Atrioventricular Conduction in Patients with Incomplete Endocardial Cushion Defect

By Daniel J. Goodman, M.D., Donald C. Harrison, M.D.
and David S. Cannom, M.D.

SUMMARY

The cause of left axis deviation noted on the electrocardiogram (ECG) in patients with ostium primum atrial septal defect has been attributed to two differing mechanisms: left ventricular overload secondary to mitral insufficiency and a congenitally anomalous atrioventricular conduction system. In order to define the conduction characteristics of the atrioventricular conduction system in patients free from the hemodynamic abnormalities of ostium primum atrial septal defect, seven patients with an ostium primum atrial septal defect were studied in the late postoperative period. Each ECG showed persisting left axis deviation with no new conduction abnormalities. Hemodynamic evaluation, including left ventriculography, showed no intra-atrial shunting and only minimal mitral insufficiency in three patients. His bundle recording techniques were utilized to study the atrioventricular conduction system. The P–A interval was normal in four patients (25–45 msec) and prolonged in three (50, 60 and 60 msec), while the A–H interval was normal in all seven patients (60–130 msec). This is compatible with abnormal intra-atrial conduction and normal conduction through the atrioventricular node. The H–V interval was normal in four patients (35–55 msec) and short in three (20, 25, and 25 msec). Atrial pacing produced the normally expected A–H prolongation, P–A and H–V remaining constant. These results and the persisting left axis deviation can be explained on the basis of the known anatomy of the atrioventricular conduction system in patients with ostium primum atrial septal defect. The left axis deviation is most probably related to early activation of the postero-basal left ventricular wall (patients with short H–V) and/or late activation of the antero-lateral left ventricular wall (patients with normal H–V). Hemodynamics are not important in producing these findings.

Additional Indexing Words:
Ostium primum atrial septal defect  Congenital heart disease  His bundle electrograms
Left axis deviation  Postoperative hemodynamics

The incomplete endocardial cushion defect (ostium primum atrial septal defect) incorporates features of two lesions—an atrial septal defect, usually moderate to large in size, and mitral insufficiency which occurs through a cleft mitral valve. In this condition, the typical electrocardiogram (ECG) shows left axis deviation with counterclockwise inscription of the frontal plane vector.1, 2, 3

The genesis of these electrocardiographic findings remains a matter of controversy. Blount et al.1 suggested that the left axis deviation found on electrocardiography was a consequence of left ventricular hypertrophy secondary to the mitral insufficiency. In contrast, others have considered that a congenital anomaly of the conduction system is the underlying cause of the axis deviation.2, 3

To our knowledge, no prior studies have analyzed atrioventricular conduction in the postoperative patient with incomplete endocardial cushion defect, when the hemodynamic effects on cardiac conduction are largely eliminated, but in whom the typical conduction abnormalities persist. We are reporting the studies of seven such patients utilizing His bundle electrograms for elucidating the electrophysiology of ostium primum atrial septal defect.

Materials and Methods

Seven patients who had undergone successful repair
of an incomplete endocardial cushion defect at Stanford University Hospital had electrophysiologic studies performed at the time of a follow-up cardiac catheterization. A similar surgical technique was employed in all patients for repair of the intracardiac defects. On cardiopulmonary bypass, the cleft in the mitral valve was repaired by multiple horizontal mattress sutures and the atrial septal defect was then closed utilizing a pericardial patch. Closure of the lowing atrial septal defect was accomplished with the heart beating spontaneously so that heart block, due to a misplaced suture, could be identified immediately and corrected. In no case did this occur during surgery, and comparison of pre- and postoperative ECGs showed no evidence of new atrioventricular conduction disturbances (table 1).

Table 1
Pre- and Postoperative Electrocardiogram

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<th>Pt.</th>
<th>HR (sec)</th>
<th>Rhythm</th>
<th>P-R (sec)</th>
<th>Axis (degrees)</th>
<th>QRS (sec)</th>
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</table>

Abbreviations: HR = heart rate; Axis = frontal plane axis; QRS = QRS duration; V1 = R-R'; deflections in mm/msec; S:5.

The follow-up study was done from five to 90 months postoperatively, with a mean of 37 months. The patients ranged in age from 17 to 33 years and included four females and three males. None of the patients were taking any medication known to affect atrioventricular conduction, and all patients were on a symptomatic basis class I (New York Heart Association) at the time of follow-up. The nature of the catheterization was explained to each patient and informed consent was obtained. Our studies included right and left heart catheterization with hydrogen-sensitive catheters for detection of residual shunting, and oxygen saturation studies for quantitation of shunt flow. Left ventriculograms were performed for assessment of mitral valve competence.

Electrophysiologic studies were made at the conclusion of the hemodynamic evaluation using standard His bundle recording techniques.4, 5 The right basilic vein pressure catheter was replaced by a quadrupolar electrode catheter (United States Catheter Instruments) which was positioned high in the right atrium with the two distal poles of the catheter in the region of the sinus node. A stable bipolar atrial electrogram was recorded from the two proximal poles. The distal pair, attached to a battery powered pacemaker (Medtronic 5837) which delivered impulses two msec in duration and twice diastolic threshold, was used for right atrial pacing. A tripolar electrode catheter (United States Catheter Instruments) with an interelectrode distance of 1 cm was then introduced via the femoral vein and positioned to record the His bundle electrogram.

The His bundle and atrial electrograms were transmitted in the A–C input of an Electronics for Medicine amplifier and filtered at 40 to 500 Hz. A standard ECG lead II was recorded and all signals were displayed simultaneously on a switched-beam oscilloscope and recorded on photographic paper at a paper speed of 100 mm/sec. The resting baseline measurements were also recorded at a 200 mm/sec paper speed.

Baseline resting measurements of each patient's heart rate (cycle length), P wave—low atrial (P–A) conduction time, atrium–His bundle (A–H) conduction time and His bundle-ventricular (H–V) conduction time were made. The P–A interval was measured between the onset of the P wave in the surface ECG recording and the onset of the atrial depolarization as recorded by the His bundle catheter. The normal P–A interval has been reported to range from 10–45° to 25–45 msec.7, 8 During atrial pacing the P–A interval was measured between the stimulus artifact onset and the onset of atrial depolarization in the His bundle catheter. The A–H interval was measured from the first rapid deflection of atrial depolarization to the first rapid deflection of the bundle of His electrogram recording; both were recorded from the tripolar catheter. The normal range has been reported to be from 70–110° to 55–130 msec.8 The H–V interval was measured from the first rapid deflection of the bundle of His electrogram, as recorded from the tripolar catheter, to the earliest onset of ventricular depolarization recorded on either a surface ECG lead or an intracardiac electrode. This has been reported to normally measure from 35–45°8 to 30–55
msec.\textsuperscript{6} In our laboratory we have used the following values as normal: P-A, 25–45 msec; A-H, 60–130 msec; and H-V, 35–55 msec.

Right atrial pacing was performed, beginning at a rate slightly faster than the resting heart rate. The pacing rate was gradually increased in increments of 10 beats/min until a rate of 150 beats/min was reached. A stable pacing position could not be found in patients 3 and 7, and this part of the study was not performed in these patients.

The electrogram recorded from the His bundle catheter, which represented activation of the His bundle, was accepted as such if the following criteria were met: the His recording catheter was in a typical position for recording the His deflection during all studies; the catheter position was stable; the His electrogram was a rapid bi- or triphasic deflection which was consistently present; an atrial electrogram was recorded with the His deflection; no other deflection which would give a longer H-V interval was recorded at any time from any bipolar lead pair. A longer H-V interval was especially sought during slow catheter withdrawals from the position in which the short H-V interval was obtained.

Results

Clinical and hemodynamic data are summarized in table 2. Postoperatively, right and left heart catheterization revealed normal intracardiac pressures and cardiac output in six of the seven patients. In patient 3, moderate pulmonary hypertension was present five months after surgery, but at a reduced level compared to the preoperative study. No residual shunt was detected by either oxygen saturation or hydrogen-sensitive catheter studies in any patient. Three patients continued to have a small amount of mitral insufficiency, as demonstrated by left ventriculography, but the degree of insufficiency was less than on preoperative angiograms.

A descriptive analysis of the pre- and postoperative ECGs is summarized in table 1. Typical tracings are shown in figure 1. All patients were in sinus rhythm, both pre- and postoperatively. The preoperative tracings showed the typical pattern associated with partial endocardial cushion defect-left axis deviation ($-20^\circ$ to $-80^\circ$).\textsuperscript{2,3} Right ventricular hypertrophy, right ventricular conduction disturbances and prolongation of the P-R interval were found in three patients. This has been noted previously in preoperative tracings.\textsuperscript{6} Postoperatively, the ECGs continued to reveal left axis deviation, while prolongation of the P-R interval and right ventricular conduction disturbances were less common.

The results of the His bundle studies are summarized in table 3. The P-A interval was normal in four patients and long in three. The A-H time was normal in all patients. In contrast, the H-V interval was not prolonged in any patient, and three patients had H-V times which were shorter than normal (fig. 2).

Right atrial pacing was performed in five of the

Table 2

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<th>Pt</th>
<th>Age (yr)</th>
<th>Sex</th>
<th>Follow-up (months)</th>
<th>Medications</th>
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Abbreviations: Age = age at time of follow-up; RA = right atrial pressure A wave/V wave/mean; PA = pulmonary artery pressure systolic/diastolic/mean; PAW = pulmonary artery wedge mean pressure; CI = cardiac index; NYHA = New York Heart Association functional classification.
seven patients. The A–H time gradually lengthened in a normal fashion in all five patients, but no episode of atrioventricular block was observed, as is frequently the case in normal hearts paced to this rate.\(^5\) The P–A and H–V intervals remained unchanged compared to control recordings. Figure 3 demonstrates the response to pacing in patient 4, who had a short H–V interval in the resting recordings.

**Discussion**

These studies were undertaken to determine the pattern of atrioventricular conduction in patients with incomplete endocardial cushion defects. In the past, the origin of the characteristic left axis deviation in the frontal plane has been a point of controversy. Blount et al.\(^3\) favored a hemodynamic explanation related to the mitral insufficiency usually found in these patients. Toscano-Barbosa et

| Table 3 |

**His Bundle Recording Results: Resting and Atrial Pacing**

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Abbreviations: HR = heart rate; R–R = cycle length between succeeding R waves; P–A = P wave-atrial interval; A–H = atrial-His interval; H–V = His-ventricular interval.
al.² favored an explanation based on a congenital anomaly of the atrioventricular conduction system. In order to eliminate, as much as possible, any hemodynamic effect of the atrial septal defect and mitral insufficiency, we performed our electrophysiologic studies in the late postoperative period. The hemodynamic studies at follow-up confirmed that only minor amounts of mitral insufficiency were present in three patients. All other patients had complete functional repair of the mitral valve, and no patient demonstrated a residual shunt. One patient did have persistent pulmonary hypertension five months after surgery, but the level of pressure elevation was markedly decreased compared to the preoperative study. While it is conceivable that the long-standing hemodynamic abnormalities led to permanent change in the conduction system, we think this is unlikely. In most patients, at least 2½ years elapsed between surgery and restudy to allow ample time for recovery from the hemodynamic insult. Therefore, we conclude that hemodynamic abnormalities did not influence atrioventricular conduction.

In view of the fact that the H-V interval was short in three of our patients, an objection to our results might be raised that the His recording catheter electrodes did not record a His deflection, but a right bundle branch depolarization. We utilized a number of criteria, as outlined in the Methods section, attempting to establish that our His deflection did come from this structure. Unfortunately, we did not attempt to confirm the recording site utilizing pacing by way of the His bundle catheter. However, it would be uncommon to record only a right bundle branch deflection and no second deflection representing His bundle activation, when recording from two pairs of bipolar leads simultaneously.¹⁰ This would be especially true since the area expected to yield the His deflection was repeatedly explored with the catheter. At no time was a second deflection representing right bundle branch depolarization recorded.

Intra-atrial conduction, as determined by the P–A interval, was completely normal in four patients, while three patients had abnormally prolonged atrial conduction intervals. Since none of the patients were taking any medication at the time of study, it is not possible to invoke a drug effect as causing these abnormalities. In addition, the A–H interval was normal in all patients, while atrial pacing in five patients at gradually increasing rates produced a graded increase in the A–H interval in all of the patients and no episodes of heart block occurred. This gradual prolongation of the A–H interval is the normally expected response to atrial pacing⁶ and is compatible with completely normal A–V nodal function.

These findings suggest that intra-atrial and atrioventricular conduction in the postoperative patient with an endocardial cushion defect is not always normal, in spite of the fact that the P–R interval on the ECG may be within normal limits. The fact that increases in the P–A interval may be minimal, coupled with the short H–V interval found in some of these patients, may effectively mask the prolonged intra-atrial conduction, as demonstrated in patients 4 and 5.

The disordered intra-atrial conduction found in this type of patient is probably related to multiple factors. The trauma of surgery, including an
atriotomy and pericardial patching of the atrial septal defect, might interfere with normal intra-atrial conduction. However, relief of the hemodynamic burden might be expected to reduce right atrial size, and therefore decrease internodal conduction time.

Preoperatively, P–R prolongation is a common finding in these patients. At this time, disordered conduction might be related to at least four factors: there may be anomalies of the specialized atrial conduction pathways existing as an intrinsic part of the incomplete endocardial cushion defect, the large atrial septal defect might interfere with conduction through the regular atrial musculature, the pathway between the sinus and atrioventricular nodes might be anatomically long. Indeed, Feldt et al. summarized previous anatomical studies and did studies of their own showing that the A–V node in patients with both the incomplete (partial) and complete forms of endocardial cushion defect is displaced in a postero-inferior direction, which would tend to increase the internodal distance. Finally, the long-standing hemodynamic burden imposed on the heart by the large interatrial shunt and mitral insufficiency would tend to produce an enlarged right atrium, again leading to a longer internodal conduction distance.

Recently, Waldo et al. demonstrated by right atrial recording during surgery that the prolonged P–R interval in patients with an endocardial cushion defect is due to abnormal internodal conduction; and Anderson et al. using His bundle recording, showed that intra-atrial conduction defects account for P–R interval prolongation in patients with ostium secundum defects.

The studies of Feldt et al. which have demonstrated anomalies of the ventricular specialized conduction system, help to explain the short H–V interval found in three of our patients (table 3). These investigators have shown that there is a relatively short distance between the atrioventricular node and the origins of the entire left bundle branch system. In addition, there is a marked postero-inferior displacement of the left bundle branch system and the anterior branches of the left bundle are "relatively hypoplastic."

These anatomical findings are directly applicable to the electrophysiologic results and electrocardiographic findings in our patients. The H–V interval represents the conduction time through the shortest functional pathway from the His bundle to that portion of the ventricles activated first. In three of our patients, the H–V interval was distinctly short. This finding was described by Miller et al. in preoperative studies of patients with ostium primum defect. In the anatomical setting in which there is a relatively short distance between the A–V node and the origin of the left bundle branch system, it might be expected that activation of the left ventricle would occur early. In addition, the left bundle branch system is markedly displaced postero-inferiorly. This would effectively shorten the length of the posterior fascicle so that excitation of the ventricular muscle normally activated by this structure, i.e., the postero-basal wall of the left ventricle, would occur earlier than in any other part of the ventricles, producing a pre-excitation pattern. Durrer, Roos and Van Dam demonstrated early epicardial excitation of the postero-basal region of the left ventricle in the hearts of four patients with ostium primum defects studied at the time of surgical correction. They suggested that the left axis deviation in the frontal plane of the ECG recording was caused "after the disappearance of outward spreading excitation forces in the postero-basal part of the left ventricular wall by now unopposed excitatory forces in the lateral and anterior parts of this ventricle."

The finding of a normal H–V interval in four of the patients is consistent with lack of early activation of the postero-basal wall of the left ventricle. As discussed above, Feldt et al. have shown that the anterior branches of the left bundle branch system are "relatively hypoplastic." Conduction through these branches might be expected to result in the distribution of excitation to a relatively smaller area of myocardium at any given time, when compared to the normal. This would be the functional equivalent of a left anterior fascicular block which is characteristically associated with left axis deviation. Burchell, DuShane and Brandenburg, using epicardial recording techniques during surgery on patients with endocardial cushion defects, found late activation in the region of the anterior interventricular groove on the left ventricular surface. They could not demonstrate early excitation of any part of the left ventricle. These results would be expected in the situation of left anterior fascicular block.

Thus, two separate but related mechanisms appear responsible for the left axis deviation typically present in the ECG of a patient with an ostium primum defect. Both have anatomic bases and, as we have shown, are clearly unrelated to associated hemodynamics. In the patient with a
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short H–V interval, early activation of the postero-
basal wall of the left ventricle, in effect, results in a
relatively late activation of the anterior wall and
therefore a pattern of left axis deviation. In those
patients having a normal H–V time, the relatively
hypoplastic anterior branches of the left bundle
branch would be invoked as the cause of the left
axis deviation, again resulting in the relatively later
activation of the anterior, as compared to the
postero-basal wall of the left ventricle. Obviously,
in patients with a short H–V interval, both early
activation of the postero-basal left ventricle and
hypoplasia of the anterior branches of the left
bundle branch may be operant factors. Although it
might be expected that the degree of left axis devi-
ation would be greater, this was not true of our
patients.

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DANIEL J. GOODMAN, DONALD C. HARRISON and DAVID S. CANNOM

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