Electrophysiologic Spectrum of Concealed Intranodal Conduction During Atrial Rate Acceleration in a Model of 2:1 Atrioventricular Block

James McKinnie, MD, Boaz Avitall, MD, Jose Caceres, MD, Mohammad Jazayeri, MD, Patrick Tchou, MD, and Masood Akhtar, MD

Concealed anterograde penetration of the atrioventricular (AV) node has been used to explain a wide variety of electrocardiographic findings. The effects of atrial rate acceleration on this phenomenon remain undefined. To examine the dynamic interrelations between conducted and nonconducted beats at different atrial rates, a unique atrial pacing protocol of functional 2:1 AV block was used in 10 patients. The pacing protocol involved abrupt transitions from 2:1 to 1:1 AV conduction and enabled quantification of conduction delay produced by nonpropagated impulses over extremes of atrial rate. Stable 2:1 AV conduction was maintained over a mean range of atrial paced cycle lengths of 289±29.6 to 223±33.0 msec, respectively. The mean AV conduction time during 2:1 and corresponding 1:1 drives at the longest atrial paced rates were 169±33.5 and 136.5±26.9 msec, respectively—revealing a significant effect of nonpropagated impulses on subsequent conduction. Surprisingly, at the shortest atrial paced rates, the mean AV conduction times were 191.5±31.8 and 161.0±23.3 msec, respectively. The lack of significant changes in conduction time between 2:1 and 1:1 drives at the extremes of atrial rate (32.5 vs. 30 msec, p=NS) suggests that the effect of concealed conduction is “fixed” and independent of rate. Clinical implications and postulated electrophysiologic mechanisms are discussed. (Circulation 1989;80:43–50)

An often-observed accompaniment of rapid atrial pacing and supraventricular tachycardias is the production of functional block at the atrioventricular (AV) nodal level, resulting in a 2:1 atrioventricular response.1 Lewis and Master2 first noted that the AV nodal conduction time of propagated impulses during 2:1 response was longer than that during 1:1 conduction at exactly half the atrial rate. This phenomenon has been attributed to partial anterograde penetration of the AV node during nonconducted impulses; an effect later termed “concealed conduction” by Langendorf.3

The 2:1 AV nodal response offers a unique opportunity to evaluate the electrophysiology of alternately conducted and nonconducted supraventricular impulses. The purpose of our study was, first, to investigate the stability of the 2:1 AV nodal response over the extremes of atrial rate encountered clinically and, second, to quantify the effects of nonpropagated impulses on subsequent conduction over a wide range of atrial rates. A unique pacing protocol was designed to achieve the latter objective. The data obtained demonstrate the manner in which concealed AV nodal conduction are operative over extremes of atrial rate. These findings provide new insight on complex AV nodal behavior.

Methods

Electrophysiologic studies were performed on patients in the nonsedated, postabsorptive state after informed consent was obtained. All antiarrhythmic medications and drugs with known AV nodal effects were discontinued for at least five half-lives before the procedure. Multipolar electrode catheters were percutaneously introduced into peripheral veins under local anesthesia and fluoroscopically positioned in the regions of the His bundle, high right atrium, and right ventricular apex. The catheters were used for local bipolar intracardiac electrogram recordings and stimulation

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using previously described techniques. Intracardiac electrograms were filtered at 30–500 Hz and simultaneously displayed with surface electrocardiographic leads I, II, and V1 and time lines on a multichannel oscilloscope (Electronics For Medicine VR16) and recorded on an FM tape recorder (Honeywell 101). Hard copy was obtained using a Mingograph ink jet recorder (Siemens) at a paper speed of 100 mm/sec. Electrical stimulation was performed with a digital programmable stimulator (Bloom Associates LTD) that delivered rectangular pulses of twice diastolic threshold and a pulse duration of 2 msec. All stimuli were delivered through an isolation unit, and the electrical equipment was grounded. The pacing protocol is depicted in Figure 1. The protocol was started by pacing the high right atrium at the longest paced cycle length, producing a stable 2:1 conduction pattern. This cycle length was determined by prior incremental atrial pacing. Stimulator rates were then abruptly halved to produce a 1:1 conduction pattern at exactly half the atrial rate. This entire sequence was repeated at 10-msec decrements until one of the following events occurred: 1) a stable 2:1 conduction ratio was lost, 2) atrial latency was noted, or 3) atrial arrhythmias were seen. At each paced cycle length, the duration of pacing was equal to or more than 30 seconds. The abrupt transition from 2:1 to 1:1 conduction was accomplished as follows.

During pacing that resulted in 2:1 AV nodal conduction, the stimulator was set to the AV sequential mode of operation with both outputs delivered through the same high right atrial pacing site. When one of the outputs was switched off, the net effect was an atrial train at twice the prior cycle length and this train resulted in 1:1 AV nodal conduction. Thus, for each atrial cycle length producing stable 2:1 conduction, its corresponding cycle length producing 1:1 AV nodal conduction was also examined. Whether the first beat of the 1:1 train followed a blocked or conducted beat was a random event. The rationale for comparing the 1:1 train directly after the 2:1 train was to minimize the time for changes in autonomic tone to occur.

**Data Analysis**

At each atrial paced cycle length, AV nodal conduction times were estimated as stimulus-to-His (SH) intervals measured from the onset of the atrial stimulus artifact to onset of the His bundle deflection in the His electrogram. Although this is not an exact measure of AV nodal conduction time, it is valid for comparative purposes as long as the atrial latency is excluded. This interval was chosen to minimize errors in measurement. To ensure stability during 2:1 conduction, SH intervals for the last 10 beats of the 2:1 train were measured and were excluded from data analysis if they differed by more than 5 msec.

**Analysis of 1:1 Cycles**

All SH intervals beginning from the first beat of the train were measured until they differed by 5 msec or less; this always occurred within three to five cycles of the onset of 1:1 conduction.

**Inclusion Criteria**

To be included in the study, patients had to meet the following criteria: 1) baseline rhythm was sinus rhythm; 2) no evidence of either dual AV nodal or accessory pathways was present; and 3) stable 2:1 train in terms of pattern and SH intervals variation by no more than 5 msec throughout the duration of pacing were present.

**Statistical Analysis**

Values are given as mean±SD. Comparison of SH intervals between 2:1 and 1:1 trains was by a paired t test. Statistical significance was defined as p<0.05.

**Results**

Eleven consecutive patients were studied for documented or suspected dysrhythmias. One patient was excluded from analysis because of the frequent induction of nonsustained atrial fibrillation. Thus, 10 consecutive patients satisfying the inclusion criteria comprised the study population. No patient was excluded because of instability of conduction during 2:1 drives. Their baseline clinical, electrocardiographic, and electrophysiologic data are shown in Table 1. There were nine men and one woman (age, 19–81 years; mean age, 50.9±20.2 years). Five patients had structural heart disease of atherosclerotic origin, one patient had idiopathic pulmonary hypertension, and four patients had structurally normal hearts as determined by physical examination and two-dimensional echocardiography. All patients had normal baseline PR (150±20 msec) and AH (98.5±11.6 msec) intervals during sinus rhythm. Thorough electrophysiologic evaluation including incremental atrial and ventricular pac-
**AV Response** which occurred during testing were carried out in all patients. However, only data pertinent to this report will be described.

**Cycle Length of Wenckebach AV Block and 2:1 AV Response**

Listed in Table 2 by patient number are the longest cycle lengths at which Wenckebach AV block was noted, the longest and shortest cycle lengths exhibiting stable 2:1 conduction, and the range of cycle lengths at which 2:1 conduction and Wenckebach AV block were noted. Wenckebach AV block occurred over a wide range of intervals from 360 to 280 msec with a mean value of 325.0±27.6 msec. The longest paced cycle length at which 2:1 AV nodal block was encountered had a mean value of 289±29.6 msec and varied from 350 to 250 msec. The 100-msec margin illustrates the wide range of cycle lengths over which the 2:1 response was initiated.

The shortest atrial paced cycle length at which a stable 2:1 response could be maintained had a mean value of 223±33.0 msec and varied from 270 to 180 msec. Cycle lengths shorter than the ones shown in the table resulted in nonsustained atrial fibrillation and the development of conduction ratios greater than 2:1. Changes in atrial latency were not seen over the range of cycle lengths used in the study. It is interesting to note that only three patients (patients 2, 5, and 10) were able to sustain a stable 2:1 conduction ratio below a cycle length of 200 msec corresponding to an atrial rate of 300 beats/min. At these atrial rates, our data suggest that in the supine state the AV node is operating near its limit for stable 2:1 conduction and may help explain the frequency of atypical conduction ratios. The 2:1 response usually manifested itself over a wide range of intervals varying from 30 to 120 msec with a mean value of 66±25.5 msec. In contrast, Wenckebach AV block was manifested over a significantly shorter range of intervals varying from 10 to 70 msec with a mean value of 35±20.7 msec (p<0.05).

**Effect of Atrial Rate Acceleration**

Table 3 lists for each patient the minimum and maximum AV nodal conduction times expressed as SH intervals for both 2:1 and 1:1 conduction over the range of cycle lengths demonstrating stable 2:1 conduction. Also, for each extreme of atrial rate, the value for the magnitude of concealment is listed. For any given atrial paced cycle length, the magnitude of concealment represents a difference of SH

<table>
<thead>
<tr>
<th>Patient</th>
<th>1:1</th>
<th>WAVB</th>
<th>2:1 Range</th>
<th>Range of WAVB</th>
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<tbody>
<tr>
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<td>340</td>
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<td>260–210</td>
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<td>2</td>
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</tr>
<tr>
<td>10</td>
<td>350</td>
<td>340</td>
<td>300–180</td>
<td>120</td>
</tr>
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</table>

All values in msec.

WAVB, Wenckebach atrioventricular block.
time between 2:1 conduction and the corresponding 1:1 conduction. The minimum SH mean value during 2:1 conduction was 169 ± 33.5 msec and during 1:1 conduction was 136.5 ± 26.9 msec. The difference in conduction times from 2:1 to 1:1 conduction was statistically significant (p < 0.05) and confirms the findings of previous investigators.\cite{3,6}

The mean value of magnitude of concealment corresponding to the minimum SH was 32.5 ± 20.3 msec. The maximum SH mean value during 2:1 conduction was 191.5 ± 31.8 msec and during 1:1 conduction was 161.0 ± 23.3 msec. The difference in these values was also significant (p < 0.05); however, the mean value of magnitude of concealment at these cycle lengths was 32.0 ± 17.0 msec. Thus, it can be appreciated that the difference in magnitude of concealment at the two extremes of atrial rate (32.5 ± 20.31 vs. 32.0 ± 17.0 msec) was not statistically significant. The effect of concealment as defined appears to be a fixed increment of conduction time over a range of atrial rates. Data from a representative patient are shown in Figure 2, as are transitions from 2:1 to 1:1 conduction at three atrial paced cycle lengths. Conduction times are labeled for each. At a paced cycle length of 280 msec, the SH interval is 140 msec and decreases to 125 msec when the corresponding 1:1 conduction ratio is examined. The difference in these SH intervals representing the magnitude of concealment is 15 msec. As atrial rate is increased, SH intervals also increase during 2:1 conduction; however, they also increased during 1:1 conduction such that the magnitude of concealment remained relatively constant. At a paced cycle length of 190, which is near the shortest cycle length allowing stable 2:1 conduction, it is seen that the magnitude of concealment remains 15 msec. Thus, while conduction times generally increased during 2:1 conduction as atrial rate acceleration occurs, conduction times also increased during the corresponding 1:1 conduction in a parallel fashion such that the magnitude of concealment remains relatively constant.

**Table 3. Comparative Conduction Data During Atrial Rate Acceleration**

<table>
<thead>
<tr>
<th>Patient</th>
<th>Minimum SH</th>
<th>Maximum SH</th>
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<tr>
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<td>9</td>
<td>210</td>
<td>135</td>
</tr>
<tr>
<td>10</td>
<td>120</td>
<td>110</td>
</tr>
</tbody>
</table>

All values in msec.

MOC, magnitude of concealment.

**Figure 2.** Transitions from 2:1 to 1:1 conduction at three different paced lengths. Electrocardiographic leads V1, I, II, and intracardiac electrograms from the high right atrium (HRA) and His bundle (HB) are shown in that order in all panels (patient 5). *The last paced complex of the 2:1 drive. Panel A: Transition from 2:1 AV conduction pattern at paced cycle length 280 msec to 1:1 conduction at cycle length of 560 msec. The 15-msec difference in SH time (from 140 to 125 msec) reflects the effects of concealed anterograde atrioventricular nodal penetration of nonpropagated impulse. Panel B: Effects of atrial rate acceleration of 250 msec. The increase of SH time to 150 msec during 2:1 conduction is paralleled by increased SH time during 1:1 conduction to 130 msec. Panel C: At the fastest atrial rates at which the 2:1 response is observed, SH times during 2:1 conduction continue to be paralleled by those seen during 1:1 conduction. S, stimulus artifact; T, time lines; S-H (SH), stimulus to His times in msec.
A graphic representation of this fact is illustrated by data from patient 6 (Figure 3). Plotted are conduction times expressed as SH intervals versus atrial paced cycle length for 2:1 and corresponding 1:1 trains. As shown by the graph, the conduction time increases during 2:1 conduction were paralleled by increases during the corresponding 1:1 conduction. Although conduction time increases during 2:1 conduction were generally paralleled by increases during the corresponding 1:1 conduction, individual differences were seen. This difference is best illustrated in patient 9 whose conduction times at the extremes of stable 2:1 conduction remained fairly flat, whereas conduction times during the corresponding 1:1 conduction increased in the usual fashion. The result in this patient was actually a decrease in the magnitude of concealment as atrial rate was accelerated. A similar effect although of lesser magnitude was seen in patient 7. In patient 4, a 20-msec increase in conduction times during 2:1 conduction was not paralleled by corresponding increases in 1:1 conduction such that the magnitude of concealment increased by 20 msec.

Surface Electrocardiographic Data

In all patients the stimulus-to-R interval of the conducted beat during 2:1 conduction exceeded the stimulus-to-R interval during the corresponding 1:1 AV conduction. An example of the relation between stimulus artifact and QRS location during atrial rate acceleration is shown in Figure 4. It can be seen that the relative position of the QRS complex and the nonconducted beat change remarkably during atrial rate acceleration; this results in a progressive incorporation of the nonconducted atrial beat into the QRS complex. In contrast, the relative position of the conducted P wave and the corresponding QRS complex change by only a small amount.

Discussion

Cycle Lengths of 2:1 AV Response

The electrophysiologic spectrum of ventricular responses to incremental atrial pacing includes a wide variety of simple and complex conduction patterns determined not only by the intrinsic state of the AV node but also by autonomic and neurohumoral factors. Typically, as atrial rate exceeds the ability of the AV node to sustain a 1:1 response, the first response observed is that of Wenckebach AV block in which periodic atrial impulses are nonconducted after PR interval prolongation. The resultant pacing induced Wenckebach cycles fre-
sequently have an atypical pattern. With further increases of atrial rate, a stable period of 2:1 AV conduction usually ensues. Although the AV nodal propensity to sustain 2:1 conduction ratios has been known for sometime, the range of intervals over which this occurs has not been well defined. In our study, this 2:1 response could be sustained over a wide range of cycle lengths having a mean value of 66±25.5 msec. In contrast, Wenckebach AV block was noted over a span of cycle lengths having a mean value of 35±20.7 msec. These differences were statistically significant (p<0.05). The stability of the 2:1 AV nodal response may be explained by beat-to-beat alternations in AV nodal refractoriness. Steinman and Lehmann⁶ used a pacing induced model of 2:1 AV conduction to demonstrate dramatic shortening of AV nodal effective refractory period after the blocked beat. Unlike the situation with Wenckebach AV block where refractory periods are increased on each beat of the cycle,⁷ the alternate long-short changes in refractory period during stable 2:1 conduction are probably instrumental in sustaining this conduction ratio. Increases of atrial rate beyond the limits of a stable 2:1 response frequently lead to the initiation of nonsustained atrial fibrillation. In patients with otherwise normal AV nodal function, the ability of the node to sustain a 2:1 response at atrial rates associated with the induction of atrial fibrillation may account for the relative rarity of conduction ratios of more than 2:1. Only three of the 10 patients were able to sustain a stable 2:1 response at atrial paced cycle lengths commonly seen during clinical atrial flutter.

The transition from Wenckebach AV block to stable 2:1 conduction was variable and, in five of 10 patients, occurred within 30 msec of the onset of Wenckebach AV block. Although study design precluded a beat-to-beat analysis of conduction ratios in the transition zone between Wenckebach and the longest cycle length of 2:1 conduction, several additional findings were observed in the majority of patients. First, as atrial rate increased, the length of Wenckebach periods decreased and conduction ratios increased. It was not unusual to see conduction ratios of 4:3 progressing to 3:2 before becoming 2:1 as the atrial rate increased. Second, in patients in whom the range of Wenckebach AV block exceeded 30 msec, alterations of 3:2 and 2:1 conduction ratios were frequent. These data are consistent with the recent work of Shrier et al;⁸ these authors were able to predict the conduction ratios at various atrial paced cycle lengths using an iterative technique based on data obtained from atrial refractory period determinations.

Effect of Increasing Atrial Rate on Concealment

The 2:1 AV conduction pacing model provides a stable means of assessing the interactions of conducted and nonconducted beats through the AV node. In our study, differences in conduction times observed during abrupt transitions between 2:1 and 1:1 drives were used to quantitate the effects of anterograde concealed AV nodal penetration at multiple atrial rates. Lewis and Master⁹ first demonstrated that nonconducted beats prolong the AV nodal conduction time of subsequently propagated impulses during 2:1 conduction. However, the effects of increasing atrial rate on this phenomenon were not examined. In addition, Steinman and Lehmann⁶ investigated the beat-to-beat alterations of conduction and refractoriness in a steady-state 2:1 pacing model using the extrastimulus technique. Significant oscillation in AV nodal effective refractory period was demonstrated. Also, a greater prolonging effect of prior concealment on AV nodal conduction time versus AV nodal effective refractory period for the propagated beat was attributed to the electrotropic effects of the blocked beat on action potential duration and recovery of excitability. Our study extends these observations by examining the effects of nonpropagated impulses over the extremes of atrial rate that sustain a stable 2:1 AV nodal response. A major finding of our study is that concealed anterograde penetration of the AV node produces a prolongation of subsequent conduction time that is relatively fixed and independent of the atrial paced cycle length. The magnitude of the increase in conduction time between 2:1 and 1:1 drives, here termed magnitude of concealment for definitional purposes, had a mean value of 32.5±20.3 msec and was not significantly different over the extremes of atrial paced cycle length. Because the exact cellular and geometric factors responsible for cycle length dependent changes in AV nodal propagation have not been completely elucidated, the mechanisms responsible for this “fixed” concealment effect cannot be determined. Results of previous experimental studies do, however, suggest several potential mechanisms.

Role of “Step Delays” and Discontinuous Conduction

The concept of concealed conduction has been used to explain a wide variety of electrocardiographic findings.⁹ Several alternative mechanisms have been proposed to explain the prolongation of AV nodal conduction time with atrial rate acceleration. Rosenblueth⁰,¹¹ has suggested that the cycle length–dependent properties of AV nodal tissue may be due to slowing of impulse propagation at a localized region of the AV node. Conduction across such a “step delay” is thought to be electrotropic in nature. Presumptive evidence for this hypothesis was found in the experimental study of Billette¹² in rabbit AV node in which he demonstrated that the major fraction of cycle length–dependent AV nodal delay occurred between the N and NH regions. This region was devoid of action potential upstrokes, suggesting propagation was electrotropic in nature. In addition, Young et al,¹³–¹⁵ using a one-step delay analysis of AV nodal transmission, was able to predict conduction patterns associated with sudden
acceleration of atrial rate, pacing-induced atypical Wenckebach patterns, and reversed alternating Wenckebach patterns. The electrophysiologic consequences associated with electrotonic transmission across such a step delay have been well studied in the sucrose gap preparation. In this preparation, impulses that were unable to conduct across a region of depressed excitability were nonetheless able to produce low-amplitude electrotonic responses in distal tissue capable of active propagation. The electrotonic image of the nonconducted impulse was shown to exert an important inhibitory effect on subsequent impulse propagation. This effect that demonstrates both voltage and time dependence has been termed “electrotonic inhibition.” Thus, the prolongation of conduction time of a propagated impulse after a nonpropagated impulse is not due to proximal delay across relatively refractory tissue but to the inhibitory effects of the nonpropagated impulse on the source-sink characteristics of tissue distal to the site of block. Therefore, the functional consequence of the blocked beat is a horizontal dissociation of the AV node into two regions. Our results indicate that the effects of the blocked beat on subsequent propagation time are relatively independent of atrial paced cycle length. It is conceivable that the observed propagation times may actually represent different phenomena at the extremes of atrial paced cycle length. For example, at the longest atrial paced cycle length, the amplitude of the electrotonic voltage at the distal site is high, resulting in maximal electrotonic inhibition. As atrial rate is accelerated, the voltage dependent effects on the distal side are diminished; however, increasing delay at more proximal sites results in relatively constant differences in propagation observed between 2:1 and 1:1 drives. This increment in conduction time due to proximal nodal delay at short cycle lengths has also been demonstrated in rabbit AV node. Differences in the relative magnitude of these two effects may account for the individual differences seen in this study.

Although we believe that an interpretation of data as detailed above best fits the known electrophysiology of the node, there are several alternative possibilities. First, it is conceivable that the observed differences in propagation time do not reflect the inhibitory effect of a nonpropagated impulse at a more distal AV nodal site but rather a change in propagation pathway. This explanation need not imply engagement of fibers that are functionally disparate but rather may reflect anisotropic conduction characteristics through the AV node. Prior studies dealing with this phenomena have been performed in tissues relying predominately on the fast inward sodium current, and the application of this concept to AV nodal conduction has not been well studied. A second possibility is that the blocked beat occurs at an AV nodal site proximal to the region of step delay. The effect of the blocked beat then will be the production of two step delays. The step delay at the more proximal level, then, would add a fixed increment of conduction time as well as temporally shift the input to the more distal step delay. This would explain many features of the data seen in the present study. However, our proposal that the site of block occurs at the proximal border of a step delay is more compatible with experimental observations. A third possibility is that during 2:1 AV nodal conduction the site of block is not in the AV node proper but instead in one of the atrial inputs to the node. This phenomena has been well demonstrated in the rabbit AV node. Conduction time increases may, therefore, represent a failure of summation phenomena at a critical AV nodal site.

Limitations

An unresolved aspect of the study is the applicability of sequential comparisons of 2:1 and 1:1 drives to quantify the effect of blocked beats on subsequent conduction. The present comparison may be an oversimplification considering the complex interactions likely to be present between conducted and nonconducted impulses. A study examining the refractory characteristics of the AV nodal pacing model concluded that the effect of the blocked beat may extend past the next propagated impulse. This effect is consistent with inhibitory phenomena observed in the sucrose gap preparation. Such consideration should not detract from the behavior demonstrated in this model. Also, we cannot totally exclude autonomic changes; however, the transitions from 2:1 to 1:1 conduction were made very rapidly to minimize these changes. In addition, the pacing sequences were varied with alternation from 2:1 to 1:1 and then back to 2:1 and there were no significant changes noted. Also, atrial-to-His (AH) times before and after pacing were compared and there were no significant changes. Thus, we do not feel that autonomic changes were responsible for the observed phenomena in a significant way.

Surface Electrocardiographic Manifestations

During tachycardias with a 2:1 AV nodal response, the question frequently arises as to which of the ectopic P waves are conducted and which are blocked. Over the span of cycle lengths supporting AV nodal conduction in this study, the blocked P wave was usually visible after the QRS complex at the longest paced cycle lengths and gradually merged into the QRS complex with decreasing paced cycle lengths. At the shortest paced cycle length, the blocked P wave was visible just before the QRS. It could be argued that because of autonomic changes that occur during tachycardia, the conducted atrial beat may have a PR interval shorter than that during sinus rhythm. However, in no instance was an ectopic P wave with a PR interval less than the PR interval during sinus rhythm conducted to the ventricle considering the site of stimulation being the high right atrium.
Clinical Implications

In addition to the significance of the surface electrocardiographic findings discussed above, the study is of theoretic and conceptual interest. For the first time, the manner in which conducted beats interact with blocked beats over a range of cycle lengths has been demonstrated. These data provide a framework for understanding the human AV nodal responses during a variety of atrial tachyarrhythmias.

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References


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