Elimination of Atrioventricular Nodal Reentrant Tachycardia Using Discrete Slow Potentials to Guide Application of Radiofrequency Energy

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Background. Ablation of the slow pathway has been performed to eliminate atrioventricular (AV) nodal reentrant tachycardia (AVNRT) either by a surgical approach or by using radiofrequency catheter technique guided by retrograde slow pathway activation mapping. From previous experience of midseptal and posteroseptal mapping, we were aware of the existence of peculiar slow potentials in most humans. Postulating their role in AVNRT, we studied these potentials and the effects of radiofrequency energy.

Methods and Results. Sixty-four patients (mean age, 48±19 years) with the usual form of AVNRT were studied. Slow, low-amplitude potentials were recorded when using the anterograde AV conducting system. Slow potentials occupied all (giving a continuum of electrograms) or some of the time between the atrial and ventricular electrograms. Their most specific patterns were their progressive response to increasing atrial rates, which resulted in a dramatic decline in amplitude and slope, a corresponding increase in duration, and a separation from preceding atrial potentials until the disappearance of any consistent activity. Slow potentials were recorded along a vertical band at the mid or posterior part of the septum near the tricuspid annulus. Radiofrequency energy applied at the slow potential site resulted in interruption of induced tachycardia within a few seconds and rendered tachycardia noninducible in all patients. A median of two impulses was delivered to each patient. In 69% of patients, postablation atrial stimulation cannot achieve a long atrial–His interval, which previously was critical for tachycardia induction or maintenance. No patient had AVNRT over a follow-up period of 1–16 months, and all had preserved AV conduction. In all except two patients, the PR interval was unchanged. In 47 patients, long-term electrophysiological studies confirmed the efficacy of ablation and the nonreversibility of results by isoproterenol; however, echo beats remained inducible in 40% of patients.

Conclusions. An area showing slow potentials is present at the perinodal region in humans. In patients with AVNRT, application of radiofrequency energy renders tachycardia noninducible through the preferential modification of the anterograde slow pathway. With present clinical methods, the exact origin and significance of these physiological potentials cannot be specified. (Circulation 1992;85:2162–2175)

Key Words • slow potentials • atrioventricular nodal reentry • catheter ablation

Atrioventricular (AV) nodal reentry (AVNRT) is the most common cause of regular paroxysmal supraventricular tachycardia.1–4 In patients with disabling drug-refractory tachycardias, various nonpharmacological procedures have been considered.3–25 Ablation of the AV junction3 has been suggested, with the drawback of the subsequent need for permanent cardiac pacing. The first curative therapies were surgical, using dissection6 or cryodestruction7 of the perinodal area within Koch’s triangle. More limited cryolesions placed between the coronary sinus and the AV node were also efficient in abolishing AVNRT.8 Successful surgery was usually accompanied by unchanged fast retrograde conduction, suggesting that the mechanism of cure was a preferential impairment of the anterograde slow pathway. Catheter ablation techniques have been reported with similar efficacy, first using fulguration,9–12 then using radiofrequency energy.13–19 In most reports, ablative energy was applied at the right anterior septum, resulting in preferential alteration of the retrograde fast conducting system. This technique was associated with a slight but definitive prolongation of the atrial–His (AH) interval and a risk of complete AV block close to 10%. In 1981, Sung et al20 demonstrated in seven patients that the slow AV nodal pathway has a retrograde exit located posterior and inferior to that of the fast AV nodal pathway. From mapping of the retrograde slow pathway21–25 and the effects of radiofrequency energy,22 Jackman et al first demonstrated in June 1990 the feasibility of catheter ablation of the slow pathway with a very low risk of AV block. Other preliminary work confirmed this finding.26–28 The earlier studies were the starting point for the present work in September 1990. From our experi-

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TABLE 1. Atrioventricular Nodal Function Before and After Ablation

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<tr>
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<th>Before ablation</th>
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<td>AVHmax (WCL)</td>
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Retrograde function

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Values are mean±SD and expressed in msec. AH, atrial–His interval; AV, atrioventricular; WCL, entire Wenckebach cycle length of the AV node when either pathway is used; Duality, dual AV nodal physiology (jump in the AH interval >50 msec); AVN FP, fast pathway of the AV node; ERP and FRP, effective and functional refractory periods, respectively; AVN SP, slow pathway of the AV node; Amax, maximal value of AH interval during either WCL or ERP assessment; AH t, AH interval during an ongoing stable tachycardia; VA, ventricular; AVN-FRP, AV nodal function-refractory period; AVN-FR, AV nodal function-refractory period; AVN-ERP, AV nodal function-ERP.

*Signifies p<0.01 before vs. after ablation, postablation vs. basal days 8–10 study, basal days 8–10 study vs. basal late study. During isoproterenol infusion, all values were significantly lower than during basal state except VA minimum and HA interval.

eence of ablative techniques,9,10,29 we were aware of the existence of peculiar slow potentials recorded from the perinodal region in most humans. These potentials could be electrophysiologically separated from ordinary atrial, hussian, or ventricular activities and were not caused by artifacts.30 We analyzed these potentials and investigated the effects of radiofrequency energy in patients affected by the common form of AVNRT.

Methods

Patient Characteristics

The study population included 64 patients: 44 women and 20 men with a mean age of 48±19 years (range, 11–85 years). All patients were suffering from disabling recurrent AVNRT. Tachycardias occurred daily in 12 patients. Six patients had experienced syncpe caused by high ventricular rates during tachycardia. A structural heart disease was present in eight patients: coronary heart disease in three, dilated cardiomyopathy in two, hypertrophic cardiomyopathy in one, and cor pulmonare in two.

Electrophysiological Study: Acquisition and Analysis of Endocardial Signals

Patients studied from September 1990 to December 1991 who had the usual (slow–fast) form of AVNRT were included. Diagnosis was based on classic criteria.3,4 Patients having atypical AVNRT were not included.

All except five studies were performed after all cardioactive drugs had been discontinued for at least five elimination half-lives. A class I drug was continued in five patients who also had spontaneous documented atrial fibrillation. Three 6F multielectrode catheters with spaces of 2 or 5 mm between electrodes (USCI, Billerica, Mass.) were introduced percutaneously through the right femoral and subclavian veins. Two were left in a stable position to record His bundle and proximal coronary sinus activities; the third was used to pace either the lateral right atrium or the right ventricular apex. A fourth electrode was introduced for ablation. This was either a 7F steerable catheter (Mansfield-Webster catheters, Mansfield, Mass.) or a modified USCI 6F catheter, both with a distal electrode 4 mm in length and an interelectrode space of 2 or 5 mm. Parameters of anterograde and retrograde AV nodal conduction and refractoriness were assessed, in particular the highest values of AH interval obtainable through the anterograde conducting system. An anterograde AV nodal duality (jump >50 msec in the AH interval) was found in 34 patients, and a retrograde duality was found in five patients. In some patients, the AH interval from the second beat of tachycardia was notably longer (>50 msec) than the maximal AH interval observed at its induction, unlike what would be expected by both AV nodal accommodation and change in autonomic tone. Baseline values are described in Table 1. Our attention was then focused on slow potentials recordable along the mid or posterior part of the septum.30 Slow potentials were defined as a low-amplitude activity with a slow rate of rise. They occupied the diastolic interval between atrial and ventricular electrograms. During this study, we analyzed 1) the incidence of slow potentials in patients with AVNRT and in a control group of 20 normal patients who were demonstrated to have no AV nodal tachycardia or echo beats; 2) the easiest way to consistently record these
potentials; 3) their characteristics and electrophysiological behavior during anterograde AV nodal conduction and vagal maneuvers: To exclude any alteration caused by ablation lesions, analysis of electrogroms was made before energy delivery; 4) their radiological location relative to the classic septal hallmarks, i.e., the His bundle and the coronary sinus ostium; and 5) the effects of radiofrequency energy applied at the slow potential site in patients with AVNRT. The sites were classified as anterior, median, or posterior within the septum divided into three equal zones from the site of the maximal His bundle potential to the coronary sinus ostium.

Three electrocardiographic leads (usually DI, II, and III) were displayed and recorded simultaneously with three intracardiac electrograms at a paper speed of 100 mm/sec with a polygraph (model VR12, Electronics-For-Medicine, Pleasantville, N.Y.). High amplification in the range of either 0.2 or 0.1 mV/cm was used to record the potentials. The results were obtained with filter sets of 30–500 Hz through 2-mm or 5-mm bipoles. As long as stable potentials were recorded, atrial stimulation at progressively increasing rates or shorter atrial extrastimuli were performed until the initiation of tachycardia or echo beat. During all these stimulations, great care was taken to obtain concomitant stable atrial and ventricular electrograms. Various stimulation techniques were used to distinguish slow potentials from atrial, Hisian, or artifact potentials and were repeated several times to ensure reproducibility of the patterns observed. Furthermore, during atrial stimulation, sinocarotidian massage or adenosine triphosphate injection was performed to obtain an AV nodal block. Finally, in the first patients, a slow activity was also sought during diastole in tachycardia. Programmed ventricular stimulation was performed during tachycardia in an attempt to advance ventricular activity and therefore to highlight a virtually isolated atrial electrogram.

Ablation Procedure

Before application of radiofrequency energy, electrograms were performed in the anterior, left anterior oblique (60°), and right anterior oblique (30°) views. A left lateral view was performed in some patients. Radiofrequency energy was delivered as a continuous unmodulated sine wave output. The generator was the HAT 100 (first five patients) or the HAT 200 (last 59 patients) (Osypta GmbH) delivering a 500-kHz current between the distal electrode of the ablation catheter and a 110-cm² cutaneous patch electrode placed over the left scapula. A power setting of 30 W was used in the first 26 patients and 40 W in the following 38 patients. Actual delivered root-mean-squared current and voltage were continuously observed on the HAT 200 during energy delivery, but these data were not stored. The electrocardiogram and the catheter position marked on the fluoroscopic screen were continuously monitored during energy delivery. The ablation site was selected as the site showing the most prominent (higher amplitude) slow potential in the absence of a significant (>0.1 mV) His bundle potential as demonstrated during rapid atrial stimulation. When similar slow potentials were recorded at contiguous sites, the first ablation site was the most posterior. In 26 patients, energy was applied during a sustained tachycardia to assess the time for tachycardia interruption. After tachycardia interruption, energy delivery was pursued provided that the position of the ablation catheter had not significantly changed. In 38 patients, energy was delivered either during a regular atrial stimulation or during sinus rhythm, depending on catheter stability. When a sustained junctional rhythm was observed, energy application was stopped in some patients within seconds to assess AV conduction. Radiofrequency energy was delivered during an overall period of 60 seconds at each ablation attempt (except in five patients in whom energy was delivered for 90 seconds). We defined one attempt as a 60-second total duration of energy delivered at one site either continuously or intermittently. The end point for the procedure was the inability to reinduce AVNRT. If either nonsustained tachycardia or sustained tachycardia with a longer AH interval remained inducible after the first current delivery, a new application was performed for 60 seconds at the same site. If no alteration of tachycardia anterograde parameters was observed, energy was applied at contiguous sites where slow potentials had also been previously recorded. After catheterization in all patients, anticoagulation was obtained with subcutaneous calcium heparinate. Twenty-four-hour ECG Holter recordings were taken for each patient both before and at days 1 and 5 after ablation. Blood was sampled for cardiac enzyme levels 6 hours after ablation. An electrophysiological study similar to baseline study was performed on day 8, 9, or 10 before and during isoproterenol infusion (2–4 μg/min). Patients were discharged at day 10 with a β-blocking drug empirically prescribed for a 4-week postablation period to prevent hypothetical arrhythmias originating from the ablation site. A long-term electrophysiological study was performed in 47 patients at 2–9 months after ablation before and during isoproterenol infusion (2–4 μg/min).

Statistical Analysis

Results are expressed as group mean±SD. Values of blood cardiac enzymes and electrophysiological parameters before and after ablation were compared using the Wilcoxon test for matched series. Differences between patients with or without echo beats were compared by the Mann-Whitney test. Because of the numerous variables studied, statistical significance was set at p<0.01.

Results

Localization of Slow Potentials

Slow potentials could be recorded by catheters introduced either by a subclavian or a femoral vein. Slow potentials were rapidly found at high amplification by using a femoral catheter with an incurved tip and withdrawing the catheter 10–20 mm from the His bundle recording site along the tricuspid annulus (evidenced by the presence of a significant ventricular electrogram). Usually, catheter withdrawal showed the progressive transition of a sharp His bundle potential to widened quasi-Hissian potentials and then to slow potentials more posteriorly. Therefore, slow potentials were recorded at the mid or posterior septum, anterior to the coronary sinus ostium (but not in or posterior to this structure); usually, the site of more vivid slow
potentials was projected at the two thirds anterior—one third posterior of the area between the His bundle to the coronary sinus ostium. The 60° left anterior oblique view was optimal because it showed the maximal amplitude of mapping catheter movements. In this view, potentials were found at contiguous sites along a band vertical to the His bundle and were absent a few millimeters away on either side or even from beat to beat because of rate variations or respiratory movements (Figure 1). The atrial-to-ventricular electrogram amplitude ratio and slow potential morphology varied according to the position of the catheters along this band. In three patients, the His bundle and coronary sinus ostium were anatomically close, so the site of the potential was constantly close (5–10 mm) to the site of the His bundle recording. In four patients, slow potentials were found in a wide area.

**Endocardial Recordings**

**Recordings during sinus rhythm and atrial stimulation.** In the control group, slow potentials with an amplitude >0.05 mV were found in 16 of 20 patients. Their morphology and electrophysiological behavior were similar to those in patients with AVNRT (Figure 2). In 61 patients with AVNRT, slow potentials were recorded during anterograde AV conduction (Figure 1). During sinus rhythm, slow potential morphology varied in the same patient and from one patient to another. Their amplitude was 0.05–0.5 mV. They could be uniphasic and rounded (hump), biphasic (sinus wave), or inscribed as a double hump. In some cases, the potentials were relatively sharp and resembled an atrial or hissian potential. At the most posteroseptal sites, slow potentials were rather hump-shaped and sometimes accompanied by an embryonic His bundle potential, whereas at the midseptum, slow potentials were more rapid, less broad, and often biphasic with a significant superimposed His bundle potential. The onset of slow potential coincided (or was included) with the end of the atrial electrogram, which was often slurred and fractionated. At low-rate stimulation, the slow potentials occupied all

**FIGURE 1.** Radiogram shows in a left anterior oblique 60° view the usual recording site of slow potentials marked in a frame. The frame is located at the mid third and posterior third of the septal space between the His bundle (H) and the coronary sinus ostium (CS), vertical to the His bundle recording site. Moving of the recording catheter to the right or left of this frame results in the disappearance of slow potentials. Recordings obtained during sinus rhythm from six different patients are shown around the radiogram. Arrows show various slow potentials occupying some or all of the atrioventricular interval. Note the variable atrial-to-ventricular amplitude electrogram ratio. The polarity is set to obtain positive potentials (as in other figures).
Mid or low right atrial stimulation shortened the double-spike interval (Figure 7), and a reversal in their relative sequence of activation was obtained with a caudocranial activation of the right atrium (Figure 7). As demonstrated in atrial flutter, this phenomenon observed overall in 11 patients was interpreted as evidence for functional dissociation (different routes of atrial inputs activating independently each potential) in the septal atrial activation.

**Dissection of slow potentials from other activities.** Slow potentials may be confused with atrial, hisian, or ventricular potentials, or artifact potentials. At atrial paced rhythms transmitted with relatively short AH intervals, slow potentials frequently bridged the whole atrioventricular interval so that their end occurred after the inscription of the His bundle potential (Figures 5 and 6), thereby suggesting an independent activation of both structures. During atrial cycles transmitted with a long AH interval, the slow activity then preceded the His bundle activation (Figures 3 and 4). Slow potentials could occur after the completion of atrial depolarization (P wave) and could be clearly separated from atrial electrograms by stimulation techniques (see above) and by vagal maneuvers in some cases. Likewise, slow potentials were clearly distinguishable from atrial repolarization waves, as the latter occurred 360–400 msec after depolarization (Figure 3) and could not be separated from atrial electrograms at such a magnitude, unlike slow potentials; furthermore, in rare instances, slow potentials were inscribed before atrial electrograms concomitantly with a reversal of atrial activation sequence. That the recordings did not represent the ventricular repolarization wave was evidenced by their inscription before the ventricular depolarization; however, such a confusion was possible at fast rates in which slow potentials could superimpose on the preceding ventricular repolarization wave. Last, elimination of an artifactual potential was established by the following arguments: The slow potential was present only after the atrial electrograms but was not observed after inefficient stimuli; it was recorded in a limited region and was not found when the catheter was moved even a few millimeters away; most importantly, we consistently reproduced the findings in different patients at similar recording sites.

**Recordings during tachycardia.** During tachycardia with 1:1 conduction, all attempts to depict a consistent slow activity were unsuccessful (as during atrial stimulation at similar high rates), but a low-amplitude activity could sometimes be recorded superimposed on the ventricular repolarization wave. An embryonic His bundle potential was frequently present. Ventricular beats elicited during tachycardia showed isolated atrial electrograms that were frequently fractionated and very similar to those recorded during premature atrial stimulation (Figure 6).

**Ablation Procedure.**

The ablation site had the following characteristics. The atrial-to-ventricular electrogram amplitude ratio was 0.8±0.7. In 26 patients, tachycardia ceased 15±16 seconds after the application of radiofrequency current on its anterograde course (after inscription of the retrograde P wave). Only one attempt was made in 25
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Figure 3. Recordings of slow potentials during sinus rhythm (first and second complexes) and during slow right lateral atrial stimulation associated with a long atriohisssian transmission time. Three surface electrocardiographic leads and two high-amplified (0.1 mV=1 cm) intracardiac leads (lateral right atrium, LRA, and ablation site) are shown. During sinus rhythm, the lower complex on first panel is recorded from the ventricular side of the tricuspid annulus, whereas the following one is obtained by a slight withdrawal of the recording catheter (higher atrial-to-ventricular ratio). On the left, the slow potential is notched and has a 0.15-mV amplitude, whereas on the right, it forms a hump with a 0.4-mV amplitude. Note the polyphasic pattern of atrial complexes. Onset of atrial stimulation yields a long atrioventricular delay, clearly showing an embryonic His bundle potential. The slow potential initially prolonged the atrial complex and progressively separated from it. The atrial repolarization wave is clearly inscribed on the LRA lead, its top (T) occurring 360 msec after depolarization. This timing is not influenced by atrial rate. (Figure 4 shows the effects of pursued atrial stimulation.)

patients (39%), two attempts in 21 (31%), three to seven in 16, and eight and 12 in one each. No clear difference in slow potential patterns appeared between successful and unsuccessful sites. Two successive impulses were applied at the same site in nine patients after a first transiently successful impulse, presumably because of the insufficient endocardial contact of the catheter. In the four patients with diffuse slow potentials, only the impulses delivered at a relative proximity (10–20 mm) of the nodohsiians area were efficient. Overall, a median of two attempts was made with a mean duration of 63±50 seconds per attempt. In one patient, the impulse duration was only 10 seconds with a successful outcome (Figure 8). In two patients, a previously undocumented fast–slow AVNRT was inducible after the delivery of the first impulse; additional impulses resulted in abolition of tachycardias. In 50 patients (78%), a junctional, usually slow rhythm occurred during successful energy delivery, requiring its intermittent curtailment in midseptal ablation sites for assessing AV conduction. We did not observe any AV block within the seconds after the occurrence of junctional rhythm, unlike that reported with radiofrequency applied at the anterior septum. However, in two patients, energy applied at the midseptum produced a prolongation of the PR interval. In no patient there was a sudden drop in current observed caused by an impedance rise.

No significant side effects were noted during energy application. The mean creatine kinase and creatine kinase–MB isoenzyme levels after ablation were 110±50 IU/l (normal, 24–195 IU/l) and 17±4 IU/l (normal, <25 IU/l), respectively. Both values were slightly but significantly (p<0.01) greater than before ablation: 63±32 and 13±4 IU/l, respectively. The overall duration of each study was 3–7 hours, most of which was to obtain stable recordings and confirm their reproducibility.

Immediate postablation results. No patient had inducible AVNRT after ablation, and all had preserved AV conduction. Echo beats were inducible in 40% of patients but were not inducible in the others. All echo beats were isolated; i.e., the atrial electrogram (after the His potential) could not capture the ventricle because of a block in the AV nodal slow pathway. The electrophysiological effects of ablation are summarized in Table 1. The main changes concerned the maximal values of the AH interval without any alteration of anterograde or retrograde basic values or of dynamic retrograde conduction. Dual AV nodal physiology was persistent in nine patients. The maximal values of the AH interval during continuous and premature stimulation were 209±64 and 213±70 msec, respectively (p<0.001 versus baseline values). Only 31% of patients attained AH intervals similar (within 50 msec) to preablation values. Therefore, the inability to obtain long AH intervals yielded an increase in the postablation effective refrac-
tory period of the entire AV conducting system, which in fact attained the preablation value of the effective refractory period of the fast pathway (Figure 9). However, the ablation produced no modification of the functional refractory period (shortest H1–H2 interval) of the AV conducting system. In two patients, radiofrequency application yielded an abolition of both anterograde slow pathway conduction and retrograde fast pathway conduction without any change in the basic AH interval. Last, ablation produced an abolition (in four patients) or significant alteration (in one patient) of the retrograde slow pathway in the five patients who had a retrograde duality.

**Results at days 8–10.** No patient had tachycardia recurrence or a change in ECG parameters. In one patient, a transient, Wenckebach-type AV block occurred at day 3 and disappeared at day 7, but electrophysiological parameters were changed in comparison with immediate postablation results (AV anterograde block, 390 versus 315 msec, abolition of retrograde conduction). A longer AH interval and a retrograde conduction block or alteration also appeared in two other patients because of a progression of tissue lesions. No patient had inducible slow–fast AVNRT even under isoproterenol infusion. Isolated echo beats were induced in 41% of patients at baseline and in 54% during isoproterenol infusion.

Differences between patients with or without echo beats are shown in Table 2. Two patients had no baseline echo beats but had an atypical AVNRT (with the His bundle region preexcited in the retrograde limb) inducible under isoproterenol; their follow-up was uneventful. Compared with immediate postablation results, electrophysiological parameters (Table 1) showed one significant change, an increase in the anterograde Wenckebach cycle length from 350±68 to 370±83 msec ($p=0.008$). In patients with residual anterograde duality, the maximal AH interval was shorter than baseline value in most patients but similar or longer in others. The effective refractory period of the slow pathway was slightly but not significantly increased. It was in fact modified because the extrastimuli frequently gave an inconsistent long AH interval (Figure 9).

**Follow-up and late electrophysiological results.** All patients were free of tachycardia in the absence of treatment over a follow-up period of 1–16 months. No long-term clinical consequences of ablation procedure were observed. The ECG showed a PR interval longer

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**Figure 4.** Recordings of atrial stimulation at progressively increasing rates in same patient as in Figure 3. The three strands are continuous. The increase in atrial rate is accompanied by a progressive widening and decline in amplitude of slow activity (arrow) parallel to the increase in AH interval until this activity cannot be distinguished from baseline. At a more rapid atrial pacing (not shown) or during diastole in tachycardia, recordings at this site are unable to detect a consistent activity because of too low an amplitude. Note potential resembling the slow potential after QRS complexes: This is an artifact because it is not constant (absent in Figure 3) and is variable in its morphology from beat to beat. Note that pacing modifies the atrial-to-ventricular electrogram amplitude ratio, suggesting that a displacement of the catheter may contribute to alterations of the potential. Nevertheless, the maximal alteration of the potential occurs between a pacing rate of 520 and 500 msec, during which atrial and ventricular electrograms are perfectly stable (patient in apnea). LRA, lateral right atrial lead.
than baseline in two patients (0.2–0.24 second). Parameters of anterograde and retrograde AV nodal conduction and refractoriness were unchanged in the 47 patients who underwent electrophysiological study at 2–9 months (Table 1) compared with those obtained 8–10 days after ablation. Modification of retrograde conduction persisted in the five patients. No patient had slow–fast AVNRT induced before or during isoproterenol infusion. Only one of the two patients with inducible atypical AVNRT continued to behave so. Echo beats were induced in 18% of patients at baseline and 36% during isoproterenol infusion. These echo beats were always isolated, except in two patients who had two consecutive echo beats and one patient who had three consecutive echo beats.

The ablation site was scanned in the last 17 patients (Figure 10). In three patients, slow potentials were either not found or had too low an amplitude (<0.05 mV); the atrial but not the ventricular electrogram was also changed. In the 14 other patients, slow potentials higher than 0.05 mV were recorded with slight or no perceptible change in most cases. Unchanged slow potentials could also be recorded at contiguous (non-ablated) sites. There was no relation between the persistence of slow potentials and the inducibility of echo beats or long AH intervals.

Discussion

This study demonstrates the characteristics and behavior of discrete slow potentials recorded in the proximal atrionodal region and the effects of radiofrequency energy applied at this site in patients with AVNRT.

Efficacy and Safety of Slow Pathway Modification

Our study confirms the results obtained by a limited posteroseptal cryosurgical approach and the feasibility of catheter-mediated selective impairment of the slow pathway shown by Jackman et al and others. Our technique is somewhat different from that previously reported. Initially, the original investigators used retrograde slow pathway activation mapping, and more recently they were guided by a spike potential that was thought to represent slow pathway potential. The ablation site was in the vicinity of the coronary sinus ostium, sometimes in or behind this structure. Guided by recordings of slow poten-

FIGURE 5. Recordings of slow potentials during premature (ps) atrial stimulation (s) associated with short atriohisian transmission times. Panel A: The slow potential (black arrow) occurs during the atrioventricular interval in sinus rhythm. Initiation of a regular (610-msec) atrial stimulation produces a displacement of the slow potential toward the ventricular electrogram. Note that the atrial complex (A) is composed of two deflections. Panel B: Premature extrastimulus ps (530 msec) produces a greater displacement of the slow potential, which occurs 85 msec after the second atrial deflection and 25 msec after inscription of the His bundle potential. During the following sinus rhythm complex, the atrial depolarization is unchanged but its end is notably slurred (open arrow) by a slow activity. Panel C: A more premature extrastimulus (500 msec) produces a still greater displacement of the slow potential, which occurs here 115 msec after the second atrial deflection and 40 msec after the His bundle potential. With more premature extra-stimulus (not shown), the slow potential was not evidenced. Note that the slow potential retained its pattern without significant widening and alteration, which is uncommon.
tials, we have obtained similar results with ablative energy applied more anteriorly, sometimes at the midseptum. This suggests that slow pathway conduction may be modified at different points on its course. The validity of our mapping approach is empirically confirmed in 64 consecutive patients by the complete clinical success of the procedure and more meaningfully, the low number of radiofrequency impulses required to achieve these results.

Although a direct, nonspecific injury to the AV node cannot be ruled out, particularly in midseptal ablation sites, the overall electrophysiological results reflect a preferential impairment of anterograde slow pathway. Indeed, energy applied at the potential site during AVNRT interrupts the rhythm within a few seconds after injection of the retrograde P wave. Furthermore, the inability of atrial stimulation to prolong the AH interval at a point previously critical for tachycardia induction or maintenance appears to us to be the clearest criterion indicating slow pathway modification. This criterion was present in 69% of our patients. In the other 31%, premature beats were transmitted with a long AH interval and thus produced echo beats. However, these echo beats were isolated, and the interruption of the circuit always occurred on the anterograde limb. The disappearance of tachycardias despite the persistence of echo beats argues against the concept of a circumscribed pathway interrupted by a discrete lesion. This dissociation in the circuit between induction and maintenance of AVNRT was clearly shown by the different AH intervals observed at the initial and subsequent cycles of the tachycardia in some patients.

Finally, in most patients, the ablation of slow pathway conduction led to an increase in the anterograde effective (but not functional) refractory period of the entire AV node conducting system, which then approximated that of the fast pathway.

Targeting delivery of radiofrequency energy to alter slow pathway conduction offers clear advantages over fast pathway ablation. The success rate is higher and the effects of ablation on tachycardia inducibility were not significantly reversed by isoproterenol. The risk of complete AV block is clearly lesser. Indeed, no instance of AV block was observed. However, a slight increase both in Wenckebach point and AV nodal effective refractory period were induced and, particularly in midseptal sites, an attentive monitoring of AV conduction is essential. Furthermore, a residual prolongation of the AH interval was not observed, and the normal PR interval is hemodynamically more favorable for patients, particularly during effort. Hypothetically, on a long-term basis, less deterioration of AV conduction may be envisaged.

**Origin and Significance of Slow Potentials**

The main characteristic of slow potentials described in this investigation is their rate-dependent alterations, resulting in the inability with current methods to perceive any activity at high rates or during tachycardia. On the other hand, they provide a simple electrophysiological target to guide slow pathway modification. With present clinical methods, the exact origin and significance of these potentials cannot be specified. The
FIGURE 7. Recordings (middle panel) and anteroposterior view (upper panel) at ablation site (midseptal, M) and at sites more anterior (A) or more posterior (P) in a 33-year-old patient. On the anterior site, the three electrograms present during sinus rhythm are of atrial (A1 and A2) or hessian (H, small arrow) origin. Atrial potential A3 occurs after the His bundle activation. On the midseptal site, the same atrial double potential (corresponding to A1 and A3) is clearly observed because of distinctly different patterns, but no His bundle potential is present. On the posterior site, the atrial electrogram labeled A2 is activated in sinus rhythm after A1 (A1−A2=45 msec) and 10 msec before A3. During sinus rhythm (i.e., a cephalocaudal atrial activation), the excitation process activates the anterior (ant) and mid sites (A1) and activates the posterior (post) site 45 msec later (A3); the presence of A3 can be explained by either a sequential activation from A1 with a local activation delay (A1−A3=55 msec) or an independent activation (two wave fronts) for A1 and A3. With low lateral right atrial stimulation (first and last complexes), A3 is activated earlier from the nearer stimulation site (A1−A2=10 msec). A3 still follows A2 from 10 msec; hence, there is a fusion of electrograms A1 and A3, i.e., a decrease in A1−A3 interval (20 msec). This fusion is clearly observed at the mid site, whereas it gives the appearance of a single biphasic electrogram at the ant site. No change occurs in the timing of H relative to A1. This reproducible observation suggests that A3 is not dependent on A1 activation but is dependent on A2. Bottom panel: electrocardiographic leads are the same except for the sixth because the recording catheter moved slightly downward during tachycardia and entered the proximal coronary sinus. Termination of tachycardia by rapid low right atrial pacing (only the last two stimuli capture the right atrium) produces a beat with a negative P wave (P') in V1 lead before sinus rhythm resumes. This beat is associated with a reversal of the activation sequence (A1−A3=35 msec), whereas H still follows A1. Note on the following sinus beat that A3 is activated late because it occurs simultaneously with the onset of the ventricular electrogram, 75 msec after the main deflection of A1. These observations clearly show the functional dissociation of activation process of two atrial electrograms recorded at midseptal and anterior septal sites. Each electrogram is activated by wave fronts coming from different directions, A1 probably from the interatrial septum and A3 from the posterior inferior right atrium; the double potential represents the collision of these wave fronts similar to observations demonstrated during experimental atrial flutter.
FIGURE 8. Recordings and right anterior oblique views at ablation sites in two patients. Slow potentials are shown during sinus rhythm and atrial stimulation (s). Slow potentials occupy the atrioventricular interval and consist of a hump that separated from atrial electrograms during atrial stimulation (arrows). In the upper panel, the potential is relatively sharp during sinus rhythm and can be confused with a late atrial potential, but it becomes rounded with higher atrial rate. Radiographic positions of ablation sites are shown relative to multipolar catheter left in the coronary sinus and quadripolar His bundle catheter. Ablation catheter (arrow) is introduced through either the left subclavian vein (upper panel image) or a femoral vein (lower panel image) and is positioned in the mid and posterior part of the septum, respectively. In both patients, a single radiofrequency impulse delivered for 10 and 60 seconds, respectively, abolished slow pathway conduction.

Recording site of slow potentials anterior to the coronary sinus ostium clearly corresponds to the proximal atrionodal region. In this region, the terminal atrial tissue is not interdigitated with but overlaps the AV node, and the transition from atrial to AV nodal activity is smooth rather than abrupt. This complex region is also the starting point of a band of atrial tissue running along the septal cusp of the tricuspid valve, which could propagate atrial impulses to the AV node (posterior input), has the lowest velocity among cardiac tissues (including the AV node), and has a high level of automaticity. Anderson et al found evidence in
the atrium (probably its deep layers owing to the low amplitude of signals), AV node (for midseptal potentials), or both. This specification is difficult because the separation of the anatomic and electrophysiological AV node is not clearly defined. However, potentials occurring after the His bundle activity cannot be nodal.

Similarly, the functional significance of slow potentials is debatable and may have several meanings. Their presence in most humans, their recording sometimes in a wide area, and their persistence after successful ablation all show that they are not direct and specific markers per se for a distinct slow pathway. Nevertheless, the complete clinical ablation success with few radiofrequency impulses demonstrates that these indirect markers are reliable. The slow rate of potential changes and their rate-dependent alterations clearly indicate underlying heterogeneous slow and anisotropic conduction.38-41 Thus, slow potentials in humans may reflect the functional complexity in the atrial myocardium of the posterior space and therefore represent an epiphenomenon. Alternately, slow potentials may be related to a specialized slow-conducting tissue, a hypothesis considerably supported both in human anatomic studies42,43 and in multiple experimental reports32-37 showing the conductile properties of myocardial structures entering the AV node. The posterior structure conducts slowly and generates extracellular potentials strikingly similar to slow potentials reported here.44-51 The modification of this tissue that contributes to physiological AV propagation would explain both the mechanism for ablation success similar to surgical5-8 or experimental35,37 ablation and the modifications of AV “nodal” parameters also inducible in patients without AVNRT.30 The fact that

![Diagram of atrioventricular (AV) nodal function curves during basal state before (solid squares) and after (open circles) ablation in two patients. The drive cycle length was 600 msec. A1A2, coupling interval of atrial extrastimulus; A2H2, atriohisian conduction time of premature beat; AH max SP or FP, maximal value of A2H2 through slow pathway or fast pathway, respectively; ERP, effective refractory period. Bottom panel shows abolition of dual AV nodal physiology. Before ablation, a decrease in A1A2 from 330 to 320 msec yields a jump in A2H2 from 120 to 300 msec. After ablation, the maximal A2H2 is 120 msec (abolition of slow conduction), resulting in an increase in the ERP of the entire AV conducting system (attaining that of the fast pathway). Top panel shows persistent residual slow conduction. Before ablation, A1A2 coupling intervals from 340 to 280 msec consistently induce long A2H2 attaining 320 to 380 msec. After ablation, A1A2 coupling intervals from 320 to 310 msec are inconsistently transmitted to the His bundle with resulting A2H2 between 290 and 325 msec. In fact, there is no typical pattern of slow pathway modification because the postablation maximal AH interval can be shorter, similar, or longer than baseline value.

![Recordings of slow potentials at the ablation site (PRE, preablation; POST, postablation at late electrophysiological study) in two patients. Top recording shows virtually unchanged electrograms in a young woman who has been cured for 13 months after one impulse delivered at the site. Bottom recording shows a decrease in the slow potential amplitude, which is lower than 0.05 mV. Note the significant modification of the atrial potential. This patient received three impulses at the site area and is now cured.](http://circ.ahajournals.org/doi/abs/10.1161/01.CIR.103.8.2173)
Table 2. Electrophysiological Data in Patients With or Without Echo Beats

<table>
<thead>
<tr>
<th>baseline</th>
<th>isoproterenol</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anterograde function</td>
<td></td>
</tr>
<tr>
<td>Echo beats present</td>
<td>Echo beats absent</td>
</tr>
<tr>
<td>AH</td>
<td>79±27</td>
</tr>
<tr>
<td>WCLAV</td>
<td>394±100</td>
</tr>
<tr>
<td>AHmax (WCL)</td>
<td>209±77</td>
</tr>
<tr>
<td>AVN FP-ERP</td>
<td>352±101</td>
</tr>
<tr>
<td>AVN-FRP</td>
<td>447±98</td>
</tr>
<tr>
<td>AHmax (ERP)</td>
<td>198±74</td>
</tr>
<tr>
<td>Retrograde function</td>
<td></td>
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<tr>
<td>WCL VA</td>
<td>391±120</td>
</tr>
<tr>
<td>VA minimum</td>
<td>135±32</td>
</tr>
<tr>
<td>VA ERP</td>
<td>285±127</td>
</tr>
<tr>
<td>HA</td>
<td>72±22</td>
</tr>
</tbody>
</table>

Values are mean±SD and expressed in msec; p>0.05.

AH, atrial–His interval; WCLAV, Wenckebach cycle length of the atrioventricular node when either pathway is used; AHmax, maximal value of AH interval during either WCL or ERP (effective refractory period) assessment; AVN FP, fast pathway of the AV node; FRP, functional refractory period; VA, ventriculoatrial; HA, interval from His bundle to local atrium during ventricular extrastimulation.

slow potentials may occur after His bundle activation would reflect the use of an anterior, more rapid conduction system; it is not surprising that this would represent the anterograde counterpart of the dual conducting system demonstrated in humans to function retrogradely20 with a posterior septal exit from the slow structure.

Limitations

Use of the standard intracardiac recording technique has limitations, particularly in the region under study. First, respiratory and cardiac movements associated with programmed electrical stimulation easily displace the catheter electrodes. The position of the catheters had to be readjusted during this work, thereby explaining the length of each study in order to obtain reproducible results. The instability of the catheters hampered the study of electrophysiological events, and insufficient catheter endocardial contact was also an impediment to radiofrequency energy transmission. This required us to deliver repeated energy impulses at the same site. Second, conventional high amplification increases electrical interference in electrograms. Technical refinements may be required to clarify these physiological signals and provide a more detailed confirmation of our findings. Experimental studies are necessary for determining the specificity and functional significance of slow potentials present in most humans and used here to target energy delivery for AVNRT ablation.

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