Effects of Oral Disopyramide Phosphate on Induction of Paroxysmal Supraventricular Tachycardia

STEVEN SWIRYN, M.D., ROBERT A. BAUERNFEIND, M.D., CHRISTOPHER R. C. WYNDHAM, M.D., RAMESH C. DHINGRA, M.D., EDWIN PALILEO, M.D., BORIS STRASBERG, M.D., AND KENNETH M. ROSEN, M.D.

SUMMARY The effects of oral disopyramide phosphate on laboratory induction of paroxysmal supraventricular tachycardia (PSVT) were studied in 16 patients with clinical PSVT. After control electrophyslogic study to determine the inducibility and mechanism of PSVT, patients were given 200–300 mg (275 ± 45 mg, mean ± SD) of disopyramide for three to five doses over 24 hours and were then restudied. All patients had inducible, sustained PSVT during the control study. After disopyramide, PSVT was noninducible in eight patients (50%), including six of nine with atrioventricular nodal reentrance and two of seven with atrioventricular reentrance; inducible but nonsustained in two (12.5%) (both with atrioventricular reentrance); and inducible and sustained in six (37.5%). The benefit of disopyramide seemed predominantly to reflect depression of conduction in the retrograde limb of the circus movements, although effects upon the antegrade limb were also observed. In the eight patients with inducible PSVT before and after disopyramide, tachycardia cycle length increased from 348 ± 33 to 404 ± 29 msec (mean ± SEM) (p < 0.001).

These results suggest that disopyramide would be effective in preventing recurrence of clinical PSVT in selected patients.

INDUCTION AND TERMINATION of paroxysmal supraventricular tachycardia (PSVT) in the catheterization laboratory have been used to study the effects of antiarrhythmic drugs on induction of this rhythm disturbance.1–3 Demonstration of prevention by a drug of induction of laboratory PSVT has been used to imply a likelihood of prevention by that drug of clinical PSVT.1–5 Other studies have reported the effects of ouabain,6–8 propranolol,7, 8 verapamil,9, 10 procainamide,11–14 and quinidine8, 11, 18 on laboratory induction of PSVT.

Disopyramide phosphate is a quinidine-like agent recently approved for management of ventricular dysrhythmia.15–18 The effectiveness of this drug for management of supraventricular dysrhythmia is less certain. Preliminary studies have shown the ability of i.v. disopyramide to terminate acute attacks of PSVT,17, 18–21 and of oral disopyramide to decrease the frequency of recurrence of PSVT.16, 22, 23 Spurrell et al.18 and Bennett19 demonstrated the effects of disopyramide in a small number of patients with PSVT complicating the Wolff-Parkinson-White (WPW) syndrome.

In the present study, we examined the effects of oral disopyramide on laboratory induction of PSVT in 16 patients with clinical PSVT, including a group with atrioventricular (AV) nodal reentrance and a group with AV reentrance. Included are observations regarding the site of action of the drug when tachycardia induction was prevented, and the effect upon tachycardia cycle length when it was not.

Method

Patient Selection

Criteria for inclusion in this study were (1) a history of recurrent, electrocardiographically documented PSVT; (2) signed, informed consent for electrophysiologic study, including drug testing; (3) control electrophysiologic study, including determination of PSVT mechanism; (4) inducible, sustained PSVT during two or more control studies on separate days; and (5) repeat electrophysiologic study while taking disopyramide phosphate.

Electrophysiologic Studies

Each electrophysiologic study was performed in the postabsorptive, nonsedated state. For initial study, a quadripolar catheter was introduced percutaneously through a femoral vein and positioned across the tricuspid valve for His bundle recording. A second quadripolar catheter was introduced similarly and positioned in the high right atrium for atrial stimulation and recording. A third quadripolar catheter was introduced through a left antecubital vein and positioned in the coronary sinus. The distal poles of the His bundle catheter were used for ventricular pacing. Catheter interelectrode distances were 1 cm. Electrocardiographic leads I, II, III and V₁ and
multiple intracardiac electrograms were recorded simultaneously using a multichannel oscilloscopic photographic recorder (Electronics for Medicine, DR-16) at paper speeds of 100 and 200 mm/sec. Stimuli were provided by a programmable digital pulse generator (M. Bloom) and were 2 msec long, with a strength of approximately twice diastolic threshold.

Electrophysiologic properties were evaluated with incremental atrial pacing, atrial extrastimulus testing during sinus and one or more atrial driven cycle lengths, incremental ventricular pacing, ventricular extrastimulus testing during a ventricular driven cycle length shorter than sinus, mapping of retrograde atrial activation sequence during induced PSVT, and ventricular extrastimulus testing during induced PSVT. Using these data, the PSVT mechanism was defined as previously described.2,3 In patients 2, 7 and 13, i.v. atropine was required to induce sustained PSVT. In these instances, atropine was also given for the second control study and for the disopyramide study. All data reported for these three patients, both control and disopyramide, reflect concomitant administration of atropine.

Chronic Study — Disopyramide Phosphate

Immediately after the initial electrophysiologic study, the femoral vein catheters were removed and the antecubital vein catheter replaced with a hexapolar catheter positioned with the two distal poles in the right ventricular apex and the four proximal poles in the right atrium. This catheter was used for subsequent study of disopyramide. Most patients had electrophysiologic study with a number of antiarrhythmic drugs between the initial study and the disopyramide study. In all cases, disopyramide was tested after five or more half-lives of the previously administered drugs had passed. Oral disopyramide was given in three to five doses of 200–300 mg (275 ± 45 mg, mean ± sp) during the 24 hours before study.

Electrophysiologic Definitions

In all cases, an attempt was made to assess the effect of disopyramide on the determinants of reentry.24,25 In patients with AV nodal reentrant PSVT, the antegrade limb (slow pathway) was studied by noting the atrial paced rate that produced AV block. The retrograde limb (fast pathway) was studied by noting the ventricular paced rate that produced ventriculoatrial (VA) block. In patients with orthodromic AV reentry the antegrade limb (AV node and His-Purkinje system) was studied by noting the AV block rate. The retrograde limb (accessory pathway) was studied by noting the VA block rate. In one patient with antidromic AV reentry the antegrade limb (accessory pathway) and retrograde limb (AV node and His-Purkinje system) were studied using the corresponding block rates. Antegrade (AV) and retrograde (VA) block rates were defined as the lowest paced rates (atrial and ventricular, respectively) at which impulses failed to conduct to the distal chamber. Occasionally during the control study, rapid pacing was carried out to high rates without achieving block. In such cases, the highest paced rate tested was considered the “block rate” for purposes of analysis. This only occurred during control and not during disopyramide studies, so decreases in block rates attributed to disopyramide in these cases are minimum values.

The time from the earliest high-frequency deflection in the high right atrial electrogram to the earliest deflection of the QRS complex in any surface lead (HRA-V) was taken as an approximation of antegrade conduction time during PSVT. Similarly, the time from the earliest deflection of the QRS to the earliest high-frequency deflection of the high right atrial electrogram (V-HRA) was taken as an approximation of retrograde conduction time during PSVT. Clearly these sites are not part of the reentrant circuits and do not provide direct or exact measures of these conduction times. Further, we could not insure that catheter movement between initial and disopyramide studies would not cause the HRA position to be recorded from a slightly different right atrial site. More precise determination of these conduction times would require recording of specific, local atrial and ventricular electrograms, which are only available on the first day of a chronic electrophysiologic study.

Results

Patient Population

Sixteen patients underwent electrophysiologic testing during control conditions and after oral disopyramide (table I). They were 17–65 years old (mean 40 ± 14 years). Eight were men and eight were women. In nine patients, the mechanism of PSVT was AV nodal reentry with dual AV nodal pathways. Of these nine, one had mitral valve prolapse, three had hypertension (one accompanied by left bundle branch block) and five had no evidence of organic heart disease.

In seven patients, the mechanism of PSVT was AV reentry using an extranodal pathway. The extranodal pathway was manifest in four (WPW syndrome) and concealed in three. Two patients had mitral valve prolapse and five had no evidence of organic heart disease. Six patients with AV reentry had orthodromic PSVT; that is, PSVT using the AV node for antegrade conduction and the extranodal pathway for retrograde conduction. The remaining patient had antidromic PSVT, using the extranodal pathway for antegrade conduction and the AV node for retrograde conduction.

Effects on Induction of PSVT

By study design, all patients had inducible, sustained PSVT on two control days. After disopyramide, PSVT was inducible and sustained in six patients (37.5%), inducible but nonsustained in two (12.5%), and noninducible in eight (50%) (table I). In all eight patients with inducible PSVT after disopyramide, the cycle length of tachycardia increased from 348 ± 33 msec to 404 ± 29 msec (p < 0.001)
TABLE 1. Effects of Disopyramide on Induction of Paroxysmal Supraventricular Tachycardia

<table>
<thead>
<tr>
<th>Pt</th>
<th>Sex</th>
<th>Age (years)</th>
<th>Heart disease</th>
<th>PSVT mechanism</th>
<th>PSVT induced</th>
<th>PSVT sustained</th>
<th>Antegrade block rate</th>
<th>Retrograde block rate</th>
<th>HRA-V</th>
<th>V-HRA</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>M</td>
<td>57</td>
<td>MVP</td>
<td>AVNR</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>&gt; 240</td>
<td>160</td>
<td>230</td>
</tr>
<tr>
<td>2</td>
<td>M</td>
<td>34</td>
<td>-</td>
<td>AVNR</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>290</td>
<td>200</td>
<td>210</td>
</tr>
<tr>
<td>3</td>
<td>M</td>
<td>41</td>
<td>HCVD</td>
<td>AVNR</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>390</td>
<td>210</td>
<td>220</td>
</tr>
<tr>
<td>4</td>
<td>F</td>
<td>47</td>
<td>-</td>
<td>AVNR</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>342</td>
<td>190</td>
<td>160</td>
</tr>
<tr>
<td>5</td>
<td>F</td>
<td>35</td>
<td>-</td>
<td>AVNR</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>320</td>
<td>230</td>
<td>150</td>
</tr>
<tr>
<td>6</td>
<td>F</td>
<td>27</td>
<td>-</td>
<td>AVNR</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>320</td>
<td>200</td>
<td>190</td>
</tr>
<tr>
<td>7</td>
<td>M</td>
<td>49</td>
<td>HCVD</td>
<td>AVNR</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>340</td>
<td>200</td>
<td>190</td>
</tr>
<tr>
<td>8</td>
<td>M</td>
<td>65</td>
<td>HCVD</td>
<td>AVNR</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>570</td>
<td>160</td>
<td>210</td>
</tr>
<tr>
<td>9</td>
<td>F</td>
<td>45</td>
<td>-</td>
<td>AVNR</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>375</td>
<td>190</td>
<td>160</td>
</tr>
<tr>
<td>10</td>
<td>M</td>
<td>20</td>
<td>MVP</td>
<td>AVR(C)</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>280</td>
<td>200</td>
<td>200</td>
</tr>
<tr>
<td>11</td>
<td>F</td>
<td>17</td>
<td>-</td>
<td>AVR(M)*</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>302</td>
<td>240</td>
<td>NP</td>
</tr>
<tr>
<td>12</td>
<td>F</td>
<td>59</td>
<td>-</td>
<td>AVR(M)</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>355</td>
<td>250</td>
<td>140</td>
</tr>
<tr>
<td>13</td>
<td>M</td>
<td>25</td>
<td>AVR(C)</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>315</td>
<td>200</td>
<td>170</td>
</tr>
<tr>
<td>14</td>
<td>F</td>
<td>35</td>
<td>AVR(M)</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>265</td>
<td>300</td>
<td>180</td>
</tr>
<tr>
<td>15</td>
<td>M</td>
<td>52</td>
<td>AVR(M)</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>340</td>
<td>&gt; 220</td>
<td>140</td>
</tr>
<tr>
<td>16</td>
<td>F</td>
<td>33</td>
<td>MVP</td>
<td>AVR(C)</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>345</td>
<td>180</td>
<td>140</td>
</tr>
</tbody>
</table>

*Antidromic PSVT.
Abbreviations: MVP = mitral valve prolapse; HCVD = hypertensive cardiovascular disease; PSVT = paroxysmal supraventricular tachycardia; AVNR = atrioventricular nodal reentry; AVR = atrioventricular reentry; (C) = concealed accessory pathway; (M) = manifest accessory pathway; Con = control; Diso = disopyramide; NP = not preexcited; HRA-V = high right atrium to ventricle interval; V-HRA = ventricle to high right atrium interval.

(fig. 1). This corresponded to a decrease in heart rate during tachycardia from a mean of 172 to 149 beats/min.

To determine the possible site of increased conduction time during PSVT after disopyramide, HRA-V and V-HRA intervals before and after the drug were examined (table 1). HRA-V interval increased after disopyramide, from 221 ± 55 to 248 ± 56 msec (p < 0.05). The V-HRA interval also increased, from 127 ± 27 to 156 ± 32 msec (p < 0.02). Thus, in-

![Figure 1](image-url)
increased cycle length of PSVT after disopyramide seemed to reflect increased conduction times in both the antegrade and retrograde limbs of the reentrant circuits involved.

**AV Nodal Reentrance**

Disopyramide prevented induction of sustained tachycardia in six of nine patients with AV nodal reentrant PSVT. Of these six, three had single AV nodal reentrant atrial echoes conducted to the ventricle but not followed by a second atrial echo (fig. 2). One patient had single and double AV nodal reentrant echoes, but the limb in which reentrance terminated was unclear because of a large ventricular electrogram in the HRA lead. Two patients had no echoes, despite attaining long AV intervals, that resulted in induction of PSVT during control conditions. Thus, in patients with AV nodal reentrance, disopyramide seemed to prevent induction of PSVT by its effect on the retrograde limb of the circuit (retrograde fast AV nodal pathway).

However, examination of AV and VA block rates demonstrated significant effects of disopyramide on both limbs of the reentrant circuit (table 1). The control antegrade block rate was 208 ± 9 beats/min. After disopyramide, the antegrade block rate decreased to 166 ± 4 beats/min (p < 0.005). The control retrograde block rate was 208 ± 8 beats/min. After disopyramide, the retrograde block rate decreased to 147 ± 8 beats/min (p < 0.001).

Of three patients with AV nodal reentrance who continued to have inducible PSVT on disopyramide, cycle length of tachycardia increased in all (342 to 430 msec, 320 to 360 msec, and 570 to 585 msec, respectively). Conduction time in the antegrade limb as assessed by HRA-V intervals also increased in all three (from 290 to 350 msec, 280 to 300 msec, and 550 to 565 msec). Conduction time in the retrograde limb as assessed by V-HRA intervals increased in two patients (from 50 to 80 msec and 40 to 60 msec) and remained constant in the third (20 msec). Thus, slowing of PSVT after disopyramide seemed to reflect increases in both antegrade and retrograde limb conduction times in two patients and increased antegrade limb conduction time in the third.

**AV Reentrance**

Disopyramide prevented induction of sustained PSVT in four of seven patients with AV reentrance. In one of the four patients, PSVT was not inducible because of absent VA conduction after disopyramide (fig. 3). In a second patient, PSVT was nonsustained, with spontaneous termination of the tachycardia with a ventricular complex not followed by an atrial electrogram. Thus, in these two patients, disopyramide prevented sustained PSVT by its effect on the retrograde limb of the reentrant circuit. In a third patient, AV reentrant PSVT induced atrial reentrant PSVT. This resulted in termination of AV reentrant PSVT because of block in the antegrade limb. Because atrial reentrant PSVT was itself nonsustained, it resulted in termination of the tachycardia. In the fourth patient, who had antidromic PSVT, no preexcitation was present after disopyramide and a marked reduction in retrograde block rate (from 210 to 130 beats/min) was noted. Therefore, PSVT could not be induced in this patient due to drug effects on both the antegrade and retrograde limbs of the reentrant circuit.

Antegrade block rates in patients with atrioventricular reentrant tachycardia usually reflected antegrade conduction in the bundle of Kent and thus were not directly related to PSVT. Retrograde block rates decreased in all patients from a mean of 247 ± 15 beats/min during control to 159 ± 18 beats/min after disopyramide (p < 0.001).

Of five patients with AV reentrance who continued to have inducible PSVT on disopyramide (three sustained and two nonsustained), the cycle length of tachycardia increased in all (from 280 to 327 msec, 355 to 420 msec, 315 to 355 msec, 265 to 345 msec, 355 to 420 msec, and 570 to 585 msec).
and 340 to 410 msec, respectively). Conduction time in the antegrade limb as assessed by HRA-V intervals increased in three (from 90 to 110 msec, 80 to 120 msec, and 190 to 260 msec). HRA-V remained constant in one patient (115 msec) and decreased in one (175 to 160 msec). Conduction time in the retrograde limb as assessed by V-HRA intervals increased in four patients (from 190 to 210 msec, 180 to 260 msec, 200 to 240 msec, and 185 to 225 msec) and remained constant in one (150 msec). Thus, slowing of PSVT after disopyramide seemed to reflect increases in both antegrade and retrograde conduction times in three patients, an increase in retrograde limb conduction time only in one, and an increase in antegrade limb conduction time only in one.

**Effects on Conduction Time vs Effects on Refractoriness**

Inducibility of sustained PSVT after disopyramide reflected the balance between the drug’s effect on conduction time within the reentrant circuit and its effect on refractoriness. The retrograde limb of the reentrant circuit was the most common “weak link” after disopyramide, so retrograde block rate after disopyramide (a measure of refactoriness) was compared to control PSVT heart rate (a measure of conduction time). In two of 16 patients, disopyramide failed to lower retrograde block rate below the control PSVT rate (table 1). These two patients continued to have inducible, sustained PSVT after the drug. However, four additional patients had inducible, sustained PSVT after disopyramide despite a retrograde block rate below the control PSVT rate (table 1). This was a result of a drug-related increase in conduction time that counterbalanced the increased refactoriness, resulting in slower, but still sustained, PSVT.

**Discussion**

Chronic electrophysiologic study, with one or more control studies and testing of individual antiarrhythmic drugs on subsequent days, has been used in patients with paroxysmal sustained ventricular, or supraventricular tachycardias. In addition to the feasibility of such a study, a high likelihood of concordance between laboratory and subsequent clinical results has been demonstrated. A drug found to prevent laboratory induction of tachycardia can be predicted to prevent recurrence of clinical tachycardia. Previous studies have described the effects of ouabain, propranolol, verapamil, procainamide, and quinidine in patients with PSVT.

Disopyramide has been advocated primarily for the control of ventricular dysrhythmia. However, many studies suggest that it is useful in terminating acute attacks of PSVT and in decreasing the frequency of their recurrence. These studies did not determine the mechanism of PSVT that was terminated or prevented, nor did they demonstrate the mechanism by which disopyramide exerted its effect.

There is some evidence that disopyramide increases AV conduction time and AV functional refractory periods in dogs. However, Josephson et al. found no prolongation of AV nodal conduction time or functional refractory periods in 12 human subjects. Further, sinus node automaticity and His-Purkinje function were unaffected. The AV nodal effective refractory period shortened slightly while the atrial effective refractory period lengthened slightly. In the two patients in that study who had PSVT (one with AV nodal reentrance and one with unknown mechanism), i.e. disopyramide caused slowing of PSVT in one and had no effect in the other.
In a study of electrophysiologic effects of disopyramide in 10 patients by Befeler et al., sinus node recovery time was significantly shortened by the drug. Atrial refractoriness and AV nodal effective refractory period were not significantly changed. AV nodal functional refractory period was significantly increased by disopyramide. Birkhead and Williams showed in 14 patients that, when pretreatment with atropine was used to control for the vagolytic effects of disopyramide, this drug resulted in a direct depressant effect on sinus node automaticity and atrial refractoriness.

Spurrell et al. reported the effects of i.v. administration of disopyramide in seven patients with normal control electrophysiologic findings and three with WPW syndrome. These authors noted increased intraatrial conduction times and atrial effective refractory periods. Minimal increases in AV nodal effective and functional refractory periods were demonstrated in a few patients. Ventricular effective refractory periods increased in all patients.

Of the three patients with preexcitation, antegrade accessory pathway conduction time was increased in two and complete antegrade block occurred in the other. Antegrade accessory pathway effective refractory periods were lengthened in both patients in whom it could be measured. Retrograde accessory pathway effective refractory periods also increased in two patients. Retrograde accessory pathway block occurred in the patient with antegrade block.

All three of Spurrell's patients had PSVT during control study. The cycle length of PSVT increased in one, decreased in one and was unchanged in the third after disopyramide. VA conduction times during PSVT increased in all patients, while AV conduction times increased in one and decreased in two. PSVT terminated in two of three patients, ending with failure of conduction in the retrograde limb of the circus movement.

Spurrell concluded that disopyramide would be useful in termination of AV reentrant PSVT in some patients. He stated that, although this drug might prevent AV nodal reentrant PSVT by suppressing premature atrial complexes, it was doubtful that it would be effective in terminating this tachycardia because of its minimal effect on AV nodal conduction.

Bennett also reported effects of disopyramide on PSVT in four patients with the WPW syndrome. In two patients PSVT could not be induced and one patient had slower, and one slightly faster heart rates during PSVT after the drug.

We further examined the mechanism of action of disopyramide in patients with PSVT. Although disopyramide had demonstrable effects on both the antegrade and retrograde limbs of the reentrant pathways, prevention of PSVT was usually the result of inability of the retrograde limb to support a circus movement. Such asymmetric effects of an antiarrhythmic drug on antegrade and retrograde AV nodal conduction have been recently described. This was evidenced in patients with AV nodal reentrance by single echoes ending with conduction to the ventricle but not returning to the atrium (three patients) or absence of echoes despite attaining long AV conduction times associated with induction of PSVT in the control state (two patients). In one patient, the limb in which reentrance terminated was unclear.

In patients with AV reentrance, the sites of the preventive effect of disopyramide were more diverse. One patient had absent VA conduction after disopyramide. One patient had only nonsustained PSVT always terminating with absence of conduction to the atrium. A second patient with nonsustained PSVT seemed to benefit from an effect of disopyramide on the atrium. In this patient, PSVT was always terminated by spontaneous induction of nonsustained atrial reentrant PSVT, a rhythm disturbance not seen in the control state. In the only patient with antiodromic AV reentrant PSVT, disopyramide prevented arrhythmia induction by both antegrade and retrograde limb effects.

When disopyramide failed to prevent induction of PSVT (eight of 16 patients), the tachycardia rate was reduced in each case, from an average of 172 to 149 beats/min. Examination of indirect measures of antegrade and retrograde conduction times during PSVT disclosed that the slower rate was a function of increased conduction time in both limbs of the circus movement. In only two of 16 patients did disopyramide fail to lower the retrograde block rate to below the rate of control PSVT. These two patients had sustained PSVT after disopyramide. However, four additional patients had sustained PSVT despite disopyramide's ability to lower retrograde block rate below the rate of control PSVT. In these patients, total conduction time in the reentrant circuit was increased by the drug more than retrograde refractoriness. The result was slower, but still sustained PSVT.

In conclusion, the present study demonstrates that oral disopyramide, in the doses used, prevented laboratory induction of sustained PSVT in 10 of 16 patients with a history of clinical PSVT, which was reliably inducible under control conditions. Disopyramide prevented induction in patients with AV or AV nodal reentrant PSVT. This effect was usually related to an increase in refractoriness in the retrograde limb of the circus movement. These results imply that this drug would be useful in selected patients in preventing recurrence of clinical tachycardia. PSVT that was not prevented by disopyramide was slowed as a result of increases in conduction time in both the antegrade and retrograde limbs of the reentrant circuit.

References


DISOPYRAMIDE FOR PSVT/Swiryn et al. 175

Effects of oral disopyramide phosphate on induction of paroxysmal supraventricular tachycardia.

S Swiryn, R A Bauernfeind, C R Wyndham, R C Dhingra, E Palileo, B Strasberg and K M Rosen

_Circulation_. 1981;64:169-175
doi: 10.1161/01.CIR.64.1.169

_Circulation_ is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1981 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/64/1/169