The Effects of Propranolol on Induction of A-V Nodal Reentrant Paroxysmal Tachycardia

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SUMMARY

Twelve patients with paroxysmal supraventricular tachycardia (PSVT) were studied before and after administration of 0.1 mg/kg i.v. propranolol. Echo zones for inducing atrioventricular (A-V) nodal reentry were determined using His bundle recording and the atrial extrastimulus technique. After propranolol the echo zone was abolished in two patients, decreased in one, unchanged in five, increased in two. In two patients echo zones appeared only after propranolol. Nine patients had episodes of sustained PSVT prior to propranolol. Following propranolol PSVT persisted in only five. In these five patients propranolol slowed the rate of PSVT.

The data were analyzed by plotting $A_1$-$A_2$ and $H_1$-$H_2$ interval curves. On the basis of these curves the patients were separated into those with "dual pathways" and those with "reflection." The effects of propranolol on both conduction patterns are discussed.

In summary, the actions of propranolol in PSVT patients were variable. Potentially beneficial effects included slowing of induced PSVT, loss of the ability to sustain PSVT, and decrease or total elimination of echo zones. Potentially deleterious effects included potentiation of the echo phenomenon with either increase or development of echo zones.

Additional Indexing Words:

Echo zones                   Critical A-H
Reflection                   Atrial extrastimulus
His bundle electrogram      Effective refractory period
Dual atrioventricular nodal pathways
                           Functional refractory period

MOST CASES of paroxysmal supraventricular tachycardia (PSVT) reflect atrioventricular (A-V) nodal reentry.1,2 In patients with A-V nodal reentrant PSVT, this arrhythmia may usually be reproduced in the catheterization laboratory with coupled atrial stimulation. Atrial echoes and/or PSVT occurs when a critical A-H interval is achieved with premature atrial stimulation or during pacing-induced Wenckebach periods.3

Propranolol has been used in the management of PSVT for both conversion of the arrhythmia and as prophylaxis to prevent recurrence.4,5 Since this agent depresses A-V nodal conduction,7 theoretically, it could either potentiate or prevent the occurrence of A-V nodal reentry. To further understand the mechanism of action of this agent in PSVT, we performed electrophysiological studies in 12 patients with A-V nodal reentrant PSVT before and after the administration of propranolol.

Methods

The study group consisted of 12 patients with documented recurrent PSVT. All patients had normal P-R intervals and narrow QRS complexes. The clinical features of the group are summarized in table 1. Only one patient was taking medication at the time of the study; case 12 was receiving alpha methyl DOPA and hydrochlorothiazide.

Electrophysiological Studies

Electrophysiological studies were performed in the postabsorptive, nonsedated state. Informed consent was obtained. His bundle recordings were obtained using a tripolar electrode catheter introduced percutaneously into the right femoral vein and placed across the tricuspid valve.8 A quadripolar electrode catheter was introduced into an antecubital vein and fluoroscopically positioned against the lateral wall of the high right atrium. The distal two electrodes were used to pace the atrium and the proximal two electrodes were used to record high right atrial electrograms. Electrocardiographic leads I, II, III, and V1, as well as high right atrial and His bundle electrograms were simultaneously recorded on a multichannel oscillographic photographic recorder (Electronics for Medicine DR-16, American Micronics, Niles, Ill.).
White Plains, New York) at paper speeds of 100 and 200 mm/sec. Recordings were also recorded on an 8 channel tape system (Honeywell 5600) to facilitate subsequent analysis. The stimulus was a rectangular wave of 2 msec in duration, approximately twice diastolic threshold, and was supplied by a programmable digital pulse generator (manufactured by M. Bloom, Philadelphia, Pa.). Stimuli were delivered through a stimulus isolation unit (Bioelectronics 212092).

The atria were paced at rates slightly faster than spontaneous sinus rhythm. Pacing rates were increased in 10 beats/min increments until A-V nodal Wenckebach periods were observed. Refractory periods of atrial extrastimulus technique. The test stimulus (S2) was introduced after every eighth driven (S1) or spontaneous sinus beat. The coupling interval was decreased in 5–10 msec decrements. Test stimuli were repeated at least three times at critical coupling intervals to identify echo zones accurately.

After the control measurements were performed a 0.1 mg/kg dose of propranolol was given intravenously over a 5 min period. Electrophysiological studies were repeated beginning 10 min later.

Electrophysiological Definitions

HRA2, A2, H2, V1 were respectively the high right atrial, the low right atrial, the His bundle, and ventricular electrograms of sinus or driven beats. S1 was the stimulus artifact of driven beats. HRA2, A2, H2, and V1 were the electrograms in response to the extrastimulus (S2). A-V nodal reentry was defined as previously described. A-V nodal reentry was noted both with the extrastimulus technique and during pacing-induced Wenckebach periods. Atrial echoes were characterized by retrograde P wave morphology; if P waves were visible, and by a reversal of the normal high-to-low sequence of atrial electrograms.

The echo zone was defined as the zone in which atrial extrastimuli induced atrial echoes with or without PSVT. The outer limit of this zone was the longest A2-A2 followed by an atrial echo. The inner limit of this zone was the shortest A2-A2 followed by an atrial echo. Thus, echo zones could be characterized by absolute total duration as well as by outer and inner limits. Critical A-H interval was defined as shortest A2-H2 inducing an atrial echo (figs. 1B, 1E, and 2D).

In all patients curves were constructed by plotting H2-H2 responses against A2-A2 coupling intervals. Patients were divided into two categories, those with reflection and those with dual pathways on the basis of these curves. Reflection cases were characterized by smooth A2-A2, H2-H2 curves, resembling those previously described by Wit et al. In these patients, A-V nodal effective and functional refractory periods (ERP and FRP) were defined as previously described. Dual pathway cases were characterized by A2-A2, H2-H2 curves which differed markedly from those described by Wit et al., and were characterized by a sudden jump in H2-H2 at a critical A2-A2 coupling interval. The portion of the curve to the right of the jump was considered the fast pathway curve, and that to the left, the slow pathway curve. The slope of the slow pathway curve differed from that of the fast pathway curve. In addition, curve-fitting analysis was performed to objectively delineate dual pathway and reflection cases. Those points considered to represent fast pathway conduction could be described by a second degree polynomial equation. A diagnosis of dual pathways was made when a second set of points appeared above the 95% confidence limits (± two standard deviations) of predicted values for the fast pathway curve.

Table 1

Table 1: Clinical Features and Electrocardiographic Findings

<table>
<thead>
<tr>
<th>Case no.</th>
<th>Age (yrs)</th>
<th>Sex</th>
<th>Cardiovascular diagnosis</th>
<th>Medications</th>
<th>ECG</th>
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<td>No OHD</td>
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<td>Normal</td>
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<tr>
<td>2</td>
<td>50</td>
<td>Male</td>
<td>No OHD</td>
<td>None</td>
<td>Nonspecific T wave changes</td>
</tr>
<tr>
<td>3</td>
<td>52</td>
<td>Male</td>
<td>No OHD</td>
<td>None</td>
<td>Normal</td>
</tr>
<tr>
<td>4</td>
<td>58</td>
<td>Female</td>
<td>No OHD</td>
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<td>Nonspecific ST-T changes</td>
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<tr>
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<td>No OHD</td>
<td>None</td>
<td>Nonspecific ST-T changes</td>
</tr>
<tr>
<td>6</td>
<td>50</td>
<td>Female</td>
<td>Hypothyroidism</td>
<td>None</td>
<td>Nonspecific ST-T changes</td>
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<tr>
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<td>No OHD</td>
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<tr>
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<td>58</td>
<td>Female</td>
<td>No OHD</td>
<td>None</td>
<td>Normal</td>
</tr>
<tr>
<td>9</td>
<td>55</td>
<td>Male</td>
<td>No OHD</td>
<td>None</td>
<td>Nonspecific ST-T changes</td>
</tr>
<tr>
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<td>64</td>
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<td>ST-T changes</td>
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<td>11</td>
<td>65</td>
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<td>No OHD</td>
<td>Aldomet 1 g/day</td>
<td>Normal</td>
</tr>
<tr>
<td>12</td>
<td>45</td>
<td>Male</td>
<td>HHD</td>
<td>Hydrochlorothiazide 100 mg/day</td>
<td>Sinus bradycardia</td>
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</table>

Abbreviations: OHD = organic heart disease; ASHD = arteriosclerotic heart disease; HHD = hypertensive heart disease; LVH = left ventricular hypertrophy.
The fast pathway ERP was defined as the longest $A_1-A_2$ that failed to conduct via the fast pathway. The fast pathway FRP was defined as the shortest attainable $H_2-H_1$ interval on the fast pathway curve. The ERP of the slow pathway was the longest $A_1-A_2$ not conducted via the slow pathway. The FRP of the slow pathway was the shortest attainable $H_1-H_2$ on the slow pathway curve. When atrial FRP limited slow pathway conduction, the slow pathway ERP could not be measured but was considered less than the shortest conducted $A_1-A_2$ interval.

Sustained PSVT was defined as an episode of induced PSVT lasting longer than 2 min. These episodes were converted using single or coupled pairs of atrial stimuli.

**Results**

**Echo Zones and Wenckebach-Induced Echoes (tables 2 and 3)**

Echo zones could be demonstrated in ten of the patients prior to propranolol administration. Following propranolol, echo zones were totally abolished in two patients (cases 1 and 2; figure 3); decreased in total duration in one patient (case 8; fig. 6); unchanged in total duration (a change of 10 msec or less) in five patients (cases 3–6 and 9; figs. 1 and 4); and increased in total duration in two patients (cases 7 and 12; fig. 5). In two patients (cases 10 and 11), echo zones were demonstrated only after propranolol administration (figs. 2 and 7).

Nine patients (cases 3–10 and 12) manifested echoes with or without PSVT during pacing-induced Wenckebach periods both prior to and following propranolol administration. Following the drug this was generally at a slightly slower paced rate.

**Ability to Sustain PSVT and Rate of PSVT (tables 2 and 3)**

Nine patients (1, 3–8, 10, and 12) had episodes of sustained PSVT prior to propranolol administration. Following propranolol, PSVT persisted in only five of these patients (cases 5–7, 10, and 12). One patient had no echoes (case 1), two patients had single echoes only (cases 3 and 4; fig. 1), and one patient had nonsustained PSVT (case 8).

In the five patients with sustained PSVT before and after propranolol, the rates of PSVT could be compared. The mean ± SEM rate of PSVT in these five patients was $142 ± 7$ beats/min (range 119 to 160 beats/min) prior to propranolol and $132 ± 6$ beats/min after propranolol administration.
Figure 2

Recordings with reflection from case 11 showing A-V nodal reentry only after propranolol. The driving cycle length (CL) was 600 msec. Panels A to C show control recordings before propranolol. Atrial echoes were not observed. The maximum $A_1-H_1$ achieved was 280 msec. Panels D to F are the recordings after propranolol at similar coupling intervals to those in panels A–C. Note that atrial echoes (E) occur at $A_1-A_2$ between 320 to 305 msec. The critical $A-H$ interval of 430 msec was achieved only after propranolol.

beats/min (range 111 to 150 beats/min) after propranolol. The rate of PSVT was decreased in each of the five patients following propranolol administration.

Dual Pathway Cases (table 2; figs. 1, 3–5)

Six of the patients (cases 1–6) had $A_1-A_2$, $H_1-H_2$ curves suggestive of dual pathways. These were characterized by a sudden jump of $H_1-H_2$ interval at a critical $A_1-A_2$ coupling interval and the demonstration of two $A_1-A_2$, $H_1-H_2$ curves with different slopes (figs. 3 and 4). Scanning with atrial test stimuli at $A_1-A_2$ intervals close to the jump revealed overlaps of the two curves without intermediate $H_1-H_2$ values in three patients (cases 1, 4, and 6), two distinct separate $H_1-H_2$ responses without intermediate values at a similar $A_1-A_2$ interval in one patient (case 5; fig. 4), and shift of curve at a slightly shorter $A_1-A_2$ interval without intermediate $H_1-H_2$ responses in two patients (cases 2 and 3). In six patients, curve-fitting analysis supported the diagnosis of dual pathways. In one additional patient (case 7) the $A_1-A_2$, $H_1-H_2$ curves were
PROPRANOLOL AND A-V NODAL REENTRY

A-V conduction curves in case 2 with dual A-V nodal pathways, before and after propranolol administration. Panel A shows $H_1$-$H_2$ responses plotted against $A_1$-$A_2$ coupling intervals. Panel B shows $A_2$-$H_2$ responses plotted against $A_1$-$A_2$ coupling intervals. Control responses are shown as circles and post propranolol responses as squares. Open circles and squares reflect responses followed by echo beats. Echo zones are designated in the A panel. The basic driving cycle length (CL) was 667 msec. A: Before propranolol, the ERP of the fast pathway was 305 msec, which coincided with the onset of the slow pathway curve. The echo zone was noted on the inner side of the slow pathway curve at $A_1$-$A_2$ between 275 to 265 msec. The atrial FRP of 265 msec limited slow pathway conduction. After propranolol, both the slow and the fast pathway curves shifted rightward and upward indicating increase of the ERP and FRP of both pathways. The echo zone was abolished. Overlaps of the fast and slow pathway curves were also noted. B: Before propranolol, the critical A-H interval was 305 msec. After propranolol, there was no critical A-H interval even at the longest attainable $A_1$-$H_2$ of 400 msec, suggesting increased refractoriness of the fast pathway for retrograde conduction. $A_1$-$H_2$ lengthened at any given $A_1$-$A_2$ intervals.

An increase in fast pathway ERP was noted in five of the seven patients after propranolol, mean fast pathway ERP increasing from 357 ± 18 msec to 401 ± 21 msec after this drug ($P < 0.05$) as seen in figures 3–5. An increase in fast pathway FRP was noted in five of the seven patients with dual pathways after propranolol, mean FRP increasing from 454 ± 22 msec to 498 ± 22 msec (figs. 3A, 4A, and 5A).

The slow pathway ERP could be measured in only four patients (cases 1, 4, 5, and 6) and increased in three of these following propranolol (cases 1, 5, and 6; fig. 4). In the remaining three dual pathway cases atrial FRP limited A-V conduction. Slow pathway FRP increased in six of the seven patients following propranolol, mean slow pathway FRP increasing from 645 ± 39 msec to 694 ± 38 msec ($P < 0.01$) as seen in figures 3A, 4A, and 5A.

In most patients the total duration of the slow pathway curve increased, reflecting a greater increase in the outer limit of this curve (fast pathway ERP) than in the inner limit (slow pathway ERP or atrial FRP) following propranolol administration (figs. 3 and 5).

Echo zones coincided with either part or all of the slow pathway curve. In two of the patients echo zones were abolished after propranolol administration, suggesting that this drug increased retrograde fast pathway refractoriness (fig. 3). In four of the patients (cases 3–6), the echo zone was unchanged in total duration after propranolol (change of less than 10 msec). In two of these (cases 5 and 6), the echo zone was shifted to the right following propranolol (fig. 4). In one of the dual pathway patients there was a marked increase in echo zone following propranolol (case 7). In this patient, the echo zone coincided with the total slow pathway curve. Following propranolol
Table 2

Electrophysiological Findings (in msec) Before and After Propranolol in Patients With Dual Pathways

<table>
<thead>
<tr>
<th>Case no.</th>
<th>Cycle length</th>
<th>Fast pathway</th>
<th>Slow pathway</th>
<th>Atrial FRP</th>
<th>Echo zones (range)</th>
<th>Critical A-H interval</th>
<th>Echo + PSVT during WP</th>
<th>Ability to sustain PSVT</th>
<th>Rate of PSVT</th>
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<tr>
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<td>Control</td>
<td>920</td>
<td>425</td>
<td>540</td>
<td>340</td>
<td>290</td>
<td>460-345 (115)</td>
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<td>Yes</td>
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<td></td>
<td>Propranolol</td>
<td>960</td>
<td>600</td>
<td></td>
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<td>No</td>
<td>—</td>
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<td>Control</td>
<td>667</td>
<td>305</td>
<td>415</td>
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<td>275-265 (10)</td>
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<td>490</td>
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<td>—</td>
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<tr>
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<td>Control</td>
<td>840</td>
<td>420</td>
<td>520</td>
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<td>530</td>
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<td>320</td>
<td>PSVT</td>
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Abbreviations: ERP = effective refractory period; FRP = functional refractory period; WP = Wenckebach period; PSVT = paroxysmal supraventricular tachycardia.
Table 3
Electrophysiological Findings (in msec) Before and After Propranolol in Patients With Reflection

<table>
<thead>
<tr>
<th>Case no.</th>
<th>Cycle length</th>
<th>A-V nodal ERP</th>
<th>A-V nodal FRP</th>
<th>Atrial FRP</th>
<th>Echo zone (range)</th>
<th>Critical A-H interval (msec)</th>
<th>Echo ± PSVT during WP</th>
<th>Ability to sustain</th>
<th>Rate of PSVT</th>
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<td>Echo</td>
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<td>590</td>
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<tr>
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<td>&lt;320</td>
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<td>PSVT</td>
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<td>570</td>
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Abbreviations: ERP = effective refractory period; FRP = functional refractory period; WP = Wenekebach period; PSVT = paroxysmal supraventricular tachycardia.
the inner limit decreased slightly, due to a slight decrease in atrial FRP, while the outer limit (fast pathway ERP) increased (fig. 5).

Critical A-H interval in dual pathway patients reflected the shortest slow pathway conduction time allowing recovery of the fast pathway for retrograde conduction. Critical A-H interval can best be recognized by noting the graphs depicting $A_2-H_2$ responses to $A_1-A_2$ coupling intervals (figs. 3B, 4B, and 5B). In the five dual pathway patients with echoes before and after propranolol, critical A-H interval increased in three (cases 3, 6, and 7) and was unchanged in two (cases 4 and 5).

**Reflection Cases (table 3; figs. 2, 6-7)**

Five of the patients (cases 8-12) had $A_1-A_2$, $H_1-H_2$ curves suggesting reflection. In all five patients, curve-fitting analysis did not separate two curves. A-V nodal ERP could only be measured in one of these (case 9) and increased from less than 310 to 330 msec after propranolol. A-V nodal FRP could be measured in all five patients before and after propranolol and increased in four of the five patients, mean A-V nodal FRP being 498 ± 30 msec prior to and 532 ± 31 msec after propranolol (figs. 6A and 7A).

In two of the patients (cases 10 and 11), echo zones could only be demonstrated after propranolol administration. In one of these patients (case 10), echoes were demonstrated during induced Wenckebach periods with an A-H interval of 340 msec prior to propranolol. The maximum $A_2-H_2$ attainable with extrastimulus technique prior to propranolol was only 285 msec. Following propranolol significantly longer $A_2-H_2$ responses could be obtained at all coupling intervals, allowing the critical A-H interval to be achieved at close coupling intervals. In case 11, the maximum $A_2-H_2$ obtained prior to propranolol was 280 msec (figs. 2 and 7). After propranolol $A_2-H_2$ responses were longer, allowing achievement of a critical A-H of 430 msec (figs. 2 and 7). Results of studies in these two patients suggested that propranolol increased the ability of extrastimuli to

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*Figure 4*

Atrioventricular conduction curves before and after propranolol in case 5 with dual A-V nodal pathways. Conventions in this and subsequent illustrations are similar to those in figure 3. The driving cycle length (CL) was 765 msec. A: Propranolol increased the ERP (from 350 to 370 msec) and the FRP (from 415 to 450 msec) of the fast pathway. It shifted the slow pathway curve slightly rightward and upward indicating slight increase in the ERP (from 290 to 310 msec) and the FRP (from 625 to 670 msec) of the slow pathway. Echo zone (EZ) coincided with the total slow pathway curve and was shifted slightly to the right although the total duration was unchanged. B: Propranolol increased the fast pathway conduction time only at shorter coupling intervals. It lengthened the slow pathway conduction time at all coupling intervals. Critical A-H interval of 360 msec was unchanged.
achieve critical A-H interval. In one of the reflection patients (case 12), echo zones increased after propranolol with a shift of the echo zone to the right. In one of the reflection cases (case 9), the echo zones were not significantly changed in duration although this zone was shifted slightly to the right. In only one reflection case (case 8) was the echo zone significantly decreased (fig. 6). In four of the reflection cases, H₁-H₂ responses for a given A₁-A₂ coupling interval were increased following propranolol.

Critical A-H interval could be measured before and after propranolol in three of the five reflection cases and was increased in all three patients (cases 8, 9, and 12; figs. 6B and 7B).

Discussion

Most cases of PSVT appear to reflect A-V nodal reentrance. Criteria for the demonstration of A-V nodal reentrance in the catheterization laboratory include: 1) induction of atrial echoes with or without PSVT following timed premature atrial extrastimuli, allowing delineation of an echo zone,² 3) demonstration that the atrial echoes have a low to high sequence of atrial depolarization and retrograde P wave morphology (if the P wave is visible),² 3) termination of induced PSVT with critically timed atrial extrastimuli,¹ ² and 5) induction of the echo phenomenon with or without PSVT during pacing-induced A-V nodal Wenckebach periods.² ³ All of the patients in the present series had previously documented PSVT and fulfilled criteria 1–3 at the time of electrophysiological study, although in two of the patients these criteria were fulfilled only following propranolol administration. Most of the patients also fulfilled criteria 4 and 5.

Plotting of A₁-A₂, H₁-H₂ curves have allowed us to separate the 12 patients into those with dual pathways and those with reflection. This separation helped us to characterize the pharmacological effects of propranolol on the mechanisms of reentry. Seven patients had dual pathway conduction patterns and five had reflection conduction patterns. Analysis of the clinical and electrophysiological data in the two groups revealed...
no significant difference in age, sex, presence or absence of organic heart disease, electrocardiographic findings, or the rate of PSVT.

Dual A-V Nodal Pathways and Propranolol

The concept of functional or anatomic division of the A-V node into two pathways with different refrac-

Figure 6
Atrioventricular conduction curves before and after propranolol in case 8 with reflection. The driving cycle length (CL) was 680 msec. A: After propranolol, H₁-H₂ curve was unchanged. However, the total duration of the echo zone (EZ) shortened from 45 to 10 msec. B: Critical A-H interval increased slightly from 285 to 310 msec after propranolol. A₂-H₂ was not significantly increased at all coupling intervals.

Figure 7
Atrioventricular conduction curves before and after propranolol in case 11 with reflection. The driving cycle length (CL) was 600 msec. A: H₁-H₂, A₁-A₂ curve shifted upward and atrial echoes were demonstrated only after propranolol. B: A₂-H₂ lengthened at all coupling intervals and the critical A-H interval was achieved only after propranolol.
Propranolol and A-V Nodal Reentry

Propranolol and A-V nodal reentry has been postulated to explain A-V nodal reentry. An impulse may encounter the effective refractory period of one pathway, and conduct slowly but successfully in the other pathway, and then return in retrograde fashion utilizing the blocked pathway to reexcite the atrium. Both animal and human studies, using indirect or direct recording methods, presented evidence suggesting the presence of dual A-V pathways. Recently, Denes et al., utilizing His bundle recording and the atrial extrastimulus technique, provided strong evidence for dual A-V nodal pathways in two patients with A-V nodal reentrant PSVT. The two patients manifested sudden jumps in H1-H2 intervals at critical A1-A2 coupling intervals with occurrence of echo beats. The outer limits of the slow pathway curve and the echo zone coincided with the ERP of the fast pathway. The inner limit of the echo zone and slow pathway curve determined by either the ERP of the slow pathway or the FRP of the atrium.

The present study extends these observations concerning dual pathways and A-V nodal reentrant PSVT. First, A1-A2, H1-H2 curves suggestive of dual pathways were frequent in patients with A-V nodal reentrant PSVT. Second, it was demonstrated that the overlap of fast and slow pathways could occur. This overlap of slow and fast pathway curves without intermediate H1-H2 responses (which occurred in five patients either before and/or after propranolol) provided strong evidence that both pathways. If the slow pathway curve reflected the relative refractory period of a single A-V nodal pathway, intermediate H1-H2 responses would have been expected. Third, it was demonstrated that the slow pathway curve slope could be either a positive, negative, or flat slope. In the case of the negative slope slow pathway curve, one could question whether or not the slow pathway curve could reflect the relative refractory period of a single A-V nodal pathway. However, these A1-A2, H1-H2 curves were unlike the normal curves previously reported by Wit et al. and unlike those seen in our own experience in patients with normal conduction. Assuming that our PSVT patients had dual A-V nodal pathways, most of the slow pathway curve was concealed by the fast pathway curve. The negative slope of the slow pathway curve being exposed after the fast pathway ERP was achieved could reflect relative slow pathway refractoriness.

Propranolol shifted both fast and slow pathway curves upward and to the right with an increase in the ERP and FRP of both the fast and slow pathways. This presumably reflects the effects of beta-adrenergic receptor blockade on two pathways, both located within the A-V node. The slow pathway curve became apparent in some patients after propranolol administration since there was more rightward shift of the fast pathway ERP when compared to the slow pathway. The changes in absolute duration of echo zones were partially determined by whether the outer or inner limits of the slow pathway curve was shifted more to the right.

Critical A-H interval for induction of A-V nodal reentry in the dual pathway case is the shortest slow pathway conduction time allowing recovery of the fast pathway for retrograde conduction. Propranolol probably increased retrograde fast pathway refractoriness and also slowed slow pathway antegrade conduction time, resulting in an increase in critical A-H interval. The total elimination of echo zones in two of the dual pathway patients following administration of propranolol suggested an increase in retrograde fast pathway refractoriness due to the drug. Since ventricular coupled pacing was not utilized in this study, this hypothesis cannot be proven.

Paroxysmal supraventricular tachycardia occurs when dual A-V nodal pathways sustain a circus movement. The rate of the PSVT would therefore reflect conduction times in the two pathways. Since fast and slow antegrade A-H intervals increased after propranolol, the slowing in PSVT following this drug would appear to reflect slowing of conduction in both pathways. The loss of ability to sustain PSVT in some of the dual pathway patients following administration of propranolol could reflect a critical increase in either retrograde fast pathway refractoriness or antegrade slow pathway refractoriness. An increase in refractoriness in one pathway could be offset by an increase in conduction time in the opposing pathway. For example, an increase in retrograde fast pathway refractoriness could be offset by slowing of antegrade conduction in the slow pathway. Since propranolol appears to affect conduction times and refractory periods of both pathways to varying degrees, the drug can result in continued ability or loss of ability to sustain PSVT. The loss of ability to sustain PSVT could also reflect changes in refractoriness that occur after several cycles of PSVT have occurred. For example, type one block, or type two block, in either pathway would result in loss of ability to sustain PSVT. In the present study, no specific electrophysiological action of propranolol could be identified as being directly related to loss of ability to sustain PSVT.

Reflection and Propranolol

Cranefield et al., working with depressed strands of Purkinje tissue, introduced the term "reflection" to describe a phenomenon in which an impulse entered a region of depressed conduction and, after considerable delay, was reflected in a retrograde direc-

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tion. Whether the reflected impulse occurred in a single conducting pathway or a small area of longitudinal dissociation had to be assumed was not clarified.\textsuperscript{22, 23} If reflection occurred in the A-V node, one would expect a smooth $A_1-A_2$, $H_1-H_2$ curve, with an echo zone coinciding with $A_1-A_2$, coupling intervals that achieve a critical A-H interval. Moe et al.\textsuperscript{14} suggested that the refractory period of a slow pathway could be longer than that of a fast pathway and that A-V nodal reentry could occur by using the slow pathway for retrograde conduction. If this occurred, it would also result in a smooth $A_1-A_2$, $H_1-H_2$ curve. In addition, a smooth $A_1-A_2$, $H_1-H_2$ curve would also result from two pathways with different refractory periods and similar conduction times. In such a case, A-V nodal reentry would occur when conduction delay in one pathway was sufficient for retrograde recovery of a blocked pathway. In the present study, we have utilized the term "reflection" only in a descriptive sense in order to describe patients with A-V nodal reentrant PSVT and smooth conduction curves. We did not use the term reflection to imply a specific underlying mechanism of A-V nodal reentry.

The reflection cases resembled the dual pathway cases in response to propranolol in several respects: 1) sustained PSVT was slower following propranolol, 2) critical A-H interval was increased following the drug, and 3) ability to sustain PSVT was lost in one of these patients with sustained PSVT prior to the drug. However, a striking finding in two of the reflection cases was the inability to demonstrate echo zones prior to propranolol and the ability to demonstrate these after the drug was administered. This would appear to reflect an increased ability to achieve the degree of A-V nodal delay necessary for reflection to occur. Although the total number of dual pathway and reflection cases was small, it appeared that the reflection cases were less likely to benefit from propranolol and more likely to manifest A-V nodal reentry following this drug. This impression needs further substantiation in a larger series of cases. In addition, our data of the reflection cases does not suggest specific mechanisms to explain the pharmacological effects of propranolol on the induction of reentry.

Clinical Implications

This study does not answer the question as to whether propranolol is or is not a useful drug in the management of PSVT. The actions of propranolol varied, with both beneficial and deleterious actions being observed. Potentially beneficial effects included slowing of induced PSVT, loss of the ability to sustain PSVT, and decrease or total elimination of echo zones. Slowing the rate of PSVT probably has little clinical significance since the slowing is only minimal. Loss of ability to sustain PSVT, if this occurred during chronic oral propranolol therapy, would eliminate attacks of PSVT. Decrease in echo zones could be beneficial, if the laboratory induction of PSVT reflects the clinical occurrence of PSVT, since random premature beats would be less likely to induce PSVT.

Potentially deleterious effects of propranolol included potentiation of the echo phenomenon, with increase in echo zones or development of echo zones when these were not demonstrable prior to propranolol administration. There was no apparent means of predicting which patients would manifest beneficial effects and which patients would manifest deleterious effects.

Our study does suggest that the use of propranolol in order to prevent PSVT needs further study.

References

PROPRANOLOL AND A-V NODAL REENTRY

The Effects of Propranolol on Induction of A-V Nodal Reentrant Paroxysmal Tachycardia

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