Surface Potentials From the Region of the Atrioventricular Node and Their Relation to Dual Pathway Electrophysiology

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Background—Clinical applications of the principles of dual atrioventricular nodal (AVN) electrophysiology in the treatment of AVN reentrant tachycardias rely on empirical findings, such as discontinued conduction curves or the presence of specific catheter-recorded signals. However, neither the shape of the conduction curve nor the surface electrograms have been validated as functionally related to the presence of slow or fast wavefronts.

Methods and Results—We performed in vitro studies using 10 rabbit atrial-AVN preparations. A bipolar roving electrode was used to explore the endocardial surface of the triangle of Koch during programmed electrical stimulation. Microelectrodes were impaled in AVN cells to correlate surface and intracellular responses. In 7 preparations, a specific area near the compact cell region produced surface electrograms that were dissociated in 2 distinct components, with progressive shortening of prematurity. Similar dissociation was demonstrated during Wenckebach periodicity and increased vagal tone. Cellular recordings supported the presence of early (“fast”) and late (“slow”) wavefronts, with different refractory properties. Although the fast-slow transition was a basis for discontinued propagation, the AVN conduction curves were smooth in the majority of cases.

Conclusions—Exploration of the triangle of Koch during programmed pacing reveals the presence of dual-wavefront surface potentials. Clinical confirmation of these AVN potentials could provide a new, sensitive tool in defining dual AVN electrophysiology. (Circulation. 2000;101:2110-2117.)

Key Words: atrioventricular node ■ conduction ■ dual pathways ■ potentials

The concept of dual atrioventricular node (AVN) physiology was constructed on the foundation of deductions made from clinical electrocardiograms,1 experimental studies using His bundle recordings,2 and from transmembrane potentials of cells in the atrioventricular (AV) junction.3 However, direct extracellular recordings from the AVN have not been correlated with various functional properties of nodal electrophysiology.

On the basis of the early results obtained with microelectrodes, it has been suggested that recordings of electrical activity with surface electrodes should fail to show rapid transients.4 Indeed, several investigators provided evidence that slow, low-amplitude waves could be recorded between atrial and His bundle potentials.5-7 In the process of developing radiofrequency catheter ablation for the termination and cure of AVN reentrant tachycardia, potentials recorded in the inferior-posterior portion of Koch’s triangle, both in animals8 and in man,5,6 served as the targets for successful ablation of slow AVN input. However, it remains unclear if surface potentials from the AVN can carry the distinctive signatures of “slow” and “fast” wavefronts that form the foundation of dual AVN physiology.

In the present study, we made in vitro observations in rabbit AV junctions; these observations suggest that extracellular recordings can be directly associated with dual AVN physiology. These findings provide a new investigational tool and may help to clarify the functional components necessary for the occurrence of smooth or discontinuous conduction curves.

Methods

Rabbit AVN Preparations In Vitro
We used 10 New Zealand White rabbit atrial-AVN preparations, as previously described.8 Briefly, after pentobarbital anesthesia, the heart was removed, and all ventricular tissues were discarded. The preparation was mounted in a thermostatically controlled superfusion system (35.5°C), and it contained the IAS, crista terminalis, and the whole triangle of Koch enclosed between the coronary sinus, the tendon of Todaro, and the septal leaflet of the tricuspid valve (Figure 1A). Tyrode solution (in mmol/L: NaCl 128.5, KCl 4.7, CaCl2 1.3, MgCl2 1.05, NaHCO3 25, NaH2PO4 1.19, and glucose 11.1) was saturated with 95% O2/5% CO2 (pH, 7.3 to 7.4) and delivered at 35 mL/min.

Electrical Recordings and Stimulation
Custom-made, 250-µm Ag-AgCl Teflon-isolated bipolar electrodes spaced 0.2 to 0.5 mm apart recorded atrial (at crista terminalis and IAS) and His bundle electrograms. A bipolar platinum-iridium electrode of similar design was used for pacing on the septal side.10,11 The AVN conduction curve, A2H2 conduction times versus A1A2...
prematurities, was generated by periodically interrupting the basic drive (A1A2 of 300 ms) with progressively shorter prematurity stimuli until the occurrence of AVN block. Time intervals were determined off-line with 1 ms resolution. Incremental pacing was implemented in 4 preparations to induce Wenckebach periodicity.

A roving bipolar electrode (125-μm wires spaced at 0.2 mm) explored the endocardial surface of the AVN in the triangle of Koch in search of AVNP. The latter were defined as responses occurring between the atrial and His electrograms that were functionally related to propagation through the AVN. They were found in an oblique patch adjacent to the AV ring located 2 to 3 mm inferior to the septum (Figure 1A). The AVNP had an average amplitude of 0.9±0.5 mV and a maximum derivative of 0.06 to 0.25 V/s. They differed strongly from the atrial or His bundle electrograms (Figure 1B). These low-amplitude responses were filtered (5 to 500 Hz or, in several experiments, 1 to 1000 Hz). In all Figures except Figure 1, AVNP traces were enlarged to show details.

We used standard glass microelectrodes to record APs from single AVN cells. Anatomical location, AP morphology and amplitude (65±4 mV; range, 59 to 72 mV), and dAP/dt (<10 V/s), as well as cycle-length dependency were used to identify cellular signals originating from the vicinity of the compact cell region (CCR).4 We also recorded from distal nodal-His (NH) cells.

The Working Hypothesis

The simplified longitudinal cross-section in Figure 1C illustrates our working hypothesis. The CCR is electrically connected to an envelope of transitional cells and to deeper inferior nodal extensions. The latter 2 structures belong to the atrial part of the specialized axis.12 The transitional cell envelope and inferior nodal extensions are not sheath-isolated conduction cables in human and dog hearts,14 is located just after the penetration of the gap9 developed: no His bundle activity existed (block), and AVNP amplitude declined to just 40% (Figure 2C). They differed strongly from the atrial or His bundle electrograms (Figure 1B). These low-amplitude responses were filtered (5 to 500 Hz or, in several experiments, 1 to 1000 Hz). In all Figures except Figure 1, AVNP traces were enlarged to show details.

We assumed that the bilayer structure (shown in Figure 1C) supports 2 distinct wavefronts. An earlier (fast) wavefront propagates via the transitional cell envelope and, through a shortcut in the anterodistal CCR, reaches the penetrating bundle (white arrow). A later (slow) wavefront propagates via the deeper inferior nodal extensions and the CCR to reach the penetrating bundle (black arrow). Neither the fast nor the slow wavefront travels via isolated channels; they are both considered interactive functional entities. This model does not specify the connections between the AVN and the atrial inputs (crista terminalis and IAS).

Electrical recordings obtained from the endocardial surface-patch should reveal the “signatures” of the propagating wavefronts, provided that the wavefronts do not arrive simultaneously under the recording electrode (dots in Figure 1C). Assuming that the wavefronts have different functional properties, programmed electrical stimulation, incremental pacing, or autonomic influence can achieve a dissociation. We found support for this hypothesis in 7 of the studied preparations (70%).

Results

Surface Signals from the AVN Region and Their Dynamic Properties

Typical changes in the morphology of AVN potentials (AVNPs) during programmed stimulation are illustrated in Figure 2. The AVNP amplitude decreased in parallel with the shortening of A1A2 prematurity and reached 80% of control (Figure 2A) when A1A2 was 150 ms (Figure 2B). With A1A2 intervals between 150 and 119 ms, AVN conduction gap9 developed: no His bundle activity existed (block), and the AVNP amplitude declined to just 40% (Figure 2C). However, when the A1A2 was 118 ms (Figure 2D), conduc-

Figure 1. A, Drawing of major structures around the triangle of Koch (dotted lines) in a real rabbit heart, including septal leaflet of tricuspid valve (TrV), tendon of Todaro (tT), inferior vena cava (IVC), and coronary sinus (CS). Large dots indicate atrial and His bundle electrodes, and small dots, AVNP electrodes in proximity to CCR (black area). B, Typical atrial (crista terminalis [CrT] and IAS), AVNP, and His bundle signals. A1 and H1 indicate last basic beat, and A2 and H2, premature beat. C, Diagram showing bilayer AVN structure and illustrating working hypothesis. TE indicates envelope of transitional cells; INE, inferior nodal extension; CFB, central fibrous body; PB, penetrating bundle; white arrow, fast wavefront; and black arrow, slow wavefront.

tion was unexpectedly restored, and the AVNP revealed a distinct second component (arrow). For A1A2<118 ms (Figure 2, E through I), conduction was associated with this delayed second component, which reached near-normal (83%) amplitude. The earlier component deteriorated to small electrotonic spikes (arrowheads). Atrial echoes followed all A2 beats at these short prematurities, and up to 3 consecutive reentry loops were documented.

Figure 3A shows the superimposed AVNPs that followed beat A2 in the same experiment. Note that AVNP components occupied 2 distinct time domains. The early components were decremental, with shortening of prematurity toward an A1A2 of 118 ms. In contrast, robust late components were associated with restored conduction at an A1A2 of 118 ms. At prematurities close to 118 ms, both AVNP components were present (arrows).

Figure 3B shows the conduction curve A2H2 versus A1A2. In addition, we plotted the delay from A2 to the occurrence of the early or late components of the AVNP. Note that early AVNPs (for A1A2<150 ms) were inscribed ~50 ms after A2 and, therefore, the prematurity-dependent A2H2 delay in this A1A2 range was generated mostly between the AVNP and H2. In contrast, at the shortest A1A2, the delay between the
late AVNP and H2 remained near constant, so that most of the A2H2 conduction time was generated before the late AVNP.

Dissociation of the surface AVNP into 2 components in the course of programmed stimulation was evident in 7 of 10 preparations, and it occurred at atrial prematurities A1A2 from 160 to 100 ms. The conduction curves in these experiments were with a gap (n = 3), with a jump (n = 1), or smooth (n = 6).

Surface Signals From the AVN Region and Their Relation to Nodal Cellular Activity

To verify that the observed phenomena reflected the underlying AVN cellular responses, we performed experiments in which glass microelectrodes were impaled in N-type cells (in the CCR) close to the AVNP electrode (Figure 1C). One such impalement is illustrated in Figure 4. In this experiment, conduction gap was observed. Note that AVN block at an A1A2 of 165 or 150 ms (Figure 4, A and B) was associated with decremental early AVNP (arrowheads) and cellular action potentials (AP), suggesting that the available driving force was insufficient to produce full depolarization of this critical region. When A1A2 was shortened to 145 ms (Figure 4C), conduction was restored. This was accompanied by a disappearance of the early AVNP (arrowhead) and by the inscription of a delayed component with an increased amplitude (arrow) and a full action potential. At an A1A2 of 140 ms (Figure 4D), the AVNP delay reached 209 ms, and reentry was initiated.

Note that the reentrant beat produced an early AVNP component (Figure 4D, arrowhead), suggesting that the subsequent block was via the fast wavefront (similar to Figure 4A). Note further that the slow wavefront, illustrated in Figures 4C and 4D (arrows), was associated with larger His amplitudes.

In 3 hearts, double-component AVNPs were associated with smooth conduction curves. One such experiment is illustrated in Figure 5. In this experiment, APs were recorded from the compact cell region (CCR), which was ≈1 mm distal to the AVNP recording site. The AVNPs in response to the A2 beat were decremental (Figure 5, A through C), and a notch became evident for A1A2 < 125 ms (Figure 5, D through F, diagonal arrows). Subsequently, 2 distinct components of the AVNP were present (Figure 5, G through I, double arrows). The second one became dominant at the shortest prematurity. Two reentry events (Figure 5, J and K, curved arrows) were seen in this experiment.

The APs recorded from the single N-fiber downstream of the AVNP electrode exhibited progressive reductions in amplitudes.
amplitude and upstroke velocity that mirrored the changes in the earlier component of AVNP (Figure 5, A through G). The time interval between the fastest downstroke of AVNP and the maximal first derivative, dAP/dt max, of the cellular AP (thin lines) progressively increased from 11 to 33 ms (Figure 5, A through F). Based on a distance of 1 mm, the calculated apparent conduction velocity for the fast wavefront declined from 9.1 to 3 cm/s.

The transition between the 2 wavefronts could be traced in Figures 5D through 5F, where the approaching later wavefront produced electrotonic humps in the AP (vertical arrows). Only the end-tail portions of these humps are seen because their start is obscured by the earlier upstroke of the AP. At an A1A2 of 110 ms (Figure 5G), the transition from a predominantly fast to a predominantly slow wavefront is completed. At even shorter A1A2 intervals (Figure 5, H through K), cellular signals revealed only one late AP that followed the second component of the AVNP. In contrast to Figures 5A through 5F, the amplitude and dAP/dt of the AP increased, despite the shortening of A1A2 (Figure 5, G through K). Interestingly, the time interval between the second component of the AVNP and the upstroke of the AP (thin lines) decreased from 43 ms to 16, 15, and 13 ms, respectively, for Figures 5G through 5K. Thus the calculated apparent conduction velocity of the slow wavefront increased from 2.3 to 7.7 cm/s.

The conduction curve A2H2 versus A1A2 (Figure 5L) had no jump. However, the curve showing the delay (A2 to AP) in the arrival of the wavefronts at the impaled fiber exhibited a discontinuity of 44 ms at an A1A2 of 115 ms. This apparent discrepancy can be easily explained. At this prematurity (Figure 5F), the fast wavefront arrives at the impaled cell in 68 ms, but it reaches the His bundle in just 44 ms (Figure 5G), the slow wavefront arrives 22 ms later (A2 to AP = 112 ms), but it reaches the His bundle after an additional 15 ms, for a total A2H2 of 126 ms. In contrast, at an A1A2 of 110 ms (Figure 5G), the slow wavefront arrives 44 ms later (A2 to AP = 112 ms), but it reaches the His bundle in just an additional 27 ms, for a total A2H2 of 139 ms, which results in A2H2 = A2H2 of 13 ms.

On the basis of the above observations, we hypothesized that transition from the fast to slow wavefront takes place at prematurities where the decremental proximal driving force provided by the fast wavefront fails to depolarize the distal AV axis and is replaced by the later and stronger slow wavefront. In Figure 6, the APs were recorded from a distal NH cell (see Figure 1C). Note that a declining electrotonic transmission produced foot formations (Figure 6B, horizontal arrow) preceding the AP upstrokes at A1A2 intervals of 175, 170, and 165 ms. In addition, the AP upstrokes declined to a dAP/dt max of 15 V/s at an A1A2 of 165 ms (Figure 6B). At an A1A2 of 160 ms, the electrotonic hump did not reach the threshold for distal excitation. AVN block would have occurred if not for the arrival, although after a substantial delay (Figure 6B, *), of the slow wavefront. The slow wavefront was responsible for the inscription of the last 4 APs (A1A2 of 160, 155, 150, and 130 ms). Note that despite the shorter prematurities, the AP upstrokes were without preceding foot formations and they were faster, reaching a dAP/dt max of 22 V/s at an A1A2 of 130 ms. Thus, the slow wavefront provided a stronger driving force. However, its delayed arrival was responsible for the jump in the A2H2 conduction curve (Figure 6C, star). It is worth noticing that the transition to the slow wavefront was associated with an increase in the amplitude of the His electrogram (Figure 6B). This finding, which is similar to that in Figure 4, suggests that the His bundle was engaged in a different spatial fashion by the 2 wavefronts.

**Surface Signals from the AVN Region During Fast Atrial Rates or Increased Autonomic Tone**

Dissociation of the AVNP signals into early and late components was also observed during high-rate pacing. In Figure 7, a shortening of the cycle length from 160 to 100 ms resulted in stable Wenckebach paradigms (numbers in brackets). The early AVNP components (arrowheads) occurred 62 ms after the electrogram of the interatrial septum (IAS), whereas the later components (arrows) were inscribed at 75 to 77 ms or even later. Note that the low and high amplitude His electrograms were associated with the early and late AVNPs, respectively.

On the basis of these criteria, one can conclude that, in Figure 7A during 3:2 Wenckebach, the fast wavefront generated a full AVNP in conducted beat 2, but it produced only early electrotonic glitches in beats 1 (blocked) and 3 (conducted via the slow wavefront).

In Figure 7B at 5:3 conduction, beat 2 was conducted via the fast wavefront (arrowhead), whereas the slow wavefront...
produced the later AVNP components (arrows) in conducted beats 3 (176 ms after IAS) and 5 (75 ms after IAS).

In Figure 7C at 2:1 conduction, all AVNPs were inscribed 62 ms after the IAS electrogram (arrowheads), suggesting that they were generated by the earlier wavefront. In Figure 7D, the pattern was again 2:1; however, now conduction to the His bundle was supported by the slow wavefront (late AVNP components, arrow).

Finally, we determined that autonomic maneuvers produced a differential effect on the dual AVN electrophysiology, as reflected in the AVNP. In this study, brief bursts of subthreshold postganglionic vagal stimulation with an amplitude of 50 μA were delivered through the same electrode that recorded the AVNP. The observations shown in Figure 8 were made at prematurities of the fast-slow wavefront transition. At an A1A2 of 125 ms (Figure 8A), the decremental fast wavefront signature was still present (arrowhead), whereas at an A1A2 of 123 ms, it had faded away (Figure 8B, arrowhead) and the delayed AVNP component was inscribed at 156 ms (arrow). When returning back to an A1A2 of 125 ms (Figure 8C), we applied postganglionic vagal stimulation. The fast wavefront seen in Figure 8A was now fully abolished (arrowhead), and the AVNP of the slow wavefront (arrow) was inscribed after a jump in the delay to 177 ms. The direct vagal effect on the slow wavefront can be determined by comparing Figure 8B and Figure 8D, where at an A1A2 of 123 ms, the slow wavefront (arrow) was delayed by postganglionic vagal stimulation with only 22 ms (178 versus 156 ms). Thus, the AVNP revealed that in this experiment, minute shortening of atrial prematurity from 125 to 123 ms produced a block of the fast wavefront (Figure 8B). A similar result was obtained with vagal stimulation (Figure 8C). The slow wavefront was insensitive to this minute shortening of A1A2 (Figure 8, C and D) and was also less sensitive to vagal stimulation (Figure 8, B and D).

**Discussion**

A major finding in this study was the observation that electrical signals recorded from the endocardial surface of the triangle of Koch revealed the presence of distinct, separate

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**Figure 5.** Validation of functional dynamic relationship between surface AVNP and AVN cellular activity from CCR. A through K, gradual dissociation of both AVNP and AP into 2 components during progressive shortening of A1A2. Numbers, in milliseconds, are as in Figure 2. CrT indicates crista terminalis; thin vertical lines, interval between fastest downstroke of AVNP and dAP/dt; diagonal arrows in D through F, notch in AVNPs in response to A1A2<125 ms; vertical arrows in D through F, electrotonic humps in AP produced by approaching wavefront; double arrows in G through I, 2 distinct components of AVNP; and curved arrows in J and K, reentry events. In L, ● indicates AVN conduction curve, and △ and □, delays from A2 to subsequent AP. Calibration bar in A indicates AP=40 mV and dAP/dt=10V/s.
wavefronts that likely represent the fast and slow components of dual AVN electrophysiology. Simultaneous analysis of these AVN potentials and the nodal cellular responses indicated that the transition from fast to slow wavefront conduction could produce either smooth or discontinuous conduction curves.

**Origin of the AVNP**

On the basis of the location of the bipolar electrodes and of the cellular activity recorded with microelectrodes, it seemed that the AVNPs were generated by the excitation of fibers located in the vicinity of the CCR. A nonuniform anisotropy has been demonstrated in the superficial layers of the triangle of Koch. This produces a preferential conduction parallel to the tricuspid valve annulus. The delay in these layers was relatively short (typically 40 ms) and, in contrast to atrial-His delay, had minimal dependence on prematurity. According to our model (Figure 1C), fast pathway conduction occurred from the envelope of transitional cells via a relatively short transverse route in the anterior triangle of Koch that connects the septum with the penetrating bundle and is likely to transverse at least a portion of the CCR.

Our model (Figure 1) further assumed that slow pathway conduction originated in the deeper nodal layers that include the inferior-nodal extensions. The wavefront in this domain would propagate longitudinally through the inferior nodal extensions and the entire CCR. The functional difference between the fast and slow pathway domains may reflect preferential directional propagation across and along, respectively, the fibers that form the complex nodal architecture. Propagation in such a milieu can be either depressed or enhanced by the combined action of the branching strands as current load or source, respectively (“pull-push effects”). At present, it is not clear if and how the above factors determine the functional fast and slow wavefront domains.

**Fast and Slow Versus Early and Late Wavefronts**

The terms fast and slow were originally introduced to describe differences in the observed time intervals during common AVN reentrant tachycardia, not velocities. In fact, it has never been demonstrated that the wavefronts deserve their adjectives.

In determining propagation, one must first consider the time needed for the atrial wavefront(s) to arrive in and then to transverse the CCR. The subsequent transmission from the CCR into the penetrating bundle occurs between neighboring cells, nodal and NH, which have distinct morphological and electrical differences. Such electromorphological mismatches may serve as a functional barrier over which critical electrotonic transmission can take place. This principle has been applied to the AVN, and the so-called Rosenblueth barrier has been proposed to explain marginal conduction, such as at shorter prematurities or Wenckebach periodicity. The model in Figure 1 depicts such an entity as a (functional) cleft between the CCR and penetrating bundle.

Our results suggest that the fast wavefront arrived promptly in the CCR and was therefore dominant at long prematurities. As seen in Figures 5A through 5F, the cellular coupling interval in the CCR was very close to atrial prematurity A1A2. Therefore, progressive shortening of A1A2 produced decremental cellular responses, as expected from the refractory properties of the nodal cells. The reduced AP amplitude led to a progressive delay of the electrotonic transmission over the Rosenblueth barrier (Figure 6, foot formations) and eventually to failure (Figure 6, at an A1A2 of 160 ms). Thus, the earlier wavefront quickly reached the CCR but, with the shortening of A1A2, produced increment in the upstroke. Thus, the later wavefront was slow in reaching the CCR, but...
it resulted in a more robust driving force for subsequent prompt transmission to the His bundle.

The above analysis suggests that conduction velocity may not be constant along the entire pathway length of either wavefront. This, plus the unknown lengths of the particular portions of the 2 pathways, makes it impossible to precisely determine velocities.

**Dual AVN Physiology and the Discontinuity of Conduction**

One should consider 2 types of discontinuity in AVN conduction. Type 1 is the functional microdiscontinuity where the impulse “stops” proximal to a barrier and proceeds on the distal side after a distinct delay. This was illustrated by the foot formations in the NH cell (Figure 6). Type 2 is the discontinuity produced by the existence of, and the transition between, fast and slow wavefronts. Although the conduction along the AVN axis does not stop, a distinct delay may be observed between the times of arrival at the CCR of the failing fast wavefront and the approaching slow wavefront (Figures 2 through 6).

The A2H2 conduction curves could be smooth (Figure 5), have a jump (Figure 6), or contain a gap (Figure 3). The jump, and especially the gap, refers to the duality of AVN electrophysiology. The smooth conduction curve, however, can coexist with either of the above-described discontinuities. For example, the conduction curve in Figure 6C was smooth for all A1A2>160 ms, despite the presence of type 1 discontinuity. Similarly, a smooth A2H2 curve in Figure 5L was observed, despite the type 2 discontinuity seen in the AVNP/AP records. In the latter case, this was due to the equalizing effect of the sum of the proximal and distal delays for each wavefront at the A1A2 prematurity, at which the transition between the 2 wave fronts occurred. Only when an excessively long proximal delay of the slow wavefront existed was a jump-curve observed (Figure 6C).

Thus, although dual wavefront conduction is apparently a universal feature of the AVN, it cannot be reliably deduced from the shape of the conduction curve. Moreover, interaction between the 2 wavefronts may further modulate the discontinuous pattern of AVN conduction.\textsuperscript{10,20}
Dual AVN Physiology and Reentry Beats

Reentry loops were frequently observed in these experiments (Figures 2 through 5) in the presence of either smooth (Figure 5) or discontinuous (Figure 2) conduction curves. Although the common sequence of atrial excitation during reentry was IAS followed by crista terminalis, in several experiments, both the crista terminalis and the IAS inputs to the AVN were simultaneously and retrogradely engaged (Figure 5). The anterograde part of the reentry loop could be completed either by the slow (Figures 2H and 5J) or the fast (Figures 3G and 3I) wavefront. This intermittent usage may reflect the current refactoriness of each domain and may have functional importance during high atrial rates (Figure 7) or atrial fibrillation. Therefore, the assumption that the fast wavefront should be ignored in such conditions may be incorrect.

Clinical Considerations and Limitations

Recording AVNP in patients should be achievable using existing bipolar catheters. The effect of ablation procedures on the AVNP, if shown to exist, could serve as a new endpoint in evaluating the efficacy of the intervention.

However, one should be aware of important interspecies differences. The CCR in humans is part of the atrial component of the conduction axis. In rabbits, it is, to a large extent, part of the penetrating bundle. Furthermore, the inferior nodal extension may not always be a prominent feature of human and dog hearts, and lesions identified as ablating the slow pathway in humans may involve only the working atrial myocardium. The inferior location of the CCR in humans (and dogs) outside the fibrous body may accentuate the differences between the slow and fast wavefronts by providing the latter with an easier connection between the envelope of transitional cells and the penetrating bundle. Although speculative, such morphological differences in the structure of the specialized axis may also underlie the higher likelihood for the initiation of AVN reentry in humans.

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