Reentry Within the Atrioventricular Node: Surgical Cure with Preservation of Atrioventricular Conduction

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SUMMARY Paroxysmal supraventricular tachycardia (PSVT) is commonly caused by reentry within the atrioventricular (AV) node. This arrhythmia was abolished by operative dissection of the AV junction in a patient with disabling tachycardia that was not controlled by drugs. The operation was intended to create complete AV block, but AV conduction persisted after surgery. An electrophysiologic study 1 year after the operation revealed that the operation changed AV conduction in both the antegrade and retrograde directions, which may explain the absence of tachycardia. The patient has been free of arrhythmias for 18 months.

Paroxysmal supraventricular tachycardia (PSVT) caused by reentry using an accessory atrioventricular (AV) pathway has been successfully treated with surgical division of the accessory pathway when drug therapy has failed. This operation is performed without damaging the normal AV conduction system. In contrast, some patients who have PSVT caused by reentry within the AV node have been treated with destruction of the normal AV conduction system. These patients require permanent ventricular pacemakers. We recently operated on a patient who had tachycardia caused by reentry within the AV node that was refractory to medical management. The patient has been free of arrhythmias since surgery, despite the fact that AV block did not result from the surgery.

Case Report

The patient was a 50-year-old male. Palpitations began in 1966 and were shown to be due to PSVT (fig. 1). He was treated with digoxin (0.25 mg/day), propranolol (240 mg/day), and quinidine sulfate (1200 mg/day). No combination of these drugs was found which could prevent PSVT. During attacks of PSVT, he was unable to perform his customary duties as a construction worker and had to quit his job.

In 1974, angiography of the coronary arteries and left ventricle revealed no abnormalities. The tachycardia was studied by programmed stimulation in 1975 and was attributed to reentry within the AV node. An ECG recorded on his first admission to Duke University in December 1976 showed sinus rhythm, first-degree AV block, and nonspecific ST-T-wave changes. On the third hospital day an electrophysiologic study was performed.

Preoperative Electrophysiologic Study

Antiarrhythmic medication was discontinued 1 week before the study. No premedication was given. Multipolar electrode catheters were used to record simultaneously from the right atrium, right ventricle, bundle of His, and coronary sinus. Programmed electrical stimulation was used to perform fixed-rate pacing and measure refractory periods with the extrastimulus technique.

Conduction intervals recorded at the beginning of the study are shown in table 1. Wenckebach conduction occurred during fixed-rate pacing of the right atrium at cycle length 490 msec and during fixed-rate pacing of the coronary sinus (left atrium) at cycle length 480 msec. Single atrial echo beats were initiated during Wenckebach cycles. Sustained tachycardia was not initiated by fixed-rate pacing of either atrium. During fixed-rate pacing of the right ventricle between cycle lengths 700 msec and 320 msec the ventricular atrial interval (measured from the earliest recorded ventricular electrogram to the lateral atrium) increased only from 235 msec to 270 msec. With a cycle length of 300 msec, 2:1 retrograde block occurred.

Refractory periods of the AV conduction system in the antegrade direction were measured using the ex-
trastimulus technique (fig. 2). Single atrial echo beats were induced at many coupling intervals, but their occurrence did not always coincide with the longest H1-H2 intervals observed at that coupling interval. Premature beats that were not conducted to the His bundle (i.e., were blocked in the AV node) never initiated atrial echo beats. The refractory period of the AV conduction system in the retrograde direction was measured during right ventricular stimulation at a cycle length of 600 msec. Ventriculoatrial intervals were nearly constant at coupling intervals longer than 400 msec. With coupling intervals shorter than 400 msec, the ventriculoatrial interval gradually increased until ventricular refractoriness was reached. No echo beats were initiated by right ventricular extrastimuli, and no retrograde His bundle electrograms were recorded.

Since sustained tachycardia was not initiated, an endocardial map of the sequence of right atrial activation was obtained by repetitively inducing single echo beats with coronary sinus extrastimuli at a coupling interval of 640 msec. The shortest ventriculoatrial interval of the echo beats was 40 msec (H: Ae interval, 95 msec); this was recorded when the modified Brockenbrough mapping catheter was positioned against the interatrial septum below the foramen ovale. The remainder of the tricuspid annulus and the coronary sinus were activated later, a pattern consistent with maps obtained during ventricular pacing in individuals who have retrograde conduction over the AV node.

Atropine 1.0 mg was given intravenously in two equal boluses. Brief runs of tachycardia were initiated by bursts of rapid atrial pacing (fig. 3). The ventriculoatrial interval measured to the lateral right atrium was 50 msec. The atrial electrogram recorded from the catheter recording the bundle of His was obscured by ventricular depolarization. The tachycardia terminated by block of an atrial echo beat in the AV node.

Table 1. Basic Conduction Intervals

<table>
<thead>
<tr>
<th></th>
<th>12-21-76—Preoperative (msec)</th>
<th>2-10-77—Postoperative (msec)</th>
<th>1-18-78—Postoperative (msec)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sinus cycle length</td>
<td>880</td>
<td>610</td>
<td>600</td>
</tr>
<tr>
<td>PA interval</td>
<td>35</td>
<td>80</td>
<td>50</td>
</tr>
<tr>
<td>AH interval</td>
<td>180</td>
<td>205</td>
<td>140</td>
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<tr>
<td>HV interval</td>
<td>55</td>
<td>55</td>
<td>60</td>
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<tr>
<td>His duration</td>
<td>15</td>
<td>15</td>
<td>15</td>
</tr>
<tr>
<td>QRS morphology</td>
<td>Normal</td>
<td>Normal</td>
<td>Right intraventricular conduction delay</td>
</tr>
</tbody>
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node. The tachycardia was not sustained long enough to study the ability of induced premature ventricular depolarizations to induce atrial preexcitation.

Operative Findings

Because the tachycardia had disabled the patient he elected to undergo operative creation of AV block. On January 31, 1977, we operated on the patient. Partial cardiopulmonary bypass was begun, atropine 2.0 mg was administered, and tachycardia was initiated. The earliest point of atrial activation was identified by epicardial mapping, and it was the medial right atrium anterior to its junction with the atrial septum.

The patient was then placed on complete cardiopulmonary bypass, and a right atriotomy was performed. A small His bundle electrogram was recorded transiently. An incision was then made in the septal portion of the right atrial wall about 3 mm above the annulus fibrosus, beginning anterior to the ostium of the coronary sinus and continuing in the posterior direction for a distance of 1.5 cm. This incision exposed the triangular space on top of the muscular ventricular septum. With this exposure we again attempted to locate the His bundle with the probe electrode, but a His bundle electrogram was not recordable. The incision was extended in the anterior direction until the endocardium was divided beyond the union of the atrial septum and the free wall of the right atrium. This incision exposed a small area of the right fibrous trigone (fig. 4). Despite this dissection, AV conduction in the antegrade direction persisted. The PR interval was now longer than that recorded at the beginning of the study. The surgical team decided to do no more dissection because they did not want to extend further the patient's time on cardiopulmonary bypass. A permanent ventricular pacemaker was inserted as prophylactic treatment for postoperative AV block. The atrium and chest wall were closed using routine methods.

Postoperative Observations

An ECG recorded the night after the operation showed sinus rhythm and normal QRS morphology. The PR interval was 0.22 second. Subsequent ECGs

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**Figure 2. Refractory period of the atrio-ventricular node. A) Right atrial stimulation (RA stim) at cycle length 800 msec. Note that with coupling interval 600 msec the shortest H1-H2 intervals initiated echoes. B) Coronary sinus stimulation (CS stim), cycle length 800 msec.**
FIGURE 3. Tachycardia due to reentry within the atrioventricular node initiated by rapid atrial stimulation. RV = right ventricular electrogram; RA = right atrial electrogram; HBE = His bundle electrogram; prox. CS = proximal coronary sinus electrogram; dist. CS = distal coronary sinus electrogram. Time = time intervals of 10 and 100 msec. The atrial electrogram recorded from the catheter recording the His bundle electrogram was obscured by ventricular depolarization. The ventriculoatrial interval measured to the lateral right atrium was 50 msec.

FIGURE 4. Operative dissection of the atrioventricular (AV) junction. CS = coronary sinus; VS = ventricular septum; LA = left atrium; AS = atrial septum; RFT = right fibrous trigone; AMVS = atrial extension of the membranous ventricular septum. The incision began posterior to the orifice of the coronary sinus and extended in the anterior direction beyond the beginning of the free wall of the right atrium. The dotted line indicates the left annulus fibrosus and the anterior right annulus fibrosus. The AV node joins the penetrating His bundle at the level of the annulus fibrosus.

showed a variety of rhythmic patterns: a junctional rhythm with AV dissociation, sinus rhythm with a prolonged PR interval, and ventricular pacing during periods of sinus slowing.

On February 10, 1977, a limited electrophysiologic study was performed. The conduction intervals are shown in table 1. Compared with the preoperative study, the PA interval had increased from 35 to 80 msec. No stimulation studies were performed. No antiarrhythmic drugs were prescribed.

In the 18 months after discharge no spontaneous arrhythmias were observed by the patient. He returned for repeat examination in January 1978. Sinus rhythm was present, and the PR interval was 0.20 second. The QRS duration was 0.09 second, and a QR complex was recorded from lead V1.

On January 18 an electrophysiologic study was performed using the techniques previously described. Conduction intervals at the beginning of this study are shown in table 1. During fixed-rate pacing of the right atrium, Wenckebach conduction in the AV node occurred at a cycle length of 530 msec. No echo beats were initiated during Wenckebach cycles. During fixed rate pacing of the right ventricle, an unexpected phenomenon was observed. The curve of the relationship between ventriculoatrial intervals and pacing cycle length was flatter between cycle lengths 600 msec and 420 msec. After 16 beats of pacing at cycle length 400 msec, the ventriculoatrial interval suddenly shortened. Subsequent beats were conducted with alternating long and short ventriculoatrial intervals (fig. 5). This alternation persisted at all cycle lengths down through 320 msec. At a cycle length of 300 msec, we saw only the shorter of the two intervals. Cy-
FIGURE 5. Fixed-rate pacing of the right ventricle (RV), cycle length (CL) 400 msec, postoperative study. Shown are ECG leads I, II, V₁, and V₆. LLRA = low lateral right atrium; HBE = His bundle electrogram; S = stimulus. Ventriculoatrial (VA) intervals are indicated above the LLRA electrogram. This segment was recorded after pacing for 16 beats at CL 400 msec. The fourth ventricular beat was conducted to the atrium with a shortened ventriculoatrial interval. Subsequent alternate ventriculoatrial intervals were similarly abbreviated. Pacing cycle lengths longer than 400 msec showed constant VA intervals. Cycle lengths shorter than 300 msec were not tested. Repeated observations during fixed-rate pacing were made to confirm the alternation of these intervals. During fixed-rate pacing of the right ventricle, the ventriculoatrial interval increased from 200 msec at a cycle length of 600 msec to a maximum of 255 msec at a cycle length of 320 msec. Neither echo beats nor tachycardia was induced by right ventricular pacing at a fixed rate. A retrograde His bundle electrogram was not recorded.

FIGURE 6. Refractory period of the atrioventricular conduction system in the retrograde direction, postoperative study. Two distinct populations of ventriculoatrial intervals were observed with coupling intervals shorter than 550 msec. RV Extra-stim = right ventricular extrastimulus; CL = cycle length; LLRA = low lateral right atrium.
The extrastimulus technique was used to measure the refractory period of the AV conduction system in the antegrade direction at a cycle length of 600 msec. The preoperative pacing cycle of 800 msec was not used because the patient’s sinus cycle was now 690 msec. Unlike the preoperative study, no atrial echo beats were initiated.

The refractory period of the AV conduction system in the retrograde direction was measured using the extrastimulus technique during pacing of the right ventricle. With coupling intervals shorter than 550 msec, two distinct clusters of ventriculoatrial intervals were observed (fig. 6). Repeated coupling intervals within a narrow range produced two distinct ventriculoatrial intervals. Coupling intervals longer than 540 msec were accompanied by longer ventriculoatrial intervals than coupling intervals between 540 msec and 400 msec. With coupling intervals shorter than 400 msec there was a gradual increase in ventriculoatrial intervals. No echo beats were initiated by extrastimuli in the right ventricle, and no retrograde His bundle electrograms were recorded.

Discussion

The etiology of the tachycardia in this patient probably was reentry within the AV node. The sequence of atrial activation during tachycardia was determined using endocardial catheter mapping, and was consistent with activation in the retrograde direction beginning at the AV node. The ventriculoatrial interval (recorded below the foramen ovale) was 40 msec, which excluded the possibility that an accessory AV pathway was the retrograde limb of the reentry circuit. However, we did not find dual AV nodal pathways that are found in many patients with PSVT and reentry within the AV node. Reentry within the atrium seems unlikely, because premature depolarizations that were blocked in the AV node never initiated echo beats.

An increased PA interval was recorded in the postoperative study. This abnormality indicates prolonged intra-atrial conduction time, probably secondary to the dissection of the medial right atrium. If the atrium had been an essential link in the reentry circuit, then the dissection might have disrupted the delicate time relations required to maintain tachycardia. This observation is, therefore, one explanation for the abolition of tachycardia postoperatively. However, the atrium may not have been a necessary link in the PSVT circuit.

The postoperative studies also showed an incomplete right bundle branch and an abnormality of AV conduction in the retrograde direction. This abnormality was a type of conduction called bradycardia-dependent block. No such finding was observed in the preoperative study. The exact site of the abnormality could not be determined, but possibilities were the right bundle branch, the AV node and the atrium. Since the postoperative ECG showed a right intraventricular conduction delay, the most probable site of bradycardia-dependent block in the retrograde direction was the right bundle branch. However, an isolated abnormality of the right bundle branch does not explain the absence of tachycardia. If the site of phase 4 block were the AV node and included the retrograde limb of the tachycardia circuit, then that abnormality could explain the abolition of the tachycardia. Unfortunately, the inability to record a retrograde His bundle electrogram prevented precise localization of the site of block. We do not know which conduction abnormality recorded in the postoperative study accounted for the cure of the tachycardia.

The complete cure of the tachycardia in this patient was fortuitous, but the postoperative electrophysiologic data provides plausible explanations for this result. Our findings suggest that surgery may abolish supraventricular arrhythmias without simultaneously creating complete AV block and the need for a pacemaker. Operating on specific portions of the conduction system for correction of intractable arrhythmias, not associated with an accessory pathway, depends on development of more precise techniques of intraoperative mapping and more precise operative methods.

Acknowledgments

The authors express their gratitude to the many cardiology fellows who participated in these studies; to Laura Cook, R.N. and to Donald Kopp, L.P.N., the staff of The Clinical Electrophysiology Laboratory; to Robert L. Blake, Don Powell, and David Huggett who prepared the illustrations; to Ann Clayton who prepared the manuscript; and to Lou Ann Brewer for editorial assistance.

References

Early Closure of the Tricuspid Valve in a Case of Ebstein’s Anomaly with Type B Wolff-Parkinson-White Syndrome

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SUMMARY Echocardiographic studies of a 19-year-old female with Ebstein’s anomaly and type B Wolff-Parkinson-White syndrome showed an earlier closure of the anterior tricuspid leaflet (ATL) than of the anterior mitral leaflet (AML), contrary to the previous reports. The interval between the closure of the ATL and the AML was 20 msec and 30 msec, respectively, before and after administration of edrophonium chloride. However, closure of the AML preceded that of the ATL after administration of atropine sulfate and during supraventricular tachycardia by 10 msec and 60 msec, respectively, concomitant with the shortening of the duration of the QRS complex. We conclude that early closure of the ATL may depend on preexcitation of the right ventricle.

ECHOCARDIOGRAPHIC FEATURES of Ebstein’s anomaly include: 1) abnormally delayed closure of the anterior tricuspid leaflet (ATL); 2) increased amplitude of the ATL with abnormal E-F slope; 3) dilated right atrium; 4) ability to record the ATL over most of the left precordium; and 5) abnormally normal ventricular septal motion. The delayed closure of the ATL is the only feature specific for this anomaly, because the other findings may be present in the other diseases that produce right-sided volume overload. Usually, the interval between the closure of the anterior mitral leaflet (AML) and the ATL in Ebstein’s anomaly is greater than 30 msec. The abnormally delayed closure of the ATL is not considered due to right bundle branch block (RBBB) frequently present in this anomaly; it is considered a specific feature of the anomaly.

We report a case of Ebstein’s anomaly with the early closure of the ATL and investigate the etiology of this finding.

Case Report

A 19-year-old female was referred to our clinic for cardiac evaluation. She had been faintly cyanotic since infancy. She had had episodes of tachycardia approximately 10 times a year, but no episodes of heart failure.

At admission, height was 156 cm, weight was 50 kg, body temperature was 36.5°C, pulse rate was 60 beats/min, and blood pressure was 116/74 mm Hg. Cyanosis of nail beds with faint clubbing and malar flush were noted. Jugular veins were not distended. The apex beat was in the left fifth intercostal space midway between the midclavicular and anterior axillary line. Two components of the first heart sound and a single second heart sound were confirmed by phonocardiogram. Third and fourth heart sounds were also recorded. A grade 3/6 early systolic murmur was best heard in the fourth intercostal space at the left sternal border. A chest roentgenogram revealed normal pulmonary markings, a marked convexity of the
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Circulation. 1979;60:440-446
doi: 10.1161/01.CIR.60.2.440

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

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