

## Demonstration of a Posterior Atrial Input to the Atrioventricular Node During Sustained Anterograde Slow Pathway Conduction

CHARLIE YOUNG, MD, FACC, MICHAEL R. LAUER, MD, PhD, L. BING LIEM, DO,  
HINGSON CHUN, MD, RUEY J. SUNG, MD, FACC

San Jose and Stanford, California

**Objectives.** This study sought to demonstrate electrophysiologic evidence for the existence of different anatomic atrial input sites of fast and slow conduction pathways in patients with dual atrioventricular (AV) node physiology.

**Background.** Although a separate posterior exit site exists for a retrograde slow AV node pathway, it remains unresolved whether a separate atrial input site into the AV node actually exists in patients with dual anterograde AV node pathway physiology.

**Methods.** In 10 patients with dual AV node pathway physiology, atrial pacing at three chosen drive cycle lengths (DCL1, DCL2 and DCL3) was performed at an anterior site (A) just above the His bundle recording site and at a posterior atrial site (P) just below the coronary sinus ostium. DCL3 was chosen as the one cycle length that resulted in a long AH interval consistent with slow pathway conduction. The stimulus to His bundle conduction times (SH) at both sites (SH<sub>P</sub> and SH<sub>A</sub>, respectively) and their

differences ( $\Delta SH = SH_P - SH_A$ ) at each of the three drive cycle lengths were analyzed.

**Results.** The mean  $\pm$  SD  $\Delta SH$  values for DCL1 and DCL2 measured  $9 \pm 16$  and  $8 \pm 18$  ms, respectively, and the mean  $\Delta SH$  value at DCL3 measured  $-34 \pm 24$  ms, which was significantly different from the mean  $\Delta SH$  values at DCL1 and DCL2 (both  $p < 0.05$ ).

**Conclusions.** The significant change in the  $\Delta SH$  (SH<sub>P</sub> - SH<sub>A</sub>) value during slow pathway conduction could be accounted for by a corresponding shift of anterograde input from an anterior to a posterior entry site to the AV node. These findings support the notion that a separate anterograde entry site of the slow pathway does exist in patients with dual AV node pathway physiology.

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Atrioventricular (AV) node modification by selective radiofrequency energy catheter ablation of the slow conducting AV node pathway is an effective curative treatment for patients with AV node reentrant tachycardia (1-4). In 1981, Sung et al. (5) showed that the retrograde exit site of the slow pathway was located near the coronary sinus ostium, posteriorly and inferiorly to that of the fast pathway. Many investigators (1-3,6-9) have subsequently shown that catheter ablation lesions placed along the tricuspid valve annulus in the posterior aspect of the interatrial septum near the coronary sinus ostium can selectively eliminate or modify anterograde slow AV node conduction without affecting anterograde fast AV node conduction. These findings confirm that the slow conducting pathway is anatomically distinct from the fast pathway. However, it remains unresolved whether a separate anterograde atrial input into the AV node actually exists in humans.

We hypothesized that the fast and slow pathways may

indeed have anatomically different atrial entry sites, with the input to the slow pathway located more posterior and inferior than the input to the fast pathway. In the present study, we designed a unique atrial pacing protocol with differential site pacing to electrophysiologically demonstrate the presence of separate atrial entry sites of fast and slow pathways.

### Method

**Patients.** Twenty consecutive patients undergoing radiofrequency catheter ablation for AV node reentrant tachycardia were evaluated. Standard intracardiac electrode catheters were positioned in the coronary sinus (CS), high right atrium, right ventricular apex and the His bundle region. Intracardiac and surface electrocardiographic leads I, aVF and V<sub>1</sub> were continuously recorded and stored for analysis (ART, Inc.). Interval measurements were performed using the computerized calipers at 100-mm/s sweep speed. The baseline electrophysiologic study was performed with atrial and ventricular stimulation, respectively, at twice diastolic threshold and 2.0 ms in duration through a programmable stimulator (model DTU-125, Bloom Associates, Ltd.)

**Study Protocol.** Ten of the 20 patients with inducible AV node reentrant tachycardia were selected for study. All 10 patients manifested dual AV node physiology with discontinuous AV node conduction curves (A<sub>1</sub>A<sub>2</sub>, A<sub>2</sub>H<sub>2</sub>; A<sub>1</sub>A<sub>2</sub>, H<sub>1</sub>H<sub>2</sub>)

From the Cardiac Electrophysiology Laboratories, Kaiser Foundation Hospital-Santa Teresa, San Jose; and Stanford University Medical Center, Stanford, California. This report was presented in part at the 67th Scientific Sessions of the American Heart Association, Dallas, Texas, November 1994.

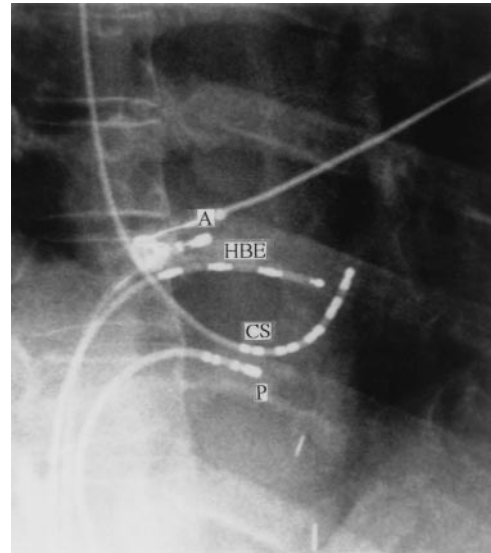
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Address for correspondence: Dr. Charlie Young, Cardiac Electrophysiology Service, Santa Teresa Community Medical Center, 275 Hospital Parkway, Suite 355, San Jose, California 95119. E-mail: [charlie.young@ncai.kaiser.org](mailto:charlie.young@ncai.kaiser.org).

**Abbreviations and Acronyms**

A	= anterior site
AH	= atrio-His interval measured at the His recording catheter
AV	= atrioventricular
CS	= coronary sinus
DCL	= drive cycle length
P	= posterior site
SH <sub>A</sub>	= stimulus to His interval measured at site A during each pacing study
SH <sub>P</sub>	= stimulus to His interval measured at site P during each pacing study
ΔSH	= SH <sub>P</sub> - SH <sub>A</sub> -derived difference of SH values measured at sites P and A for each drive cycle length studied

(i.e., an increment of at least 50 ms in the AH interval after a shift from fast to slow pathway conduction). In addition, during burst atrial pacing, a greater than 50 ms increment in the A<sub>1</sub>H<sub>1</sub> interval resulting from a critical 10-ms decrement in the S<sub>1</sub>S<sub>1</sub> interval was also demonstrated. Furthermore, patients were specifically selected on the basis of their ability to achieve a stable “steady state” AV node conduction during continuous atrial pacing at various cycle lengths. Steady state conduction was achieved when the AH interval varied <5 ms during at least 10 s of atrial pacing. Patients were also excluded if the induction of AV node reentrant tachycardia during pacing interfered with the study or if the AV node Wenckebach phenomenon had occurred before stable AV node conduction was achieved. After the baseline electrophysiologic study was completed, the high right atrial and right ventricular apex catheters were replaced with two 4-mm large-tipped deflectable 7F quadripolar catheters with 2-mm interelectrode distance. These two electrode catheters were positioned to obtain clear bipolar atrial electrograms and stable pacing thresholds at two selected sites. One catheter was placed at the anterior atrial septum (site A), just above the His bundle recording site, and the other was placed at the posterior atrium (site P), just below the CS ostium (Fig. 1). The distal pair of electrodes was used for stimulation and the proximal pair for recording the local electrogram. During baseline conditions, three progressively shorter fixed pacing drive cycle lengths (DCLs), were chosen for study. The DCLs were specifically chosen such that the two longer DCLs resulted in constant short AH intervals representative of fast pathway conduction, and the third, or shortest DCL produced the longest constant AH interval representing slow pathway conduction without inducing the AV node Wenckebach phenomenon or AV node reentrant tachycardia. The AH interval at this shortest DCL was within the range of slow pathway conduction as defined by the A<sub>1</sub>A<sub>2</sub> versus A<sub>2</sub>H<sub>2</sub> curve measured during the baseline atrial extra-stimulation study (Table 1), ensuring that the drive chosen indeed resulted in slow pathway conduction. These preselected DCLs (DCL1, DCL2, DCL3, e.g., 660, 530, 500 ms) were then used to perform the three pacing trials in each patient. Pacing from each site was performed at a 2-ms pulse width and at



**Figure 1.** Fluoroscopic image (30° right anterior oblique projection) indicating the catheter positions during the pacing studies. Sites A and P are displayed relative to the location of the His bundle electrode (HBE) and CS catheters.

twice diastolic threshold of the pacing site with the greater threshold. All pacing studies were completed with <3.0-V maximal pulse amplitude. During each pacing trial, pacing was initially begun at site A, and after achieving steady state (at least 15 beats), the pacing site was abruptly switched to site P.

**Defined measurement intervals.** The conduction time from the pacing sites (A or P) through the AV node to the His bundle was measured as the interval from the stimulus artifact to the His bundle potential (SH interval). SH intervals were measured over 5 consecutive beats with <5 ms of absolute variation for all beats measured. During each pacing trial, the SH conduction times recorded at site A (SH<sub>A</sub>) and site P (SH<sub>P</sub>) were compared, and the difference in conduction time between these two sites was calculated as  $\Delta SH = SH_P - SH_A$ . Figure 2 illustrates an example of measured data from Patient 10. The three  $\Delta SH$  values for each of the pacing trials performed in each patient were then compared for statistically significant differences.

**Statistical Analysis.** Results are expressed as mean value  $\pm$  SD. Statistical analysis among the three groups was performed with the use of one-way analysis of variance, with multiple comparisons performed with the two-tailed Student-Newman-Keuls test. Statistical significance was accepted at the  $p < 0.05$  level.

## Results

**Electrophysiologic characteristics.** The baseline electrophysiologic data of the 10 patients are summarized in Table 1. In addition to effective refractory periods of fast and slow pathways, the range of AH intervals of both pathways derived from A<sub>1</sub>A<sub>2</sub> versus A<sub>2</sub>H<sub>2</sub> at 600-ms drive are displayed. In all 10 patients, the longest AH interval (A<sub>2</sub>H<sub>2</sub> at 600 ms drive) for

**Table 1.** Baseline Electrophysiologic Data for 10 Patients

Pt No.	AH (ms)	HV (ms)	AV BCL (ms)	Fast Pathway		Slow Pathway		VA BCL (ms)	SVT CL (ms)
				ERP (600) (ms)	AH Range (ms)	ERP (600) (ms)	AH Range (ms)		
1	83	55	400	370	83-180	260	250-450	300	370
2	90	35	450	430	90-150	<240	210-380	450	320
3	90	45	350	460	90-190	290	270-350	350	300
4	85	35	240	450	85-175	250	240-370	280	300
5	92	55	310	300	92-220	<220	280-320	240	420
6	75	40	400	420	75-200	280	250-330	350	320
7	95	40	450	500	95-210	290	300-400	350	340
8	80	45	350	360	80-160	240	240-410	340	350
9	100	40	380	440	100-180	300	280-390	700	320
10	79	47	450	420	79-175	320	256-452	450	360

AH Range = measured range of AH intervals recorded during fast or slow pathway conduction; AV BCL = atrioventricular (AV) node block cycle length; ERP (600) = effective refractory period of the AV node of the indicated conduction pathway at a drive cycle length of 600 ms; Pt = patient; SVT CL = cycle length of the supraventricular tachycardia; VA BCL = ventriculoatrial conduction block cycle length through the normal AV node pathway.

fast pathway conduction was  $\leq 220$  ms (range 150 to 220 ms). In contrast, the longest AH intervals for slow pathway conduction measured ranged from 320 to 450 ms. The shortest slow pathway conduction time measured at the shift from fast to slow pathway conduction was never  $< 210$  ms (range 210 to 330 ms). Retrograde AV node conduction during ventricular pacing at corresponding DCLs was always through the fast pathway in these 10 patients. Typical slow-fast AV node reentrant tachycardia was inducible in all patients. Selective catheter ablation of slow pathway conduction was performed successfully in all 10 patients by means of previously described techniques (1-3,6-9).

**Patterns of AV node conduction and measurement of conduction time.** Figure 2 shows how all SH conduction times were measured over 5 consecutive beats with  $< 5$  ms of absolute variation. However, as noted in Figure 2C, after the switch from pacing at site A with an SH of 385 ms, there was persistence of a long SH interval of 381 ms before the adjustment to a new and stable shorter SH of 335 ms. This phenomenon was noted in all 10 patients during pacing at DCL3 and was not noted to be significant in any patients at DCL1 or DCL2.

The SH conduction times from each patient recorded from sites A and P during pacing with each of the three cycle lengths (DCL1 to DCL3) are displayed in Table 2. Longer SH times were seen at successively shorter DCLs in all patients. The SH times for DCL1 and DCL2 were both short (mean  $SH_A = 126 \pm 29$  ms,  $SH_P = 135 \pm 25$  ms for DCL1; mean  $SH_A = 146 \pm 26$  ms,  $SH_P = 154 \pm 26$  ms for DCL2) compared with the much longer SH time measured at the DCL3 (mean  $SH_A = 350 \pm 67$  ms,  $SH_P = 315 \pm 76$  ms).

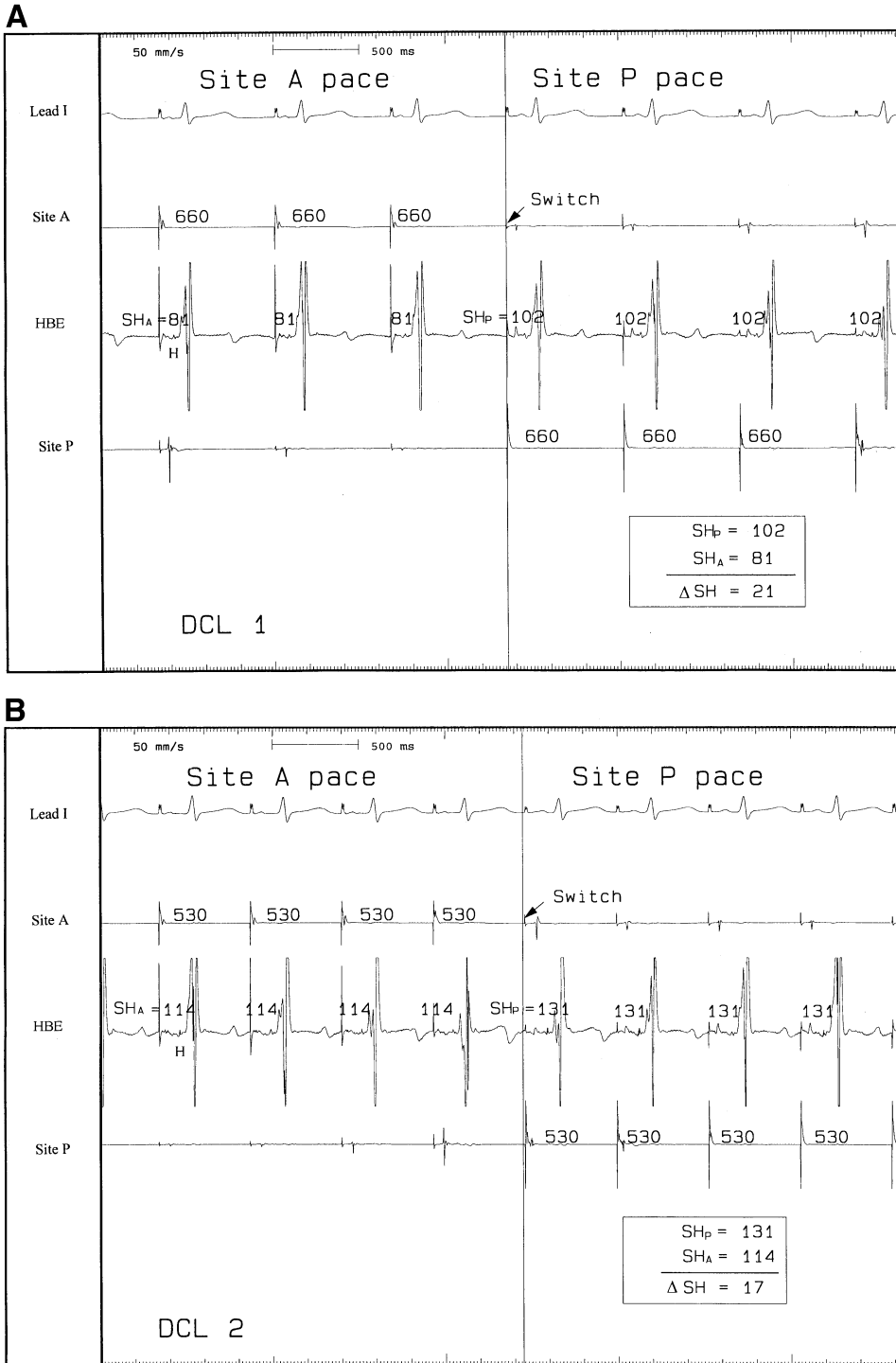
The  $\Delta SH$  values ( $SH_P - SH_A$ ) for all patients at each DCL are also displayed in Table 2. The range of  $\Delta SH$  values was noted to be wide in each of the DCLs ( $-20$  to  $25$  ms,  $-30$  to  $26$  ms,  $-80$  to  $5$  ms for DCL1, DCL2, DCL3, respectively). The difference in mean  $\Delta SH$  values between DCL1 and DCL2 ( $9 \pm 16$  and  $8 \pm 18$  ms) was not statistically different. However, the

mean  $\Delta SH$  at DCL3 measured  $-34 \pm 24$  ms, which was significantly different from the mean  $\Delta SH$  at DCL1 and DCL2 (both  $p < 0.05$ ). Figure 3 shows a summary plot of the data displayed in Table 2 and compares  $\Delta SH$  values among all three DCLs for each patient. Again note that  $\Delta SH$  is similar for DCL1 and DCL2 but is markedly different from DCL3 within each individual patient, as well as for the mean values of the entire group.

## Discussion

**AV node conduction patterns during drive trains.** All patients studied had dual AV node pathway conduction patterns demonstrable at baseline by discontinuous  $A_1A_2 A_2H_2$ ;  $A_1A_2, H_1H_2$  curves (Table 1). Evidence of a dual pathway conduction pattern was also demonstrated during incremental atrial pacing by a sudden increment in  $A_1H_1$  ( $> 50$  ms) associated with a critical 10-ms decrease in pacing cycle length. A sudden increase in conduction time with AH intervals in the slow pathway range strongly supported that slow pathway conduction was present in these patients when the pacing cycle length was changed from DCL2 to DCL3. Thus, AV node conduction time measured during DCL3 pacing trial represented slow pathway conduction, whereas during DCL1 and DCL2 pacing trials, AV node conduction was through the fast pathway.

**Changes in  $\Delta SH$  related to differential site of pacing.** In the present study, a geometric argument is put forth such that if a separate slow pathway input is located at the posterior inferior aspect of the AV node region, then the conduction time measured during slow AV node conduction from stimulus to His bundle recording should be shorter when pacing is performed closer to the slow pathway input posteriorly at site P ( $SH_P$ ) and longer when pacing further away anteriorly at site A ( $SH_A$ ). Conversely, during fast pathway conduction, because the input to the fast pathway is now more anterior, the measured conduction time at site P ( $SH_P$ ) is now longer than



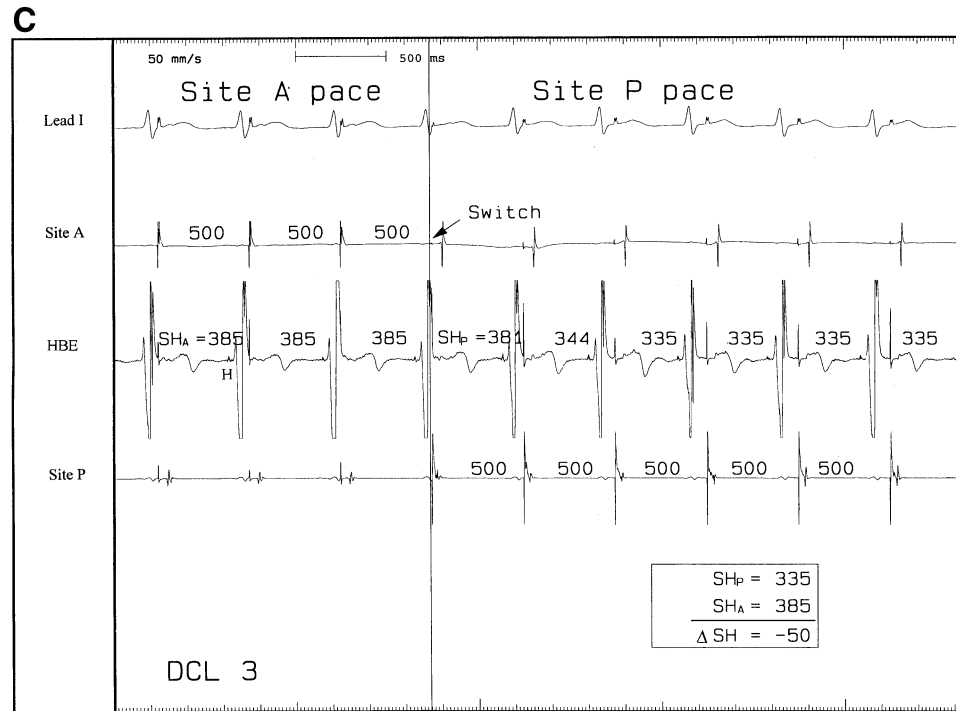
**Figure 2.** Intracardiac recordings of atrial pacing study from Patient 10 at three DCLs are shown. Pacing was performed at drive cycle lengths of 660 ms (DCL1) (A), 530 ms (DCL2) (B) and 500 ms (DCL3) (C). Surface lead I, with intracardiac channels from the His and sites A and P, is displayed. In each panel, the **left side of the tracing shows pacing from site A** at a given drive cycle length (DCL1, DCL2 or DCL3). At the label “Switch,” the pacing site was changed instantaneously to site P without changing the DCL. The measured SH times (SH<sub>A</sub> or SH<sub>P</sub>) are displayed on the His recording channel. Note the different but constant SH conduction times recorded from both sites after the switch in the pacing site. The SH times measured at sites P and A are compared at each DCL, and the difference is represented as a ΔSH value for that DCL and is shown in each panel.

at site A (SH<sub>A</sub>). Given this hypothesis, if the relation between SH<sub>P</sub> and SH<sub>A</sub> is then represented by a single variable ΔSH (ΔSH = SH<sub>P</sub> - SH<sub>A</sub>), it is therefore expected that ΔSH will be different during fast compared with slow pathway conduction.

Results of the present study (Table 2, Fig. 3) showed that ΔSH was not significantly different during fast pathway conduction at both DCL1 and DCL2 but that it was significantly different during slow pathway conduction at DCL3. Because

conditions that may affect intranode conduction should be the same for all three DCLs, this change in ΔSH during slow pathway conduction (DCL3) is best explained by an actual change in the atrial entry site to the slow pathway itself.

This unique pacing protocol, whereby an instantaneous switch in pacing site was accomplished without interrupting the continuity of the drive train, was designed to minimize the moment to moment variability in AV node conduction time



**Figure 2 (continued).** In panel C, for the first beat after the switch to site P, the SH interval (381 ms) is persistently long before it stabilizes to a shorter SH interval (335 ms) after the second beat. Also note that it is only at this transitional beat that the  $A_1A_1$  interval is shorter than the  $S_1S_1$  interval at site P ( $S_1S_1 = 500$  ms;  $A_1A_1 = 470$  ms).

resulting from variation in influences of autonomic tone during the course of the electrophysiologic study. Thus, with other factors affecting AV node conduction minimized, this pacing protocol allowed for an easier method of studying the direct effect of differential site pacing on measured conduction times during fast and slow pathway conduction.

As was previously noted in the results section, Figure 2C illustrates an interesting phenomenon of a persistent long SH interval 1 beat after the switch from pacing site A to site P during DCL3. This phenomenon may be inherent in the study

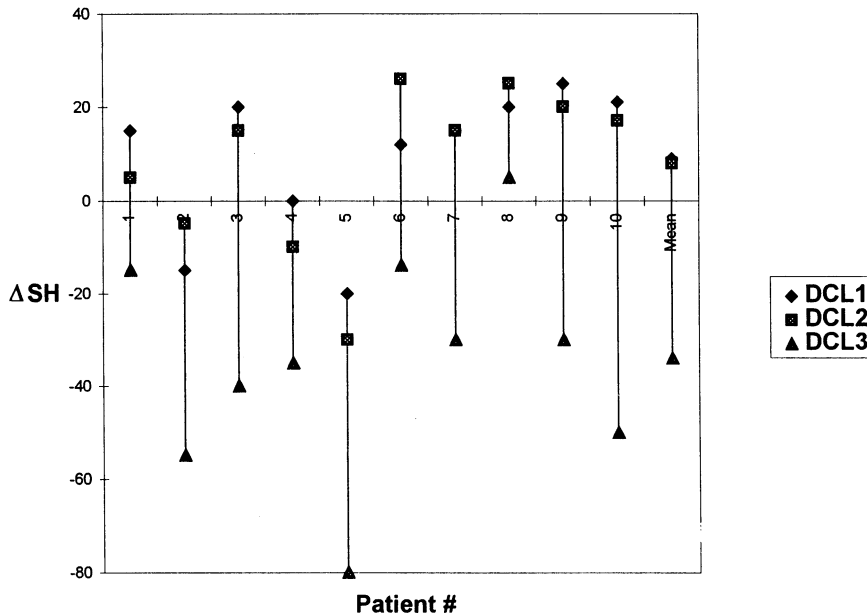
design, which requires a transition where one pacing site is instantly switched to another. At this transition point, stimulation occurs uninterrupted and in sequence from two separate points (site A and then site P, respectively). Because there is a latency for arrival of the first stimulus from site A to site P when pacing is then suddenly switched to site P, a shorter local  $A_1A_1$  interval than the intended  $S_1S_1$  interval results for this transitional beat (Fig. 2C,  $S_1S_1 = 500$ ,  $A_1A_1 = 470$  at site P). Furthermore, given that the putative entry site of the slow pathway may be more posterior, a shorter  $A_1A_1$  interval at this

**Table 2.** Summary Data of Relative Conduction Times

Pt No.	Drive Cycle Length 1				Drive Cycle Length 2				Drive Cycle Length 3			
	DCL (ms)	SH <sub>P</sub> (ms)	SH <sub>A</sub> (ms)	ΔSH (ms)	DCL (ms)	SH <sub>P</sub> (ms)	SH <sub>A</sub> (ms)	ΔSH (ms)	DCL (ms)	SH <sub>P</sub> (ms)	SH <sub>A</sub> (ms)	ΔSH (ms)
1	600	170	155	15	500	190	185	5	430	475	490	-15
2	700	130	145	-15	500	130	135	-5	480	265	320	-55
3	500	125	105	20	400	160	145	15	380	340	380	-40
4	800	125	125	0	750	135	145	-10	500	275	310	-35
5	600	130	150	-20	500	140	170	-30	450	210	290	-80
6	750	182	170	12	650	206	180	26	400	237	251	-14
7	700	140	125	15	600	160	145	15	450	370	400	-30
8	600	110	90	20	400	135	110	25	350	345	340	5
9	600	135	110	25	500	150	130	20	410	300	330	-30
10	600	102	81	21	500	131	114	17	430	335	385	-50
Mean	645	135	126	9*	530	154	146	8*	428	315	350	-34†
±SD	±90	±25	±29	±16*	±109	±26	±26	±18*	±45	±76	±67	±24†

\*p = NS, DCL1 versus DCL2. †p < 0.05, DCL1 versus DCL3 and DCL2 versus DCL3. DCL = drive cycle length used during each of three pacing studies in each patient; SH<sub>P</sub> = stimulus to His time recorded during pacing at site P; SH<sub>A</sub> = stimulus to His time recorded during pacing from site A; ΔSH = difference of SH<sub>P</sub> and SH<sub>A</sub> (SH<sub>P</sub> - SH<sub>A</sub>), at drive cycle length studied.





**Figure 3.** Graphic representation of  $\Delta$ SH determined at DCL1, DCL2 and DCL3 for 10 patients. Note that  $\Delta$ SH values measured at DCL3 are significantly different from those calculated at DCL 1 and DCL 2.

transitional beat will have the likely effect of prolonging the SH interval and canceling any shortening that might be expected from pacing at site P. This phenomenon was not observed at DCL1 and DCL2, possibly because the effect of this different transitional  $A_1A_1$  interval on AV node conduction may not be as noticeable at these two DCLs, which are associated with shorter SH times. Because this phenomenon occurred only at the transitional beat, and may be a direct result of the pacing design, we do not consider that other mechanisms such as variable vagal influences are needed to account for these findings.

During slow pathway conduction (DCL3), the mean  $\Delta$ SH value of  $-34 \pm 24$  ms (range  $+5$  to  $-80$ ) suggests that site P is on average  $34 \pm 24$  ms closer to the slow pathway atrial entry point than site A. During fast pathway conduction at DCL1 and DCL2, the mean  $\Delta$ SH values ( $9 \pm 16$ ,  $8 \pm 18$ , respectively) were positive. However, 3 of 10 patients did show negative values (Table 2), suggesting that the fast pathway input was actually closer to site P than site A in these patients. The location of the putative input of the fast pathway may be variable and even more posterior in some patients (10,11). Therefore, in the three patients in whom  $\Delta$ SH was a negative value, the entry site of the fast pathway may have been located between the pacing sites and actually closer to site P. Despite the negative value found during fast pathway conduction, all three patients showed significantly more negative  $\Delta$ SH values during slow pathway conduction, suggesting that a shift to a more posterior input site had occurred as well. Finally, although a wide range of  $\Delta$ SH values were noted in the results, this variability should not affect the study, which was designed to have each patient serve as his or her own control for comparison of fast and slow pathway conduction.

**Electrophysiologic implications.** Although many reports do support separate fast and slow pathways, few reports offer

more direct evidence regarding the existence of separate atrial inputs to the AV node in humans (3,8,12-14). Keim et al. (15) used intraoperative ice mapping around the AV node region to determine the critical areas of fast and slow pathway conduction during AV node reentrant tachycardia. They found that despite being able to terminate the tachycardia by affecting slow pathway conduction at sites peripheral to the compact AV node posteriorly, they were unable to affect the tachycardia within a large area located in between. On the basis of these findings, they suggested that during typical slow-fast AV node reentrant tachycardia, the septal wave front that has exited the fast pathway to the atrial septum superiorly cannot enter the slow pathway at sites near the compact node because of either anatomic absence of connection or functional refractoriness of these fibers. The wave front enters more distally into the slow pathway located along the tricuspid annulus near the CS os and propagates along the pathway into the compact AV node (15). Satoh et al. (16) demonstrated orthodromic rather than antidromic capture of the atrial myocardium adjacent to the fast pathway exit site during entrainment of typical slow-fast AV node reentrant tachycardia with pacing from the CS os. They argued that this finding would support the existence of a posterior input to the slow pathway during AV node reentrant tachycardia. That is, only an impulse that originates near the entry site of the slow pathway would have access to the widest excitable gap that would allow entrainment of the tachycardia in such a unique manner (16). Our present study, although very different in design, provides further electrophysiologic evidence for the existence of separate atrial inputs to the AV node that are used during fast and slow AV node conduction. Furthermore, these inputs may be anatomically fixed in location and are accessible as separate fast and slow entry sites used for anterograde AV node conduction during atrial pacing.

**Study limitations.** The present study was designed to evaluate the patterns of AV node conduction by means of a simple geometric argument. This argument assumes that conduction within the AV node region is constant and that the measured differences in the conduction times only reflect differences in location of input sites during fast and slow pathway conduction. Differences in conduction velocity within this region that may result from the effect of fiber orientation relative to the wave front of impulse conduction were not considered in the design of the study. That is, slow pathway fibers may be oriented such that the wave front of impulse conduction is more perpendicular to fiber orientation when pacing is performed at site A than at site P. This configuration would then result in selectively longer measured slow pathway conduction times at site A, and in this way,  $SH_A$  during DCL3 would be consistently longer than  $SH_P$ , as seen in the present study. The present study does not exclude this alternative explanation for the results. However, because separate retrograde fast and slow pathway exit sites are known to exist, it would seem more likely that separate input sites to fast and slow pathways may also exist.

Patients in our study were selected to meet the requirements of the study protocol, which may have resulted in selection for a limited cohort of patients with AV node reentrant tachycardia. Some variability in AV node anatomy of fast and slow pathway exit points among a small number of patients has been described (17,18). Because our study group was small, our findings may not apply to all patients with AV node reentrant tachycardia, particularly those with smooth AV node conduction curves.

Without using autonomic blockade, this study was designed to minimize the variation in AV node conduction that may occur during the study. Although we expect no significant change in the data, it is not known how inclusion of autonomic blockade would additionally affect the data.

The current study was not designed to evaluate the AV node reentry circuit involved in the genesis of the tachycardia. Therefore, the findings obtained with differential sites of atrial pacing may not truly reflect the functional circuit during AV node reentrant tachycardia and cannot be used to address the true nature or location of the upper turnaround portion of the AV node reentrant circuit. Finally, the study cannot answer whether anterograde and retrograde exit sites are the same or different because of the inherent limitations of the study protocol.

**Conclusions.** We used a unique pacing protocol specifically to overcome the problem of variable conduction in the AV node due to moment to moment changes in autonomic tone and were able to stabilize AV node conduction times for differential site pacing at the AV junction. On the basis of the results of the study, we believe that we provide evidence for the existence of distinctly different anatomic atrial entry sites for the fast and slow conduction pathways. During atrial pacing,

entry to the slow conduction pathway occurs at a posterior and inferior site separate from the anterior and superior entry site of the fast conduction pathway in patients with dual AV node pathway physiology.

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