Reentrant Circuits in the Canine Atrioventricular Node During Atrial and Ventricular Echoes
Electrophysiological and Histological Correlation

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Background—The anatomic-electrophysiological correlation of AV nodal reentry is unclear. To localize reentrant circuits during atrial and ventricular echoes and to characterize sites of slow conduction and block, we correlated histology with electrophysiology of the AV node.

Methods and Results—In 10 isolated dog hearts, extracellular electrical activity was recorded in Koch’s triangle at 208 or 247 sites (interelectrode distance, 0.5 and 0.3 mm) after removal of 0.7 to 1.5 mm of overlying atrial tissue. Resection did not affect refractory periods. Five hearts were subjected to histology. Complete atrial echoes were induced in 1 heart, incomplete atrial echoes in 5 hearts. Unidirectional conduction block occurred at the atrial–transitional cell junction in the superior area. Zones of slow conduction arose at the atrial–transitional or the transitional–compact node junction in the inferior area. Complete reentrant circuits of ventricular echoes were obtained in 5 hearts. Unidirectional conduction block occurred at the compact node–transitional cell junction in the superior area. Localized zones of slow conduction arose at the junctions between the different types of tissue in the inferior area.

Conclusions—In the dog heart, tissue architecture and functional dissociation between the inferior and the superior region of the AV node enable dual physiology and reentry. Slow conduction and functional conduction block occur at the junctions between the different types of tissue in the AV nodal area. Atrial echoes were enabled by conduction block at the atrial-transitional cell junction, whereas during ventricular echoes conduction block occurred at the compact node-transitional cell junction. (Circulation. 2003;108:231-238.)

Key Words: atrioventricular node ■ reentry ■ mapping ■ electrophysiology

The original description of the atrioventricular (AV) node dates back to 1906.1 The first evidence for AV nodal reentry was provided 7 years later.2 Today, the substrate for AV nodal reentry is still unclear. Experimental and clinical data suggest intranodal reentry or reentry circuits outside the nodal area.3–7 Part of this controversy may be ascribed to an inconsistent definition of the AV node. The term “AV node” is often used for the compact node alone. However, morphological, electrophysiological, and clinical observations suggest that the AV node includes both of the cell groups that determine its functional properties, namely, transitional and compact nodal cells.8,9 We recently showed that in canine hearts, reentry during ventricular echoes is confined to the AV node.10

The underlying mechanisms of unidirectional conduction block and slow conduction are also controversial. Recent morphological and electrophysiological studies reemphasized the role of the inferior nodal extension.11–13 A comprehensive work on these issues has been published.14

Direct recording of AV nodal activation is hampered by its particular location and complex structure. In 1955, Scher15 provided the first recordings of AV nodal depolarization using needle electrodes. Sequential mapping with microelectrodes is fragmentary and challenging.3,4,8,16 Spach et al17 recorded extracellular AV nodal potentials in rabbit and dog hearts with a 50-μm tungsten electrode. Recent studies used optical imaging techniques, but no correlation with the underlying anatomy was made.18,19

In the present study, we exposed the AV node by resecting the overlying atrial myocardium and recorded the extracellular activity of the AV node during atrial and ventricular echo beats. The activation maps were correlated with the underlying histology.

Methods
This study conformed to the guiding principles of the American Physiological Society. Hearts were obtained from 10 mature mongrel dogs.
dogs (18 to 30 kg; A.E. Man in’t Veld BV, Apeldoorn, the Netherlands) of either sex. The methods of preparation and Landegordorff perfusion of isolated hearts have been described previously. The roof of the coronary sinus (CS) was cut away, and the interatrial septum was trimmed down to the AV junction. Unipolar and bipolar recordings from a roving electrode identified the approximate position of the compact node–penetrating bundle interface: the largest H-V interval in the bipolar and an initially negative His deflection in the unipolar recording. With watchmaker forceps and scissors, a thin layer of atrial myocardium overlying the AV node was carefully resected. The area was explored with a bipolar electrode, and resection was continued until low-frequency, low-amplitude signals were detected. Incremental atrial pacing was performed to ensure that the slow potentials were related to A-H conduction.

Programmed stimulation before and after resection ensured that the resection did not influence AV nodal functioning. If the refractory periods or the Wenckebach cycle length increased more than 30 ms because of direct damage of the AV node, the heart was excluded. A change of refractory periods or Wenckebach cycle length up to 30 ms was accepted because of the known procedure time–related changes in Langendorff-perfused hearts.

In 8 hearts, diacetyl monoxime (10 to 15 mmol/L) was added to the perfusate to dampen contraction.20

Radiofrequency Ablation of the Right and Left Bundle Branches

The small AV nodal potentials may be masked by depolarization of the ventricles. Therefore, the right and left bundle branches were ablated with a conventional ablation catheter. Retrograde conduction was achieved by stimulating the penetrating His bundle at the level of the central fibrous body.

Stimulation Protocol

Bipolar hook electrodes (interelectrode distance, 1 mm) were placed close to the CS ostium and over the His bundle. Stimulation was achieved with 2-ms-long pulses at twice diastolic threshold. Up to 2 premature stimuli after every eighth beat of a paced rhythm were applied to induce AV nodal echoes. If the AV node did not conduct retrogradely at baseline (4 hearts), isoproterenol was added to the perfusate at a stable infusion rate and titrated upward (0.05 to 0.5 μg/min) until stable VA conduction was achieved.

Mapping Plaques

Two multiterminal electrodes were used. One contained 208 unipolar recording sites (silver wires; diameter, 100 μm; interelectrode distances, 0.5 mm), arranged in a 16×13 matrix with a recording area of 7.5×6.0 mm. The second electrode contained 247 unipolar sites (silver wires; diameter, 100 μm; interelectrode distances, 0.3 mm), arranged in a 19×13 matrix with a recording area of 5.4×3.6 mm. The indifferent pole was placed either at the aortic root or, to reach a higher level of common-mode rejection, close to the plaque in the fluid covering the endocardium.

Signal Processing and Analysis of Electrical Activation

A customized data acquisition system allowed simultaneous recording of all channels at a sample frequency of 1 kHz/channel. Signals were amplified 40 times and bandpass filtered with lower and upper cutoff frequencies of 0.1 and 500 Hz, respectively. Recordings were stored on a hard disk of an IBM-compatible computer system.

The point of maximum negative dV/dt was selected as the time of local activation. In case of doubt, the laplacian analysis (difference between the electrogram and the weighted sum of electrograms recorded at surrounding sites) was calculated to suppress deflections caused by remote activation. Isochronal maps were constructed by connecting points with the same time of local activation.

Histology

Five hearts were prepared for histology. The position of the mapping electrode initially marked with fine needles (Figure 1) was retraced with India ink. Serial sections at 10-μm thickness, perpendicular to the plane of the septum, were cut. Every 10th section was stained with Masson’s trichrome technique. On examination of each section under the microscope, the penetrating AV bundle, compact AV node, zone of transitional cells, and ordinary atrial myocardium were identified according to the definitions of Tawara.1 In this plane of sectioning, the compact node consisted of a half-oval area of small cells adherent to the atrial aspect of the central fibrous body. Transitional cells were palely stained, slender, and frequently separated into small fascicles by connective tissue septa.21 The AV node–penetrating bundle interface was defined as the point distal to the last contact with transitional fibers, where the conduction axis became completely surrounded by fibrous tissue from the central fibrous body.22 Using the electrode position and the section number as references, the components of the AV nodal area were reconstructed as if the hearts were being viewed from the right aspect. After the reconstruction, the extent of the resection of atrial tissue overlying the AV node was estimated.

Results

Histology

The endocardium, which measured ≈0.3 mm, was completely removed in all 5 hearts. The total depth of the resections was between 0.7 and 1.5 mm. The specialized conduction tissue could be distinguished in all hearts.

Figure 1, top, shows a photograph of Koch’s triangle in heart 9 before it was processed for histology. The reconstructed AV nodal area of this heart is shown in Figure 2. The compact AV node reveals 2 inferior extensions. Transitional cells overlie the compact node and extend inferiorly toward the CS, where they are in continuity with deeper atrial cells. Only a few strands of superficial atrial cells are left.

Electrophysiological Parameters

The Table shows the functional parameters of all hearts before and after resection. Typical examples of antegrade AV nodal conduction curves before and after resection and of retrograde AV nodal conduction are displayed in Figure 1, bottom. None of the hearts showed discontinuous antegrade or retrograde conduction at baseline or after resection. Antegrade dual AV nodal pathways manifested as atrial echoes in 1 heart and as incomplete or “aborted” atrial echoes that did not lead to atrial activation in 5. Retrograde dual AV nodal physiology manifested as ventricular echoes in all 10 hearts.

Aborted Atrial Echoes

Figure 3 shows antegrade activation and an aborted atrial echo in heart 1. The inset at lower left illustrates the corresponding histological reconstruction. The compact node reveals a single extension and is covered by transitional cells and deep and superficial residual atrial cells. The black rectangle indicates the size and position of the mapping electrode. The contours of the compact node and the transitional zone are indicated in the activation maps.

In the middle part of the figure, selected electrograms from the activation maps shown in A and C are displayed. White circles in the activation maps indicate the respective positions of the corresponding electrodes. The recordings show the stimulus artifact (S) followed by a low-amplitude electrogram.
that represents local activation. The last deflection is generated by distant ventricular activation (ventricle). The right and left bundle branches were still intact in this preparation.

During baseline stimulation (A), the earliest local activation arises 30 ms after the stimulus (open arrowhead in tracings), enters the transitional cell zone after 60 ms, and reaches the proximal His bundle after 130 ms. An area of crowded isochronal lines at the superior junction between the atrial and transitional cells indicates a zone of hampered conduction (bold arrow). At atrial extrastimulation (B), this zone extends progressively along the atrial–transitional cell interface in the direction of the tricuspid valve annulus (TVA, bold arrows). At a shorter coupling interval of the premature stimulus (C), complete conduction block develops at the superior aspect of this zone (bold arrows). Conduction block extends along the inferior aspect of the atrial–transitional cell interface just beyond the middle of the recording area, from where it bends and continues toward the His bundle.

Activation runs along this zone of conduction block toward the TVA and turns toward the compact AV node. Between 180 and 240 ms after the stimulus, a zone of slow conduction arises as indicated by crowded isochrones (C, open arrow). Activation reaches the proximal His bundle after 260 ms and follows its course. However, another activation front passes the distal AV node–proximal His bundle (C, bold arrowheads in tracings) and reenters the superior septal area distal to the zone of antegrade block. Reentrant activation proceeds in the inferior direction but blocks after 330 ms, preventing completion of the reentrant circuit and the occurrence of an atrial echo.

**Atrial Echoes**

In the heart of Figures 1A and 2 (heart 9), a complete atrial reentrant circuit was determined (Figure 4). During baseline stimulation, low-amplitude–low-frequency electrograms indicate local activation of the AV nodal area (A, open arrowheads in tracings). A zone of slow conduction arises at the inferior junction between transitional and compact nodal cells (A, bold arrow).

At an extrastimulus, unidirectional conduction block occurs in the superior segment of the junction between atrial...
and transitional cells (B, bold arrows). At the inferior segment of the atrial–transitional cell interface, activation proceeds slowly, as indicated by densely packed isochrones between 80 and 130 ms after the stimulus. Double components in the corresponding tracings (marked with an asterisk) show nonhomogeneous conduction through this zone. Activation reaches the proximal His bundle 190 ms after the stimulus.

In the upper 4 tracings, late components (bold arrowheads) indicate reentrant activation distal to the zone of conduction block. Panel C illustrates the spread of reentrant activation, which runs superiorly in the transitional cells along the inferior aspect of the compact node, traverses the area of antegrade conduction block (B, bold arrows), which has regained excitability, and generates an atrial echo.

In contrast to the aborted reentrant circuit described in Figure 3 where activation first reached the compact AV node before it reentered the superior area, reciprocation occurs more proximal in the A-H conduction axis.

Ventricular Echoes

Ventricular echoes were inducible in all 10 hearts, and the complete reentrant circuit could be identified in 5 hearts. In the remaining 5 hearts, the reentrant circuit could not be entirely reconstructed.

Retrograde activation of the AV node in the heart of Figure 4 during His bundle stimulation is shown in Figure 5. Baseline retrograde activation spreads through the compact AV node and enters the transitional cell zone (A). Crowded isochrones indicate slow conduction at the superior junction between compact node and transitional/atrial cells (A, bold arrows). This zone of slow conduction extends inferiorly through the transitional cells and turns toward the TVA at the inferior junction between transitional and atrial cells.

<p>| Antegrade AV Nodal Conduction Parameters Before and After Resection |
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<th>FRP&lt;sub&gt;av&lt;/sub&gt;, ms</th>
<th>WCL, ms</th>
<th>A-H, ms</th>
<th>ERP&lt;sub&gt;av&lt;/sub&gt;, ms</th>
<th>FRP&lt;sub&gt;av&lt;/sub&gt;, ms</th>
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Mean±SD 66.5±10 167±12 285±33 256±25 77.5±13 176±19 293±33 266±22

ERP indicates effective refractory period; FRP, functional refractory period; WCL, Wenckebach cycle length; AVN, atrioventricular node; and Hist., histology.
At an extrastimulus, unidirectional conduction block occurs at the superior junction between compact node and transitional cells (B, bold arrow). A zone of slow conduction extends along the inferior aspect of the compact node toward the TVA (white arrows). Activation runs in the inferior direction and proceeds along the TVA. At 80 ms after the stimulus, the impulse encounters a zone of slow conduction at the transitional–atrial cell interface. The corresponding electrograms at 80 and 130 ms reveal multiple components (B, marked with asterisks). Activation then turns in the superior direction. The zone of retrograde block has regained excitability, and activation reenters the distal AV node 300 ms after the stimulus (C). The inset at lower left shows a ventricular echo in the same heart before resection. Activation of the His bundle occurred 310 ms after the stimulus, indicating that the time course of reentrant activation was virtually the same before and after resection.

**Figure 3.** Activation maps (247-terminal electrode) in heart 1 during atrial stimulation near CS ostium. Isochrones are drawn at 10-ms intervals. Bar at right indicates color-coding of activation time, early activation is red, late activation is blue. Outline of compact AV node (AVN) is indicated in yellow. Inset at lower left shows histological reconstruction of AV nodal area. A, Baseline atrial stimulation, basic cycle length (BCL) 500 ms. White arrow illustrates main spread of activation. Selected electrograms are shown at bottom. Respective recording sites are indicated by white circles; numbers indicate local activation time in milliseconds. B, Atrial extrastimulation, S1–S2 240 ms, BCL 500 ms. C, Aborted atrial echo at premature atrial stimulation, S1–S2 180 ms, BCL 500 ms. See text for discussion.

**Figure 4.** Activation maps (208-terminal electrode) in heart 9 during atrial stimulation near CS ostium. Isochrones are drawn at 5-ms intervals. A, Baseline atrial stimulation, basic cycle length (BCL) 500 ms. B, Atrial echo at atrial extrastimulation, S1–S2 190 ms, BCL 500 ms. C, Spread of reentrant activation of atrial echo in B. See text for discussion.
Figure 6 shows retrograde activation of the AV node during baseline and extrastimulation in heart 8. The inset at lower left shows the histological reconstruction of the AV nodal area. During baseline stimulation (A), local activation spreads in the inferior and superior directions, as illustrated by the red/yellow–colored areas. The activation front that spreads inferiorly exhibits slow conduction at the inferior transitional–atrial cell interface and at the inferior junction between transitional and compact nodal cells. The activation front that spreads superiorly slows down at the superior interface between transitional and atrial cells (A, bold arrows). The compact node becomes activated slightly later (30 ms), and activation proceeds toward the atrium.

Figure 5. Activation maps in heart 9 during His-bundle stimulation. Isochrones are drawn at 5-ms intervals. See text for discussion. A, Baseline His-bundle stimulation, basic cycle length (BCL) 500 ms. B, Ventricular echo after extrastimulation, S₁–S₂ 270 ms, BCL 500 ms. C, Spread of reentrant activation of ventricular echo in B. Inset at lower left shows a ventricular echo before resection. A, atrium; HBE, His-bundle electrogram; H, retrograde His activation; He, His activation during echo; V, ventricular activation; Ve, ventricular echo.

Figure 6. Activation maps (208-terminal electrode) in heart 8 during His-bundle stimulation. Reentrant activation completes 1.5 circuits before blocking in superior area. Isochrones are drawn at 5-ms intervals. Inset at lower left shows histological reconstruction of AV nodal area. A, Baseline His-bundle stimulation, basic cycle length (BCL) 500 ms. B, Ventricular echo after extrastimulation, S₁–S₂ 280 ms, BCL 500 ms. C, Spread of reentrant activation during second reentrant circuit of ventricular echo in B. See text for discussion.
At an extrastimulus, unidirectional conduction block arises at the superior junction between compact node and transitional cells (B, bold arrows). Activation proceeds inferiorly and encounters a first zone of slow conduction 80 ms after the stimulus at the inferior compact node–transitional cell interface. A second area of densely packed isochrones arises at the junction between transitional and atrial cells (white arrows). Activation then runs in the superior direction along the inferior aspect of the transitional zone and reenters the superior area. The junction between transitional and compact nodal cells has regained excitability, and activation reenters the distal AV node. Late deflections in the electrograms (B, bold arrowheads) indicate the initiation of a second reentrant circuit. The inferior area is again activated in the retrograde direction (C). Activation blocks in the superior area 590 ms after the stimulus.

**Discussion**

**Site of AV Nodal Reentry**

Tawara1 described the “atrioventricular connecting system” as a “relatively large, complicated network of muscular tissues. . . .” Short bundles of muscle fibers, arranged more or less parallel to each other, posteriorly extend approximately to the front end of the CS, where they connect with the ordinary atrial muscle fibers.” Later, combined histological and electrophysiological studies described a zone of transitional cells bordering the compact AV node.8,9

In the present study, direct recording of AV nodal electrograms and correlation with histology show that AV nodal reentry occurs in this complex network of nodal and transitional cells and in the rim of surrounding atrial cells. McGuire et al23 found that atrial cells contiguous with the AV node reveal nodal-type electrophysiology. An electrophysiological characterization of the AV node might, therefore, define a larger area than light microscopy. A reentrant circuit outside the “morphological” AV node might still be confined to the “electrophysiological” AV node.

Using optical mapping, Wu et al18 suggested that unidirectional conduction block during AV nodal reentry occurred at the junction between the fast pathway area and the AV node. This is in keeping with our findings, which, however, further unravel the mechanisms of reentry: functional block occurs at the atrial–transitional cell interface during atrial echoes and at the junction between compact nodal cells and transitional cells during ventricular echoes.

**Slow Conduction**

To date, the exact site of conduction delay in the AV node is unclear. This is mainly because of difficulties in correlating AV nodal activation time to a “morphological” cell type.8,9 A slow conducting “pathway” is considered a prerequisite for AV nodal reentry. However, a histological equivalent has never been found, and one can only speculate about the mechanism of slow conduction: normal conduction simply through a longer path, slowing of conduction because of the characteristics of depolarizing membrane currents, reduced intercellular coupling, and discontinuous conduction because of branching tissue architecture have been discussed as possible mechanisms.24 Recently, Medkour et al15 reemphasized the role of the inferior nodal extension as a possible substrate for slow conduction and reentry. Mazgalev et al25 ascribed dual physiology to separate wave fronts in functionally isolated “domains.”

The findings of this study suggest that slow conduction takes place primarily in the connecting zones between atrial, transitional, and compact nodal tissue. Localized zones of slow conduction arose after premature stimulation in regions that conducted normally during baseline stimulation. De Bakker et al26 described double-component action potentials in the inferior approach to the compact AV node. They suggested that these double components represent asynchronous propagation of activation in poorly coupled sheets or bundles of transitional cells. The interconnection network between different types of tissue forms regions of potential electrical inhomogeneity that may allow for slow conduction and block and thereby provide the substrate for reentry.

**Functional Dissociation**

During both ventricular and atrial echoes, unidirectional conduction block occurred in the superior AV node: at the superior junction between atrial and transitional tissue during premature atrial stimulation and at the superior junction between compact node and transitional cells during ventricular premature stimulation. Zones of slow conduction arose in the inferior AV node. This is in keeping with the historical concept of functional dissociation of the AV node as described by Mendez and Moe.27 Experimental and clinical studies have described a lower safety factor of conduction in the superior approach to the AV node.28

**Canine Dual AV Nodal Physiology**

In earlier experiments, we had to speculate as to why atrial echoes were such a rare finding in canine hearts, whereas they are a common finding in the human heart.10,29 The present study shows that the occurrence of antegrade dual physiology in canine hearts is comparable to that in humans: atrial echoes were inducible in 6 of 10 hearts. Echoes were, however, “aborted” and therewith concealed to endocardial mapping in 5 of these 6 hearts.

**Clinical Implications**

Our data add further aspects to a new understanding of AV nodal functioning. Instead of distinct insulated pathways, tissue architecture and functional dissociation between different regions of the AV node enable dual physiology and reentry. However, sustained reentry is a rare finding in perfused canine heart preparations. We do not know yet whether single echoes and sustained reentry use the same substrate. In heart 8, however, reentrant activation completed 1.5 circuits (Figure 6). It is conceivable that reentry could have continued under appropriate functional conditions. Wu et al18 described 1 case of AV nodal reentrant tachycardia lasting >15 minutes in a perfused dog heart. The activation patterns were the same during single atrial echo beats and sustained reentry.

“Slow-pathway” ablation/modulation for AV nodal reentrant tachycardia generates lesions at the inferior approach to the AV node, close to or at the atrial–transitional cell junction.
or even the transitional–compact nodal cell junction. It is conceivable that the complex functional and structural conditions of this delicate network can be modified in such a way as to abolish sustained reentry, even if dual-pathway physiology is preserved.

Limitations
Mechanical resection of the atrial overlay of the AV node can modify the functional parameters. However, earlier experiments showed that chemical destruction of the subendocardial tissue in Koch’s triangle with phenol had no influence on AV nodal functioning. In the present study, ventricular echoes in all 10 hearts and atrial echoes in 1 heart were inducible before resection. **The “window of inducibility” (S1–S2 intervals) and the time course of reentrant activation were virtually the same before and after resection (see inset in Figure 5).** Resection of the atrial overlay never enabled reentry that could not be induced at baseline. We therefore believe that the influence of the resection on the substrate of reentry is minimal.

Despite atrial resection, the AV node remains a complex structure. A planar mapping plaque scales the 3D activation back to 2 dimensions, and part of the information might be lost. Because bipolar recordings reflect only local activity, we used unipolar recordings, which have a wider field of view and reveal part of the deeper electrical activity. Nevertheless, more refined mapping techniques will be needed to accurately reconstruct the 3D activation pattern.

The histological reconstructions of the AV node cannot be absolutely precise owing to inherent difficulties in tissue processing. Care was taken to make them as accurate as possible. The distinction between transitional and compact nodal cells was made on histological grounds according to the criteria described by Anderson et al. Electrophysiological characterization of the AV node with microelectrode recordings could have provided additional information, but sequential microelectrode recordings are time consuming, and subsequent correlation with histology is difficult. Furthermore, discrepancies between AV nodal cell morphology and electrophysiology have been described. In a combined morphological and electrophysiological study of the rabbit AV node, Anderson et al found that “action potential configuration is determined to only a limited extent by cellular morphology.”

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References
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