Effect of Digitalis in Patients with Paroxysmal Atrioventricular Nodal Tachycardia

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SUMMARY
Atroventricular (A-V) conduction, ventriculo-atrial conduction and mechanism of tachycardia were studied by programmed electrical stimulation before and after the administration of ouabain in 15 patients suffering from paroxysmal supraventricular re-entrant tachycardia. In 13 patients the tachycardia circuit was confined to the A-V node. In two patients the stimulation study showed that an accessory pathway was used in a ventriculo-atrial direction during tachycardia. Ouabain lengthened the effective and functional refractory period of the A-V node and A-V nodal transmission time in all patients in whom this could be studied. Only six patients showed lengthening in ventriculo-atrial conduction time or refractory period of the ventriculo-atrial conduction system. In seven patients no tachycardia could be initiated after ouabain. The width of the zone of atrial premature beats able to initiate tachycardia (the tachycardia zone) narrowed in five patients, showed no change in two patients, and increased in one patient. In these eight patients the tachycardia zone shifted to longer premature beat intervals. Ouabain resulted in slowing of cardiac rate during tachycardia. Both patients who used an accessory pathway during tachycardia showed no change in width of their tachycardia zone following ouabain administration. Seven patients were restudied two weeks after chronic oral administration of digoxin. The results were similar to those obtained following ouabain administration. This indicates that in patients suffering from paroxysmal A-V nodal tachycardia the effect of chronic oral digoxin administration can be predicted from the study of the effect of ouabain during programmed stimulation of the heart.

PATIENTS IN WHOM TACHYCARDIAS can be reproducibly initiated by programmed electrical stimulation of the heart are most suitable for the study of the effects of drugs on the arrhythmia. By using programmed electrical stimulation the acute action of different drugs has been evaluated in patients with the Wolff-Parkinson-White syndrome1-5 and A-V nodal tachycardia.6

This article reports on the effect of both acute and chronic administration of digitalis in patients thought to be suffering from paroxysmal A-V nodal tachycardia.

Material and Methods
Fifteen patients were studied. All suffered from recurrent attacks of (electrocardiographically documented) supraventricular tachycardia. During sinus rhythm and tachycardia 12 patients showed a normal QRS, two showed complete right bundle branch block and one complete left bundle branch block. None of the patients showed electrocardiographic evidence of the Wolff-Parkinson-White syndrome. Eleven patients were female and four male. Their ages ranged from 11 to 73 years. After obtaining informed consent, four catheters were passed through the femoral veins using the Seldinger technique. Two bipolar catheters were positioned high on the lateral wall of the right atrium. One was used for stimulation, the other for recording an intra-atrial electrogram. A tripolar catheter was placed in the region of the bundle of His to record a His bundle electrogram.7 The fourth (bipolar) catheter was positioned in the apex of the right ventricle and used for ventricular stimulation. In five patients a patent foramen ovale permitted recording of a left atrial electrogram. With help of the single test stimulus method during right atrial stimulation the functional and effective refractory period of the A-V node, the effective refractory period of the atrium and the zone of premature beat intervals resulting in atrial echoes or sustained tachycardia were carefully determined. Thereafter, using the single test stimulus method the right ventricle was paced up to its effective refractory period. The refractory period and pattern of V-A conduction were registered. Both atrial and ventricular pacing were done at rates just above the spontaneous sinus rate. Following these measurements digitalis was administered as ouabain in a dosage that ranged from 0.75 to 1.25 mg according to body weight. The drug was given directly into the right atrium through the catheter used for recording the atrial electrogram. During the hour following termination of the intra-atrial injection of ouabain the same stimulation program was repeated at least four times using identical basic cycle lengths and test stimulus intervals. In seven patients the stimulation procedure was repeated three to six weeks later, after taking oral digoxin in the interim. In these patients the digoxin levels at the time of the second catheterization varied from 1.1 to 2.4 ng/ml. In all seven patients the same basic pacing intervals were used as during their first study. Care was taken to place the stimulating and recording electrodes as nearly as possible in the same positions during the first and second catheterization to minimize the effect of
varied approaches to the A-V node on A-V nodal function. All data obtained during the stimulation studies were recorded on tape (Ampex FR 1300) and directly registered on an eight channel Elema Mangraf recorder. Leads I, II, III, V₁, V₄, the intracavitary right atrial lead, a left atrial lead if available, and the His bundle lead were all recorded simultaneously. For recording the His bundle electrogram an Elema EMT 12 was used.

S₁, A₁, H₁, and V₁ represent stimulus artifact, atrial, His bundle and ventricular electrograms of the basic atrial or ventricular paced complexes. S₂, A₂, H₂ and V₂ represent stimulus artifact, atrial, His bundle and ventricular electrograms of the premature atrial or ventricular beats.

A* represents the atrial electrogram of the atrial echo or atrial activation during tachycardia.

The effective refractory period of the A-V node was defined as the longest A₁-A* interval failing to conduct through the A-V node. The functional refractory period of the A-V node was defined as the shortest attainable H₁-H₂ interval.

Since it was not consistently possible to determine which part of the V-A conduction pathway became refractory during ventricular pacing, using the single test stimulus method, no values will be given for the effective refractory period of the different parts of this pathway.

The interval between the test stimulus and the beginning of the bundle of His electrogram is represented by V₁-H₂, and the interval between the beginning of the bundle of His and the beginning of activation of the atrium by H₂-A*.

Results

A-V Conduction

Before Drug Administration

The FRPAVN ranged from 360 to 480 msec in the 15 patients studied. The ERPAVN could be measured in 11 patients (in four patients A-V conduction was present up to the ERP of the atrium). The values ranged from 250 to 350 msec.

Following Ouabain

As shown in figure 1 following ouabain administration the A₂-H₂ interval gradually lengthened over a 45 minute period. No further increase was observed during the last 15 minutes of the study. In all patients the FRPAVN lengthened. The ERPAVN increased in all 11 patients in which this value could be measured. The increase varied individually from 40 to 130 msec (mean 90 msec) for the

Figure 1

Simultaneous recordings of lead II, an intracavitary lead from the right atrium (RA) and a His bundle lead following an atrial premature beat (A₂) at 320 msec during right atrial pacing at a basic cycle length of 700 msec. From top to bottom the events are shown prior to, and 15, 30, and 45 minutes after administration of ouabain. Note initiation of tachycardia prior to ouabain at an A₁-H₂ interval of 280 msec. Following ouabain the A₂-H₂ interval increases to a maximum of 420 msec 45 minutes after drug administration. The atrial premature beat is now only followed by a single atrial echo beat. There is no change in H₁-A* interval following ouabain administration.
FRP\textsubscript{AVN} and from 30 to 300 msec (mean 106 msec) for the ERPAVN.

When the A-H interval was compared at the ERPAVN following ouabain with the A-H interval at the same A\textsubscript{1}-A\textsubscript{2} interval prior to ouabain, all patients showed increase in A-H interval. The increase varied from 40 to 230 msec (mean 110 msec).

Following Digoxin

Similar findings were obtained as following ouabain. Figure 2 correlates the ERPAVN and FRPAVN following ouabain with those observed following digoxin administration. When A-H intervals measured at the shortest A\textsubscript{1}-A\textsubscript{2} which were conducted to the ventricle were compared following ouabain and digoxin, differences were found varying from 10 to 35 msec.

V-A Conduction

Before Drug Administration

All patients showed ventriculoatrial conduction during ventricular pacing. On applying the single test stimulation procedure ten patients showed the emergence of a retrograde His bundle electrogram at a critical test stimulus interval. This made it possible in these patients to divide the V\textsubscript{2}-A\textsubscript{2} interval into a V\textsubscript{2}-H\textsubscript{2} and an H\textsubscript{2}-A\textsubscript{2} interval.

Following premature beats given with increasing prematurity five patterns of V-A conduction could be recognized prior to ouabain administration:

A) No change in V-A conduction time up to the refractory period of the ventricle: one patient.

B) At first, no change in V-A conduction time with the His bundle electrogram approaching the atrial electrogram, followed at a critical V\textsubscript{1}-V\textsubscript{2} interval by sudden prolongation of the V-A conduction time which was found to be situated in the H\textsubscript{2}-A\textsubscript{2} interval: two patients (fig. 3).

C) Increase in V-A conduction time located in the V\textsubscript{2}-H\textsubscript{2} interval, the H\textsubscript{2}-A\textsubscript{2} interval remaining constant: five patients.

D) Increase in V-A conduction time, located both in the V\textsubscript{2}-H\textsubscript{2} interval and the H\textsubscript{2}-A\textsubscript{2} interval: two patients.

E) Increase in V-A conduction time where because of the absence of a His bundle electrogram the site of increase in V\textsubscript{2}-A\textsubscript{2} interval could not be determined: five patients.

Following Ouabain

Following ouabain nine patients showed no change or an increase of 10 msec or less in V-A refractory period and/or V\textsubscript{2}-A\textsubscript{2} conduction time: the patient with pattern A, four patients showing pattern C, both patients with pattern D and two patients with pattern E.

In six patients there was an increase in V-A refractory period or V\textsubscript{2}-A\textsubscript{2} conduction time of more than 10 msec. Both patients with pattern B, who showed a

![Figure 2](http://circ.ahajournals.org/)

Relation in seven patients between the effective refractory period (ERP) of the A-V node following ouabain and the ERP after digoxin (left), and the functional refractory period (FRP) of the A-V node following ouabain and the FRP after digoxin (right).

![Figure 3](http://circ.ahajournals.org/)

V-A conduction pattern while driving the right ventricle at a basic cycle length of 500 msec. A premature ventricular beat given after an interval of 290 msec is conducted to the atrium. As shown in the His bundle lead, activation of the His bundle coincides with the beginning of atrial activation. Shortening the premature beat interval to 280 msec results in sudden prolongation of V-A conduction time, with the His bundle electrogram preceding atrial activation by 150 msec. This pattern of V-A conduction continues up to the refractory period of the right ventricle (230 msec). The sequence suggests at first V-A conduction over an accessory pathway followed (on reaching the refractory period of the accessory pathway at a premature beat interval of 290 msec) by V-A conduction over the A-V node. Right bundle branch block was present during sinus rhythm and tachycardia.

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sudden increase in H2-A2 interval on reaching a critical V1-V2 interval, increased the refractory period of their A-V node after ouabain and the sudden increase in V2-A2 interval was no longer observed because of refractoriness of the area between the His bundle potential and the atrium. This meant an increase in refractory period of V-A conduction of 50 and 60 msec, respectively. V-A refractory period lengthened by 30 msec (due to increase in the refractory period of the H2-A2 interval) in one patient showing pattern C. The H2-A2 interval of the stimulus given at a V1-V2 interval 10 msec longer than the refractory period of V-A conduction following ouabain measured 20 msec longer than the H2-A2 interval following a premature beat given at the same V1-V2 interval prior to ouabain. Three patients with pattern E (no His bundle electrogram following the ventricular test stimulus) showed lengthening of the refractory period of V-A conduction by respectively 20, 50 and 110 msec. When following ouabain, V1-V2 intervals 10 msec longer than the effective refractory period of V-A conduction were compared with similar V1-V2 intervals prior to ouabain, V2-A2 intervals increased by respectively 20, 30 and 30 msec.

In the six patients showing lengthening of the effective refractory period in V-A direction the increase in length was 40 to 140 msec less than the increase in ERPAVN in antegrade direction following ouabain administration.

Following Digoxin

Similar findings as following ouabain administration were observed in six of the seven patients after digoxin. One patient (with pattern E of V-A conduction) who had shown no change following ouabain showed lengthening of the effective refractory period of V-A conduction by 50 msec.

Mechanism of Tachycardia

Before Drug Administration

Prior to ouabain, tachycardia could be initiated by a single atrial premature beat in all patients. Characteristically this occurred when a critical A2-H2 interval was reached. In eleven patients a sudden prolongation of the A2-H2 interval of 50 msec or more was observed when the A2-H2 interval of the latest premature beat able to initiate tachycardia was compared with the A2-H2 interval of the premature beat given 10 msec later. In the remaining four patients a gradual increase in A2-H2 interval was seen with initiation of tachycardia at a critical A2-H2 interval. The width of the zone during which an atrial test stimulus initiated tachycardia varied from 50 to 140 msec. As seen in the His bundle lead, in 10 patients atrial activation of the first beat of the tachycardia preceded or occurred simultaneously with ventricular activation. In five patients atrial activation occurred after ventricular activation.

The R-R interval during tachycardia varied from 410 to 260 msec. During single test stimulation from the ventricle it was possible to initiate tachycardia by a single ventricular premature beat in one patient. This was the only patient with no change in V-A conduction time during ventricular pacing (fig. 4). During tachycardia all patients showed a low to high sequence of atrial depolarization. Tachycardia could be terminated by critically timed atrial and ventricular stimuli in all patients.

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Figure 4

Upper panel: No change in V-A conduction time following a ventricular premature beat at 290 msec during pacing of the right ventricle with a basic cycle length of 550 msec. As demonstrated in the simultaneously recorded leads from the right atrium (RA), left atrium (LA) and vicinity of the bundle of His (His), left atrial activation precedes atrial activation in the His bundle lead and right atrial lead. This indicates V-A conduction over a left-sided accessory pathway. Note 1) activation of the bundle of His following the premature beat and 2) the sinus escape is followed by a QRS complex showing complete right bundle branch block. Lower panel) A premature beat given at 290 msec finds the His bundle refractory and is conducted to the atrium over the left-sided accessory pathway and followed by tachycardia. The sequence of events suggests V-A conduction over the accessory pathway and A-V conduction over the node during tachycardia.

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Following Ouabain

Following ouabain administration four different patterns could be observed as far as initiation of tachycardia by single atrial test stimuli was concerned.

A) In seven patients it was no longer possible to initiate tachycardia. An example is given in figures 5 and 6. In six of the seven patients test stimuli given close to the ERP of the A-V node after ouabain were conducted to the ventricle with marked A2-H2 prolongation and followed by single atrial echoes. The interval between H2 and the atrial echo was similar to the H2-A* interval during tachycardia prior to ouabain administration. In two patients the pattern of sudden increase in A2-H2 interval on entering the tachycardia zone (fig. 5) was no longer present when the atrial echo zone was entered following ouabain (fig. 6).

B) Five patients showed narrowing of the tachycardia zone in addition to a shift to longer A1-A2 intervals of premature beats initiating tachycardia. The width of this zone, which measured 50, 50, 60, 90 and 100 msec, was reduced to 20, 30, 20, 50 and 20 msec, respectively. Three patients showed initiation of tachycardia up to the ERP of the A-V node following ouabain. The other two showed a zone of 20 and 40 msec, respectively, between the ERP of the A-V node and the tachycardia zone. Test stimuli given with such an A1-A2 interval that they fell in between the ERP of the A-V node and the tachycardia zone were conducted with marked delay (A2-H2 interval) to the ventricle and followed by single atrial echoes but no tachycardia.

C) In two patients the tachycardia zone shifted to longer A1-A2 intervals with no significant change in width. In one patient the tachycardia zone moved from A1-A2 intervals of 290 to 350 msec to 320 to 390 msec, in the other patient from A1-A2 intervals of 270 to 360 msec to 350 to 430 msec. The first patient showed the V-A conduction pattern during ventricular pacing shown in figure 3. The second patient was the one in whom tachycardia could also be initiated by a single ventricular premature beat during ventricular pacing (fig. 4). In both patients, following ouabain, the tachycardia zone lasted to the ERP of the A-V node.

D) In one patient the tachycardia zone not only shifted to longer A1-A2 intervals but also widened considerably. This is shown in figures 7 and 8. In this patient the tachycardia zone increased by 90 msec. As shown in figures 7 and 8 the sudden increase in A2-H2 interval on entering the tachycardia zone was present both before and after ouabain administration. All patients in whom it was still possible to initiate tachycardia following ouabain administration showed slowing in heart rate during tachycardia. During tachycardia after ouabain A-H intervals were longer but H-A intervals were the same compared to tachycardia before ouabain. This prolongation of A-H without reduction of H-A produced a longer R-R in-

Figure 5
Initiation of tachycardia by a single atrial premature beat during atrial pacing at a basic cycle length of 700 msec. Five different premature beat intervals are shown. Note the gradual increase in A-V nodal transmission time (A2-H2 interval), reaching 145 msec at a premature beat interval of 340 msec. Further shortening of the premature beat interval by 10 msec is followed by marked prolongation of the A2-H2 interval (250 msec) and tachycardia.
interval and therefore a slower rate. The increases in R-R interval which individually varied from 35 to 110 msec represented a 9 to 30% increase over the original R-R intervals.

**Following Digoxin**

Four patients in whom the administration of ouabain resulted in inability to initiate tachycardias were given digoxin. In all four patients the same observations were made after oral digoxin as those after ouabain. An example from one of these patients is given in figure 9. In two patients ouabain resulted in narrowing of the tachycardia zone. The same was observed after digoxin. In one patient the tachycardia zone which measured 100 msec prior to digitalis, narrowed to 20 msec following ouabain, and became 50 msec after digoxin. One patient who showed a change in tachycardia zone to longer premature beat intervals without a change in width of the tachycardia zone following ouabain had similar findings after digoxin.

**Discussion**

If vagal maneuvers fail to terminate supraventricular tachycardia digitalis has been advocated as the first drug of choice. As shown by several workers using programmed electrical stimulation of the heart, digitalis increases the effective and functional refractory period of the A-V node and slows A-V nodal conduction time.

Our observations demonstrate that these effects can be beneficial in patients suffering from paroxysmal A-V nodal tachycardia, not only for termination but also for prevention of tachycardia. In twelve of our fifteen patients tachycardia could either no longer be initiated or the zone of premature beats able to initiate tachycardias narrowed considerably. In order to initiate A-V nodal tachycardia by a premature beat, slow conduction through the node is necessary. It has been stated that the ERP of the node should be short enough to allow the premature beat to enter the node early enough to be slowed sufficiently to start re-entry. Lengthening of the ERP of the node, thereby preventing the possibility of slow conduction through it, has been described by Wit and Cranefield as the mechanism responsible for prevention of A-V nodal tachycardia following verapamil. Following digitalis administration our patients demonstrated that in spite
of lengthening of the ERP of the node marked slowing in A-V nodal transmission time had occurred. All patients showed maximal A-H intervals which were of the same order or even longer (figs. 5 and 6) compared to those prior to digitalis. The beneficial effect of digitalis can therefore not be explained by lengthening of the ERP of the node alone. In six of the seven patients in whom digitalis prevented the initiation of tachycardia it was still possible to elicit a single atrial echo beat. Most probably, the increase in transmission time through the node in atrioventricular direction resulted from considerable prolongation of the refractory period of the antegrade part of the re-entry pathway. This prevented the impulse that followed the production of the atrial echo from returning to the ventricles and initiating tachycardia. In those patients in whom initiation of tachycardia was still possible after digitalis administration there was slowing of the tachycardia frequency associated with an increase of only the A-H interval. Since the returning H-A interval of the re-entry tachycardia did not change, we suggest that the slower rate was a result of decreased velocity of conduction in the antegrade limb of the re-entry circuit, while the retrograde limb remained unaffected. In addition, in spite of prolongation of the A-H interval after digitalis, atrial echo beats showed no change in the H-A* interval. In terms of the surface ECG none of our patients showed the inverse relation between P-R and R-P interval which is considered suggestive for A-V nodal re-entry. In the absence of knowledge of the exact location of the tachycardia pathway the following explanations can be offered to account for the increase in echo time (A-H-A*) interval without change in H-A* interval. First, the retrograde part of the re-entry circuit is located in the A-V node and is always fully excitable (even in the presence of digitalis) because of its late activation via the slowly conducting antegrade pathway. Second, V-A conduction occurs over an accessory pathway which is not affected by digitalis. Such a mechanism was postulated by Spurrell et al. observing that some patients suffering from paroxysmal supraventricular tachycardia did not increase their H2-A* interval during tachycardia following the administration of verapamil. Third, retrograde conduction through the A-V node is affected differently by digitalis administration than antegrade conduction. This proposition is extremely difficult to evaluate in the intact human heart because the single test stimulus method in several patients revealed the emergence of a retrograde His bundle electrogram following the induced premature ventricular activation indicating.
delay in conduction in the area between the site of ventricular stimulation and the bundle of His with late arrival of the impulse at the A-V node. Even in those patients who do not show the appearance of a His bundle electrogram following a critically timed ventricular premature beat we cannot exclude the possibility of delay in this area because His bundle activation might have been hidden in the QRS complex (measuring 120 to 140 msec) of the ventricular test stimulus.

Our six patients who showed a smaller increase in V-A conduction time or less lengthening of the refractory period of the A-V junction in the V-A direction after digitalis could not be counted as definite evidence for a possible divergent action of digitalis on the properties of the A-V node in the antegrade versus retrograde direction. In two of the three patients who did not show disappearance or narrowing of the tachycardia zone evidence could be assembled for A-V conduction over an accessory pathway during tachycardia. In one patient, left atrial activation preceded atrial activation in the His bundle lead by 40 msec, indicating a left sided accessory pathway only conducting from ventricle to atrium (fig. 4). During the application of test stimuli given with increasing

Figure 8
Same patient as figure 7, following ouabain. On changing the premature beat interval from 520 to 510 msec the \( A_2 \)-H2 interval increases from 180 to 390 msec. All premature beats given in the interval range 510 to 360 msec were followed by tachycardia. The ERP of the A-V node measured 360 msec. If one compares the R-R interval during tachycardia before and after ouabain an increase of 60 msec is seen.

Figure 9
Relation between the \( A_1 \)-\( A_2 \) interval and the \( H_1 \)-\( H_2 \) interval prior to and following ouabain, and after chronic oral digoxin administration. Prior to ouabain premature beats given in the interval range 320 to 260 msec resulted in tachycardia. The ERP\( _{AVN} \) and FRP\( _{AVN} \) prior to ouabain measured 290 and 410 msec, respectively. Following ouabain, no tachycardias could be initiated. The ERP\( _{AVN} \) and FRP\( _{AVN} \) increased to 330 and 470 msec, respectively. After digoxin there was a further increase in ERP\( _{AVN} \) and FRP\( _{AVN} \) to 380 and 480, respectively.
Prematurity the other patient showed no increase in V-A conduction time, with the H₂ approaching A₂, up to a critical V₁-V₂ interval when a sudden increase in H₂-A₂ interval appeared. This suggested V-A conduction over an accessory pathway with block in this pathway at a critical V₁-V₂ interval. Following digitalis V-A conduction was only seen over the accessory pathway.

The findings in these two patients demonstrate that in the presence of V-A conduction over an accessory pathway digitalis may not have a beneficial effect. This is not surprising because digitalis-induced slowing in A-V nodal conduction will increase the chance for the impulse to find the ventricular end of the accessory pathway excitable, thus favoring V-A conduction over this pathway and initiation of tachycardia.

Anatomically the tachycardia circuit will be larger in the presence of an accessory pathway compared to an intranodal re-entry circuit, increasing the chance for the A-V node to recover and to conduct the impulse in the A-V direction. The effect of digitalis administration in these patients will depend on the interplay between slowing in A-V conduction time on one hand and the increase in length of the effective refractory period of the node on the other hand.

Denes et al. in their study on the effect of propranolol on A-V nodal re-entrant tachycardia had the impression that patients showing a sudden increase in A₂-H₂ interval on entering the tachycardia zone (their “double pathway” cases) reacted more favorably to the drug than patients with a gradual increase in A₂-H₂ interval (their “reflection” cases). Prior to ouabain eleven of our patients showed a sudden prolongation of their A₂-H₂ interval on reaching the tachycardia zone. In ten the effect of ouabain was beneficial. In two of the four patients who showed gradual prolongation of the A₂-H₂ interval evidence could be obtained for V-A conduction over an accessory pathway. In both patients digitalis did not reduce the width of the tachycardia zone. It is of interest that in two patients showing a sudden increase in A₂-H₂ interval followed by tachycardia, the administration of ouabain prevented initiation of tachycardia but not of single atrial echoes which appeared at a critical A₂-H₂ interval after a gradual increase in A₂-H₂ interval (figs. 5 and 6). If one accepts the double pathway theory this might indicate that administration of ouabain resulted in a) complete refractoriness of the “fast” pathway or b) reduction in the differences in length of refractory periods of the two pathways. Studies at the cellular level are needed to clarify these changes.

Of practical importance is our observation of the good correlation between the acute effect of ouabain and chronic digoxin administration in patients with A-V nodal re-entrant tachycardia. This indicates that in patients suffering from this type of tachycardia the study of the effect of ouabain during the stimulation procedure is of great value for selecting those who will benefit from chronic oral digoxin administration.

Several authors have recently reported on the use of an accessory A-V connection, only conducting in V-A direction, in patients suffering from paroxysmal supraventricular tachycardia. Our study shows that failure to respond to digoxin therapy in patients apparently suffering from A-V nodal tachycardia may be based upon “concealed” V-A conduction over an accessory A-V connection during tachycardia.

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