Intraoperative Recording of Specialized Atrioventricular Conduction Tissue Electrograms in 47 Patients

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SUMMARY Intraoperative mapping of the specialized atrioventricular conduction system was performed in 47 patients during cardiac surgery. Specialized conduction tissue electrograms were identified in 37, and atrioventricular conduction preserved in 92%. Specialized conduction tissue was identified in 27 patients with atrioventricular canal defect; complete heart block was avoided in 25. Conduction tissue was located in six of 12 patients with complex transpositions; atrioventricular conduction was preserved in all six. Other lesions in which the technique was useful were Ebstein's anomaly and single atrium. Limitations to the technique are 1) deep hypothermia and circulatory arrest; 2) interruption in atrioventricular conduction during mapping; 3) inadequate exposure and access to probable sites of conduction tissue; 4) variation of size and spatial relations of individual malformations; and 5) limited time for identification of unusually located conduction tissue. Indications for use of this technique include patients with both forms of atrioventricular canal, complex transpositions, atrioventricular discordance, single ventricle and single atrium.

ELECTROPHYSIOLOGIC IDENTIFICATION of the specialized cardiac conduction system was introduced in 1970 to prevent major injury to the cardiac conduction tissue during open heart surgery. Several reports have described mapping the conduction system in patients with selected cardiovascular malformations, including incomplete atrioventricular (AV) canal, complete AV canal, transposition of the great arteries, atrial septal defect, ventricular septal defect, tetralogy of Fallot, corrected transposition of the great arteries with either situs solitus or situs inversus, Ebstein's anomaly, and single ventricle. In this report we review our experience with intracardiac mapping and define the limitations and indications of this electrophysiologic technique in patients with congenital heart disease.

Materials and Methods

Between March 1, 1974 and August 31, 1977, intraoperative mapping of the specialized AV conduction tissue was performed at the Children's Hospital Medical Center, Boston, in 47 patients with complex congenital heart disease. In all the patients the course of the specialized conduction system was either unpredictable, unknown or particularly vulnerable and thus at risk at surgery; identification by the electrophysiologic technique was performed in an attempt to preserve AV conduction.

Recording of the intracardiac specialized conduction tissue potentials was performed in the manner previously described. After institution of cardiopulmonary bypass the area suspected of containing...
AV conduction tissue was explored with a hand-held probe 3–5 mm in diameter, with three biopolar pairs of electrodes (1 mm apart). The patients’ esophageal temperatures were maintained between 30–37°C. Each bipolar pair of electrodes was connected to a Hewlett-Packard high impedance differential amplifier (MN8811A) and was isolated from both ground and the recording apparatus by an isolation transformer. Electrograms were recorded at frequencies between 15–300 Hz. All tracings were monitored on a Hewlett-Packard 1308A oscilloscope and recorded simultaneously on a photographic paper moving at 100 mm/sec. During the mapping procedure AV conduction was maintained by either a normal sinus rhythm or atrial pacing; in a few patients specialized conduction tissue electrograms were recorded in the presence of other mechanisms (atrial fibrillation or AV dissociation). Time required for the mapping procedure was generally no more than 3–5 minutes; in one patient unsuccessful exploration for specialized conduction time electrograms was prolonged to 10 minutes.

Table 1 shows patient diagnoses, ages, electrophysiologic findings and results. Twenty-seven patients had either partial or complete AV canal. One additional patient had ventricular septal defect of the AV canal type. Twelve patients with complex cardiac malformations had either complete or corrected transposition, with either situs solitus or inversus of viscera and atria. Two patients had single ventricle with right ventricular outflow tract chamber and corrected transposition of the great arteries;1,2,10 one of these patients had, in addition, a restricted bulbo-ventricular foramen with a hypertensive left ventricle. One patient had tricuspid atresia with d-transposition of the great arteries and restrictive bulbo-ventricular foramen. Two patients had double outlet right ventricle with ventricular septal defect and pulmonary artery band, and one patient each had Ebstein’s anomaly and single atrium with AV canal defect.

Results

In 39 of the 47 patients studied, specialized atrial ventricular conduction tissue was identified by the electrophysiologic technique (table 1).

Incomplete Atrioventricular Canal

We successfully mapped 14 patients with incomplete AV canal. The median age at surgery of this group was 9 years. None developed complete heart block postoperatively, although two patients developed symptomatic tachy-bradyarrhythmia syndrome and one (patient 11) had intermittent junctional mechanism. All three of these patients also had mitral valve replacement because of significant mitral regurgitation. Two of these three (patients 12 and 14) received their xenograft valves at a later operation, after initial unsuccessful valvuloplasty. Figure 1 illustrates the findings in a 3-year-old boy with an atrial septal defect primum type; the course of the intra-atrial conduction tissue began inferior to the coronary sinus and proceeded along the interavalva ridge between mitral and tricuspid orifices. Approaching the ventricular crest, the conduction tissue deviated slightly to the left; this pattern was observed in three additional patients. The atrial septal patch was placed to the right of this area. In two patients the conduction tissue was traced beneath the septal leaflet of the tricuspid valve onto the ventricular crest. Conduction tissue was avoided in these two cases by placing the patch on the left side of the ventricular crest. In the remainder of the patients the conduction tissue was found along the interavalva ridge in the middle of the ventricular crest. Conduction tissue was avoided by placing sutures to the right. One- to 4-year follow-up revealed normal sinus rhythm in nine patients, persistent sick sinus syndrome in two, and sinus rhythm with intermittent AV dissociation in one. One infant (patient 1) 6 months of age and weighing 4 kg, died 3 days after surgery of pulmonary complications. Postoperatively, AV conduction was intact.

Complete Atrioventricular Canal

Thirteen patients were operated on for primary repair of complete AV canal, with successful electrophysiologic delineation of the specialized conduction tissue. These patients were considerably younger at the time of surgery (median age 4 years) than those with incomplete AV canal. Two patients (15 and 16), both younger than 18 months and weighing less than 5 kg, developed complete heart block. One additional patient (21) had AV dissociation after surgery. Although hospital mortality was high in this group (four of 13, with one additional late death), conduction disturbances could be implicated as the primary cause of death in only two. Figure 2 illustrates the relationship between the recorded intracardiac electrogram and the anatomic sites within the heart of patient 26. With the probe slightly inferior and to the right of the coronary sinus orifice, both atrial and His bundle electrograms were recorded; the ventricular electrogram was small. Proceeding along the His bundle toward the ventricle, the ventricular electrogram became larger as the atrial electrogram diminished. Upon reaching the ventricular crest, both the a wave of the atrial electrogram and the His bundle potential disappeared; only a ventricular electrogram was recorded. In this manner the course of the conduction system was outlined.

In seven of the 13 patients the specialized conduction tissue could be traced onto the middle of the ventricular crest before the signal was lost. Conduction tissue was avoided by careful placement of the inferior posterior margin of the patch to the right of this area. In six of the patients the intraventricular portion of the specialized conduction tissue was identified beneath the tricuspid posterior leaflet. In these cases the septal patch was placed to the left of this area. In the two smallest infants, complete heart block developed. Satisfactory identification of the specialized conduction tissue had been accomplished
### Table 1. Clinical Data

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<tr>
<th>Pt</th>
<th>Age</th>
<th>Data Identification Postop</th>
<th>Postop rhythm</th>
<th>Postop AV conduction</th>
<th>Follow-up</th>
<th>Other</th>
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<tr>
<td>1</td>
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<tr>
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<td>18 years</td>
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<td>SSS</td>
<td>Intact, 1st degree heart block</td>
<td>4 years</td>
<td>MVR</td>
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**Incomplete atrioventricular canal**

- 15: 9 months, Yes, Pacemaker, Surgical CHB, Dead
- 16: 16 months, Yes, Pacemaker, Surgical CHB, 1 year—died
- 17: 1½ years, Yes, Sinus, Intact, Dead
- 18: 20 months, Yes, Sinus, Intact, 3 years
- 19: 23 months, Yes, Sinus, Intact, 3 years
- 20: 2 years, Yes, Sinus, Intact, Dead
- 21: 4 years, Yes, Junctional, AV dissociation, Dead
- 22: 4½ years, Yes, Sinus, Intact, 3 years
- 23: 4 years, Yes, Sinus, Intact, 2 years
- 24: 7 years, Yes, Sinus, Intact, 3 years
- 25: 7 years, Yes, Sinus, Intact, 2 years, PAB
- 26: 12 years, Yes, Sinus, Intact, 2 years, PAB, MVR
- 27: 14 years, Yes, Sinus, Intact, 2 years, MVR

**Complete AV canal**

- 28: 12 years, Yes, Sinus, Intact, 3 years

**Ventricular septal defect**

- (AV canal type)

**Transposition**

- IVS

**Transposition**

- VSD, PS, (S, D, D)

**Transposition**

- VSD, PS, (I, D, D), dextrocardia

**Corrected transposition**

- VSD, PS, (S, L, L)

**Corrected transposition**

- Left-sided AV valve regurgitation (S, L, L)

- 39: 11 years, No, Sinus, Intact, 2 years
<table>
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<tr>
<th>Pt</th>
<th>Age</th>
<th>Identification of SAVC tissue</th>
<th>Postop rhythm</th>
<th>Postop AV conduction</th>
<th>Follow-up</th>
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<td>PAH, CHF</td>
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<td>CHB, Surgical</td>
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<td>Intact</td>
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<tr>
<td>45</td>
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<td>46</td>
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<td>MVR</td>
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<td>47</td>
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<td>Sinus</td>
<td>Intact</td>
<td>1 year</td>
<td></td>
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**Abbreviations:** A-H = atrial-His bundle interval; AV = atrioventricular; CHB = complete heart block; CHF = congestive heart failure; H-V = His bundle-ventricular interval; (L,D,D) = situs inversus of viscera and atria; d-loop; aortic valve to right of pulmonary valve; (S,L,L) = situs solitus of viscera and aorta; l-loop; aortic valve to left of pulmonary valve; IVS = intact ventricular septum; MVR = mitral valve replacement; PAB = pulmonary artery band; PAH = pulmonary artery hypertension; PS = pulmonary stenosis; SAVC = specialized atrioventricular conduction tissue; (S,D,D) = situs solitus of viscera and atria; d-loop; aortic valve to right of pulmonary valve; (S,L,L) = situs solitus of viscera and atria; l-loop; aortic valve to the left of pulmonary valve; (S,L,L) = situs solitus of viscera and atria; l-loop; aortic valve to the left of pulmonary valve, mal-abnormally placed but not transposed across the ventricular septum; SSS = sick sinus syndrome; VSD = ventricular septal defect.

**Figure 1.** The left panel is a photograph taken at repair of an atrial septal defect primum type; the right panel is an intraoperative His bundle tracing obtained at surgery. The intervalvar ridge, between the mitral and tricuspid orifices, was the site of intra-atrial electrogram recordings (indicated by the dotted line). The patch was placed to the right of this ridge. A = atrial electrogram; H = His bundle electrogram; HBE = His bundle recording; HRA = high right atrial recording; P = P wave; R = R wave; V = ventricular electrogram.
Posterior interior

**FIGURE 2.** Photograph of intracardiac anatomy and intraoperative His bundle recording obtained at various sites in the atria of a 12-year-old girl with complete atrioventricular canal. With the probe high in the right atrium, both atrial and His bundle electrograms (HBE) were recorded. The ventricular electrogram was quite small. Proceeding along the His bundle toward the ventricle, the ventricular electrogram became larger as the atrial electrogram diminished; upon reaching the ventricular crest the a wave of the atrial electrogram and HBE disappeared and only a ventricular electrogram persisted. In this manner, the course of the conduction system was outlined. Abbreviations: same as in figure 1.

in both, but the atrial anatomy necessitated placement of the inferior margin of the patch on the identified sites. During the later part of this study (1976–1977), three children with complete AV canal had their defects repaired using deep hypothermia and circulatory arrest without intraoperative mapping; therefore, they were not included in this study. Two of these children weighed 8 kg and were 1½ years old; both of these children had intact AV conduction postoperatively. The third child, 11 months of age,

**FIGURE 3.** Diagrammatic sketch (left panel) of the relationship between ventricular septal defect (VSD), atrioventricular canal type and the location of His bundle electrograms (HBE). The tricuspid valve has been removed. HBEs were recorded along the superior margin of this VSD, but in the usual inferior location in relationship to the membraneous septum and parietal band. Abbreviations: same as figure 1.
weighed approximately 4 kg at surgery and was in severe congestive heart failure. Transient complete heart block persisted for 7 days after surgical repair, before AV conduction with first degree heart block returned.

A 12-year-old boy (patient 28) required closure of a large ventricular septal defect and removal of a pulmonary artery band. At surgery the defect was located posteriorly and inferiorly behind the posterior leaflet of the tricuspid valve, with no intervening muscular tissue between the margin of the ventricular septal defect and the insertion at the AV groove of the posterior leaflet, indicating an AV canal type of defect.\(^\text{14}\) Normal intra-atrial electrograms were recorded. When exploring the margin of the ventricular septal defect the specialized conduction tissue electrograms were found along the anterior-superior rim of the defect (fig. 3).

Transpositions

Five patients (30–34) with d-transposition of the great arteries; ventricular septal defect, and pulmonary stenosis were examined at surgery by the electrophysiologic technique. In d-transposition of the great arteries with situs solitus and membranous ventricular septal defect, the conduction system is located along the posterior-inferior margin of the ventricular septal defect;\(^\text{16}\) early in our experience, we studied two patients, with the expected findings. In contrast, two patients with d-transposition of the great arteries with situs inversus, ventricular septal defect, and pulmonary stenosis presented different findings. In spite of the AV discordance, and in contrast to the usual form of corrected (l) transposition of the great arteries [S,L,L], the intraventricular conduction tissue was identified along the posterior-inferior margin of the ventricular septal defect.\(^\text{9}\) In the third similar patient (34), specialized conduction tissue electrograms were not recorded.

In the four patients with corrected transposition of the great arteries (l-transposition) with ventricular septal defect and pulmonary stenosis [S,L,L],\(^\text{13}\) specialized conduction tissue electrograms were identified in two and AV conduction preserved postoperatively. In the other two, specialized conduction tissue was not identified and surgically induced complete heart block occurred in one (patient 46), probably due to the failure to explore the subpulmonic area for His bundle electrograms. The other (patient 35) had complete heart block before surgery. Figure 4 (patient 38) illustrates the location of the specialized intraventricular conduction tissue in a patient with l-transposition of the great arteries, situs solitus, ventricular septal defect and pulmonary stenosis. As previously reported,\(^\text{7, 8, 10, 11}\) specialized conduction tissue electrograms were recorded on the anterior-superior margin of the ventricular septal defect rather than the inferior margin, its usual location in transposition of the [S,D,D] configuration. However, in this patient, specialized conduction tissue electrograms were not recorded until the probe was placed on the far left-sided right ventricular (anterior-superior) margin of the ventricular septal defect. Because of this

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**Figure 4.** This figure shows the anatomy of corrected transposition in situs solitus (adapted from Kupersmith et al.\(^\text{4}\)) and the intraoperative electrograms. Specialized conduction tissue electrograms were identified along the superior-anterior margin of the ventricular septal defect, but on the right ventricular (left side) rather than left ventricular (right side) side of the anterior ventricular septum. Abbreviations: same as figure 1.
SUPERIOR

RIGHT

INFERIOR

SUPERIOR

LEFT POSTERIOR

MV

RIGHT ANTERIOR

INFERIOR

SUPERIOR

LEFT POSTERIOR

MV

RIGHT ANTERIOR

INFERIOR

Figure 5. Intraoperative recording (right) in a patient with double outlet right ventricle, ventricular septal defect (VSD), and pulmonary artery bank (not shown). Intraventricular conduction tissue was found in the expected location, along the posterior-inferior margin of the VSD. Abbreviations: same as figure 1.

finding, sutures were placed on the right side, left ventricular aspect of the ventricular septal defect. AV conduction was preserved. Follow-up catheterization demonstrated residual severe pulmonary stenosis. At reoperation 1 year later, attempts at resection of the subpulmonary obstruction without the benefit of mapping resulted in complete heart block, and the patient required a permanent pacemaker. In two patients with l-transposition, one (patient 39) with situs solitus [S,L,L] and left-sided AV valve regurgitation (intact ventricular septum) and one (patient 40) with situs inversus [I,L,L], pulmonary atresia and ventricular septal defect, specialized AV conduction tissue was not identified. AV conduction was intact postoperatively in both patients.

Other Cardiac Malformations

Two patients with single ventricle, type A,12 and I-transposition of the great arteries required surgery. One (patient 41), 6 years of age, had a restrictive bulbo-ventricular foramen and a hypertensive right ventricle. At surgery the margin of the bulbo-ventricular foramen (ventricular septal defect) was thickened.

Figure 6. The illustration (left panel) schematically outlines the atrioventricular (AV) canal anatomy viewed from above in a 12-year-old patient with single atrium. The atria have been removed. The right panel is the intraoperative His bundle recording (HBE). Note the location of the HBE posterior to the left-sided AV orifice. Also note atrial fibrillation and spontaneous His bundle rhythm. AV conduction was maintained during placement of the inferior margin of the patch. MV = mitral valve; TV = tricuspid valve; A.Fib. = atrial fibrillation; other abbreviations same as figure 1.
with fibrous tissue. Exploration of the entire margin of the ventricular defect failed to disclose specialized conduction tissue electrograms. Incision of the superior-anterior margin of the ventricular septal defect to enlarge the foramen resulted in complete heart block, and the patient required a permanent pacemaker. In the second patient (42) with similar anatomic findings, ventricular septation was unsuccessful; extensive exploration (10 minutes) for specialized conduction tissue was also unsuccessful, resulting in complete heart block and postoperative death (24 hours).

A 12-year-old boy (patient 43) with tricuspid atresia, d-transposition of the great arteries, and a restrictive bulbo-ventricular foramen (ventricular septal defect) was operated on to relieve the hypertensive left ventricle. No specialized intraventricular conduction tissue was identified. This patient died after surgery, in part related to the surgically-induced complete heart block.

Two patients with double outlet right ventricle, subaortic ventricular septal defect [S,D,D] and pulmonary artery banding required intracardiac repair; at surgery, intratrial and intraventricular specialized conduction tissue electrograms were recorded in one (patient 45) (fig. 5) at the expected atrial location adjacent to the coronary sinus, and along the posterior-inferior margin of the ventricular septal defect. In the other patient (44), specialized conduction tissue electrograms were not obtained.

An 11-year-old girl (patient 46) with single atrium and AV canal defect was repaired using the electrophysiologic technique to identify the specialized conduction tissue. Intra-atrial electrograms were recorded from the posterior margin of the mitral orifice, and traced inferiorly onto the left side of the ventricular crest (fig. 6). Conduction was maintained during placement of the inferior margin of the patch. However, the ring of the mitral valve prosthesis, by necessity, rested on the point where the electrograms were recorded; AV dissociation with intermittent intact AV conduction developed.

A 13-year-old girl (patient 47) received a heterograft valve at the tricuspid position for Ebstein's anomaly. Intra-atrial electrograms (fig. 7) were recorded for a short distance within the atrium above the inferiorly and leftwardly-displaced posterior right AV valve, and superior and anterior to the coronary sinus. Sutures for the tricuspid valve prosthesis were placed to either side of this point. After surgery, AV dissociation was present for 24 hours. Intact AV conduction with first degree heart block then returned.

Discussion

The primary objective of intraoperative electrophysiologic identification of specialized conduction tissue is the preservation of AV conduction after surgery.1-4 We were successful in identifying specialized conduction tissue in 79% of our patients (37 of 47). In 92% of these patients (34 of 37) surgically induced complete heart block was avoided. In one other (patient 32), transient complete heart block developed, but AV conduction resumed before discharge from the hospital. Intermittent AV dissociation was present in three (1, 11 and 46) and junctional rhythm in one
(patient 21). In contrast, 40% (four of 10) of the patients in whom specialized conduction tissue was not identified developed complete heart block.

Complete heart block has been reported to develop in 15% of patients during repair of incomplete AV canal,18 and is known to appear as a late complication after AV canal repair.16, 17 In all of the 27 patients with AV canal defects in this report, specialized AV conduction tissue potentials were identified and AV conduction was preserved in 95%. Little or no variation in the origin of the specialized AV conduction tissue, inferior (caudal) and anterior (ventral) to the coronary sinus, was found, in contrast to the experience of others.5, 3 We did observe variation, however, in the course of the His bundle as it reached the ventricular crest. In 11 of 13 patients with incomplete AV canal defects and seven of 14 with complete AV canal defects, AV block was avoided by placement of the inferior margin of the patch on the right side of the crest. Thus, in the majority of patients with incomplete AV canal defects, conduction tissue can be avoided by adhering to the right margin of the ventricular crest. In a few patients, however, (15% in our series, two of 13) the conduction tissue may precede beneath the right-sided tricuspid valve on to the ventricular crest. This variation can only be identified by recording His bundle electrograms, and underscores the necessity of mapping patients with incomplete AV canal. In patients with the complete form of AV canal the position of the conduction tissue on the ventricular crest was even more variable and thus, also required mapping to locate precisely its course. Even with this technique anatomic limitations such as size and spatial relations may require placement of suture at points where specialized conduction time electrograms are recorded, as occurred in two of our patients (15 and 16).

The course of the conduction system in membranous ventricular septal defect is along the inferior posterior margin of the defect.14, 18 In the AV canal type of defect, conduction tissue has been identified along the posterior margin;19 in contrast, the conduction tissue in muscular defects is often unrelated to the defect and may thus be located anterior and superior to it.18 In patient 28, the anatomic features of the defect suggested an AV canal type, but the conduction tissue was anterior, proceeding in a more normal course, indicating that this defect probably did not impinge upon the membranous septum,18 and resembled in its relationships to the conduction tissue the muscular type of defect.18

Intraventricular specialized conduction tissue electrograms were not recorded in six of the 12 patients with complex transpositions. In the single patient (29) with d-transposition with intact ventricular septum and one patient (34) with d-transposition, pulmonary stenosis, and ventricular septal defect in situus inversus [I,D,D], intraventricular mapping was not pursued for technical reasons. In one patient (36) with corrected transposition of the great arteries [S,L,L], failure to explore the anterior margin of the pulmonary outflow tract probably led to false negative findings. Resection of subpulmonary tissue containing conduction tissue6, 15 resulted in complete heart block. In this patient the transatrial approach to the ventricular septal defect was sufficient for electrophysiologic exploration of the margin of the ventricular septal defect and its subsequent closure, but inadequate for mapping of the anterior pulmonary valve area; this poor exposure probably accounted for failure of identification of the conduction tissue. Specialized conduction tissue at risk in this location may be avoided by use of a left ventricular (pulmonary ventricle) pulmonary artery valve containing conduit.19, 20 The second failure in corrected transposition occurred in a girl (patient 35) with an idioventricular rhythm arising distal to detectable conduction pathways; retrograde activation of specialized conduction tissue was lost in the ventricular electrogram.

Limited exposure and access to the conduction system may impede adequate recording of the specialized conduction electrograms. Two additional patients with l-transposition were unsuccessfully explored for specialized conduction time electrograms. One patient with l-transposition [S,L,L], intact ventricular septum and left-sided AV valve regurgitation received a xenograph valve in one left-sided tricuspid position. After native valve resection and before prosthetic valve insertion, the septal surface of the left-sided right ventricle beneath the aortic valve was explored with the probe; visualization of the area was poor, and we could not locate specialized AV conduction tissue. In the patient (40) with l-transposition [I,L,L] with pulmonary atresia, ventricular septal defect had previously been palliated with a Waterston shunt. Pulmonary artery hypertension developed and surgical repair was advised. At operation a transventricular approach through the markedly hypertrophied right-sided left ventricle (pulmonary ventricle) did not provide adequate exposure for mapping. Similar difficulty was encountered in one (patient 44) with double outlet right ventricle, ventricular septal defect, subaortic stenosis, and pulmonary artery band. In these four patients AV conduction was intact postoperatively.

In the patient with tricuspid atresia, exposure through the hypoplastic right ventricle may not have been sufficient; the intraventricular His bundle lies subendocardially on the left ventricular aspect of the ventricular septum and posterior to the ventricular septal defect.15 Although the incision to enlarge the ventricular septal defect was made anteriorly (leftward and superior), complete heart block resulted.

Patients with complex cardiac malformations — those with abnormally related great arteries, AV discordance and single ventricles — presented more difficult problems, with poorer results. The variability of the conduction system in these anatomic entities is well described.7, 8, 10, 11, 21, 22 Although intraventricular conduction tissue has been identified by the electrophysiologic technique, complete heart block is a major problem in this group21 and a major cause of
operative mortality. In our two patients with single ventricles, failure to identify specialized conduction tissue resulted in complete heart block. In one (patient 41) extensive endothelialization of the bulbo-ventricular foramen probably played a role in our inability to identify specialized conduction tissue electrograms. An incision in the anterior-superior margin of the ventricular septal defect, a probable site for the conduction tissue, \(^{15}\) resulted in complete heart block. In the second patient (42) with single ventricle, in spite of prolonged (10-minute) exploration during sinus rhythm and intact AV conduction, specialized conduction tissue potentials could not be identified.

Tricuspid valve replacement for Ebstein's anomaly has been complicated by a 30% instance of complete heart block, \(^8\) electrophysiologic identification of the specialized AV conduction system contributed to the preservation of intact AV conduction, both in our single case and that of others. \(^8\)

Single atrium is accompanied by absence of a recognizable coronary sinus orifice, an anatomic landmark of the AV node and proximal His bundle. Thus identification of the conduction tissue by the electrophysiologic technique is especially useful. In our case His bundle potentials were located posterior to the mitral valve orifice (fig. 5) and continued onto the left side of the ventricular crest. AV dissociation, however, occurred after placement of the mitral valve prosthesis, probably on the basis of local compression or edema adjacent to vulnerable underlying conduction tissue.

The major limitation of intraoperative mapping is the use of deep hypothermia and circulatory arrest. Under these conditions, although AV conduction is generally maintained to approximately 25°C, minor manipulations on the endocardial surface often lead to arrhythmias or further conduction disturbances. At deep hypothermic temperatures, below 20°C, spontaneous impulse formation and propagation cease. \(^{23}\) Other technical and theoretical considerations are important. Maintenance of intact AV conduction, either by sinus mechanism or by atrial pacing during the mapping procedure, is necessary to insure a normal activation sequence and promote detection of the specialized conduction tissue in question. Although His bundle rhythms can be recorded during atrial fibrillation during surgery (fig. 4) and specialized conduction tissue electrograms have been recorded in the presence of AV dissociation, \(^9\) the absence of intact AV conduction makes identification more difficult. Local metabolic factors such as hypokalemia, ischemia, acidosis and hypothermia also alter impulse formation and propagation sufficient to interfere with identification of underlying conduction tissue by locally recorded electrograms. \(^{24}\) The margin for error provided by the technique is 2–3 mm to either side of the specialized conduction tissue, \(^{25}\) a precision requiring careful gentle exploration.

Until rapid, nontoxic histochemical techniques become available for visualization of viable specialized conduction tissue, the electrophysiologic technique remains useful. Based on our experience and that of others, \(^{2,5}\) including those not using the technique, \(^{16,17}\) mapping should be used in all patients with both forms of AV canal, when operating conditions (near normothermia) permit. In addition, in patients who are demonstrated to have abnormally normal anatomy, such as single atrium or Ebstein's anomaly, and associated lesions, identification of specialized AV conduction tissue electrograms is necessary. In patients with complex transpositions and abnormal intersegmental relationships, where the course of the conduction system is variable and unknown, the mapping technique is also imperative. Single ventricle, when surgical intraventricular intervention is indicated, requires electrophysiologic delineation of the specialized conduction tissue. Even when mapping identifies the site of conduction tissue, individual anatomic and spatial relations of the malformation may necessitate suture placement (or insertion of prosthetic devices) on vulnerable sites of conduction tissue. Exposure and access to expected sites of special conduction tissue may be poor, preventing adequate mapping. In these situations, postoperative intact AV conduction is less likely to occur.

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