Cryosurgical Ablation of the Atrioventricular Node-His Bundle: Long-term Follow-up and Properties of the Junctional Pacemaker

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SUMMARY We used a cryosurgical technique to ablate the atrioventricular (AV) node-His bundle in twenty-two selected patients with disabling supraventricular tachyarrhythmias unresponsive to medical management. Successful AV block was achieved in seventeen. There was no intraoperative mortality and significant surgical complications were not encountered. Electrophysiologic studies performed 7-10 days after surgery revealed that the subsidiary pacemaker had a narrow QRS complex morphology and a mean cycle length of 1244 msec. Isoproterenol (1-4 μg/min iv.) significantly increased (p < 0.01) the rate of the subsidiary pacemaker (mean maximum response 1008.3 msec); atropine (2 mg iv.) had no effect on its rate. The cycle length of the subsidiary pacemaker during long-term follow-up (mean 14.8 months) showed a small but significant (p = 0.024) increase (mean cycle length 1430 msec). Treadmill exercise in seven patients did not result in the subsidiary pacemaker exceeding the rate of the implanted demand pacemaker set at 70 beats/min. The properties of the subsidiary pacemaker suggest an intra-Hisian site of impulse formation.

PATIENTS WITH RECURRENT supraventricular tachyarrhythmias sometimes cannot be managed adequately by either pharmacologic or pacemaker techniques. For such patients, surgical interruption of atrioventricular (AV) conduction followed by implantation of a permanent ventricular pacemaker offers a method to control episodes of tachycardia that has been shown to be feasible and beneficial.1-9 We have described such a method using cryosurgery.10 This report describes the results of surgery and long-term follow-up after 22 cryosurgical procedures and discusses the electrophysiologic properties of the subsidiary junctional pacemaker.

Methods

Patient Population

The patients were among a large group referred to the Clinical Electrophysiologic Laboratory of Duke University between November 1975 and December 1978 to evaluate mechanisms and potential therapy for recurrent tachycardia. Patients selected for surgical interruption of atrioventricular conduction were severely incapacitated by supraventricular arrhythmias that had been resistant for months or years to pharmacologic agents. There were 12 males (mean age 46.7 years, range 33-57 years) and 10 females (mean age 49.9 years, range 24-70 years). ECGs showing spontaneous supraventricular arrhythmias were obtained from referring physicians before electrophysiologic study.

Preoperative Assessment

All patients underwent electrophysiologic assessment to determine the functional properties of their AV conduction system, to detect accessory pathways and to define the mechanism of their arrhythmias. The details of this procedure have been published.11, 12 In all patients, the arrhythmias recorded by the referring physician before referral were induced or occurred spontaneously during our electrophysiologic studies (table 1). In addition to disabling supraventricular arrhythmias, three patients also had ventricular tachycardia. Patients older than 50 years or those with clinical evidence of significant coexistent heart disease also underwent hemodynamic catheterization and coronary angiography.

Operative Procedures

In sixteen patients, cryoablation of the AV node-His bundle was the only surgical procedure performed (table 1). Five patients had concurrent coronary artery bypass grafting or valve replacement. One patient un-
### Table 1. Patient Data Summary

<table>
<thead>
<tr>
<th>Age (yrs)</th>
<th>Arrhythmia diagnosis</th>
<th>Associated diagnosis</th>
<th>Preoperative ECG</th>
<th>Concomitant surgery</th>
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<th>Follow-up (months)</th>
<th>Cycle length (msec)</th>
<th>Junctional recovery time (msec)</th>
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</table>

*This patient received a unipolar ventricular pacemaker. It was possible to inhibit his pacemaker by chest wall stimulation and determine recovery times greater than 2000 msec.

**Abbreviations:** AF = atrial fibrillation; AFL = atrial flutter; Comm = commissurotomy; IVCD = intraventricular conduction delay; LAH = left atrial hypertrophy; LV = left ventricular; LVH = left ventricular hypertrophy; N = within normal limits; NS = not successful in producing complete atrioventricular block; S = successful production of complete atrioventricular block; SVT(AP) = supraventricular tachycardia using an accessory pathway in the reentrant circuit; SVT(AVN) = supraventricular tachycardia due to reentry in the atioventricular node; SVT(ECT) = supraventricular tachycardia due to an ectopic atrial focus; SVT(AR) = supraventricular tachycardia due to atrial reentry.

derwent intraoperative mapping of ventricular tachycardia, with a subsequent attempt at cryosurgical ablation of the myocardium showing early activation during ventricular tachycardia.

In patients with accessory AV pathways and poor ventricular function, cryoablation of the AV node-His bundle was performed to control tachycardia rather than interruption of the accessory pathway. This decision was based on the judgment that the relatively short period of cardiopulmonary bypass required for cryoablation might be critical to operative survival.

### Cryosurgery

The cryosurgical technique used in our institution has been described in detail elsewhere.10,13 Patients were placed on cardiopulmonary bypass and the right atrium was opened. The His bundle was located by recording endocardial electrograms using a hand-held probe containing two electrodes 1 mm apart. The cryoprobe was applied to this area, which was subsequently cooled to 0°C for 30 seconds. This usually led to complete AV block, which was reversible upon
rewarming. If the test cooling did not produce AV block, the area was again mapped, searching for a His deflection of greater amplitude preceded by a prominent atrial deflection. Cooling was then repeated. When AV block was achieved by cooling, the area under the probe was cooled to −60°C for 2–3 minutes. After the area thawed, the procedure was repeated. One or two additional freeze lesions were produced in a similar fashion 3–6 mm proximal to the first lesion and nearer to the coronary sinus. A rate-adjustable pacemaker was then implanted and connected to a bipolar screw-in epicardial lead implanted in the left ventricle.

Postoperative Electrophysiologic Studies

Seven to 10 days postoperatively, 16 of the 17 successfully operated patients were returned to the electrophysiology laboratory. Each patient’s paced rate was abruptly decreased to 30 cycles/min (the lowest programmable rate) using an external programmer, and the escape rhythm was observed. The emergence of the escape rhythm was also observed after pacing the ventricle at rates of 50–100 beats/min for 2 minutes and abruptly programming the pacing rate to 30 beats/min (cycle length 2000 msec). This permitted a limited noninvasive estimate of recovery time of the subsidiary pacemaker. The junctional recovery time was defined as the longest escape interval observed at any cycle length tested.

During a stable escape rhythm, the effect of a graded infusion of isoproterenol was observed (eight of 17 patients). The initial infusion rate was 1 μg/min. This rate was increased by 1 μg/min every 5 minutes to a final rate of 4 μg/min. The infusion was then discontinued and the intrinsic heart rate was allowed to return to control rate. Atropine, 0.5 mg, was then injected intravenously, and repeated after 2 minutes. An additional 1 mg was given after another 2 minutes. These studies were omitted or modified in selected patients if consent was not given or if a significant risk was involved due to coexistent myocardial disease. Three patients consented to introduction of a single tripolar catheter through the right femoral vein for recording of the His bundle electrogram.

Between September 1978 and November 1978, all referring physicians were contacted and asked to provide information regarding the functional status of the patient, electrocardiographic data during inhibition of the patient’s pacemaker, the results of exercise testing and any complications encountered.

Results

Twenty-two patients underwent open-heart surgery in an effort to control intractable supraventricular arrhythmias that were severe and unresponsive to conventional medical therapy. The antiarrhythmic agents invariably included digoxin, propranolol, quinidine and procainamide alone and in combination. All patients underwent cryoablation of the AV node-His bundle and six underwent other operative procedures for correction of coexistent conditions (table 1).

Surgical Results

Successful Procedures

Permanent disruption of AV conduction was achieved in 17 patients. No patient required antiarrhythmic therapy postoperatively, although three patients noticed abnormal neck pulsations during atrial flutter or fibrillation that continued without transmission to the ventricles.

Eight patients required digitalis or diuretics for symptoms of exertional dyspnea or congestive heart failure. In one patient the requirement for therapy was transient. Of the other seven, all had a significant preexisting myocardial problem, such as valvular disease, cardiomyopathy or coronary heart disease, and six had a preoperative history of congestive heart failure that required treatment. One patient was subsequently converted to an AV sequential pacemaker.

Two patients had pacemaker failure during follow-up. Patient 1 (table 1) was found to be in junctional rhythm with a nonfunctioning pacemaker during a routine visit to his physician. Patient 13 (table 1) presented after a syncopal episode and also was found to be in junctional rhythm with a nonfunctioning pacemaker.

Failures

Permanent AV block was not achieved in five patients. His bundle electrograms were recorded intraoperatively in all five.

In three of these patients, extensive surgical dissection of the AV node region was performed after cryosurgery and this too failed to achieve block. First-degree AV block was noted postoperatively in one patient. He has had no recurrence of reciprocating tachycardia (previously shown to be secondary to reentry in the AV node) during 23 months of follow-up.14

In the remaining two patients, conduction was blocked but returned early in the postoperative period (8 hours and 5 days, respectively). The intraoperative mapping procedure was complicated in one of these patients by intermittent function of a septal accessory pathway. Right bundle branch block was observed in this patient postoperatively.

Mortality

There were no operative deaths. One patient died 1 month after unsuccessful surgery. This patient had a prosthetic mitral valve and very poor ventricular function before surgery. A second patient died 12 months after surgery as a result of a coexisting ventricular arrhythmia that was present preoperatively.

Postoperative Electrophysiologic Studies

These studies included: 1) determination of QRS morphology during junctional rhythm; 2) determination of junctional recovery times; 3) determination of stable junctional rate; 4) determination of the effects of isoproterenol and atropine on junctional rate; 5) determination of the response of the junctional
pacemaker to treadmill exercise; and 6) recording His bundle electrograms (three patients).

**QRS Morphology**

In patients in whom successful block was achieved, the postoperative QRS complex (nonpaced) was of normal configuration or unchanged from preoperative recordings in 14 of 17 cases. Patients 7 and 16 developed incomplete right bundle branch block postoperatively (table 1) and patient 6 (table 1) developed new anterior Q waves as a result of ventriculectomy to ablate coexistent ventricular tachycardia.

**Junctional Recovery Times**

Junctional recovery times were measured noninvasively, using the implanted programmable pacemaker, which enabled recovery time determinations for paced rates of 50–100 cycles/min. Abrupt programming to 30 cycles/min enabled observation of intrinsic escape rhythms, but those appearing after 2000 msec were preempted by a paced beat and could only be recorded as greater than 2000 msec. Recovery times were generally, but not invariably, longer at faster paced rates. The longest recovery time at any paced rate was defined as the maximal junctional recovery time (table 1). We considered recovery time after a paced rate of 70 cycles/min to be of special clinical significance, as this is usually the rate at which the patient’s pacemaker is permanently set. A determination of recovery time at this paced rate would simulate response to spontaneous pacemaker failure. All patients but one had recovery times of less than 2000 msec (table 1) at this rate. The exception was patient 17, who failed to show any spontaneous rhythm when her implanted pacemaker was abruptly programmed to 30 cycles/min.

**Stable Junctional Rhythm**

In one patient, these data were not obtained due to concern about inhibiting her pacemaker in the presence of coexistent coronary disease. In the remaining 16 patients, stable junctional rhythm was invariably achieved within 3–10 cycles after inhibition of the pacemaker in the early postoperative period. The cycle length of the junctional rhythm varied from 830–1500 msec (1244 ± 178 msec [mean ± SD]). Repeat evaluation of the cycle length during long-term follow-up (14.8 ± 10.95 months [mean ± SD], range 3–33 months) showed a slight increase in cycle length for most patients (fig. 1). In one patient (not included in figure 1), the pacemaker was temporarily programmed from 70 to 30 cycles/min. No escape rhythm appeared over a sampling interval of at least 26 seconds, during which time the implanted pacemaker was pacing at 30 cycles/min.

**Effects of Isoproterenol and Atropine**

Isoproterenol, 1–4 µg/min by infusion, significantly (p < 0.01) increased the rate of the junctional pacemaker (fig. 2) in a dose-related manner (fig. 3). Atropine had no effect on the rate of the junctional pacemaker (fig. 2).

**Treadmill Exercise**

Seven patients had treadmill exercise tests 10 days to 35 months postoperatively (mean 10.9 months). The implanted pacemaker was not inhibited during treadmill exercise testing. All stress tests were limited by fatigue or dyspnea. Six of seven patients were unable to increase their heart rates above that of the implanted pacemaker. One patient was able to increase his intrinsic heart rate to exceed the paced rhythm for brief periods (two to three beats) during maximal exercise.

**Figure 1.** Cycle length of the subsidiary junctional pacemaker as a function of time. The greatest increments in cycle length over time occur in patients with shortest initial cycle lengths. Early = cycle length determined within 8–14 days of surgery. Late = cycle length determined 3–33 months (mean 14.8) months postoperatively.

**Figure 2.** The effects of atropine and isoproterenol on the cycle length of the junctional pacemakers. The control and minimum cycle length attained after the intervention are shown.
His Bundle Electrograms

His bundle electrograms were obtained from three patients postoperatively. In each case, there was AV block with His deflections preceding each QRS complex. HV intervals were equal to or slightly shorter than those observed preoperatively (fig. 4).

Pathologic Extent of the Cryolesion

Patient 6 (table 1) died 12 months after production of AV block from causes unrelated to this procedure. The heart was obtained for pathologic examination. Photomicrographs of the region of the AV node and His bundle in this patient revealed fibrosis of the common His bundle and the upper right ventricular aspect of the muscular interventricular septum (fig. 5).

Discussion

On rare occasions, patients with recurrent supraventricular tachycardia remain disabled because available antiarrhythmic agents are ineffective or poorly tolerated. Surgical interruption of the AV node-His bundle with implantation of a permanent ventricular pacemaker provides an alternative mode of therapy.

Previous investigators have used dissection, suture ligation or cautery to ablate the AV node area. Cryosurgery offers several advantages over these other methods. First, a reversible cessation of AV conduction can be achieved by first cooling the area suspected to contain the AV node and proximal His bundle. If the AV node has not been well localized, AV conduction will not be interrupted. Mapping can then be repeated to localize the area without destroying adjacent tissues. Second, the lesion produced by cryosurgery is relatively small, discrete and sharply demarcated from undamaged adjacent tissues. The lesion per se is not arrhythmogenic. Third, the chronic lesion consists of dense scar tissue, with no tendency to rupture or form aneurysms. This is largely due to the

Figure 3. The decrease in cycle length, expressed as percent decrease from control value, is plotted against infusion rate of isoproterenol. Mean values and standard error are indicated.

Figure 4. Preoperative and postoperative His bundle electrograms (HBE) (patient 9). A) The preoperative His bundle electrogram. B) The postoperative His bundle electrogram. There is complete atrioventricular block. Each QRS complex is preceded by a His potential. RA = right atrium; RV = right ventricle; CL = cycle length; CS = coronary sinus.
The relative resistance of fibroblasts and collagenous elements to cooling, elements that are uniformly destroyed by cautery.

The lesion produced by freezing the endocardium over the His deflection probably involves the AV node and the common His bundle to various degrees (fig. 5). The subsidiary pacemaker was below the AV node but proximal to the bifurcation of the bundle branches as shown by the narrow QRS complexes of the escape rhythm, identical to those observed preoperatively. In patients in whom we obtained His bundle electrograms, the QRS complexes were preceded by a His deflection with an HV interval equal to or only slightly shorter than that observed preoperatively.

The electrophysiologic properties of the subsidiary pacemaker are similar to those reported in patients and experimental animals with intra-Hisian rhythms. The mean cycle length of the escape rhythm was 1244 msec (range 830–1500 msec), comparable to that reported for patients with intra-Hisian block secondary to disease of the conduction system. The rate of the subsidiary pacemaker decreased slightly in most patients over long-term follow-up. In one notable exception, no subsidiary pacemaker was observed when the paced rate was decreased to 30 beats/min.

Atropine had no significant effect on the rate of the subsidiary pacemaker. This finding has generally been
reported in patients and experimental animals with intra-Hisian block. Isoproterenol significantly increased the rate of the junctional pacemaker in a dose-related manner.

Junctional recovery time generally, but not invariably, increased with increasing paced rate, a finding reported in patients and experimental animals. The junctional recovery time after a paced rate of 70 cycles/min was especially important, because this is the rate at which most patients undergo permanent ventricular pacing. Because the junctional recovery time was less than 2 seconds in all but one patient tested, this may suggest that most of these patients would not be prone to syncope in the event of sudden pacemaker failure.

Patients who underwent treadmill exercise were unable to increase their heart rate beyond that of the permanent pacemaker (70 cycles/min). This appears to correlate with the effect of isoproterenol infusion; the maximum rate achieved was 73 beats/min (fig. 2). This certainly would be an important factor in effort intolerance, especially in older patients and those with coexistent organic heart disease. Most of our patients who required treatment for some degree of heart failure postoperatively had significant coexistent heart disease and a preoperative history of congestive heart failure. For this reason, we now implant a permanent atrial electrode in such patients at the time of surgery, in addition to the ventricular electrode. The atrial lead is left in the pacemaker pocket for subsequent use should the need for AV sequential pacing arise. The use of AV sequential pacing would not be helpful in many patients with chronic atrial arrhythmias such as atrial fibrillation. The added expense and technical complexity associated with AV sequential pacing do not appear to justify its use in patients with normal left ventricular function. This group does not appear to experience difficulties with congestive heart failure after surgical AV block.

Permanent AV block was not achieved in five patients. In three of these patients (patients 4, 11 and 16), extensive surgical dissection in the presumed region of the AV node-His bundle similarly failed to achieve block. Scarring from previous right atrial surgery was a factor in one patient. It is possible that these patients had some variant of normal AV junctional anatomy. It has been emphasized by some investigators that the AV node is an "interatrial" structure and not a right atrial structure. Both the AV node and proximal His bundle may have extensive histologic continuity with the mitral annulus and left atrial muscle. Left atrial connections to the AV node may have been more distal than was appreciated at the time of surgery in these patients. In the remaining two patients, who had early return of AV conduction postoperatively, it is probable that the cryoprobe was not optimally positioned, and return of AV conduction was related to reversible injury to the AV junction at the margin of the cryolesion.

The development of more efficacious antiarrhythmic agents promises to minimize the need for the production of surgical AV block as a therapeutic measure. When indicated, cryosurgical ablation of the AV node and His bundle is safe and effective. It offers several advantages over other ablative techniques, including the production of a discrete, firm scar with no tendency to rupture and minimal disruption to the surrounding anatomy. The subsidiary pacemaker is within the His bundle and generally has electrophysiologic properties previously described for subsidiary pacemakers felt to be in this location.

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References

15. Wieberdink J: Experimental production of permanent heart block (total or bundle branch block) without circulatory arrest
or extracorporeal circulation. Thorax 21: 401, 1966
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