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Curative Surgery for Atrioventricular Junctional ("AV Nodal") Reentrant Tachycardia

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A new surgical approach was studied prospectively in 10 consecutive patients with atrioventricular (AV) junctional reentrant tachycardia. The aim was to abolish tachycardia yet preserve normal AV conduction. On the basis of electrophysiologic study before operation, patients were classified as type A (ventriculoatrial [VA] intervals during tachycardia ≤40 ms) (seven patients) or type B (VA intervals >40 ms) (three patients). Dual AV junctional pathways were demonstrable with single extrastimulus testing in seven patients before operation. Endocardial mapping during tachycardia at surgery revealed earliest atrial activation anteromedial to the AV node in type A patients and posterior to the node in the type B patients. The perinodal atrium in the region of earliest atrial activation during tachycardia was carefully disconnected from the AV node.

After operation, AV junctional reentrant tachycardia was not inducible at comprehensive electrophysiologic study in any patient, and no clinical recurrences have occurred during a follow-up period of 2 to 14 months (mean 8 ± 4). Normal AV conduction was preserved in all cases. Anterograde slow AV junctional pathway conduction was abolished in five of seven cases. Retrograde His to atrium conduction time was prolonged in type A patients but the capacity for retrograde VA conduction remained excellent. Retrograde His to atrium conduction was interrupted or severely compromised in the type B patients.

These data show that there are at least two types of AV junctional reentry. Perinodal atrium appears to be part of the reentrant circuit in human AV junctional reentry. Although the most consistent effect of surgery was on the retrograde limb of the circuit, anterograde slow pathway conduction was also modified. AV junctional reentry is surgically curable with a high success rate.

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The components and precise location of the tachycardia circuit used in atrioventricular (AV) junctional reentrant (AV nodal) tachycardia are unknown and current opinion is divided. Some authors (1–8) favor the use of an AV node to atrium or His bundle to atrium connection as the retrograde limb of the usual anterograde slow pathway, retrograde fast pathway type of tachycardia. Others (9–19) have presented evidence suggesting that the atrium is not a necessary part of this tachycardia circuit and that the circuit is therefore intranodal. Because AV junctional reentrant tachycardia has not previously been considered suitable for selective surgery, the standard procedure for this arrhythmia is ablation of the His bundle with creation of heart block. A permanent pacemaker is usually inserted. This approach abolishes tachycardia but leaves residual problems and therefore is not an ideal treatment. However, accidental "cures" of AV junctional reentrant tachycardia have been reported during attempted His bundle ablation despite return of AV conduction (20,21).

We believed that the reentrant circuit in AV junctional reentrant tachycardia used a retrograde AV node to atrium or His bundle to atrium connection that was extranodal and that the perinodal atrium was part of the reentrant circuit. With this idea in mind, and encouraged by previously reported surgical "accidents" showing that it was feasible, we designed a new surgical procedure for AV junctional reentrant tachycardia the aim of which was to section the presumed perinodal connection yet preserve normal AV conduction. The successful application of this approach in a prospective series of 10 consecutive patients begun in October 1983 is described in this report. In addition, new insights were obtained regarding the mechanism and location of the tachycardia circuit.

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Table 1. Patient Characteristics

Case	Age (yr) & Sex	Duration of Symptoms (yr)	Average Frequency of SVT Episodes	Other Disease or Condition
1	43F	10	3–5/week	MVP
2	31F	26	≤5/week	—
3	34M	24	2–5/day	—
4	54F	43	3–10/day	—
5	33F	24	≤3/week	—
6	39F	29	1/2 months	MVP
7	23F	18	2–3/week	—
8	21F	5	4/week	Mercury overdose, gastrectomy
9	23F	4	1/2 months	—
10	10M	6	≤5/day	—

MVP = mitral valve prolapse; SVT = supraventricular tachycardia.

Methods

Patients (Table 1). All 10 patients had highly symptomatic recurrent episodes of AV junctional reentrant tachycardia. The indication for surgery was unresponsiveness to vigorous medical therapy with a wide range of conventional antiarrhythmic drugs (nine patients) or patient preference for surgical treatment rather than long-term antiarrhythmic drug therapy (one patient). Each patient gave informed consent for the operation after the experimental nature of the surgery and its possible outcomes had been fully explained.

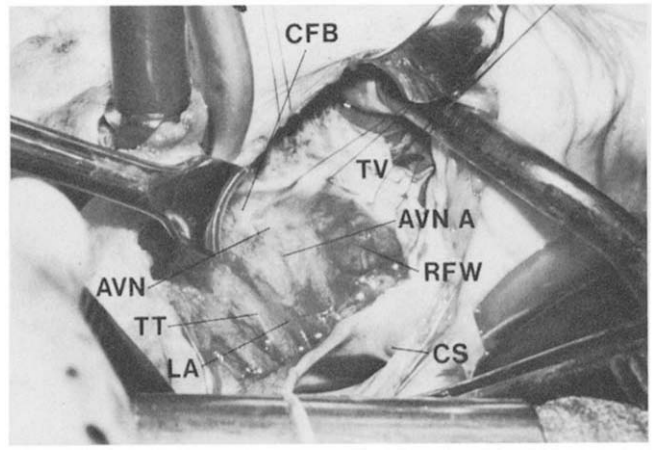
Preoperative electrophysiologic studies. Our techniques for electrophysiologic study have been described in detail (22). All cardioactive medications were stopped 1 week before electrophysiologic study. Diazepam (10 mg orally) was used for premedication. Atropine (0.5 mg intravenously) was used to facilitate induction of sustained AV junctional reentrant tachycardia if the tachycardia was not sustained at electrophysiologic study (23).

Our criteria for the diagnosis of AV junctional reentrant tachycardia and the means by which it can be distinguished from atrial and concealed accessory ventriculoatrial (VA) connection tachycardias have been described in detail (24). In brief, diagnosis required 1) a septal sequence of atrial activation during tachycardia; 2) inability to advance atrial activation during tachycardia by a right ventricular apical extrastimulus delivered 50 ms or less before anterograde tachycardia His bundle activation; and 3) the continued linking of tachycardia atrial activation to prior His bundle activation despite prolongation of the AH interval induced by atrial extrastimuli. Onset of atrial activation during tachycardia before the onset of ventricular activation was a useful additional factor in excluding the use of a septal accessory VA connection in the tachycardia circuit when this finding was present.

Surgical technique and operative mapping. The approach was through a midline sternotomy with conventional cardiopulmonary bypass using a bubble oxygenator. A transverse submammary skin incision was used in three female patients for cosmetic reasons. Both venae cavae were cannulated using right angled cannulas to give maximal access to the right atrium. A bipolar clip electrode was attached to the left atrial appendage as an atrial reference electrode and for atrial pacing. A bipolar button was sutured to the anterior wall of the right ventricle for use as the ventricular reference electrode and for ventricular pacing. Cardiac mapping using hand-held malleable probes (bipolar electrodes 1 mm apart) was performed during normothermic cardiopulmonary bypass. Epicardial mapping during sinus rhythm and left atrial pacing was carried out to confirm that ventricular pre-excitation was not present. Epicardial atrial mapping was then performed during supraventricular tachycardia. The right atrium was then opened by a long oblique incision to expose the tricuspid valve and coronary sinus. The right atrial endocardium was then mapped during AV junctional reentrant tachycardia using a hand-held probe. A fine needle probe (0.3 mm diameter bipolar facial electromyogram needle) was then used to locate with greater accuracy the earliest site of atrial activation during tachycardia.

After location of the earliest site of atrial activation during tachycardia, the patient was cooled on bypass to 28°C, the aorta was cross-clamped and potassium cardioplegia was infused. Stay sutures were placed accurately in the posterior tricuspid anulus, and with a still, dry field an incision was made through the right atrial wall 2 mm above the tricuspid anulus, starting just lateral to the mouth of the coronary

Figure 1. Operative photograph. The right atrium has been opened and the endocardium incised just above the posterior tricuspid anulus to expose the posterior space. The posterior anulus of the tricuspid valve (TV) has been pulled forward by stay sutures, and the tendon of Todaro (TT) has been retracted medially. The central fibrous body (CFB), atrioventricular node (AVN) and its artery (AVN A), the right ventricular free wall (RFW), left atrial (LA) myocardium and the coronary sinus (CS) orifice are visible.



sinus and continuing medially toward the apex of the triangle of Koch. The coronary sinus end of this incision was then dissected immediately onto the right ventricular free wall, and once this plane was identified the wall of the coronary sinus was dissected from the fat in the posterior space. This dissection gradually exposed the wall of the left atrium, the artery to the AV node, the central fibrous body and the tendon of Todaro (Fig. 1). Subsequent dissection depended on the site of earliest activation during tachycardia. In seven patients with AV junctional reentrant tachycardia designated as type A at electrophysiologic study before operation (see later for definition), atrial activation in tachycardia was earliest at the apex of the triangle of Koch. In this group, the fat surrounding the artery to the AV node and the node itself were not dissected, but the right atrial wall was carefully reflected anteriorly to expose the central fibrous body. The tendon of Todaro was divided where it inserted into the central fibrous body. The central fibrous body anteromedial to the node and the left atrial wall medial to the tendon were scraped clean with a scalpel. This dissection was continued posteriorly along the left margin of the pyramidal space as far as the coronary sinus. The posterior approaches of the AV node were preserved.

A second subgroup of three patients with AV junctional reentrant tachycardia designated as type B at preoperative electrophysiologic study (see later) had earliest atrial activation during tachycardia along the posterior border of the triangle of Koch near the coronary sinus. In these type B patients the free wall of the right ventricle was dissected clean from the tricuspid anulus to the epicardium up to the lateral limits of the AV node and beneath the latter into the interventricular groove. The inferior wall of the coronary sinus was dissected clean to the epicardium commencing at the mouth of the sinus and continuing medially to the left atrial wall. The medial approaches of the AV node were left intact.

After dissection, the endocardial right atrial incision was closed with 4-0 Prolene taking care to avoid the conduction tissue. After closure of the right atrial free wall, rewarming and restoration of sinus rhythm, AV and VA conduction were assessed by incremental atrial and ventricular pacing. Cardiopulmonary bypass was then discontinued, and temporary atrial and ventricular pacing wires were attached.

Postoperative investigations and follow-up. Repeat electrophysiologic studies were performed 1 week after surgery before hospital discharge. All patients will have an additional postoperative electrophysiologic study within the next 12 months. Five patients have been restudied at the time of writing. At the postoperative electrophysiologic studies, anterograde and retrograde conduction studies were performed using single extrastimulus testing at three different basic cycle lengths (as close as practicable to 600, 500 and 400 ms). Incremental atrial and ventricular pacing were then performed to the point of second degree block. Atropine, 1.0 mg intravenously, was administered and anterograde and retrograde conduction studies were repeated. Burst pacing was then used in attempts to induce tachycardia. After hospital discharge, no patient received antiarrhythmic medication and all patients were followed up closely.

Results

Preoperative electrophysiologic characteristics. Dual anterograde AV junctional pathways were evident with single atrial extrastimulus testing at a basic cycle length of 600 ms in 7 of the 10 patients. Retrograde dual AV junctional pathways were evident during single ventricular extrastimulus testing at a basic cycle length of 600 ms in only one patient. The AH interval during sinus rhythm was less than 60 ms (that is, "enhanced AV nodal conduction") in 6 of the 10 patients.

Table 2. Supraventricular Tachycardia Characteristics in 10 Patients

Case	Cycle Length (ms)	AH Interval (ms)	VA Interval (ms)	Site of Earliest Atrial Activation at Catheter EPS	Latest Ventricular Extrastimulus Advancing Atrial Activation During Tachycardia (ms before expected His electrogram)
1	320	274	+10*	His bundle region	<VFRP
2	340	314	−12†	His bundle region	<VFRP
3	310	272	0	His bundle region	<VFRP
4	330	290	−8†	His bundle region	70 ms before H
5	416	360	0	His bundle region	62 ms before H
6	286	228	−16†	His bundle region	56 ms before H
7	390	336	0	His bundle region	52 ms before H
8	544	406	+64*	His bundle region	92 ms before H
9	400	264	+76*	Proximal region of CS	<VFRP
10	360	260	+48*	Proximal region of CS	84 ms before H

*After QRS onset; †before QRS onset. VA intervals were measured from onset of QRS to earliest onset of atrial activation. CS = coronary sinus; EPS = electrophysiologic study; H = His bundle electrogram; VFRP = right ventricular functional refractory period.

The characteristics of the induced AV junctional reentrant tachycardia at electrophysiologic study are shown in Table 2. Three patients required atropine, 0.5 mg intravenously, for induction of sustained tachycardia at preoperative electrophysiologic study. Patients with a VA interval of 40 ms or less during AV junctional reentrant tachycardia were designated as type A. All seven type A patients (Cases 1 to 7) had earliest atrial activation during tachycardia in the His bundle lead (Fig. 2). Patients with a VA interval greater than 40 ms were designated as type B (Cases 8 to 10). Two of these three patients had earliest atrial activation during tachycardia recorded in the proximal coronary sinus (Fig. 3).

Surgical mapping. Tachycardia was not inducible intraoperatively in one patient. Sustained AV junctional reentrant tachycardia was inducible for both epicardial and endocardial mapping in five patients, for epicardial mapping only in one patient, and for endocardial mapping only in three patients. Atropine, 0.5 to 1.0 mg, or isoproterenol, 5 to 15 μg, or both, was required to facilitate induction of tachycardia in six patients.

Figure 2. Electrophysiologic tracings before operation. Typical example of type A AV junctional reentrant tachycardia (Patient 2). Electrograms from the high right atrium (HRA), proximal (CS_p) and distal (CS_d) coronary sinus, His bundle (HIS) and right ventricular (RV) apex are shown with surface Frank electrocardiographic leads X, Y and Z. All measurements are in milliseconds. The onset of ventricular activation is shown by the **dotted line**. Atrial activation (A) during tachycardia is earliest in the His bundle lead and precedes onset of ventricular activation. A high right atrial extrastimulus (S) prolongs anterograde slow pathway conduction and delays subsequent His bundle activation. Note that subsequent atrial activation remains linked to His bundle activation (H), although the HA interval is minimally shorter after the long cycle.

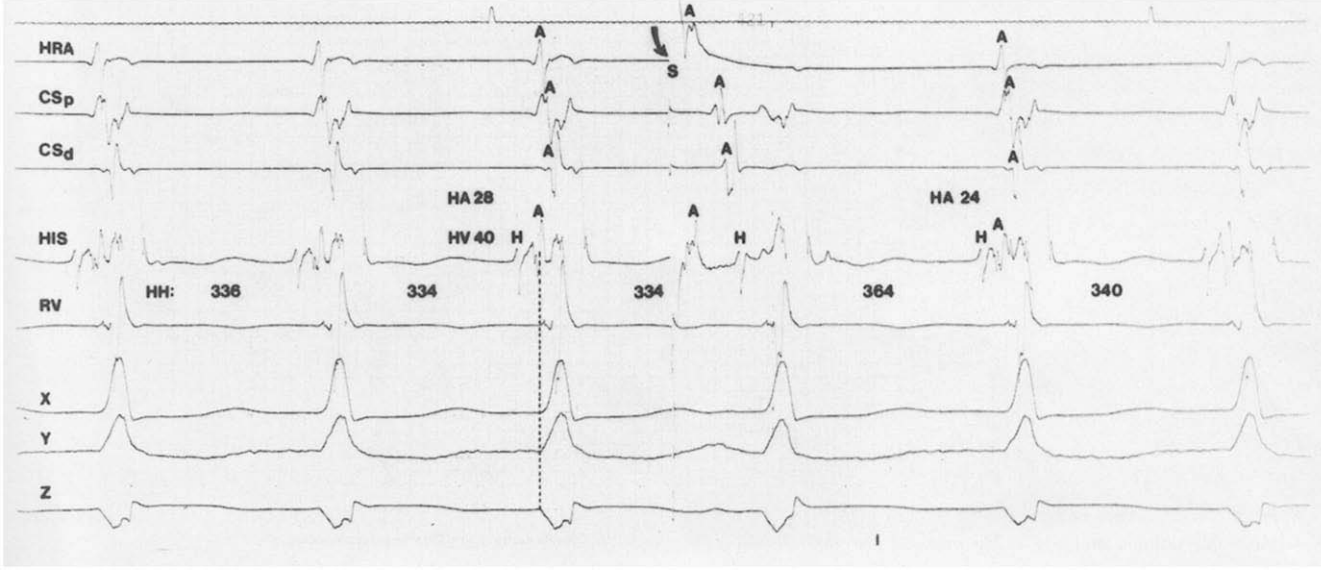
Epicardial atrial activation during tachycardia was earliest over the anterior atrial septum in four of four type A patients in whom it was recorded, and earliest over the posterior atrial septum in two of two type B patients.

The sites of earliest right atrial endocardial activation during tachycardia in the eight patients in whom it was recorded are shown in Figure 4A. Those with type A tachycardia (Cases 1 to 7) showed earliest atrial activation overlying the central fibrous body anterior or anteromedial to the AV node (Fig. 4B). Those with type B tachycardia (Cases 8 to 10) showed earliest atrial activation posterior to the AV node in the mid or posterior parts of the triangle of Koch, near the coronary sinus orifice (Fig. 4C). An unusually large thebesian vein was present at the earliest site in one patient (Case 8). In two patients (Cases 3 and 10) endocardial mapping was performed during right ventricular pacing because tachycardia could not be induced. The site of earliest atrial activation was anterior to the AV node in Patient 3 and posterior to the node in Patient 10 (Fig. 4A).

Comparison of Electrophysiologic Data Before and 1 Week After Operation

Induction of tachycardia. Whereas sustained tachycardia was inducible in all patients before operation, tachycardia (sustained or nonsustained) was not inducible after operation in any patient despite comprehensive testing and use of atropine.

Anterograde conduction. The effects of surgery were similar in all patients (Table 3). AV conduction was intact in all; the AH interval was mildly increased and the HV interval was unchanged. Slow anterograde pathway conduction was absent after operation despite intensive testing in five of the seven patients who had easily demonstrable dual AV nodal pathways before operation. The critical AH



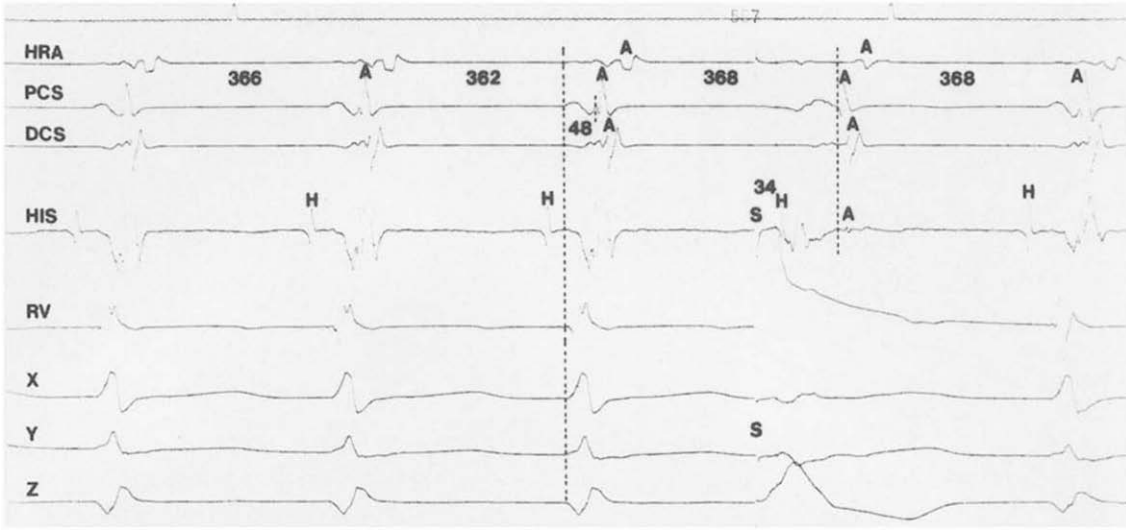


Figure 3. Example of type B AV junctional reentrant tachycardia (Patient 10). Tachycardia cycle length showed minor spontaneous variation from 360 to 372 ms. A ventricular extrastimulus delivered 34 ms before His bundle activation does not alter the timing of subsequent atrial activation (368 ms is within the range of spontaneous variability). However, it allows the sequence of atrial activation to be seen, revealing that earliest activation was in the proximal coronary sinus (PCS). Note that the VA interval is significantly longer than in type A tachycardia. DCS = distal coronary sinus electrogram; other abbreviations as in Figure 2.

interval for reentry could not be achieved after operation in 9 of 10 patients (this decreased to 8 of 10 patients when the available late postoperative data were included). Fast pathway AV nodal effective refractory period and functional refractory period were not altered. AV nodal effective refractory period was prolonged after operation because of loss of anterograde slow pathway function in most patients. The cycle length causing AV block during incremental atrial pacing was tested in only five patients before operation but in all patients after operation. AV block occurred at a cycle length of 390 ± 77 ms before operation, 424 ± 64 ms after operation (p = NS for the paired data) and 372 ± 79 ms after operation after atropine, 1.0 mg. One to one AV conduction was possible at rates faster than the rate of tachycardia in all patients after operation.

Dual AV junctional pathways remained present after operation in two patients. In one, the slow pathway AH interval was less than the "critical" AH interval necessary to induce AV junctional reentry in the preoperative study. Pre- and postoperative AV nodal function curves in the other patient (Case 4) are shown in Figure 5. Similar slow pathway AH interval and AV nodal effective refractory period could be produced postoperatively after atropine without inducing AV junctional echoes.

Retrograde conduction. *Type A patients.* In the seven patients with type A tachycardia, retrograde VA conduction was intact 1 week after operation in six patients and returned later in the seventh. Retrograde His bundle electrograms were visible during ventricular extrastimulus testing before and after operation in five of the seven patients. Retrograde His to atrium conduction times were determined at the longest ventricular extrastimulus interval that produced a clear His electrogram (Table 4). These times were prolonged after operation in four of these five patients despite similar basic cycle lengths, ventricular extrastimulus coupling intervals and S_2 to retrograde H_2 intervals.

VA effective refractory period was increased in three patients, unchanged in three and decreased in one patient (p = NS). The cycle length of ventricular pacing causing VA block was assessed before operation in only three type A patients (240, 350 and 270 ms before operation; 360, 310 and 300 ms after operation, respectively). One to one VA conduction was possible at rates faster than the preoperative rate of tachycardia in all patients. Type A patients developed second degree VA block during ventricular pacing at a cycle length of 316 ± 5 ms after operation and 265 ± 29 ms after atropine administration, 1.0 mg intravenously. VA conduction was therefore excellent.

Type B patients. VA conduction was absent after operation in two patients and VA block occurred with ventricular pacing at a cycle length of 520 ms in the third patient.

Late Postoperative Electrophysiologic Studies

These studies were performed 3 to 6 months after surgery. Only five patients have been studied so far. Tachycardia was not inducible in any patient despite intensive efforts including intravenous administration of atropine and isoproterenol. Studies to date confirm the findings of the early postoperative electrophysiologic study.

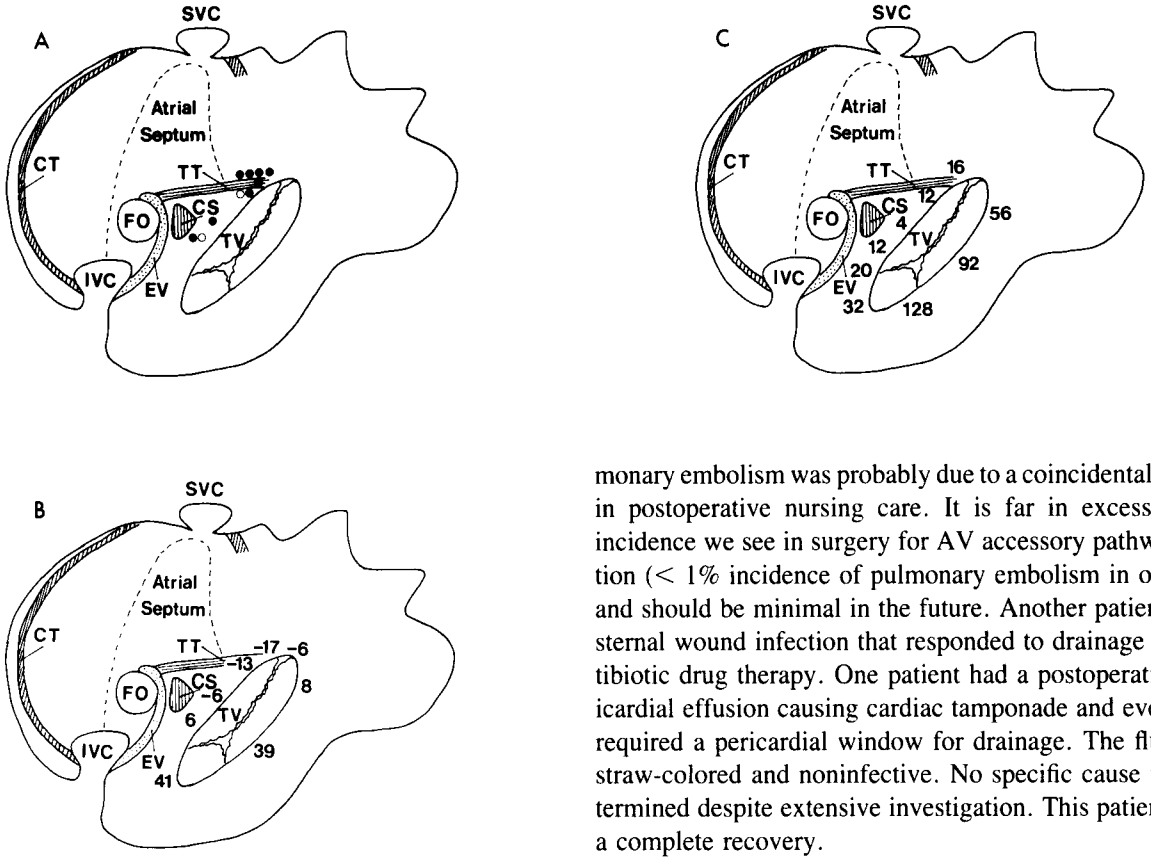


Figure 4. A, Location of the earliest sites of atrial activation during AV junctional reentrant tachycardia with right atrial endocardial mapping at surgery. All 10 patients are shown. This mapping diagram represents the endocardial surface of the right atrium as if it had been opened by an oblique incision in the anterolateral wall from the inferior vena cava (IVC) to the right atrial appendage and then laid open. **Black dots** represent early sites during tachycardia and **open circles** represent early sites recorded during ventricular pacing in those patients whose tachycardia was not inducible for endocardial mapping. Earliest atrial activation in type A tachycardia (seven patients) was clustered at the apex of the triangle of Koch, anterior or anteromedial to the AV node. Earliest atrial activation in type B tachycardia (three patients) was located posterior to the AV node, close to the coronary sinus (CS). **B,** Surgical map during type A tachycardia (Patient 2). Times represent VA intervals in milliseconds measured from onset of the QRS complex to the midpoint of the local atrial electrogram. **C,** Surgical map during type B tachycardia (Patient 8). Tachycardia was difficult to induce intraoperatively in this patient and required 1.0 mg of atropine and 15 μg of isoproterenol intravenously. Thus, the VA intervals were shorter than during the electrophysiologic study before operation. CT = crista terminalis; EV = eustachian valve; FO = foramen ovale; SVC = superior vena cava; TT = tendon of Todaro; TV = tricuspid valve.

Surgical Complications

There were no deaths. Two patients had a pulmonary embolus after operation. There were no long-term sequelae from the pulmonary emboli and both patients made an uneventful recovery. This unusually high incidence of pulmonary embolism was probably due to a coincidental change in postoperative nursing care. It is far in excess of the incidence we see in surgery for AV accessory pathway section (< 1% incidence of pulmonary embolism in our unit) and should be minimal in the future. Another patient had a sternal wound infection that responded to drainage and antibiotic drug therapy. One patient had a postoperative pericardial effusion causing cardiac tamponade and eventually required a pericardial window for drainage. The fluid was straw-colored and noninfective. No specific cause was determined despite extensive investigation. This patient made a complete recovery.

Clinical Outcome

Duration of follow-up study was 2 to 14 months (mean ± SD 8.1 ± 4). No patient was taking antiarrhythmic drugs. There have been no recurrences of AV junctional reentrant tachycardia. All patients have noted a remarkable improvement after surgery. One patient had two episodes of a slower, slightly irregular, different tachyarrhythmia. Repeat electrophysiologic study 5 months after surgery in this patient showed that AV junctional reentrant tachycardia remained noninducible. Atrial flutter was inducible (and had been inducible before operation). The induced atrial flutter reproduced her symptoms. Another patient was aware of atrial ectopic beats and felt as if the tachycardia "was about

Table 3. Comparison of Electrophysiologic Data on Anterograde Conduction in 10 Patients Before and 1 Week After Operation

	Before Operation	After Operation	p Value
Sinus cycle length (ms)	771 ± 145	626 ± 95	0.002
AH interval in SR (ms)	54 ± 15	66 ± 14	0.04
HV interval in SR (ms)	45 ± 8	43 ± 9	NS
Fast pathway AVN ERP (ms)	349 ± 81	324 ± 70	NS
AVN ERP (ms)	270 ± 32	317 ± 65	0.03
AVN FRP (ms)	420 ± 86	395 ± 74	NS

AVN = atrioventricular node; ERP = effective refractory period; FRP = functional refractory period; SR = sinus rhythm.

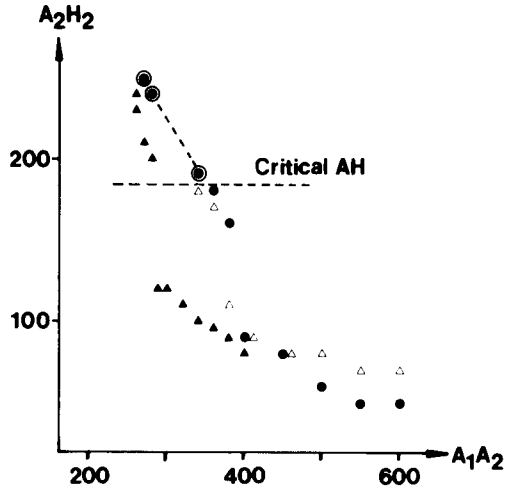


Figure 5. AV nodal function curves before and after operation (A_1A_2 versus A_2H_2 intervals) in Patient 4. **Black dots** show the curve before operation. Induction of AV junctional reentry is shown by a **circle around the dot.** When a critical AH interval of 190 ms was obtained, reentry occurred. The reentry zone and critical AH interval are represented by the **dashed line.** Postoperative data are shown by **triangles; open triangles** are baseline data and **closed triangles** represent the curve obtained after atropine. In the curve after operation, dual pathways are present, but the critical AH interval was not achieved before the AV nodal refractory period. However, after atropine AH intervals and AV nodal refractoriness similar to the curve before operation could be achieved without initiating reentry.

to start," but has remained free of tachycardia. One patient complained of recurrent episodes of palpitation (which he perceived to occur at a slower rate than before operation), but supraventricular tachycardia was never documented when he presented for medical attention. Repeat electrophysiologic study showed that AV junctional reentrant tachycardia was not inducible, and after extensive investigation the final diagnosis was anxiety-induced sinus tachycardia. This boy had emotional problems related to parental marital breakdown.

Discussion

New observations on AV junctional reentry. This study shows that there are at least two anatomically distinct types of AV junctional reentry. In type A, the earliest atrial activation in tachycardia is anteromedial to the AV node consistent with exit from the conduction system in the region of the central fibrous body. In type B, the earliest atrial activation is posterior to the AV node. Type A has a short conduction time for the retrograde limb ($\leq$ 40 ms) compared with type B ($>$ 40 ms). Other investigators (5–7,25–28) have shown that, in the majority of cases, the retrograde limb of the tachycardia circuit in AV junctional reentry has accessory pathway-like (not AV nodal) properties based on responses to drugs and perturbations of tachycardia cycle length. Their patients had short HA conduction times and are similar to our type A patients. In a minority of patients

Table 4. His to Atrium Conduction Times During Ventricular Extrastimulus Testing

	HA Interval (ms)		
Case	Before Operation	1 Week After Operation	Late Postoperation
1	80	130	No retro H
2	80	AVN block	110
3	80	No retro H (VA block)	110
4	60	90	—
5	70	70	—
6	No retro H	No retro H	—
7	No retro H	60	—
8	180	AVN block	—
9	200	AVN block	AVN block
10	100	AVN block	AVN block

H = His bundle electrogram; HA interval = onset of the His electrogram to midpoint of the atrial deflection; No retro H = retrograde His bundle electrogram not visible with ventricular extrastimulus testing; VA = ventriculoatrial; other abbreviations as in Table 3.

with AV junctional reentry, Gomes et al. (6) described decremental properties of the retrograde limb. The conduction times in their patients were generally long and suggestive of our type B patients and increased further after ouabain administration.

Sung and coworkers (29) described differing patterns of atrial activation in patients with retrograde dual AV junctional pathways. Fast pathway conduction activated the atrium earliest in the region of the His bundle, whereas slow pathway conduction activated the atrium earliest in the region of the coronary sinus. These differing exit sites correspond to our findings with type A versus type B AV junctional reentrant tachycardia.

Our surgical technique was directed at the perinodal atrium. Although some superficial damage to the AV node cannot be excluded, the total abolition of AV junctional reentry with preservation of normal AV conduction strongly suggests that the perinodal atrium is part of the reentrant circuit in AV junctional reentry. Iinuma et al. (8) showed utilization of perinodal atrium in the reentrant circuit of AV junctional echoes in the isolated rabbit heart, with exit points from the AV node adjacent to the interatrial septum in one type (similar to our type A) and adjacent to the crista terminalis and coronary sinus in the other type (similar to our type B). Earlier studies in dog and rabbit hearts (1–4) also concluded that the atrium was an essential part of the circuit in AV junctional reentry.

Mechanism of surgical cure of tachycardia. Long-term modification of the retrograde limb of the tachycardia circuit appears to be the major mechanism of cure in type B tachycardia. Although the usual type of VA accessory pathway was ruled out in these cases at electrophysiologic study before operation, the possibility needs to be considered of a VA accessory pathway with decremental properties, such as that used in "permanent" AV junctional reentry (30).

JACC Vol. 6, No. 6
December 1985:1383–92

With current electrophysiologic techniques it may be impossible to distinguish these pathways from others arising from the His bundle or above unless AV block develops during tachycardia. This latter phenomenon was not observed in our type B patients. However, the type B VA intervals in tachycardia were much shorter than those seen in "permanent" AV junctional reentry.

The mechanism of cure in type A tachycardia is not certain, but possible mechanisms are:

1. *Ablation or modification of an AV nodal or His bundle to atrium connection.* Observations in favor of this mechanism are: a) AV junctional reentry was totally abolished in all patients, yet AV and VA conduction were possible after operation at rates in excess of the tachycardia rate before operation. Thus, tachycardia was cured without major compromise of AV nodal function. b) His bundle to atrium conduction times after operation were prolonged at ventricular extrastimulus testing in four of the five patients in whom this could be measured both before and after operation. This finding is consistent with a different or modified route of postoperative His bundle to atrium conduction. c) Modification of anterograde slow pathway conduction was not necessary for operative success in one patient (Case 4). In this patient, critical slow pathway AH interval and AV nodal effective refractory period could be achieved postoperatively after atropine administration (Fig. 5). VA conduction in this patient was excellent postoperatively. The only detectable electrophysiologic difference, apart from noninducibility of tachycardia, was prolongation of the HA interval with extrastimulus testing, consistent with a different route of His bundle to atrium conduction. These findings strongly favor this hypothesis, but only occurred in one patient.
2. *Selective modification of anterograde slow AV junctional pathway conduction.* This was abolished or modified such that the critical AH interval for reentry was not achieved in four of five type A patients with easily demonstrable dual pathways at single extrastimulus testing before operation. The only exception was Patient 4. Thus, modification of the slow anterograde AV junctional pathway would explain most but not all operative successes. Holman et al. (31) showed that cryoablation of the perimeter of the triangle of Koch in the dog heart abolished slow anterograde pathway function in three animals with preoperative dual pathways. AV nodal effective refractory period was markedly prolonged in two of these three dogs, but was unchanged in the third. Our findings in the human heart are therefore similar.
3. *Nonspecific trauma to the AV junction.* The major effects of surgery were on anterograde slow AV junctional pathway function and retrograde fast pathway function. Anterograde fast pathway function was spared. AV conduction before operation was normal and permitted heart rates in excess of preoperative tachycardia rates, indicating no major damage to the AV node. The type A operation preserved excellent VA conduction after surgery, whereas this was absent or poor after the type B operation. These selective effects argue against nonspecific trauma to the region. It is also very unlikely that nonspecific trauma would be uniformly successful in abolishing tachycardia yet preserving AV conduction in 10 consecutive patients. The critical AH interval for reentry before operation could still be achieved after operation in two patients (one type A and one type B) without induction of reentry, suggesting that minor modification of AV nodal conduction was not the cause of success.
4. *Denervation of the AV junction.* AV nodal function after operation changed appropriately when challenged with atropine, demonstrating intact vagal innervation. Sympathetic nervous system function was not tested, but isoprenaline infusion produced expected effects on the AV node.

Clinical implications. Intentional selective abolition of AV junctional reentrant tachycardia at surgery with preservation of normal AV conduction has not previously been considered feasible. This report shows that such an operation can be performed with a high long-term success rate in a representative cross section of patients with AV junctional reentrant tachycardia. Thus, surgical cure of almost all patients with reentrant supraventricular tachycardia is now possible. Accessory pathway tachycardias, atrial tachycardias, nodoventricular fiber tachycardias and "permanent" tachycardias using a posterior septal VA accessory pathway with a long conduction time have been shown to be surgically curable (22,30,32–39). The addition to this list of AV junctional reentrant tachycardia, which comprises 45% of cases of supraventricular tachycardia, using an operation that results in minimal mortality and long-term morbidity, may well make surgery or other permanent ablative methods the treatment of choice in patients with significant symptoms. In our experience with AV and VA accessory pathway tachycardias, the majority of patients with significant symptoms prefer safe and definitive surgery over long-term antiarrhythmic drug therapy.

We should introduce one cautionary note. A very small subgroup of patients with AV junctional reentrant tachycardia may be unsuitable for this type of surgery. Those rare patients who show retrograde block to the atrium during AV junctional reentrant tachycardia (12,13,40–42) may indeed have intranodal reentry and may not be suitable for the approach we have described.

Conclusions. AV junctional reentrant tachycardia has at least two subtypes which can be distinguished at both preoperative and operative electrophysiologic study. Both may

be cured surgically with excellent long-term results. Normal anterograde AV nodal conduction is preserved. The mechanism of surgical cure is inconclusive at present, but modification of retrograde His to atrium conduction or anterograde slow pathway conduction, or both, is important. The precise requirements for surgical success are now the focus of further investigation. The perinodal atrium appears to be a component of the reentrant circuit in human AV junctional reentrant tachycardia.

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