the A-V node). From the onset of the A wave on the HBE to the first rapid deflection of the His bundle potential.

H-V: (conduction time from the HB to the ventricular septum). From the first rapid deflection of the HB potential to the earliest activation of the ventricle in either the surface or the intracardiac electrograms.

The threshold of pacing-induced second degree A-V block (PAVB) was defined as the lowest atrial stimulation rate at which second degree A-V block developed, independently of the mode of pacing. The site and the type of second degree A-V block was observed.

Sinus node recovery time, or post pacing depression of the SA node, was measured from the last paced A wave to the onset of atrial activity apparently due to SA node discharge.

The corrected sinus node recovery time (CSRT) was obtained by subtracting the mean of three P-P intervals preceding pacing from the SA node recovery time; the values given here are the longest CSRT intervals measured for each patient.

Pharmacological Tests

Three patients with abnormal sinus node or A-V node function were given 0.5-1 mg of atropine intravenously and the electrophysiologic studies were repeated.

Results

Postoperative conduction defects and arrhythmias as seen on the surface ECG appear in Table 1. There was no case of permanent CHB, either early or late postoperatively. Transient CHB was seen in three patients and lasted from 1 hour to 2½ days.

Six children had complete RBBB with LAD, in two of whom LAD was transient, lasting 2 weeks and 4 weeks respectively. At the time of study, 14 children showed complete RBBB and five had no conduction defect at all on surface ECG.

During investigation 19 children were in sinus rhythm and one was in A-V dissociation with in-
Table 2

Control Group

<table>
<thead>
<tr>
<th>Case no.</th>
<th>Age (yr)</th>
<th>Sex</th>
<th>Cardiovasc. diagnosis</th>
<th>Medication</th>
<th>Clinical data</th>
<th>Electrophysiological data</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>At rest</td>
<td>At rest</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Rhythm</td>
<td>PA (msec)</td>
</tr>
<tr>
<td>1</td>
<td>6</td>
<td>M</td>
<td>TF</td>
<td>Propranolol</td>
<td>sinus</td>
<td>25</td>
</tr>
<tr>
<td>2</td>
<td>12</td>
<td>M</td>
<td>TF</td>
<td></td>
<td>sinus</td>
<td>15</td>
</tr>
<tr>
<td>3</td>
<td>3½</td>
<td>M</td>
<td>TF, ASD</td>
<td></td>
<td>sinus</td>
<td>20</td>
</tr>
<tr>
<td>4</td>
<td>13</td>
<td>M</td>
<td>TF</td>
<td></td>
<td>sinus</td>
<td>20</td>
</tr>
<tr>
<td>5</td>
<td>12½</td>
<td>F</td>
<td>TF, ASD</td>
<td></td>
<td>sinus</td>
<td>10</td>
</tr>
<tr>
<td>6</td>
<td>12½</td>
<td>M</td>
<td>DORV, PS</td>
<td>Propranolol</td>
<td>sinus</td>
<td>30</td>
</tr>
<tr>
<td>7</td>
<td>13½</td>
<td>M</td>
<td>TF</td>
<td></td>
<td>sinus</td>
<td>10</td>
</tr>
<tr>
<td>8</td>
<td>10½</td>
<td>F</td>
<td>TF, ASD</td>
<td></td>
<td>sinus</td>
<td>30</td>
</tr>
</tbody>
</table>

Electrophysiological Findings (tables 2 and 3).

1. Intervals at Rest

Values for the control group are shown in table 2; they do not differ from those found in normal children or patients with a variety of congenital heart lesions.\(^1\)\(^2\)\(^3\) Values for the study group appear in table 3.

Table 3

Electrophysiological Findings

<table>
<thead>
<tr>
<th>Case no.</th>
<th>Rhythm</th>
<th>Atrial pacing II(^a) AV Block</th>
<th>Corrected sinus node recovery time (msec)</th>
<th>Escape rhythm</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>At rest (msec)</td>
<td>H-V (msec)</td>
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</tr>
<tr>
<td>1</td>
<td>sinus</td>
<td>30</td>
<td>130*</td>
<td>216</td>
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<tr>
<td>2</td>
<td>AV dissociation</td>
<td>30</td>
<td>80</td>
<td>210</td>
</tr>
<tr>
<td>3</td>
<td>sinus</td>
<td>15</td>
<td>80</td>
<td>210</td>
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<tr>
<td>4</td>
<td>sinus</td>
<td>25</td>
<td>115*</td>
<td>210</td>
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<tr>
<td>5</td>
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<td>70</td>
<td>230</td>
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<td>25</td>
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<td>154</td>
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<tr>
<td>9</td>
<td>sinus arrhythmia</td>
<td>25</td>
<td>95</td>
<td>200</td>
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<tr>
<td>10</td>
<td>sinus</td>
<td>15</td>
<td>70</td>
<td>240</td>
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<tr>
<td>11</td>
<td>sinus</td>
<td>25</td>
<td>95</td>
<td>218</td>
</tr>
<tr>
<td>12</td>
<td>AV dissociation, SA block</td>
<td>45</td>
<td>100</td>
<td>200</td>
</tr>
</tbody>
</table>

Note: *Values considered abnormal.
†After intravenous atropine.

Interference, due to accelerated junctional rhythm. Three other children showed episodes of A-V dissociation, due to intermittent slowing of the sinus rate in two, and to persistent sinus bradycardia in one (P-P: 1300 msec). This last case (patient 20) showed junctional and multifocal atrial escape rhythms.

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3 and are plotted against the regression lines (relating intervals to age) established by Abella et al.14

P-A interval. This was somewhat shorter than previously found by Abella et al.,14 possibly because a different surface ECG lead was used. There was no difference in P-A interval between children with and without an associated ASD.

A-H interval (fig. 1). The conduction time through the A-V node was normal in 19 and prolonged in four subjects. Two of these (patients 4 and 20) received therapeutic doses of digoxin at the time of the study, which could explain the A-H prolongation.

H-V interval (fig. 2). The H-V interval was normal for age in 22 children, including all patients with bifascicular block and the three with a history of transient postoperative heart block. Only one patient had a prolonged H-V interval. This child was in chronic congestive heart failure with a dilated right ventricle, a left ventricular end-diastolic pressure and A-V conduction disturbance (prolonged P-R, A-H and H-V intervals). It is likely that he had widespread myocardial damage.

2. Atrial Pacing

During atrial pacing the H-V interval remained unchanged in all children, including the boy with prolonged H-V interval at rest. With increasing pacing rate, the A-H interval became prolonged, until second degree A-V block occurred. In one patient the block was localized distal to the His bundle at a very high pacing rate (240/min). In all the other children the block was situated at the A-V nodal level and was of Wenckebach type.

Threshold of PAVB (fig. 3). The pacing rate at which second degree A-V block occurred ranged from 75/min to 240/min and was age dependent ($r = -0.55$). Two patients fell below the 95% confidence limits of the regression and were considered abnormal.

Corrected sinus node recovery time (CSRT) (fig. 4). Sufficient data about normal limits of CSRT in children are not yet available. In three patients from our study-group the CSRT exceeded 500 msec and was therefore higher than the generally accepted normal values for adults, which are 375–525 msec according to various authors;11, 15, 16 they also exceed the normal values for children from our laboratory (unpublished data) and those found by Gillette et al.8 The values of CSRT in these three patients are therefore considered abnormal. Moreover, junctional and low atrial escape beats, prior to SA node recovery, could be observed in all three (fig. 5). These three patients had rhythm disturbances on the surface ECG: A-V dissociation by interference, sinoatrial block or sinus bradycardia. One had episodes of paroxysmal atrial tachycardia.

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

**Figure 1**

Postoperative A-H intervals found in our study group, plotted against the graph for normal values according to Abella et al.14

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

**Figure 2**

Diagram of H-V intervals with average values and upper and lower limits of normal (+ 2 sd) after Abella et al.14 Postoperative H-V intervals found in the children of our study group are entered into the diagram of normal values. Patients with the ECG pattern of RBBB with LAD are indicated by squares.

![Figure 3](http://circ.ahajournals.org/)

**Figure 3**

The threshold of pacing-induced second degree A-V block (PAVB) for each patient is plotted against age. (regression: $HR = -0.36 \times age + 242.0; r = -0.55$). Symbols: circle = PAVB of Wenckebach type proximal to the His bundle; triangle = PAVB of Wenckebach type distal to His bundle; square = PAVB after administration of atropine intravenously. The figure indicates abnormally low PAVB in two cases (below the 95% confidence limit), one case returning to normal after atropine administration.
Pharmacological Tests

Atropine improved conduction through the A-V node in both patients with abnormally low threshold of PAVB, but in one case the threshold rate remained abnormal (fig. 3).

In two children with prolonged CSRT, atropine accelerated the junctional and low atrial (case 20) escape rhythms, but hardly accelerated sinus rhythm. Furthermore CSRT was prolonged to 5000 msec in one case.

Discussion

Conduction disorders after intracardial repair of tetralogy of Fallot and their relation to immediate and late CHB have been given increasing attention in recent years. The incidence of early postoperative permanent heart block has been greatly reduced; attention has been focused more recently on the occurrence of late CHB, which seems to bear a relationship to RBBB and LAD, a transient CHB in the early postoperative phase, or a combination of both. Godman et al. have demonstrated that in those cases, HBE may be of help in assessing the remaining conduction system, as it may demonstrate a conduction delay in the His-Purkinje system (prolonged H-V interval).

However, the His bundle and its branches are not the only part at risk during open heart surgery: the atrial incision and the placing of cannulas might damage the SA node; closure of an ASD and other reconstructive procedures in the atria could interrupt atrial pathways or injure the A-V node.

This study was designed to evaluate the whole conduction system, including sinus node function, of 23 unselected children after complete repair of tetralogy of Fallot. This group included four children with an ECG pattern of RBBB with LAD who may have an increased risk of CHB. An additional two patients

Figure 5

Left) After atrial pacing is discontinued (S-S interval: 450 msec) the sinus node recovers promptly, as indicated by a short A-A interval. I = surface ECG lead I, S = stimulus artifact, HBE = His bundle electrogram, HRA = intracardiac electrogram from the high right atrium. Bottom) After atrial pacing at a high rate (S-S interval: 270 msec; 2:1 A-V block proximal to the His bundle) is discontinued, sinus node recovery is delayed (CSRT = 540 msec). This is expressed by a marked delay of the first post-pacing A wave (visible on HRA electrogram), which is preceded by a junctional escape beat (visible on HBE). The ventricular capture (second post-pacing QRS complex) is again followed by a junctional escape beat (last QRS complex).
CONDUCTION SYSTEM AFTER TF REPAIR

presented transient bifascicular block postoperatively. All of these six children showed normal His-Purkinje conduction time at rest and during atrial pacing.

These findings may seem to be at variance with the report of Godman et al. who found H-V prolongation in 50% of patients with RBBB and LAD. However in Godman's report, all the patients with bifascicular block and H-V prolongation also had transient CHB in the postoperative period. In our series the uniformly satisfactory His-Purkinje conduction may be explained by the fact that no patient had the ominous combination of transient postoperative heart block followed by bifascicular block.

H-V conduction time was normal in children with transient CHB and RBBB with a normal axis at the time of study and this is in agreement with the observations of Godman et al.

Hence, His-Purkinje conduction time was normal in all children, except one boy who had a prolonged H-V interval in the presence of RBBB. This might indicate damage to the left bundle branch. However, in this child angiography showed considerable right ventricular dilatation with diminished contractility. We believe, along with Wen-Pin Lien et al., that ventricular dilatation may affect A-V conduction intervals, and this may explain the observed H-V prolongation. The fact that atrial pacing did not further prolong the H-V interval, nor induce a block at that level, seems to strengthen this hypothesis.

Atrial pacing represents a well known provocative stress of the conduction system, which may reveal conduction defects not apparent at rest.21-23 Pacing usually induces second degree A-V block of the Wenckebach type localized in the A-V node, whereas the His-Purkinje conduction time should remain constant.16, 21-24 On this basis, it may be stated that all children in this study showed adequate His-Purkinje conduction during atrial pacing. The only block distal to the His bundle occurred at a very high pacing rate; we believe it was due to an extremely high permeability of the A-V node, rather than to a damaged His-Purkinje system. It has been suggested that normal I:1 conduction up to a high rate of pacing does not necessarily exclude damage to the His-Purkinje system. Indeed, Narula25 and others23, 24 saw CHB appear within a short time of demonstration of excellent conduction by the method described above. However, their observations were made on aged patients with ischemic heart disease and do not necessarily apply to children. Certainly our results do not support the premise that prophylactic implantation of a demand pacemaker should be considered in patients with bifascicular block postoperatively.4

The A-H interval represents conduction time through the A-V node and therefore reflects a part of A-V node function. Atrial pacing, as previously mentioned, allows further assessment of A-V node function. The threshold rate at which PAVB occurs in adults is well established;16, 24 we are not aware of any data concerning the threshold in children, and its relation to age.

The findings in the study group patients (fig. 4) indicate that the threshold of pacing-induced second degree A-V block exceeds normal adult values and is age dependent. We believe that assessment of the threshold of PAVB, associated with pharmacological tests, is a more reliable indicator of A-V node dysfunction, than is the bare measurement of A-H interval at rest. Three out of four patients with prolonged A-H interval at rest, had an excellent response to pacing and a very high threshold of PAVB; only one had a low threshold which remained abnormal after administration of atropine. On the other hand, a low threshold of PAVB was found in a patient with a normal A-H interval at rest, but returned within normal limits after administration of atropine; this may be evidence against organic damage of the A-V node.

Intraatrial conduction is difficult to assess with accuracy, because the P-A interval is short and the onset difficult to determine. However, it is of interest that the presence of an associated ASD had no bearing on the P-A intervals, either preoperatively or after surgery. This contrasts with the observation of Anderson et al.26 who found significantly prolonged P-A intervals in children with a large ASD. This difference may be explained by the presence of a large left-to-right shunt in Anderson’s patients who thus represent a different hemodynamic situation.

An unexpected yet important finding of this study is the evidence of SA node dysfunction in three out of 23 unselected patients. The evidence is based on the presence of postoperative arrhythmias similar to those seen in the sick sinus syndrome.16, 27, 28 sinus bradycardia, sinoatrial block of second degree and supraventricular tachycardia. These three patients had an abnormal CSRT, and prolonged postpacing depression of the sinus node is now generally taken as a sign of SA node dysfunction.11, 15, 16, 29

Intravenous administration of atropine produced inadequate responses in two cases, who developed enhancement of junctional and low atrial pacemakers, isorhythmic dissociation, and further prolongation of CSRT. Such a response strengthens the diagnosis of SA node dysfunction, especially as the ectopic rhythm lasted for several minutes.30

It has been known for some time that damage to the SA node may occur after correction of transposition of the great arteries by the Mustard procedure.8, 31 This is not unexpected because this operation requires extensive reconstruction at the level of the atria; during
insertion of the baffle, damage to the SA node may be caused by direct trauma to the node, or interruption of the sinus node artery. Only recently has it become apparent that SA node dysfunction may be seen after correction of other heart defects. Indeed, Greenwood et al. found a "sick sinus syndrome" in some patients after correction of tetralogy of Fallot, atrial septal defect and ventricular septal defect; their evidence was based on clinical data and surface ECG, but electrophysiological studies were not done. In these situations, there is no extensive atrial reconstruction. We may speculate with Aberdeen and Carr that damage results from insertion of the superior vena cava cannula or sealing of the atrium around it. There is a possibility that the atrial incision may divide the node. In our series, no specimens are available, so that we cannot bring forward anatomic or histologic evidence of SA node injury.

In conclusion, the majority of patients in this study, all of whom had undergone extensive intracardiac surgery, showed little or no damage to the conduction system. The serious consequences of injury to the His bundle are now well recognized, and new surgical techniques have succeeded in avoiding it. However this study has shown that other parts of the conduction system, especially the SA and A-V nodes, may be vulnerable. These structures should also be avoided if possible during open heart surgery.

References
Sinus node function and conduction system after complete repair of tetralogy of Fallot.

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