pathologic anatomy is seemingly so varied, one would expect the physiology to be somewhat varied and that prognosis might be related to a number of factors hitherto unrecognized in these patients.

It is our feeling that a number of potential risk factors have been recognized in this study — all requiring long-term follow-up to see which, if any, do indeed increase risk. The abnormalities identified in this study, most of which were totally unsuspected from routine ECG studies and which are suggested as possible risk factors include: 1) nocturnal bradycardia mostly due to junctional exit block, noted in 7/20; 2) tachyarrhythmias (12/20) and especially major tachyarrhythmias (3/20); and 3) unresponsive or "lazy" junctional pacemakers which did not vary discharge rate. Several patients had more than one of these potential risk factors but the only patient with all three suffered a serious syncopal attack. In order to determine whether or not these really are risk factors, we would recommend that follow-up of all patients with congenital heart block should include, in addition to a His study, electromagnetic ECG tape recordings at fairly frequent intervals. The meaning of these described abnormalities is still not sufficiently understood to change recommendations for insertion of permanent pacemakers. These indications, at present, are symptomatic block or evidence of distal block.

SUMMARY Two patients who presented by scalar ECG with an A-V junctional tachycardia were demonstrated during an electrophysiologic evaluation to have an atrial tachycardia without P waves in the surface ECG. Case 1 had an atrial tachycardia that conducted through the A-V node with a Wenckebach block. Atrial activity was recorded only from the proximal portion of the coronary sinus and from right atrial areas near the tricuspid valve. Case 2 had an atrial tachycardia that abruptly began and terminated following carotid sinus massage. Atrial activity was recorded only in the coronary sinus os, and pacing at that site resulted in atrial capture, with Wenckebach conduction to the ventricles. These observations demonstrate that an atrial tachycardia without P waves can simulate A-V junctional tachycardia with or without Wenckebach block. Such findings may have a bearing on some important electrophysiologic concepts such as the origin of A-V junctional rhythms and the need for atrial participation in A-V nodal re-entry.

RHYTHMS CHARACTERIZED in the scalar electrocardiogram (ECG) by a normal QRS complex and the absence of P waves are thought to originate in or near the bundle of His. Similarly, when a regular ventricular rhythm with normal QRS complexes occurs during atrial fibril-lation, the ventricular rhythm is again considered to originate in the atrioventricular (A-V) junction. If the ventricular rhythm exhibits group beating indicative of Wenckebach periodicity, an A-V junctional tachycardia with a Wenckebach exit block is diagnosed. 1

Although the ability of one atrium or a part of one atrium to exhibit electrical activity which differs in rate and/or rhythm from the rest of the atria has been known since about 1900, 2,3 interest in the concept of dissimilar atrial rhythms 4 recently has been revived. We4 have shown with catheter electrode recordings that one atrium may be electrically silent while the other atrium, or a part of it, generates electrical activity. Since such electrical activity

References

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Atrial Tachycardia without P Waves
Masquerading as an A-V Junctional Tachycardia

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present in only one atrium or a portion of one atrium may not be detected by conventional scalar electrocardiography, it is possible that some examples of apparent A-V junctional tachycardia might in fact be due to an atrial tachycardia arising in a discrete area of one atrium. Naturally, for a small part of the atrium, in the midst of more widespread atrial quiescence, to conduct to the ventricles, the area should probably be located near the A-V node. Recently, we had the opportunity to assess atrial activity in two patients who presented with a normal QRS complex and no recordable atrial activity in the scalar ECG. In this report, we present the results of electrophysiologic evaluation of these patients.

Case 1

A 62-year-old female with rheumatic mitral stenosis and insufficiency, and tricuspid insufficiency, had atrial fibrillation for many years, adequately controlled with standard doses of digitalis. Because of severe congestive heart failure, she underwent mitral valve replacement with a porcine mitral valve and a tricuspid valve annuloplasty with a Carpentier ring. Postoperatively, the ECG failed to show evidence of atrial activity and she was thought to have an A-V junctional rhythm. The R-R cycles exhibited group beating, consistent with the diagnosis of an A-V junctional tachycardia with a Wenckebach exit block (fig. 1). She exhibited no evidence of digitalis toxicity, and the same rhythm was still present one month after surgery, at which time she underwent an electrophysiologic evaluation. She was receiving digoxin, 0.25 mg daily at the time of study.

Case 2

A 57-year-old female underwent mitral valve commissurotomy in 1965 and mitral valve replacement in 1968 because of rheumatic mitral stenosis. She had had atrial fibrillation for many years and over the past several months had experienced progressive worsening of congestive heart failure along with the development of significant tricuspid insufficiency. During this period, the ECG demonstrated no recordable atrial activity. There was a regular paroxysmal supraventricular tachycardia which would terminate and

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**Figure 1.** Case 1. Apparent A-V junctional tachycardia with a Wenckebach exit block. No P waves can be seen. The R-R intervals in the 12 lead electrocardiogram conform to a classical Wenckebach structure, except for V5 and V6. The rhythms in these leads may have been generated by the more rapid atrial tachycardia. In the rhythm strips below (lead I, top; lead V5, bottom) recorded on a different day, the R-R cycles perfectly fit the diagnosis of an A-V junctional tachycardia with a Wenckebach exit block of 4:3, 5:4 and 6:5, at an A-V junctional discharge cycle length of 465 to 475 msec.

**Figure 2.** Case 2. Apparent A-V junctional tachycardia at two different rates. No P waves can be seen in the 12 lead electrocardiogram. In the monitor strip below (continuous recording), a deflection in the early portion of the ST segment appears at times to be a P wave but its inconsistent presence in this portion of the cardiac cycle, and its presence during the periods of asystole, indicate that it is an artifact in the monitor recording lead. Carotid sinus massage abruptly terminated the tachycardia, which then began again. The monitor strip was recorded immediately prior to the electrophysiologic study, which confirmed the absence of P waves or atrial fibrillation.
begin suddenly following carotid sinus massage (fig. 2). No evidence of digitalis toxicity was present. The patient was receiving digoxin, 0.25 mg daily at the time of the electrophysiologic study.

Methods

The electrophysiologic study was performed using standard methods,2 after obtaining informed consent. Case 1 had had a bipolar pacing catheter electrode positioned at the apex of the right ventricle and at the time of study, two quadripolar electrode catheters were inserted into the right femoral vein and were used to record activity from the atria and bundle of His. In case 2, quadripolar electrode catheters were inserted via the right femoral vein and left antecubital vein into the right atrium and coronary sinus, respectively. A tripolar electrode catheter was inserted into the left femoral vein and positioned to record His bundle activity. The distal tip electrode of the quadripolar catheters was separated from the first ring electrode by 10 mm; each of the other ring electrodes were separated by 10 mm. Tracings were recorded on a multichannel oscilloscopic recorder (Electronics for Medicine DR 8) at a paper speed of 100 mm/sec, using filter settings between 40 and 500 Hz for the electrograms, and 0.1–20 Hz for the surface ECG. The right atrium, apex of the right ventricle, and the left atrium via the coronary sinus were each stimulated in turn through an isolation transformer (Digipulser, model 830; Isopulser, model 850) with pulses of 2 msec duration at 2–4 volts intensity for the ventricle and up to 75 volts for the atria.

Results

Case 1

One quadripolar catheter was inserted into the coronary sinus, while the second was used to search the right atrium for electrical activity. No atrial activity could be recorded in the distal coronary sinus or in any portion of the right atrium, except at a position near the tricuspid valve. At that site, large amplitude deflections were registered (fig. 3, upper panel). Movement of the atrial catheter to a position closer to the tricuspid valve diminished the amplitude of the atrial deflections but enabled us to simultaneously record His bundle activity (fig. 3, lower panel). The H-V interval remained constant at 50 msec. During this portion of the study the atrial cycle length was about 325 msec and atrial impulses conducted through the A-V node with a varying degree of block. The R-R interval was irregular and did not conform to strict Wenckebach group beating, probably due to concealed conduction, but the block was proximal to the His bundle. Occasional ventricular extrasystoles were present and provided a pause during which consecutive atrial cycles were clearly seen.

It was difficult to maintain ideal catheter position from which both atrial and His activity could be recorded simultaneously. The catheter could not be held within the orifice of the tricuspid valve because of the large right atrium and the presence of tricuspid insufficiency, and the catheter was constantly expelled back into the right atrium. The catheter within the coronary sinus was more stable. Catheter positions were documented radiographically while simultaneously recording electrical activity, at the time of a coronary sinus "pullback" (fig. 4). In the BAE lead (top panel), right atrial activity is no longer recorded as a sharp deflection because of a very slight catheter shift, but it was from this general area or slightly closer to the tricuspid valve ring, that atrial and His activity were recorded in figures 3 and 5. Note the fractionated appearance and prolonged duration of the right atrial electrogram at this site. The coronary sinus catheter has been pulled back a few millimeters from its position at the time figures 3 and 5 were recorded and very small atrial deflections can be seen in the electrogram recorded from the two proximal poles (CSb, arrows). In the middle panel, the coronary sinus catheter has been pulled

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

**Figure 3.** Case 1. Atrial tachycardia without P waves at a cycle length in the range of 325 msec, producing an irregular ventricular response due to block at the A-V node. BAE, bipolar right atrial electrogram; CSb, bipolar recording from the proximal two electrodes of the catheter deep in the coronary sinus; CS, bipolar recording from the distal two electrodes of the catheter in the coronary sinus; I, II, III and V₁, scalar leads I, II, III and V₁; A, atrial activity; V, ventricular activity; H, His bundle activity. All numbers in msec. Paper speed, 100 mm/sec. Horizontal bar, 200 msec.
back further and fairly large atrial deflections were recorded in CSb, but none in CSa. Finally, when the catheter was withdrawn so that the distal two electrodes lay within the coronary sinus os, atrial activity was recorded from both CSb and CSa (bottom panel). These figures illustrate that bipolar electrodes with one of the pair only 10 mm away from recordable atrial activity may not register that activity.

The atrial cycle length remained fairly constant within 5–15 msec at any one time. However, the tachycardia occurred at two different cycle lengths of about 325 and 385 msec during the electrophysiologic study. At the slower atrial rate, classical Wenkebach conduction was present and conduction ratios varied between 3:2, 4:3 and 5:4 (fig. 5; the last QRS complex in the top panel is repeated as the first QRS complex in the bottom panel). In this example, during the 5:4 grouping, the length of the final R-R cycle exceeded the length of the preceding R-R cycle and is characteristic of "atypical Wenkebach."

The right and left atria (via the coronary sinus) were totally nonexcitable at voltages up to 75 volts. During ventricular pacing, complete retrograde block was present.

Case 2

One quadripolar catheter was inserted deep within the coronary sinus, and the second was used to search the right atrium for electrical activity. No atrial activity could be recorded in the distal coronary sinus, or in any portion of the cavity of the right atrium. When the coronary sinus catheter was withdrawn so that the distal two poles were at the coronary sinus os, distinct atrial activity was registered (fig. 6A). Note the absence of atrial activity in both the His (BHE) and right atrial (BAE) electrograms. The H-V interval remained constant at 50 msec. The tripoal catheter was used to record His bundle activity but, as in case 1, tricuspid insufficiency prevented positioning the catheter across the tricuspid valve, and the best recordings were made with the catheters in the positions seen in figure 6B.

At the beginning of the study, prior to recording definitive atrial activity, the R-R cycle length ranged between 800 and 1200 msec. Then, tachycardia began spontaneously and remained throughout the period of investigation, except when transiently interrupted by carotid sinus massage or pacing. The atrial cycle length was 490–530 msec during the tachycardia. Carotid sinus massage resulted in abrupt slowing of the ventricular rate, as seen in figure 7. The atrial rate also slowed, though not as markedly.

The nature of this response to carotid sinus stimulation can be interpreted in several ways, depending on the mechanism thought to be responsible for the PSVT. First, if the PSVT was due to discharge of an automatic ectopic
atrial pacemaker which conducted to the ventricle with a long A-H conduction time, enhanced vagal tone could have transiently slowed the discharge rate of the pacemaker, causing a number of atrial impulses to block, and thus slowed the ventricular rate. The second possibility is that atrial activity was due to retrograde conduction during a PSVT, which was maintained by A-V nodal re-entry. Carotid massage abruptly terminated the A-V node re-entry and PSVT, which was replaced by an "atrial escape rhythm" with coupled (460-470 msec) premature atrial extrasystoles. When one of the coupled premature atrial extrasystoles conducted through the A-V node with a sufficiently long A-H interval, it restarted the A-V nodal re-entry and PSVT. A third possibility, that of a tachycardia originating in or near the His bundle with retrograde atrial capture, also must be considered. The fact that atrial activity precedes,
and appears to conduct to the ventricles when the tachycardia terminates at least establishes that atrial activity during tachycardia could be conducting anterogradely to the ventricles with a long A-H interval, but does not eliminate the possibility of a His bundle pacemaker during tachycardia.

The premature ventricular extrasystole shown in figure 6A occurred 350 msec after the onset of the preceding QRS complex, at least 80 msec in advance of the next expected His bundle discharge. Therefore, the premature ventricular extrasystole probably depolarized the His bundle retrogradely. If retrograde activation of the His bundle in fact occurred without altering the A-A cycle ("compensatory pause") or interrupting the tachycardia, then a tachycardia originating in the atria would appear most probable. If, however, the premature ventricular extrasystole did not excite the His bundle retrogradely, then the possibility of a tachycardia originating in the His bundle or due to A-V nodal re-entry cannot be eliminated.

Ordinarily, the contour of the P wave in the surface ECG could be used to help differentiate between these possibilities. For example, if the contour of the P waves remained abnormal at the time of ventricular slowing (fig. 7), and both the "escape" and coupled premature P waves resembled the P wave during the PSVT, then an automatic pacemaker in the atrium would seem likely. If, however, the contour of the "escape" P wave, at the time of ventricular slowing, resembled a normal sinus P wave, the coupled premature P wave was abnormal, and the P wave during the PSVT was retrograde in contour, then a tachycardia due to His bundle discharge or A-V nodal re-entry might be likely. It is difficult to ascribe significance to the contour of a single electrogram recording because of catheter shifts and the use of closely spaced bipolar electrodes. A marked change in the P contour might occur with a minimal change in electrogram shape. Thus, we cannot absolutely differentiate between the possibilities of automaticity and re-entry.

The presence of the premature ventricular extrasystole at the time of slowing in this example (fig. 7) is probably fortuitous because premature ventricular extrasystoles with the same coupling interval occurred at times when the PSVT did not terminate (fig. 6) and were not present at times when the PSVT did terminate (fig. 2).

The right atrium and the left atrium (via the coronary sinus) were totally nonexcitable at voltages up to 75 volts, except when stimulating at the precise site in the coronary sinus os where the atrial activity had been recorded. At that site, atrial pacing at a cycle length slightly shorter than the spontaneous cycle length resulted in capture of the atrium and conduction to the ventricles with a Wenckebach block. We were unable to record atrial activity with the proximal pair of electrodes (CSp), which were only 10 millimeters from the pacing site, or in the surface ECG (fig. 8). Atrial pacing at cycle lengths of 400 msec decreased the ventricular rate by increasing the A-V block, and resulted in termination of the tachycardia on two occasions when pacing was abruptly stopped. These latter observations provide evidence to support the fact that atrial pacing was conducting to the ventricles without producing P waves.

Discussion

Validation

In both patients, the entire length of the coronary sinus and multiple sites in the right atrium were explored with the tip of the catheter electrode, searching for electrical activity, which was recorded only near the tricuspid valve. In the first patient, electrical activity was recorded from an area definitely located in the right atrium, while in the second patient electrical activity was recorded from an area located near the os of the coronary sinus, which conceivably could have represented either right or left atrial activity, or activity from the coronary sinus itself. Repeated exploration of these areas many times throughout the procedure confirmed the presence of consistent, localized atrial activity. In both patients, no atrial activity could be recorded from deep within the coronary sinus but could be recorded from more proximal portions. In case 2, localized atrial excitability was established by demonstrating capture of the atrium with conduction to the ventricles when stimulating in the specific area where atrial potentials were recorded. No P waves in the scalar ECG or atrial activity in bipolar electrodes only
10 mm away from the pacing site were registered. Pacing at other sites in the right atrium and from the coronary sinus in both patients failed to demonstrate atrial excitability. From these data, it would appear quite certain that we were recording actual atrial activity.

It is of interest that the recordings from these two patients resemble somewhat the recordings obtained from experimental animals during sinoventricular conduction. In both instances, a pacemaker site in the atrium controls the ventricles without manifest P waves.

**Interpretation**

We interpret these data to indicate that, in spite of the absence of a P wave in the scalar electrocardiogram or recordable atrial activity from the majority of the right atrium and length of the coronary sinus, localized atrial activity can still control the cardiac rhythm or participate in an A-V nodal re-entrant circuit. Without a careful exploration for electrical activity in both atria, the diagnosis of atrial quiescence would have been made. Therefore, the relationship of atrial activity to the cardiac rhythm cannot be unequivocally established unless such a search is performed.

**Significance**

These data indicate that the diagnosis of an A-V junctional rhythm or an A-V junctional tachycardia with or without exit block cannot be made with complete certainty from the scalar electrocardiogram since an area of atrial activity can control or participate in the cardiac rhythm and not be recorded in the ECG. Whether such a concept can be extended to patients without atrial disease cannot be stated on the basis of our data. However, it is possible that premature atrial excitation in a normal heart could result in activity in a portion of one atrium only. If the mass of this portion were sufficiently small, a P wave might not be generated in the scalar electrocardiogram. Such dissimilar atrial rhythms might also occur in the presence of atrial fibrillation or any other manifest atrial rhythm. Thus, it is possible that A-V junctional rhythms or A-V junctional tachycardias, including those with a Wenckebach exit block, may be caused by atrial rhythms or atrial tachycardias in some instances.

In addition, figures 1, 3 and 5 from case 1 document that the discharge rate of an atrial tachycardia can vary, so that at more rapid rates the ventricular response may not conform to a typical Wenckebach structure and may be misinterpreted as the ventricular response to atrial fibrillation. At the slower atrial tachycardia rate, the more usual Wenckebach grouping was present in this patient.

These observations have important implications in the interpretation of some electrophysiologic data obtained from animals or patients. For example, it has been accepted generally that A-V nodal cells lack automatic activity and therefore "A-V junctional rhythms" are thought to arise from sites very close to, or within, the bundle of His. Perhaps the origin of such rhythms in some patients lies within localized areas of atrial discharge, as shown here. Whether these localized areas of atrial activity represent a rim which lies within the anatomic boundary of the "A-V apparatus" and therefore should be considered "A-V junctional rhythms" can only be conjectured. Clearly, in case 1, atrial activity was recorded at some distance from the anatomic location of the A-V node and His bundle (BAE, fig. 4) and preceded the onset of atrial activity recorded at a position closer to the A-V node, in the coronary sinus.

Mendez et al. demonstrated the need for atrial participation in ventricular echoes caused by A-V nodal re-entrant activity in dogs. Since that time, studies in animals and patients have challenged the interpretations made by Mendez et al. and have suggested that ventricular echoes due to A-V nodal re-entry can occur without activation of the atrium. Whether case 2 had an A-V nodal re-entrant supraventricular tachycardia may be questioned. What cannot be questioned is that data from both patients indicate that a portion of the atrium may control or participate in the cardiac rhythm without recognition of this fact from the scalar ECG or, indeed, from recordings within the right atrium or the coronary sinus. Thus, whether or not the observations of Mendez et al. are applicable to man, the fact remains that data refuting the participation of the atrium as a necessary link in the re-entry pathway must be obtained from studies in which both atria are carefully searched for the presence of localized electrical activity, particularly in regions near the A-V node.

Finally, it is tempting to speculate about the relationship of our data to recent electrophysiologic observations demonstrating that automaticity can occur in canine mitral and tricuspid valve fibers. If such automaticity occurs in the human A-V valve or perhaps in the coronary sinus, it might represent a source of the electrical activity found in these two patients.
Effects of Cycle Length on Atrial Vulnerability

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SUMMARY The effect of cycle length on atrial vulnerability was studied in 14 patients manifesting reproducible repetitive atrial firing during atrial extra-stimulus (A2) testing. Repetitive atrial firing was defined as the occurrence of two or more premature atrial responses with return cycle (A1–A2) of 250 msec or less and subsequent mean cycle length of 300 msec or less, following A1. The zone of repetitive atrial firing could be defined in terms of its longest and shortest A1–A2 coupling intervals. Each patient was tested at a long cycle length (CL1) (mean 884 msec) and a short cycle length (CL2) (mean 557 msec). CL1 was sinus rhythm, and CL2, an atrial paced rhythm. Repetitive atrial firing occurred in two patients at CL1, and in all patients at CL2. Of the former two patients (group 2), the zone of repetitive atrial firing was markedly widened in one at CL1 due to a shortening of atrial functional refractory period (FRP) at CL2. In the other, zone of repetitive atrial firing could not be totally defined due to induction of sustained atrial flutter preventing definition of atrial FRP. The occurrence of repetitive atrial firing at only CL2 in 12 patients (group 1) reflected: 1) a shortening of atrial FRP from 294 ± 11 msec at CL1 to 242 ± 10 msec at CL2 (mean ± SEM; P < 0.01), allowing delivery of A2 at shorter coupling intervals (9); 2) the new occurrence of repetitive atrial firing at A1–A2 coupling intervals achievable at both cycle lengths (1); or 3) both effects (2).

In conclusion, decrease of cycle length potentiated atrial vulnerability. This demonstration implies that atrial pacing could potentiate occurrence of paroxysmal atrial fibrillation or flutter.

NONUNIFORM RECOVERY OF VENTRICULAR MUSCLE (dispersal of ventricular refractoriness) predisposes to ventricular fibrillation. Classically, cycle length has been said to affect uniformity of refractoriness, long cycle lengths causing variation in ventricular refractory periods, and short cycle lengths producing more uniform repolarization.1 It has been reported that long cycle lengths predispose to ventricular ectopic activity and ventricular fibrillation.2,4

It has recently been suggested that slow atrial rates predispose to atrial ectopic activity by a similar mechanism.4 If this hypothesis were true, then one would expect shortening of atrial cycle length to decrease atrial vulnerability to atrial fibrillation. In the present study we examine the effects of atrial cycle length on the phenomenon of atrial extra-stimulus induced atrial repetitive firing. The results demonstrated that decreasing cycle lengths predisposed the atrium...
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