Retrograde concealed conduction in the atrioventricular node: differential manifestations related to level of intranodal penetration

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ABSTRACT Although concealed conduction in the atrioventricular node (AVN) has been the focus of numerous experimental and clinical studies, little is known about the differential effects on AVN functional behavior of prior concealed retrograde impulse penetration alone vs prior anterograde or dual AVN activation. This study was undertaken specifically to investigate this aspect of human AVN physiology in patients without intact ventriculoatrial conduction to provide a model with which to analyze concealed retrograde AVN conduction. In 13 such patients AVN behavior during a subsequent anterograde input was characterized for each of three different manners of prior AVN activation: anterograde alone (method I), simultaneous anterograde and retrograde excitation (method II), or equivalently timed retrograde concealed AVN penetration alone (method III). In all patients evidence for retrograde AVN concealment was documented by longer anterograde conduction in the AVN during method III than that observed when the retrograde impulse was omitted (method IV). Furthermore, in these patients lacking intact ventriculoatrial conduction, anterograde conduction in the AVN was facilitated and refactoriness was decreased during dual excitation vs anterograde activation alone (method II vs method I). However, when each of methods I and II were compared with concealed retrograde AVN penetration alone (method III), two patterns of AVN responses were observed: In 11 of 13 patients (group A) conduction in the AVN was “worse” and refactoriness was greater both in methods I and II compared with method III; in the remaining two patients (group B) the opposite relationships were observed. The results suggest that among patients with no ventriculoatrial AVN conduction there exist at least two subpopulations whose AVN functional characteristics differ markedly when the effects of anterograde and dual excitation are compared with equivalently timed concealed retrograde activation alone. These findings may be related to differences in level of retrograde AVN concealment in groups A and B, which can be unmasked by the outlined pacing techniques.


CONCEALED CONDUCTION in the atrioventricular node (AVN) is an electrophysiologic phenomenon that has continued to stimulate considerable experimental and clinical investigation ever since its classic demonstration 60 years ago.1–13 Among the various manifestations of intranodal concealment of impulses, the effects of retrograde concealment in the AVN on subsequent anterograde conduction have been of special interest to both electrocardiographers and physiologists. From the data available it is apparent that the consequences of retrograde concealed conduction on subsequent anterograde impulse propagation may vary depending on whether (1) the AVN is penetrated by an isolated retrograde impulse or (2) the invading retrograde impulse is associated with concomitant anterograde intranodal penetration, resulting in dual AVN excitation. Both particular settings of concealed retrograde conduction have been extensively investigated.2,4–11,13

To our knowledge, however, there exists no study in animals or human beings that systematically compares within the same subject the AVN effects of concealed retrograde conduction caused by a solitary blocked impulse with the effects of dual excitation. This investigation was undertaken specifically to address this issue as well as to explore in detail the actual magnitude of alteration in anterograde AVN conduction and refactoriness engendered by a retrogradely blocked impulse. Our electrophysiologic analysis was accom-
plished with pacing protocols that permitted direct comparison of the effects of identically timed solitary vs simultaneous anterograde and (concealed) retrograde impulses on subsequent anterograde AVN conduction. We report new findings in this interesting area of human cardiac physiology and attempt to provide a unifying explanation for the observed phenomena.

Methods

Intracardiac electrophysiologic studies were performed as previously described after the nature of the procedure was explained to the patient and informed consent was obtained. Cardioactive medications were withheld for at least five half-lives before the study.

This investigation was limited to patients lacking ventriculoatrial conduction, since retrograde concealed AVN conduction would neither be readily produced nor analyzed (with a sufficiently early subsequent anterograde input) in individuals with intact retrograde AVN conduction. Specifically, patients were included if and only if (1) ventriculoatrial conduction across the AVN was absent during incremental ventricular pacing and at long ventricular coupling intervals after an atrial drive, (2) there was no evidence of either accessory atrioventricular pathways or discontinuous AVN refractory period curves, (3) an atrial basic cycle length could be identified that permitted successful accomplishment of the pacing protocols described below, and (4) AVN conduction appeared stable, i.e., steady-state AVN conduction time during the atrial basic drive was reproducible to within ± 5 msec. Data from 13 consecutive patients satisfying these inclusion criteria form the basis of this report. Clinical information as well as baseline electrocardiographic and electrophysiologic data are listed in table 1.

In each patient, assessment of retrograde concealed conduction as compared with other types of AVN activation was carried out by means of five different pacing protocols, which are depicted schematically in figure 1. Each pacing method consisted of an 8 beat basic atrial drive (S1) at constant cycle length, with an additional ninth paced beat (S1') originating from right atrium alone (method I), right atrium and right ventricle simultaneously (method II), or right ventricle alone (methods III and V), or omitted altogether (method IV). S1S1' was always programmed to equal S1S2. The diastolic interval following S1' was scanned with an atrial extrastimulus beginning at the longest possible S1S2 interval (i.e., such that S2 was not preempted by a sinus escape beat), which was then decreased in 10 msec decrements until the AVN effective (or atrial relative) refractory period was encountered. For any given S1S2 interval the same S2 was used with each of pacing methods I through IV (the order of which was varied in all cases). Stated differently, for any given S1 the same S1S2 interval (which equals S1S1' + S1'S2) was tested with methods I through IV (figure 1). S2 was omitted in method V, a pacing technique that was used at least once in all patients both to exclude "return" of ventriculoatrial conduc-

### Table 1

Baseline clinical, electrocardiographic, and electrophysiologic data

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age (yr)/sex</th>
<th>Structural heart disease</th>
<th>Reason for study</th>
<th>Sinus CL</th>
<th>PR</th>
<th>QRS</th>
<th>AH</th>
<th>HV</th>
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CL = cycle length; AT = atrial tachycardia; VF = ventricular fibrillation; VT-NS = nonsustained ventricular tachycardia; VT-Sust. = sustained ventricular tachycardia.

*aElectrocardiographic and electrophysiologic data are expressed in milliseconds.

*bNonspecific intraventricular conduction delay.

*cRight bundle branch block.
tion occurring in association with the atrial drive (but not apparent during ventricular pacing) and to rule out intranodal reciprocation in instances when the $S_2$ impulse conducted anterogradely across the AVN during method III despite having retrogradely blocked with either method I or II.

The basic atrial cycle length that was chosen in a given patient had to satisfy the following two requirements: (1) during methods II, III, and V the $S_1'$ stimulus consistently captured the ventricle and (2) during both methods I and II at least one $S_1'/S_2$ interval existed such that $S_2$ preempted a sinus escape beat and yet also fell outside the atrial relative refractory period (following $S_1'$).

**Definitions**

Anterograde AVN conduction time, in association with $S_1$, $S_1'$, or $S_2$, was determined indirectly by measurement of the SH interval (onset of stimulus artifact to onset of His bundle deflection). Since the atrial relative refractory period was deliberately avoided, changes in SH values for any given patient should have paralleled differences in anterograde AVN conduction time.

AVN effective refractory period (ERP) for methods I through III is the longest $S_1'S_2$ coupling interval for each respective pacing method so that $S_2$ failed to conduct completely across the AVN.

Atrial functional refractory period for methods I and II is the shortest attainable $A_1'A_2$ interval, for any $S_1'S_2$ coupling interval, during methods I and II.

**Statistical analysis.** Except for actual measured values (and ranges), all data are expressed as mean ± SD. Statistical comparisons were made with a paired t test, with significance defined at the 5% level.

### TABLE 2

**Electrophysiologic measurements of AVN conduction and refractoriness**

<table>
<thead>
<tr>
<th>Patient</th>
<th>$S_1'$ $S_2$</th>
<th>$S_1'H_1$</th>
<th>$S_2'H_2$ at longest comparable or attainable $S_1'S_2$ assoc. with AVN cond. of $S_2$ in M-IV</th>
<th>$S_2'H_2$ at shortest comparable $S_1'S_2$ for methods II–IV</th>
<th>AVN ERP</th>
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</table>

All values are expressed in milliseconds.

**cond.** = conduction; **M** = method of pacing (as described in text); **AFRP** = atrial functional refractory period.

*Since $S_1'$ was omitted in method IV, each value in this column corresponds to an $S_1'S_2$ interval identical to that used with methods I through III (see figure 1).*

*Values not shown if $S_1'S_2$ coupling interval is the same as that corresponding to $S_1'H_2$ values already tabulated in columns immediately to the left.*

*Values not shown if $S_1'S_2$ coupling interval is the same as that corresponding to $S_2'H_2$ values tabulated in columns immediately to the left (patients 1 to 5 and 12) or if no shortest $S_1'S_2$ interval existed such that $S_2$ conducted anterogradely across the AVN in each of methods II to IV (patients 11 and 13).*

*Sinus escape beat precluded use of longer $S_1'S_2$ test interval.*

**Results**

Complete electrophysiologic assessment was performed in every patient; however, only data pertinent to this report will be presented. In any given patient the range of basic cycle lengths that satisfied the criteria outlined above varied by no more than 50 to 100 msec. The actual basic ($S_1'S_1'$) cycle lengths used as well as the corresponding $S_1'H_1$ values are listed in table 2. For comparing methods I through IV, $S_2$ scanned fully a 49 ± 37 msec (range 20 to 140) interval extending beyond $S_1'$. It should be mentioned that retrograde His bundle deflections were not seen after any $V_1'$ electrogram. Moreover, in methods II, III, and V the interval from last anterogradely activated $V_1$ to onset of pacing stimulus $S_1'$ (which induced the retrogradely traveling impulse $V_1'$) represented 61 ± 7% (range 51% to 74%) of the $S_1'S_1'$ (or $V_1'V_r$) cycle length. Thus it is unlikely that functional His-Purkinje delays attended retrograde propagation of the $V_1'$ impulse.

Evidence for concealed retrograde penetration of the AVN by the $V_1'$ impulse and magnitude of change in subsequent anterograde AVN conduction. Since by study design $S_1'$ was omitted in method IV, the latter served as a control for method III. Comparison of $S_2'H_2$ in meth-

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od III vs method IV therefore permitted quantification of the change in AVN conduction time (of the S₂ impulse) caused by V₃' at each S₁'S₂ (or corresponding S₁S₂) coupling interval (table 2). In 12 patients (Nos. 1 to 12) S₂H₂ with method III exceeded that obtained with method IV by 64 ± 40 msec (range 15 to 150, 42 ± 29% increase; p < .0001) at the longest and by 77 ± 49 msec (range 20 to 160, 47 ± 30% increase; p < .0002) at the shortest comparable S₁'S₂ coupling intervals. Anterograde AVN block was observed in the remaining patient (No. 13) at all S₁'S₂ intervals used in method III, whereas the S₂ impulses traversed the AVN with method IV (table 2). Thus, in all patients it was possible to document both the occurrence of concealed retrograde AVN penetration and the magnitude of altered subsequent AVN conduction engendered by the V₃' impulse.

Anterograde AVN conduction after dual excitation from anterograde and retrograde impulses (method II) vs concealed retrograde impulse penetration alone (method III). Overall, S₂H₂ with method II was longer than the value obtained with method III by 18 ± 17 msec (range −5 to +60; p < .005) at the longest and by 34 ± 47 msec (range −85 to +65; p < .02) at the shortest comparable S₁'S₂ coupling intervals. Inspection of table 2, however, reveals that in 10 patients (Nos. 1 to 10), S₂H₂ during method II exceeded (or equaled, as in patient 5) the corresponding value measured in method III by 21 ± 16 msec (range 0 to 60, 9 ± 6% increase; p < .002) at the longest and by 46 ± 26 msec (range 10 to 65, 18 ± 9% increase; p < .002) at the shortest comparable coupling intervals (figure 2). Additionally, in patient 11 the S₂ impulse blocked in the AVN with method II even at the longest attainable S₁'S₂ coupling interval, whereas successful anterograde AVN propagation was observed with method III (table 2).

Therefore, in 11 of the 13 patients studied (Nos. 1 to 11; designated group A in table 2), anterograde AVN conduction was “worse” in method II than in method III. In only two patients (Nos. 12 and 13; group B in table 2) did successful AVN propagation of the S₂ impulse take longer or fail to occur with method III when compared with method II (figure 3).

Anterograde AVN conduction after anterograde excitation alone (method I) vs equivalently timed concealed retrograde impulse penetration alone (method III). A similar divergence in behavior between groups A and B became apparent when AVN conduction after an anterograde impulse (method I) was compared with that occurring in the wake of an equivalently timed concealed retrograde impulse (method III). Specifically, in all of patients 1 to 9 from group A, S₂H₂ with method I exceeded the value measured in method III by 39 ± 19 msec (range 10 to 70, 17 ± 6% increase; p < .001) at the longest and by 65 ± 31 msec (range 15 to 110, 29

![FIGURE 2](attachment:image.png)

**FIGURE 2.** Comparison of AVN conduction with methods I through IV at shortest comparable coupling interval in a group A patient (No. 1). Tracings from top to bottom are electrocardiogram lead 2, high right atrial (HRA) and His bundle (HB) electrogams, and time line (T). Basic atrial cycle length (S₁S₁') and S₁S₂ interval are both 500 msec. Solid arrows designate the paced atrial beats S₁ or S₂. Unfilled arrows and small black arrows designate atrial and ventricular sites of pacing, respectively, during the S₁' beat. Note identical S₁S₂ coupling interval of 270 msec (corresponding to S₁S₂ interval of 770 msec) in all panels. The index of AVN conduction time associated with the S₂ impulse (S₂H₂) is longer in panel A, in which there is an S₁' of atrial origin alone (method I), than in panel B, which depicts dual AVN excitation (simultaneous A₁' and V₁') during S₁' (method II). In turn, S₂H₂ is even shorter in panel C (method III), which shows a retrograde impulse (V₁') alone before introduction of S₂. However, when S₁' is completely omitted while S₁S₂ is unchanged (method IV), as in panel D, S₂H₂ is shortest, being even less than S₁H₁ (180 msec, as labeled in panel A). The relatively longer S₂H₂ value in panel C is therefore evidence for concealed retrograde penetration of the AVN by the V₁' impulse during method III (panel C). S = stimulus artifact; A = atrium; H = His bundle; V = ventricle.
FIGURE 3. Comparison of AVN conduction with methods I through IV at shortest comparable coupling interval in a group B patient (No. 12). Format as in figure 2 except that surface ECG lead is \( V_1 \), basic cycle length is 600 msec, and \( S_1/S_2 \) coupling interval in all panels is 390 msec (corresponding to \( S_1/S_2 \) of 990 msec). In this patient, although \( S_1H_2 \) is still shorter in method II (265 msec, panel B) than in method I (285 msec, panel A), \( S_1H_2 \) is longest with method III (350 msec, panel C). That \( S_1H_2 \) is shortest in method IV (190 msec, panel D) at an identical \( S_1/S_2 \) interval gives evidence of concealed retrograde AVN conduction occurring with method III. Abbreviations as in figure 2.

Another difference between groups A and B emerged when the magnitude of increase in \( S_1H_2 \) with method III from longest to shortest \( S_1/S_2 \) coupling intervals (for values shown in table 2) were compared. Thus there was a 16 ± 18 msec (maximum 55) increase in \( S_1H_2 \) in patients 1 to 10 from group A vs a larger 95 msec corresponding increase in patient 12 from group B.

Anterograde AVN conduction after anterograde activation alone (method I) vs dual excitation from anterograde and retrograde impulses (method II). Disparate responses between groups A and B were not apparent when findings with methods I and II were contrasted. In all 13 patients anterograde AVN conduction of the \( S_1 \) impulse with method II was “better” (or no different in patient 11) than with method I. Excluding patients 10 and 11 (in whom AVN block of A2 occurred with method I), \( S_1H_2 \) in method I exceeded the value of that parameter measured in method II by 16 ± 5 msec (range 5 to 45, 6 ± 5% increase; \( p < .005 \)) at the longest and by 24 ± 20 msec (range 5 to 55, 8 ± 6% increase; \( p < .002 \)) at the shortest comparable \( S_1/S_2 \) interval (table 2 and figures 2 and 3). In patient 13, at the shortest coupling interval, \( S_1H_2 \) was identical with methods I and II. Finally, at the longest attainable \( S_1/S_2 \) interval, AVN block was observed in patient 10 during method I but not during method II.

AVN refractoriness during methods I, II, and III. In patients 1 to 5, atrial functional refractory period exceeded and therefore prevented attainment of AVN ERP during methods I and II so that a comparison with method III was not possible. Overall, in the remaining patients, AVN ERP with method II was less than (or equal to, in patients 11 and 13) the value of that parameter with method I by 33 ± 34 msec (6 ± 6% decrease; \( p < .02 \)) (table 2 and figure 4, A, B, and D). On the other hand, AVN ERP with method II exceeded (or equaled, in patients 6 and 8) the value obtained with method III by 21 ± 71 msec, a statistically insignificant change.

When the differential effects of method II vs method III on AVN refractoriness were analyzed specifically according to patient group, however, two distinct behaviors were observed (table 2). Thus, in patients 6 to 11 from group A, AVN ERP with method III was 47 ± 46 msec less than that obtained with method II \( (p < .03; \) figure 4, D to G). In contrast, AVN ERP as determined with method III in both group B patients considerably exceeded (by 90 and 40 msec, respectively) the value measured with method II (figure 5).

A dichotomy in the patients’ responses was also observed with respect to the differential effects of methods I and III on AVN refractoriness. Thus, in all of patients 6 to 11 from group A, AVN ERP with method I exceeded the corresponding value with method III by 82 ± 72 msec (range 10 to 150; \( p < .02 \))
(figure 4, A and C). On the other hand, in both group B patients AVN ERP with method I was 40 msec shorter when compared with method III (figure 5). It should be emphasized that method V was used in all patients from group A who had a shorter AVN ERP with method III than with method I or II to exclude the possible occurrence of an AVN echo beat (induced by V',) which potentially could have simulated true anterograde AVN conduction of the S2 impulse in method III (figure 4, H).

**Discussion**

Based on their responses to the pacing protocols used in this investigation, it was possible to divide the patients studied into two basic categories. In the majority (group A) AVN conduction was “worse” and refractoriness was greater after either anterograde activation alone (method I) or dual excitation (method II) than was the case after an equivalently timed concealed retrograde impulse alone (method III). The remaining individuals (group B) manifested a fundamentally different pattern in which S1H2 and AVN ERP were more prolonged with method III than with either method I or II. In all patients, however, AVN conduction of the S2 impulse was more rapid (or at least as fast) and AVN ERP was shorter (or the same) during method II vs method I.

To our knowledge, a comparable systematic analysis of the differential effects of the various types of AVN activation that we have investigated does not exist in the experimental literature. Previously we demonstrated that in 23 of 26 patients studied, intrano-
dal collision of anterograde and retrograde impulses improved conduction and decreased refractoriness compared with prior anterograde AVN activation alone. The present results are therefore consistent with that general finding, specifically with respect to the situation of dual nodal excitation involving a retrograde impulse that by itself penetrates but fails to completely traverse the AVN.

On the other hand, we also observed in the earlier study that when the equivalent of method III was attempted in seven of 13 patients in whom a programmed retrograde impulse (Vs, the counterpart of V') blocked in the AVN, only a response pattern typical of that in our group B was seen. Thus, had the present systematic investigation not been carried out, the existence of the AVN behavior exhibited by group A would not have been appreciated. Variation in patient sampling most probably accounts for the discrepancy between our two reports with respect to the actual proportion of patients demonstrating behavior patterns typical of group A or B. It should also be pointed out that, in contrast to our previous communication, the present study was undertaken specifically to investigate concealed retrograde AVN conduction and therefore involved nearly twice as many patients (13 vs seven), used additional relevant pacing protocols (i.e., methods IV and V), and included analyses of additional electrophysiologic parameters.

Postulated electrophysiologic mechanisms. Any unifying mechanism proposed to explain our findings must also take into account the possibilities that (1) in method III the retrograde V', impulse may not block at the same AVN location in every patient and (2) in method II the AVN level of impulse collision (if the latter indeed occurs) conceivably could be different from the level of block of the retrograde impulse in method III. Whether or not the two AVN impulses actually collide, of course, will depend on a combination of factors, including level of block of the concealed retrograde impulse, relative timing of anterograde and retrograde inputs, and differences in their respective conduction velocities.

Bearing these considerations in mind, one can satisfactorily explain the different constellation of responses in groups A and B by postulating a relatively more shallow extent of retrograde AVN penetration by V' in group A with method III, i.e., a more distal level of block within the AVN, compared with group B. According to this assumption, during method III the proximal portion of the AVN would be relatively less penetrated and, hence, more fully recovered in group A vs group B. Furthermore, with method II actual impulse collision need not occur in group A, since advancement of the anterograde wavefront might be arrested some short time after occurrence of retrograde block — at either the same (presumed distal) level or perhaps somewhat more proximally because of local electrotonic inhibition. On the other hand, with the potentially deeper (and, hence, more proximal) extent of retrograde impulse penetration of the AVN in group B, true collision of impulses might occur.

These differences in "trajectory" or "history" of the concealed V' impulse subsequent to its retrograde invasion of the AVN could have the following consequences, which are compatible with our findings and which will be described first with respect to group A: Compared with anterograde AVN excitation by the A,' impulse alone, retrograde activation by the concealed

FIGURE 5. Differences in AVN refractoriness for methods I through III in a group B patient (No 12). Same format as in figure 3. Basic cycle length is 600 msec. At an S'/S2 coupling interval of 310 msec (with corresponding S'/S1 of 910 msec), S2 fails well within the ERP of the AVN during both methods I (panel A) and III (panel B) (350 and 390 msec, respectively, as indicated in table 2). With dual excitation at the same coupling interval, however, anterograde AVN conduction of the S2 impulse is now successful (panel C), reflecting decreased refractoriness associated with method II. AVN ERP with method II is encountered only when S'/S2 is decreased by an additional 10 msec (panel D). Abbreviations as in figure 2.
V', impulse alone would have the combined effects of distal AVN preexcitation and "peeling back" of refractoriness on the one hand and the granting of additional time for proximal AVN recovery (due to absence of proximal activation) on the other. This might well lead to a shorter ERP and S2H2 delays in method III vs method I. Occurrence of distal AVN preexcitation with method II would also result in more distal recovery and, hence, faster anterograde conduction and less refractoriness than would obtain with method I. Compared with method III, however, method II (with or without occurrence of true collision) would be associated with relatively greater S1H2 delays and prolonged ERP due to superimposed proximal AVN activation (and consequent delayed recovery) engendered by the A,' impulse.

On the other hand, in group B farther retrograde penetration by the concealed V', impulse to more proximal AVN regions during method III might create a situation in which the favorable effect of distal preexcitation would be exactly counterbalanced, or even outweighed, by delayed (relative to method I) proximal activation and recovery. The net overall effect, then, would be one of equivalent or greater refractoriness (and magnitude of S1H2 delays) with method III vs method I. With dual excitation, however, only the distal (conduction-enhancing) but not the proximal (conduction-retarding) effects of the V', impulse would be preserved because of a presumed collision of impulses. This in turn would result in less anterograde AVN refractoriness and conduction delay compared not only with method I but also with method III. In other words, the intranodal collision in method II would permit a "peeling back" of distal refractoriness (vs method I) and, concomitantly, would prevent proximal concealment by the V', impulse that would otherwise have occurred with method III (figure 5).

Besides being consistent with the observed phenomena, our hypothesis that retrograde AVN penetration by the V', impulse was deeper (i.e., more proximal) in group B is supported by the relatively greater magnitude of increase with method III in S2H2 values (shown in table 2) from longest to shortest S1'S2 coupling intervals in patient 12 compared with the smaller corresponding S2H2 increment in patients 1 to 10 from group A. It should also be mentioned that a summation-like phenomenon (somehow "carrying over" from S1' to S2') might have contributed to the favorable alterations in anterograde AVN conduction and refractoriness with method II, particularly in group B; however, our study was not designed specifically to evaluate such a potential mechanism.

Postulation of different levels of retrograde AVN block is certainly reasonable in light of experimental parallels in the anterograde direction. Nonetheless, the available data do not permit us to exclude categorically an alternative interpretation of the results, i.e., that retrograde AVN propagation of the V', impulse before blocking was slower in group B than in group A; nevertheless block occurred at the same level in all patients. According to this scenario, subsequent anterograde AVN conduction would be "worse" and refractoriness more prolonged in group B during method III vs method I or II because of delayed arrival (relative to group A) of the V', impulse at the putative universal level of retrograde block.

The two interpretations need not be mutually exclusive, however, since block might occur at more than one level and retrograde conduction velocity of the V', impulse before blocking might differ from patient to patient. Interestingly, by either postulated mechanism, true collision of anterograde and retrograde impulses in method II is implied in group B.

Unlikely alternative explanations. Although the mechanisms described above can account for the different group A and group B behaviors observed, it is important to examine and exclude the possibilities that methodologic variables or particular anterograde AVN characteristics may have contributed to the results. For example, patient-to-patient differences in actual timing of retrograde (V',) AVN input relative to subsequent anterograde (S2) input, even if minor in degree, conceivably could have given rise to the differences between groups A and B. Specifically, for any given S1'S2 coupling interval, delayed (vs earlier) retrograde AVN input of the concealed V', impulse (subsequent to retrograde His-Purkinje activation) would permit less time for AVN recovery before anterograde activation by the test impulse (S2). As a consequence, AVN refractoriness might be relatively greater and conduction time (of the S2 impulse) longer than would obtain with lesser temporal proximity of retrograde nodal input to the onset of S2. The data in tables 1 and 2, however, suggest that such a hypothetical consideration was probably not an important factor in our study. This conclusion may be drawn by using the HV interval as an indirect (lower-limit) estimate of retrograde conduction time across an unstressed His-Purkinje system (SH interval). According to this line of reasoning, it is apparent that retrograde AVN input of the V', impulse must have been relatively delayed in patients 3 and 4, and especially in Nos. 9 (with coexistent right bundle branch block) and 10 (with marked HV prolongation); yet, in all of these patients AVN
conduction was “better” and ERP (when measureable) was shorter with method III than with either method I or II. Conversely, patient 12 demonstrated group B behavior despite having the shortest HV interval of all the patients studied.

It is also unlikely that differences in anterograde AVN functional properties could have accounted for the disparate responses in groups A and B, since AH intervals during sinus rhythm and His bundle and AVN ERPs at comparable S1, S2 cycle lengths were of similar magnitude in the two groups. Finally, the possibility that patients 12 and 13 merely demonstrated experimentally aberrant responses can be dismissed on the basis of similar group B behavior that we observed previously in other patients.13

Electrophysiologic implications. Conceived conduction is a concept that can help to explain various common and unusual electrocardiographic patterns.2, 5, 7, 11, 24 The present investigation elucidates one facet of this phenomenon in a specific group of patients most likely to demonstrate concealed penetration of retrograde impulses into the AVN. Our results have several potential ramifications, although these cannot be proved conclusively from the results of this study. For example, whereas previous clinical and experimental studies of concealed anterograde impulse propagation in the AVN have either implicated or demonstrated the existence of multiple levels of AVN block,2, 5, 22-25 our findings suggest that cardiac impulses in humans may encounter an analogous situation in the retrograde direction as well. Moreover, the type of electrophysiologic analysis we have used might serve as one method for unmasking different levels of retrograde AVN block in patients with absent ventriculoatrial conduction. Such a technique could be of value in light of the intriguing possibility that retrograde block at one particular level in the AVN may be more (or less) likely to occur or be reversed with a given pharmacologic intervention compared with block at another level.

Besides being of theoretical interest, these considerations might have relevance in determining which patients are more likely to have retrograde AVN conduction restored, e.g., in the presence of either diminished vagal or enhanced sympathetic tone. Indeed, dynamic reversibility of retrograde AVN block in patients lacking intact ventriculoatrial conduction has already been demonstrated26 and could well account for the unexpected occurrence in such individuals of “endless loop” tachycardia (in the setting of DDD pacemakers), antidromic reciprocating tachycardia (in association with accessory pathways), and even AVN reentry.27

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References
Retrograde concealed conduction in the atrioventricular node: differential manifestations related to level of intranodal penetration.

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