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Subthreshold Atrial Pacing in Patients With a Left-Sided Accessory Pathway: An Effective New Method for Terminating Reciprocating Tachycardia

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This study investigated the possibility of terminating reciprocating atrioventricular (AV) tachycardia using subthreshold atrial pacing. Ten patients with a left-sided accessory pathway and sustained AV tachycardia underwent subthreshold atrial pacing from the coronary sinus site closest to insertion of the accessory pathway. In seven of these patients, the tachycardia could be reliably terminated with subthreshold atrial overdrive pacing. When pacing at a cycle length of 80 ± 23% of the tachycardia cycle length, the minimal subthreshold current that was effective in tachycardia termination was 64 ± 14% of threshold current and the maximal ineffective current was 49 ± 17% of threshold ($p < 0.05$). In all cases, the tachycardia was terminated by one or two instances of atrial capture that resulted in a premature atrial impulse (20 ± 4% advancement of the atrial cycle) that blocked the AV node limb of the tachycardia. Anterograde conduction over the accessory pathway never occurred, either during the tachycardia or during subthreshold pacing after a return to normal sinus rhythm. No instances of atrial fibrillation were provoked by subthreshold pacing. Possible explanations for the intermittent atrial capture with critically placed subthreshold impulses include supernormal atrial conduction or summation of impulses at the atrial insertion site of the accessory pathway.

It is concluded that subthreshold pacing is effective in selected patients with AV tachycardia due to an accessory pathway. Furthermore, because neither atrial fibrillation nor anterograde conduction over the accessory pathway is seen with subthreshold pacing, this modality may hold significant promise for permanent antitachycardia pacing in these patients.

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The initiation and termination of reentrant tachyarrhythmias have become accepted and routine procedures in the clinical electrophysiology laboratory. In recent years, significant investigative and industrial effort has been directed toward finding safe and effective permanent antitachycardia pacemakers that could be implanted in appropriate patients with troublesome reentrant supraventricular (1–4) or ventricular (5,6) tachyarrhythmias. Implanted devices have included patient-activated (7) as well as automatic (1–4,8) units capable of delivering discrete single, multiple or trains of stimuli to the appropriate cardiac chamber.

Permanent antitachycardia pacemaking is, however, not devoid of potential risks to the patient, especially the risk of pacing-induced acceleration of the tachycardia (8–10). This is particularly true of pacemakers utilizing the burst pacing mode. Another potential complication of rapid atrial pacing in patients with the Wolff-Parkinson-White syndrome is the ability of some accessory pathways to tolerate one to one conduction of rapid trains of atrial stimulation or pacemaker-induced atrial fibrillation, thereby creating the potential for ventricular tachycardia or fibrillation (8,9).

Subthreshold pacing, long a subject of interest to investigators performing in vitro and animal experiments (11–13), has recently been of interest to clinical electrophysiologists as well (14–16). Because of the potential of this mode of pacing for offering safe permanent antitachycardia pacemak-

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Table 1. Clinical Data and Descriptors of the Bypass Tract in 10 Patients

Patient No.	Age (yr) & Sex	Bypass Tract Site	Tachycardia CL (ms)	ERP of Bypass Tract (ms)	Pacing Threshold (mA)
Group I					
1	28F	Distal CS	300	≤200	1.6
2	26F	Distal CS	400	300	1.5
3	30F	Distal CS	290	NA	1.2
4	53F	Distal CS	270	200	2.0
5	35F	Distal CS	380	245	0.8
6	48M	Distal CS	360	<200	0.6
7	65M	Distal CS	420	389	1.2
Group II					
8	53F	Distal CS	350	260	0.7
9	44F	Distal CS	435	250	1.0
10	33F	Distal CS	375	290	1.1

CL = cycle length; CS = coronary sinus; ERP = effective refractory period (anterograde); F = female; Group I = patients whose tachycardia was terminated by subthreshold pacing; Group II = patients whose tachycardia was not terminated by subthreshold pacing; M = male; NA = not available.

ing without the heart rate-accelerating complications of suprathreshold pacing, we investigated the efficacy of subthreshold atrial pacing in terminating reentrant tachycardia in 10 patients with a left-sided atrioventricular (AV) accessory pathway.

Methods

Study patients (Table 1). Ten patients were studied in the electrophysiology laboratory because of an appropriate clinical history, namely, troublesome supraventricular tachyarrhythmias related to the probable presence of an accessory AV pathway. To be included in the study, patients needed to have a reliably induced reciprocating AV tachycardia utilizing a left-sided accessory pathway. The tachycardia also needed to be: 1) well tolerated by the patient, and 2) a sustained supraventricular tachycardia, that is, lasting ≥5 min. The study group consisted of 10 patients, 8 women and 2 men, with a mean age of 42 ± 13 years. Four electrode catheters were inserted percutaneously into peripheral veins and positioned under fluoroscopic guidance into the appropriate intracardiac recording and stimulating sites. For the purpose of this study, the most relevant electrode catheter was the one positioned in the coronary sinus. This catheter (Webster Laboratories) was a six to eight pole electrode catheter with a 2 mm interelectrode distance and a 10 mm distance between electrode pairs. The catheter was inserted from the left brachial vein and positioned in the coronary sinus such that at least three electrode pairs were within the body of the coronary sinus. Three orthogonal surface electrocardiographic (ECG) leads were displayed and recorded (Electronics for Medicine, VR-12) as were the intracardiac electrograms filtered at 30 to 500 Hz. Recording paper speeds were 50 and 100 mm/s.

Localization of the bypass tract. Electrophysiologic localization of the bypass tract was achieved in each patient using accepted clinical electrophysiologic maneuvers (17). Namely, pacing at several right atrial and coronary sinus sites at several paced cycle lengths was performed and the shortest stimulus to delta wave interval was determined; the earliest atrial activation site was located during supraventricular tachycardia and during ventricular pacing. When available, the effects of functional bundle branch block on tachycardia rate as well as on ventriculoatrial conduction time were also evaluated.

Pacing protocol. Before initiating subthreshold pacing, the reliability of termination of the supraventricular tachycardia with *suprathreshold* pulses was confirmed by pacing from the atrial site of the insertion of the bypass tract, that is, from the electrode pair in the coronary sinus that was judged to be closest to the bypass tract using the previously listed criteria. In addition, the anterograde effective refractory period of the bypass tract was measured (in patients with a bypass tract capable of anterograde conduction, that is, in 9 of the 10 patients) using the atrial extrastimulus technique at a paced cycle length similar to the tachycardia cycle length. All pacing was bipolar, with the more distal electrode designated as the cathode. Pacing pulses were rectangular in shape and 2 ms in duration.

The late diastolic threshold of the coronary sinus pacing site was measured at a paced rate similar to the tachycardia rate. The reciprocating supraventricular tachycardia was then initiated. When it was apparent that the induced tachycardia was a stable and sustained rhythm, a continuous train of subthreshold atrial stimuli was then delivered at a current intensity of 60 to 70% of threshold current. At the onset of the protocol, atrial pacing was performed at a rate approximately 10 to 15 beats/min faster than the tachycardia rate. The pacing train was maintained for 15 s and then terminated. If the initial current setting resulted in termination of the tachycardia, the tachycardia was reinitiated and the pacing current was decreased by 10%. This was repeated until the tachycardia could no longer be terminated by a train of subthreshold impulses. When the initial current setting was inadequate for terminating the tachycardia, the pacing intensity was increased by 10% and the 15 s pacing train was repeated. The lowest current intensity required for termination of the tachycardia with "overdrive" pacing was thus determined.

The rate of the subthreshold pacing train was also varied in each patient. The effective subthreshold current was always sought while pacing at the initial rate of 10 to 15 beats/min faster than the tachycardia rate; when no subthreshold currents were found to be effective at this pacing rate, subthreshold pacing was attempted at several faster pacing rates, typically with decrements of 50 ms. In patients

with successful termination of the tachycardia with subthreshold pacing, several pacing rates were also tried at the lowest current setting found to be *ineffective*. In two patients (Patients 2 and 7) subthreshold stimulation rates *slower* than the tachycardia rate were also tried at an effective subthreshold current setting.

Finally, in patients with successful termination of the tachycardia with subthreshold pacing, a second pacing site was also tested during sustained tachycardia using the current and pacing rate settings found to be effective at the first pacing site, that is, the presumed atrial site of the bypass tract. At the termination of the pacing protocol, the pacing threshold was measured again. Position of the pacing catheter was also fluoroscopically confirmed at the end of the pacing protocol.

Statistics. Results of interval and current measurements are expressed as mean ± standard deviation. We performed *t* tests for unpaired samples in comparing the tachycardia rate, effective refractory period of the bypass tract and the pacing threshold in Group I and Group II patients.

Results

Patient characteristics (Table 1). In all 10 patients who underwent the subthreshold pacing protocol, the location of the accessory pathway was presumed to be closest to the electrode pair located in the distal coronary sinus. The bypass tract was of the "concealed" variety in one case (Patient 3). On the basis of their response to the subthreshold pacing protocol, patients were classified into two groups: Group I, the seven patients whose tachycardia could be successfully terminated with subthreshold pacing and Group II, the three patients in whom no combination of subthreshold pacing currents or rates could be found to terminate the tachycardia. All patients exhibited reliably inducible, sustained orthodromic reciprocating supraventricular tachycardia. The tachycardia always incorporated the accessory pathway in its reentrant circuit; AV nodal reentrant tachycardia was not documented in any of these patients. Also, in none of the patients was the presence of a second accessory pathway demonstrated, although it was sought in each patient. The mean tachycardia cycle length was 358 ± 56 ms, the mean anterograde effective refractory period of the accessory pathway (when measurable) was 238 ± 90 ms and the mean pacing threshold was 1.17 ± 0.44 mA.

Mechanisms of termination of tachycardia with subthreshold pacing. In each of the seven Group I patients, subthreshold pacing effectively and reproducibly terminated the supraventricular tachycardia. In Group I a mean of 4 ± 2 successful and 3 ± 2 unsuccessful attempts at tachycardia termination with subthreshold pacing were recorded. The electrophysiologic events leading to tachycardia termination were remarkably similar in each patient. The following descriptions serve as illustrations of the mechanisms involved in tachycardia termination.

Patient 1 (Fig. 1 and 2).

Termination of an orthodromic supraventricular tachycardia with a cycle length of 300 ms was performed using a suprathreshold (twice diastolic) train of paced atrial impulses at a paced cycle length of 250 ms (Fig. 1A). The last four QRS complexes are wide as a result of ventricular activation occurring predominantly by way of a left-sided accessory pathway. Termination of the tachycardia in this instance occurred when, before the first wide QRS complex, a paced atrial impulse succeeded in capturing the accessory pathway, thereby interrupting a macroreentrant circuit that utilized the accessory pathway for retrograde activation of the atria. In Figure 1B the supraventricular tachycardia had been reinitiated. Earliest retrograde atrial activation (recorded at high paper speed) was always recorded in the distal coronary sinus electrode pair (although in Fig. 1B the mid-coronary sinus atrial electrogram was slightly earlier than the His bundle atrial electrogram whereas the latter actually preceded the "proximal coronary sinus" electrogram; this may have been due to placement of the proximal electrodes outside the coronary sinus). In this instance, subthreshold pacing had been initiated from the site of earliest atrial activation during the tachycardia, that is, from the distal coronary sinus (CS_d). Subthreshold pacing was performed at 63% of threshold current (1.0 mA) and at a paced cycle length of 250 ms. The thin arrow points to the impulse that terminated the tachycardia. The final tachycardia interval measured in the right atrial electrogram was 40 ms shorter than the tachycardia cycle length, that is, 260 ms. Advancement of the last atrial impulse (marked by a thick arrow) occurred with a left to midline activation sequence. Subsequent to termination of the tachycardia, subthreshold pacing was continued for an additional 17 impulses (4 s) *without* affecting atrial or ventricular capture. Note that the P wave vector during this period of subthreshold pacing was identical to the vector seen during normal sinus rhythm, thereby excluding the possibility that the atrial rate was somehow controlled by the subthreshold pacing. Thus the subthreshold nature of the pacing impulses was maintained before and after termination of the tachycardia.

In Figure 2, tachycardia termination in the same patient followed a slightly different electrophysiologic sequence. Subthreshold pacing at the same current intensity and at the same pacing rate was repeated during a tachycardia with a cycle length of 300 ms. This time the last *two* (high right atrial) impulses during the tachycardia were advanced and the last two tachycardia cycles measured 270 and 250 ms, respectively. Thus, the first of these two cycles was foreshortened by 30 ms and the second appears to have assumed the subthreshold pacing cycle rate. It is of interest that the first local capture in the coronary sinus did not advance the right atrial electrogram, suggesting that at least some fibers of the accessory pathway were unaffected by this first

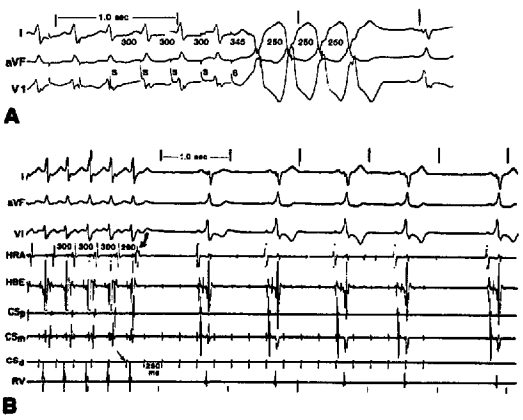

Figure 1. Patient 1. Termination of reciprocating tachycardia with supra- and subthreshold pacing. **A**, Conventional suprathreshold atrial pacing during orthodromic tachycardia (cycle length 300 ms) terminates the tachycardia during the longest cycle (345 ms) by preferentially conducting in the anterograde direction over the left-sided accessory pathway. Subsequent QRS complexes are wide and pre-excited as one to one anterograde conduction at the paced cycle length of 250 ms occurs. **B**, Subthreshold pacing at 63% of threshold current is performed from the distal coronary sinus (CS_d) at the same pacing rate (250 ms) during an identical tachycardia. The last atrial impulse during the tachycardia (**thick arrow**) has been advanced 40 ms by a "subthreshold" impulse (**thin arrow**); the tachycardia is terminated at the anterograde (AV node) limb of the circuit. See text for further details. Surface leads I, aVF and V_1 are shown, along with high right atrial (HRA), His bundle (HBE), proximal coronary sinus (CS_p), mid-coronary sinus (CS_m) and right ventricular (RV) electrograms. The distal coronary sinus (CS_d) tracing serves as the stimulus marker.

subthreshold capture or that the pacing site was not exactly at the site of atrial insertion of the bypass tract. Atrial activation sequence was again preserved during the final two rapid cycles (marked by the thick arrows). The tachycardia terminated when pacing produced an atrial impulse that was sufficiently premature (at 250 ms) to block within the AV node. The coupling intervals (PS) of the subthreshold impulses that produced the transient atrial capture illustrated in this patient ranged from 0.65 to 0.70 of the basic PP interval during the supraventricular tachycardia (Table 2). Deviations of more than approximately 10 ms in the stimulus coupling intervals failed to evoke atrial capture.

Patients 6 and 7 (Fig. 3 and 4). Figure 3 shows a termination sequence similar to that shown in Figure 1B and was chosen to illustrate the effects of very *rapid* subthreshold pacing. During a tachycardia with a cycle length of

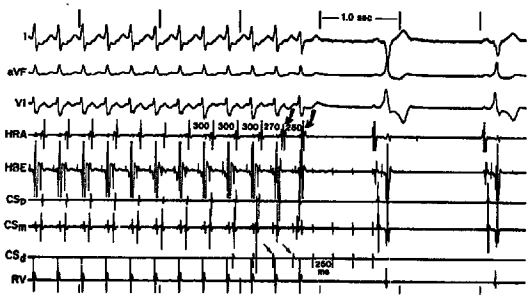

Figure 2. Patient 1. Termination of tachycardia caused by two instances of premature atrial capture during (otherwise) subthreshold atrial pacing. Subthreshold pacing is performed using the same current intensity and pacing rate during an identical tachycardia as in Figure 1. The last two atrial impulses (HRA) are foreshortened (**thick arrows**) and advancement of the last *three* atrial impulses is shown in the mid-coronary sinus electrogram. Abbreviations as in Figure 1. See text for details.

360 ms, pacing at 67% of threshold current (0.4 mA) and at a paced cycle length of 100 ms, the final tachycardia cycle was advanced by 70 ms and followed by abrupt resumption of normal sinus rhythm, again without eliciting further atrial or ventricular capture.

In contrast, Figure 4 (Patient 7) shows the effects of subthreshold *underdrive* pacing. During a tachycardia with a cycle length of 400 ms, subthreshold pacing at 67% of threshold current (0.8 mA) and at a cycle length of 500 ms produced a marked alteration in heart rate and termination of the tachycardia, again without evoking a pre-excited QRS complex. The first altered tachycardia cycle was caused by a 50 ms advancement of the atrial signal that encroached on the relative refractory period of the AV node (as evidenced by a marked prolongation of the AH interval). The next paced impulse appears also to have achieved atrial capture and the resultant atrial wave of excitation blocked in the AV node despite a PP interval that was well in excess of the effective refractory period of the AV node (335 ms at a paced cycle length of 500 ms). In this patient as in all Group I patients, subthreshold atrial pacing failed to produce *ventricular* pre-excitation although it caused termination of the reciprocating tachycardia with appropriately positioned premature atrial stimuli. Regardless of the rate of subthreshold pacing, in none of our patients did subthreshold pacing result in the induction of atrial fibrillation or flutter.

Comparison of pacing currents and rates (Table 2). Not surprisingly, when the *minimal* current intensity of the successful pacing episodes was compared in each patient with the *maximal* intensity of the unsuccessful pacing episodes, a significant difference was found: the minimal successful currents were 64 ± 14% of threshold current and the maximal unsuccessful currents were 49 ± 17% of threshold current ($p < 0.05$). Further analysis of tachycardia termination in these patients revealed that the mean subthreshold pacing rate was 82 ± 25% of the tachycardia rate and that the mean advancement of the first altered tachycardia cycle was 20 ± 4% of the tachycardia cycle length.

Table 2. Electrophysiologic Measurements During Subthreshold Atrial Pacing in Group I

Patient No.	Subthreshold Current (% TC)		PCL at Termination of SVT (% CL)	Shortening of Atrial Cycle (%)	(PS)/(PP) Ratio
	Effective	Ineffective			
1	63	47	83	20	0.65–0.70
2	87	71	85	18	0.71
3	42	29	88	21	0.76
4	75	63	93	19	0.70
5	63	32	97	26	0.55–0.68
6	59	NA	28	22	0.78
7	59	50	83	14	0.71–0.73
Mean	64 ± 14	49 ± 17*	80 ± 23	20 ± 4	0.71 ± 5

*$p < 0.05$ when compared with effective subthreshold current. For this comparison, the results of each pacing run in each patient are used rather than the mean values listed in this table. CL = cycle length of supraventricular tachycardia; PCL = paced cycle length; PP = interval between successive atrial signals, i.e., the tachycardia cycle length; PS = interval from atrial signal to stimulus; SVT = supraventricular tachycardia; TC = threshold current (mA).

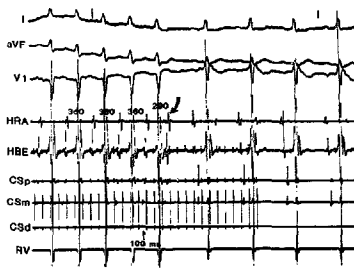

Figure 3. Patient 6. Termination of reciprocating tachycardia during rapid subthreshold atrial pacing. The effect of very rapid (100 ms cycle length) coronary sinus subthreshold pacing is shown during a reciprocating tachycardia with a cycle length of 360 ms. A single atrial capture (**thick arrow**) shortens the last atrial cycle during tachycardia by 70 ms, thereby causing termination of the tachycardia. After termination of the tachycardia, atrial capture does not recur. Abbreviations as in Figure 1.

We looked for differences in the two groups of patients that might account for observed disparity in responses to subthreshold pacing. When comparing the tachycardia rate in Group I with that in Group II, no significant difference was found (345 ± 59 versus 387 ± 44 ms, $p = 0.3$). Similarly, the anterograde effective refractory period of the accessory pathway did not significantly differ in the two groups of patients (264 ± 68 versus 267 ± 21 ms, $p = 0.9$). A comparison of atrial effective refractory periods suggested shorter refractory periods in Group I than in Group II patients (214 ± 20 versus 248 ± 43) but this difference did not achieve significance. Finally, significant difference in atrial (coronary sinus) pacing thresholds were not detectable between Groups I and II (1.3 ± 0.5 versus 0.9 ± 0.2 mA, respectively, $p = 0.2$).

Discussion

Results of subthreshold atrial pacing. Our study provides several new findings: 1) subthreshold atrial pacing is an effective method of terminating reciprocating AV tachycardia in many patients with a left-sided accessory pathway; 2) termination of orthodromic tachycardia is accomplished by an apparent transient atrial capture at the bypass tract pacing

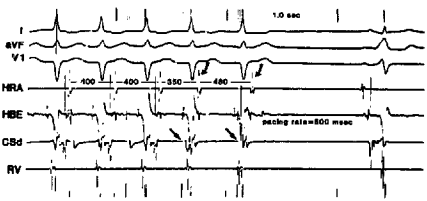

Figure 4. Patient 7. Underdrive subthreshold pacing causing tachycardia termination. Pacing at a rate (120 beats/min) slower than the tachycardia rate (150 beats/min) at 67% of threshold current produces two instances of atrial (distal coronary sinus) capture that result in termination of the tachycardia. There is absence of ventricular pre-excitation during the captured beats. Abbreviations as in Figure 1.

site that interrupts the tachycardia circuit by prematurely invading the AV node (anterograde) limb of this circuit; 3) on termination of the tachycardia, continuous atrial capture *does not* take place despite continuous subthreshold pacing; and 4) ventricular activation by way of the accessory pathway does not occur during brief episodes of subthreshold pacing such as those performed in this study.

Possible mechanisms. By what mechanisms does a continuous train of discrete subthreshold stimuli elicit transient capture of adjacent myocardial (atrial) tissue? We considered several possibilities and phenomena that could be invoked to explain our observations.

Supernormal excitation. Supernormality refers to a phase of recovery of excitability during which impulses are propagated more rapidly and less current is required to reexcite the tissue than at a later period in diastole. This phenomenon has been demonstrated in isolated preparations of the canine bundle branch Purkinje system but may not be a property of His bundle fibers or ventricular muscle fibers (18). A period of supernormal excitability has also been described in the specialized atrial fibers of Bachmann's bundle (19). In canine bundle branch preparations, supernormality is voltage dependent (most prominently at a phase 3 membrane voltage of 74 mV in one set of experiments [18]) and may be a normal property of conduction tissues as well as a result of ischemic or hypoxic injury to cardiac tissue (20). Although the existence and in vivo relevance of supernormality has been questioned by some (20), a recent preliminary report sought to demonstrate supernormal conduction in accessory pathways with slow conduction properties (21). In our Group 1 patients, subthreshold atrial stimulation resulted in atrial capture during tachycardia when the coupling interval of the pacing stimulus fell within a narrow window, usually at approximately 71% of the PP interval during tachycardia. Also, in most instance the coupling interval of the stimulus that resulted in atrial capture was close to the atrial effective refractory period measured at the stimulation site.

Still another point in favor of supernormal excitability is the absence of atrial capture by subthreshold pacing stimuli, that are not preceded by atrial depolarization, that is, during the prolonged electrical diastole in normal sinus rhythm (Fig. 1B, 2 and 3). By this explanation, impulses that were measured to be subthreshold during normal sinus rhythm may cause local atrial capture during tachycardia when they fortuitously occur at an appropriate time in atrial repolarization. Supernormal excitability offers a plausible explanation for our observations in most, but not all, of the instances of tachycardia termination during subthreshold pacing. In at least one instance, however, supernormality cannot be invoked because a "subthreshold" impulse caused atrial capture even though the impulse coupling interval was considerably longer than the atrial effective refractory period, for example, the second impulse in Figure 4 (during underdrive pacing).

Summation of electrical impulses. We also considered the electrophysiologic phenomenon of summation of electrical impulses has previously been described both in vitro and in whole animal experimental models (11,22). Cranefield and Hoffman (2) described a canine Purkinje fiber model in which conduction and responsiveness of a segment of the preparation were depressed by encasing the segment in hyperkalemic agar. Stimulation from either end of the preparation caused minimal or no responses in the depressed segment, but stimulation of *both* segments produced a normal response in the depressed segment of the preparation. Stimulation of the two pacing sites need not have been simultaneous for impulse summation to occur, as summation was observed over a wide range of relative timing of excitation of the proximal and distal sites. Extrapolating from these experimental observations to our results, one might postulate that slightly premature subthreshold stimuli, when combined with an arriving retrograde impulse from the accessory pathway, resulted in summation of impulses and caused the observed atrial capture. A problem with this explanation, however, is the marked prematurity of the captured atrial impulse. Although in the clinical setting of our study we have no way of determining the actual atrial arrival time of the impulse that traverses the accessory pathway, our observation that atrial activation was typically advanced by 30 to 50 ms makes it less plausible that the accessory pathway impulses play a significant role in causing the observed atrial capture.

Motion of coronary sinus catheter and lowering of pacing

threshold. Two other explanations for the observed atrial capture during subthreshold pacing were considered. First, motion of the coronary sinus catheter during tachycardia might have occurred and caused occasional atrial capture. This was, however, not observed to occur spontaneously in the absence of subthreshold pacing. Second, a mechanism that allows for a lowering of the pacing threshold either with changes in heart rate (that is, as a result of the tachycardia) or as a product of several seconds of atrial pacing was considered. This second explanation has been demonstrated (12) in rabbit atrial trabeculae that have been partially depolarized by barium and by a hyperkalemic environment. A gradual decline in threshold current is seen during repetitive *threshold* stimulation of this preparation ("threshold current hysteresis"). Furthermore, repetitive *subthreshold* stimulation can result in one to one activation of the preparation by causing repetitive "triggered activity." Again, neither of these mechanisms satisfactorily explains our observations, because on termination of the tachycardia atrial activation *ceased* despite continuous subthreshold pacing.

Possible mechanisms for absence of pre-excitation during subthreshold atrial pacing. Our results pose yet another problem: unlike suprathreshold pacing, which terminated episodes of tachycardia and produced ventricular preexcitation by virtue of anterograde conduction over a bypass tract (Fig. 1A), subthreshold pacing *failed* to cause manifest ventricular pre-excitation despite the isolated instances of atrial capture at the atrial insertion site of the accessory pathway. A possible explanation for this observation is that the premature atrial capture evoked by subthreshold pacing produces an action potential that is suboptimal in amplitude and rate of rise. When a relatively inadequate wave front traveling over an accessory pathway reaches the ventricular insertion point of the accessory pathway, it is likely not to be sufficient to bring the much larger volume of ventricular myocardial cells to threshold (the so-called impedance mismatch theory [23]).

Clinical implications. Permanent antitachycardia pacing for the termination of recurrent supraventricular tachycardia has not yet become widely accepted. When considering this mode of therapy electrophysiologists have been fearful of provoking atrial fibrillation with its attendant rapid AV conduction in patients with accessory pathways and, because of similar considerations, they have been reluctant to use "burst" atrial pacing, despite its superior efficacy, in patients with accessory pathways. Our study suggests that subthreshold atrial pacing may prove to be an effective and relatively risk-free mode of permanent antitachycardia pacing in selected patients who prove to be susceptible to this form of pacing in the clinical electrophysiology laboratory. Obviously, much work remains to be done in determining optimal pacing variables during subthreshold pacing, including pulse width, pacing sites, current intensity and measurement of long-term pacing thresholds.

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