Anatomic and Functional Characteristics of a Slow Posterior AV Nodal Pathway
Role in Dual-Pathway Physiology and Reentry

Djamila Medkour, MD; Anton E. Becker, MD; Karim Khalife, BSc; Jacques Billette, MD, PhD

Background—The AV node is frequently the site of reentrant rhythms. These rhythms arise from a slow and a fast pathway for which the anatomic and functional substratum remain debated. This study proposes a new explanation for dual-pathway physiology in which the posterior nodal extension (PNE) provides the substratum for the slow pathway.

Methods and Results—The anatomic and functional properties of the PNE were studied in 14 isolated rabbit heart preparations. A PNE was found in all studied preparations. It appeared as an elongated bundle of specialized tissues lying along the lower side of Koch’s triangle between the coronary sinus ostium and compact node. No well-defined boundary separated the PNE, compact node, and lower nodal cell bundle. The electric properties of the PNE were characterized with a premature protocol and surface potential recordings from histologically controlled locations. The PNE showed cycle-length–dependent posteroanterior slow activation with a shorter refractory period (minimum local cycle length) than that of the compact node. During early premature beats resulting in block in transitional tissues, the markedly delayed PNE activation could propagate to maintain or resume nodal conduction and initiate reentrant beats. A shift to PNE conduction resulted in different patterns of discontinuity on conduction curves. Transmembrane action potentials recorded from PNE cells in 6 other preparations confirmed the slow nature of PNE potentials.

Conclusions—The PNE is a normal anatomic feature of the rabbit AV node. It constitutes a cycle-length–dependent slow pathway with a shorter refractory period than that of the compact node. Propagated PNE activation can account for a discontinuity in conduction curves, markedly delayed AV nodal responses, and reentry. Finally, the PNE provides a substratum for the slow pathway in dual-pathway physiology. (Circulation. 1998;98:164-174.)

Key Words: atrioventricular node ■ tachycardia ■ reentry ■ electrophysiology

The AV node is frequently the site of reentrant rhythms. These rhythms arise from a functional and perhaps anatomic dissociation of the node into two parallel pathways with different conduction and refractory properties.1–6 This view is particularly supported by the sudden increase of AV nodal conduction time observed in the short-cycle-length range in patients suffering from AVNRT.7,8 This increase is reflected by a jump in their AV nodal recovery curve. The jump reflects the shift of the conduction from a fast pathway with a long refractory period to a slow pathway with a short refractory period. Dual-pathway physiology can also be manifested by other patterns of discontinuity in the nodal recovery curve.9,10 Some patients with documented AVNRT and thus obvious dual-pathway physiology have no apparent discontinuity in their recovery curve.10,11 Results of ablation therapy provide further convincing evidence of dual-pathway physiology; ablation carried out posteriorly to the compact node eliminates the slow pathway,12–14 whereas ablation carried out anterosuperiorly to the compact node eliminates the fast pathway.15–17

The anatomic and functional substratum underlying the different manifestations of AV nodal dual-pathway physiology and reentry remains unclear.3,4,6 Early studies indicated that the crista terminalis and interatrial septum inputs together with the proximal portion of the compact node provide a substratum for asymmetrical pathways and reentry.18,19 Several subsequent studies provided evidence for functional asymmetry between the inputs20–24 and a necessary involvement of the perinodal fibers or atrium in the reentry.25–27 However, no quantitative link has yet been established between AV nodal input asymmetry, dual-pathway physiology, and reentry. The effort to identify differences in the effective refractory period of the two pathways similar to those postulated from discontinuous recovery curves has been unsuccessful.21,22,28–30 Moreover, the functional symmetry of the inputs is also supported by studies31–33 showing that conduction and refractory values determined with local stimulation and recording at the crista terminalis input do not differ significantly from those obtained with local stimulation and recording at the septum input. The search for an anatomic...
substratum has also been unsuccessful, even in patients with documented dual-pathway physiology and AVNRT.\textsuperscript{34–36} Mapping studies thus far have not identified fast- or slow-conducting input areas.\textsuperscript{37–42} Nonuniform anisotropy at the level of the inputs remains another possibility to be confirmed formally.\textsuperscript{43} There is also convincing evidence that the compact node is involved in or could itself support the reentrant circuit.\textsuperscript{34–40}

The present study proposes an alternative anatomic and functional substratum for AV nodal dual-pathway physiology and reentrant rhythms. In the proposed scheme, the posterior extension of the AV node (PNE) acts as a slow pathway that can provide the distal node with the delayed impulse necessary for long delays and reentrant rhythms. The study characterizes the anatomic and functional properties of the PNE and provides evidence for its involvement in nodal reentry. Surface and microelectrode recordings from historically controlled PNE locations allowed for its selective characterization. A preliminary report from the present study has been presented,\textsuperscript{50} together with an anatomic study focusing on the PNEs of the compact AV node in human hearts.\textsuperscript{51,52}

**Methods**

**Preparation and Apparatus**

Experiments were performed in 20 superfused isolated rabbit heart preparations. Animal care was conducted according to guidelines of the American Physiological Society and the Université de Montréal. The preparation, perfusion system, stimulation techniques, and recording system were as previously described.\textsuperscript{2,3,13–14} Briefly, the preparation (Figure 1), which included the right atrium, AV node area, and upper portion of the right ventricle, was mounted in a tissue bath perfused at 200 mL/min with a 6-L volume of oxygenated (95% O\textsubscript{2}-5% CO\textsubscript{2}) Tyrode solution maintained at 37°C (pH 7.38). Its composition (mmol/L) was 128.2 NaCl, 4.7 KCl, 2.0 CaCl\textsubscript{2}, 1.0 MgCl\textsubscript{2}, 20 NaHCO\textsubscript{3}, 0.7 NaH\textsubscript{2}PO\textsubscript{4}, and 11.1 dextrose. Preparations were driven from the upper atrium through a bipolar platinum-iridium stimulation electrode placed on the crista terminalis near the sinus node region. Unipolar electrograms were recorded from the upper atrium, low crista, left septum, and His bundle with 250-μm PTFE-insulated silver electrodes (Figure 1). The indifferent electrode was positioned 3 cm away from the recording electrodes in the perfusion bath. Unipolar surface electrograms (E in text and Figures) were also recorded with a 125-μm PTFE-insulated silver electrode along the PNE as well as from nearby transitional tissues. The surface electrode was positioned with a micromanipulator under visual control through a dissecting microscope.\textsuperscript{53} Electrograms were recorded on a videotape along with the stimulation pulse, a time code, and a tachogram and analyzed off-line. Bandwidth was 0.1 Hz to 3 kHz. Stimulation sequences were generated with a 1-ms resolution and a 0.47-ms precision with a computer algorithm.\textsuperscript{54} Stimulation voltage pulses were twice threshold and had a 2-ms duration.

**Protocol**

The nodal conduction and refractory properties and corresponding PNE properties were characterized in 14 preparations with a standard premature protocol. A test premature impulse was introduced at every 20th basic beat with a decrement of 40, 20, 10, 5, or 1 ms in progressively shorter coupling-interval ranges. The same protocol was repeated for each nodal surface recording site. At the end of the experiments, typical electrode positions were marked by passing current until it left a small hollow around the electrode. Tissue blocks from 12 preparations were histologically studied. The tissue was fixed with 4% paraformaldehyde; dehydrated in 70%, 96%, and 100% ethanol; cleared in xylene; and infiltrated with paraffin. Each block was serially sectioned at 10-μm thickness perpendicular to the endocardium along the AV axis from the coronary sinus to the His bundle. Sections were stained with Masson’s trichrome. The relationship between surface and transmembrane PNE APs was studied in 6 other preparations. A microelectrode (2.6 mol/L KCl, 10 to 15 MΩ) recording was obtained from a PNE cell during a shortened premature protocol. The number of basic beats was reduced to 10, and scanning of coupling intervals was accelerated. Once a PNE cell impalement had been maintained during a complete protocol, a surface electrogram was obtained from the same location during a repeated protocol. Electrode position was marked and identified on serial sections.

**Interval Measurements**

Activation times at atrial (A), His bundle (H), and nodal recording sites were determined with 0.2-ms precision. Electrograms were digitized at 5 kHz per channel with the Axoscope program (Axon Instruments) and analyzed with the Data-Pac II program (Run Technologies). Nodal responses to premature protocol were represented as a recovery curve (A\textsubscript{H} versus A\textsubscript{A}, premature nodal conduction time versus atrial cycle length) and refractory curve (H\textsubscript{H} versus A\textsubscript{A}, His bundle cycle length versus atrial cycle length). The 1 and 2 subscripts identify the last basic beat and the premature beat, respectively. The crista terminalis reference was used in the reported data, but qualitatively similar observations were made from the septal reference. Local conduction and refractory properties were assessed from surface electrograms by constructing local recovery curves (A\textsubscript{E} versus A\textsubscript{A}, local activation time versus atrial cycle length) and refractory curves (E\textsubscript{E} versus A\textsubscript{A}, local cycle length versus atrial cycle length). When applicable, data are given as mean±SD.

**Definition**

The AV node included the structures known as the transitional zone, compact node, and lower nodal cell bundle.\textsuperscript{39,39,56–58} The PNE is also considered a portion of the AV node. These structures are all involved in the genesis of AV nodal delay and rate-dependent properties, including reentry. This definition of the AV node was more practical in the present anatomicophysiological correlation than one limited to the compact node. This definition also fits currently available information on the spatial distribution of the

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**Figure 1.** AV node landmarks and structures. UA indicates upper atrium; CT, crista terminalis; IAS, interatrial septum; HIS, His bundle; TT, tendon of Todaro; TV, tricuspid valve; CS, coronary sinus; TC, transitional cell zone; CN, compact node; and LNC, lower nodal cell bundle. Arrows point to posterior, middle, and anterior portion of the PNE.
different electrophysiological cell types and corresponding underlying structures.38,39,53,56

Results

Anatomic Characteristics of the Posterior Nodal Extension

Serial sections performed between the coronary sinus ostium and His bundle exposed the compact node and lower nodal cell bundle together with the transitional zone and PNE in each of the 12 specimens studied (Figure 1 and the Table). The PNE formed a continuum with the compact node and lower nodal cell bundle. It appeared as a small, elongated bundle of specialized tissues, similar to that of the compact node, located along the lower side of Koch’s triangle. The PNE extends along the AV ring passing underneath the coronary sinus ostium (Figure 1). Posteroanterior PNE length was 2488±331 μm (n=12), which corresponded to nearly half of the total AV nodal length. The PNE had a transverse ovoid shape, with its greater diameter lying along the AV axis (Figure 2). As shown in Figure 2A, 2B, 2C, and 2D, taken from the posterior, central and anterior portion of the PNE, and compact node, respectively, PNE dimension decreased at more posterior locations. The greatest and smallest PNE diameters were 617±87 and 117±29 μm, respectively (Table). Although not encapsulated, the PNE formed a well-defined continuous bundle that established contact with nearby transitional tissues (Figure 2). Figure 2C shows a burn mark left on the PNE at the recording site. Such marks, readily identifiable on serial sections, established the link between the slow potentials and underlying PNE. These findings show that the PNE is a consistent anatomic feature of the rabbit AV node identifiable on serial sections.

Functional Properties of PNE

Slow Potentials

Slow surface PNE potentials were recorded from all preparations. Figure 3A illustrates, together with reference electro-

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PNE posteroanterior length: 2488±331 μm; AV nodal posteroanterior length: 4850±608 μm. Values are mean±SD, in μm, n=12. Posterior, middle, and anterior refer to portions of the PNE indicated by arrows in Figure 1.
grams, a typical slow potential (E) recorded from the middle portion of the PNE (inset) at an $S_1S_2$ of 104 ms. Left and right potentials correspond to the last basic and premature beats, respectively. The basic E potential measured from the crista reference occurred at a local activation time ($A_2E_2$) of 34 ms and thus well within the AV nodal delay ($A_2H_2$ of 54 ms). At the premature beat, $A_2E_2$ and $A_2H_2$ increased to 79 and 121 ms, respectively. Figure 3B shows the changes in the slow potential for the different $A_1A_2$ values listed. Increasing prematurity shifted the slow potentials rightward without affecting their morphology except at the 105-ms $A_1A_2$. At this $A_1A_2$, PNE activation was markedly delayed while the surface potential increased in amplitude and downstroke velocity, a frequently observed PNE feature. Corresponding local and nodal recovery curves show that PNE activation systematically preceded His bundle activation (Figure 3C). As seen more readily in Figure 6D, the shape of the two curves differs slightly; the local curve shows less and more cycle-length dependence in the intermediate and short $A_1A_2$ ranges, respectively. This was another consistent PNE feature. Local and nodal refractory curves showed that the functional refractory period of PNE ($E_1E_2$ min) was shorter than that of the node ($H_1H_2$ min) (Figure 3D). The estimated effective refractory period ($A_1A_2$ min) coincided in this case. Recordings from nearby transitional tissues resulted in a flat local recovery curve (not shown). These findings show that PNE generates typical slow potentials with cycle-length-dependent activation times and a short refractory period.

Conduction and Refractoriness Along PNE

The conduction and refractory properties changed along the PNE. Figure 4A, 4B, and 4C shows three slow potentials (E) obtained at identical $A_1A_2$ (154 ms) but at different PNE locations (inset) in one preparation. Local activation time was 22, 45, and 52 ms in Figure 4A, 4B, and 4C, respectively, increasing along the PNE. Cycle-length dependence also increased along the PNE, as shown by the steeper rise of the local recovery curve nearer to the compact node (Figure 4D). The local refractory curves show a progressive upward shift, indicating that refractoriness also increased along the PNE (Figure 4E). The PNE functional refractory period was always shorter than that of the node. Qualitatively similar observations of PNE conduction and refractory properties were made in all studied preparations. In brief, PNE activation progressed in a posteroanterior direction. Cycle-length dependence and refractoriness increased along the PNE.

PNE Activation and Discontinuous Recovery Curve

A jump or a discontinuity in a recovery curve signals a change in conduction pathway. Figure 5 illustrates such discontinuity in PNE premature activation. $S_1S_2$ of 113, 108, and 98 ms (Figure 5A, 5B, and 5C) resulted in $A_2H_2$ of 128 ms, a nodal block, and $A_2H_2$ of 149 ms with reentry, respectively. Corresponding $E_1E_2$ values were 82, 87, and 130 ms. The block and resumed conduction resulted in a gap
and discontinuity in the nodal recovery curve (closed circles in Figure 5D). However, there were no discontinuities in the local PNE recovery curve (open circles); PNE remained activated at all coupling intervals, including the one that resulted in nodal block. Conduction stopped at the shortest A1 A2 in both curves (Figure 5D). Thus, conduction may fail in the compact node while persisting in the PNE; further PNE delay may then result in resumed conduction to the His bundle and trigger reentry.

Transitional Block, PNE Activation, and Reentry

A different example of PNE activation during reentry is illustrated in Figure 6. These data are from the same premature protocol that is illustrated in Figure 3. Figure 6A, 6B, and 6C shows electrograms recorded at S1 S2 of 104, 101, and 99 ms that resulted in prolonged A2 H2, sudden increase in A2 H2 with reentry, and block, respectively. They correspond to the 108-, 105-, and 104-ms A1 A2 of Figure 3B. A jump in delay with reentry (Figure 6B) and block (Figure 6C) occurred simultaneously in the PNE and the His bundle. The relationship between PNE and His bundle activation is illustrated in more detail in Figure 6D. This graph comes from the superimposition of the local and nodal recovery curve (same as in Figure 3C) on a common zero baseline. It shows that the PNE and His bundle operate along different functional curves that converge in the short-coupling-interval range. The jump in delay coincides in the two curves. These data support a relationship between a jump in A3 H3 and PNE-delayed activation.

Evidence that a transitional block favors PNE-induced nodal activation and reentry is provided in Figure 7. Surface potentials were recorded from two sites at the junction between transitional tissues and compact node in the same preparation. Figure 7A and 7B shows surface potentials from site 1 (inset) at S1 S2 of 120 and 110 ms, respectively. Figure 7D and 7E shows surface potentials from site 2 (inset) at the same S1 S2. S1 S2 of 120 ms resulted in an A2 E2 of 42 and 46 ms at sites 1 and 2, respectively. S1 S2 of 110 ms resulted in local block at both sites, yet the impulse reached the His bundle with a slightly prolonged A2 H2 and reentered at both sites. These events coincided with the occurrence of a minute discontinuity in the nodal recovery curve (arrows in Figure 7C and 7F). Reentry occurred at all coupling intervals beyond this point (not shown). The flat local A2 E2 curves from transitional sites showed their cycle-length independence. These data show that AV nodal prolonged delay, discontinuous recovery curve, and reentry can be associated with a block over a broad area of the transitional cell zone.

Slow Transmembrane Action Potentials From PNE Cells

Transmembrane APs were obtained from PNE cells in six preparations and correlated with corresponding surface potentials at the same site (Figure 8). Figure 8A shows the two superimposed records obtained at an S1 S2 of 110 ms from a
histologically confirmed PNE site (inset). Transmembrane and surface PNE potentials differ in morphology but correspond in activation time. The reference electrograms are virtually identical. The local and nodal recovery curves also correspond nicely (Figure 8B). Basic and premature APs are superimposed in reference to the last S1 in Figure 8C. Basic APs are nearly identical and establish the stability of the impalement. Premature potentials occur increasingly earlier in the recovery cycle until the cell cycle length reaches a minimum. Surface potentials show the same pattern of changes in activation (Figure 8D). PNE APs were systematically slow; the maximum rate of rise was 12±2 and 6±2 V/s (n=6) at the basic and shortest cell cycle lengths, respectively. The AP decreased slightly in amplitude and duration but did not dissociate with prematurity. Local activation time and refractory period increased in a posteroanterior direction along the PNE whether assessed by microelectrode or surface recordings. Recordings obtained slightly higher on the septum were always of the transitional type. In brief, PNE conduction and refractory properties are similarly reflected on microelectrode and surface recordings.

Discussion

Our findings support a new substratum for AV nodal dual-pathway physiology and reentry. They indicate that PNE anatomic and functional properties account for the slow pathway in this physiology. These properties include a posterior location, appropriate contact with transitional and compact node tissues, cycle-length–dependent slow activation, posteroanterior propagation, and short refractoriness. The study also shows that the PNE can provide the node with the delayed impulse necessary for delayed activation and reentry. In the presence of a transitional block, this delayed impulse could propagate to the His bundle and reenter the atrium. The shift from compact node to PNE conduction was often associated with a discontinuity in the recovery curve and reentry. In brief, the PNE provides a substratum for the slow pathway in dual-pathway physiology.

Anatomy of the Posterior Nodal Extension

A PNE was found in all studied preparations and is thus a consistent anatomic feature of the normal AV node. Another important PNE characteristic was its compact node–like histology and the formation of a continuum without well-defined boundaries with the compact node and lower nodal cell bundle. This arrangement is certainly compatible with functional communication between these structures. The PNE also established links with nearby transitional tissues and could therefore be activated in this manner. However, the exact nature of the interconnection between the PNE, transitional tissues, the compact node, and the lower nodal cell bundle could not be resolved with our data. The posterior location of the PNE along the AV ring was another very
consistent PNE feature with obvious consequential implications in the context of ablation therapy.

These anatomic findings were qualitatively similar to those made in a parallel study conducted in the human heart.\textsuperscript{51,52} Besides dimension, the only substantial difference between human and rabbit PNE was the presence of a left limb in human PNE. The PNE or some equivalent has been previously identified.\textsuperscript{56,57,59} Anderson et al\textsuperscript{56} described a PNE that was in continuity with the lower nodal cell bundle. In the present study, we identify the lower nodal cell bundle as the portion of the node that connects the compact node to the His bundle (Figure 1). The specific anatomic and functional characteristics of the PNE rather than its boundaries distinguished it from the compact node and lower nodal cell bundle. Others\textsuperscript{60–62} have proposed a link between slow potentials recorded in the posterior region of the node and underlying transitional tissues. Although we found transitional tissues above the PNE (Figure 2), they differed from the compact nodal-like tissues of the PNE. Moreover, transitional responses resulted in early activation and a flat local recovery curve. The present study establishes the PNE as an independent substructure of the normal AV node with specific anatomic and functional characteristics.

Is the Posterior Nodal Extension a Dead-end Pathway, a Slow Pathway, or Both?

The PNE can likely act as a dead-end pathway.\textsuperscript{38,56} A dead-end pathway is a sidetrack structure connected to the mainstream of tissues involved in nodal conduction and manifested by a discrepancy in delay on comparison of antegrade versus retrograde conduction. We have not assessed this possibility. However, the fact that the PNE followed a different functional curve than the node, particularly at long and intermediate coupling intervals (Figures 3 through 6), suggests that an activation could enter the PNE and vanish.\textsuperscript{38,51,52} Contact between the PNE and nearby transitional cells (Figure 2) would support such a possibility. At short coupling intervals resulting in transitional blocks (Figure 7), the PNE becomes an integral part of the antegrade circuit. It then provides the compact node and distal node with a delayed input (Figures 5 and 6). McGuire et al\textsuperscript{62} postulated a variant of the above in which the “AV junctional cells in the posterior AV nodal approaches appear to participate in slow pathway connection.” Our data suggest that transitional activation may indeed trigger PNE activation. However, transitional activation was fast and did not participate per se in the slow pathway. In other words, PNE conduction is slow, but nearby crista terminalis conduction is fast. In addition, the transitional cells did not appear as an extension of the compact node. In our opinion, the PNE alone is the slow pathway but can also act as a dead-end pathway.

Functional Properties of the Posterior Nodal Extension and AV Nodal Reentrant Circuit

Our findings support the following explanation for dual-pathway physiology and reentry. The slow pathway arises from PNE activation. The apparently faster pathway arises from conduction initiated from the crista terminalis or interatrial septum input and propagated through the compact node. Fast pathway conduction would prevail at intermediate and long coupling intervals but fail at some critical short coupling interval. When this occurs in the transitional cell zone (Figure 9A), PNE cycle-length-dependent slow activation further delays but maintains conduction to the His bundle (Figure...
This extra delay also allows the recovery of excitability in transitional tissues and the initiation of a reentrant beat. The shorter refractory period of PNE versus the compact node favors this sequence of events. The close association between jumps or discontinuities in recovery curves, transitional block, reentry, and PNE-delayed activation (Figures 3 through 7) supports this explanation. This scheme is also in agreement with input functional symmetry. This hypothesis is certainly compatible with the various forms of discontinuities observed in human recovery curves but also with the following characteristics of dual-pathway physiology. Posterior ablation therapy eliminates slow-pathway physiology and AVNRT; although the posterior input is the current target of this procedure, ablation of the nearby PNE could be a more critical factor for success than ablation of the input itself. This would also explain why the slow potential guidance and pure anatomic approach are similarly effective; they are both primarily targeted toward the same posterior area that corresponds to PNE location. The upward shift of the baseline of the recovery curve caused by fast-pathway (septal) ablation can also be explained in the context of the proposed hypothesis; the impulse entering the node from the septum input has to go around the ablation obstacle to reach the crista input and activate the node. This prolongs the nodal delay measured from the “a” complex of the His bundle derivation and makes retrograde septal invasion from PNE activation less likely. These possibilities will obviously require further testing.

Our findings support a compact node involvement in the reentrant circuit. Such involvement of at least the proximal portion of the compact node has been suggested by a number of studies. The possibility of a compact node reentrant circuit in responses initiated from the ventricles was also recently demonstrated. The present study supports such a phenomenon and suggests that the PNE provides a substratum for such an invasion.

Limitations

The nodal origin of AVNRT was initially demonstrated in the rabbit AV node, in which reentrant beats can be readily initiated. However, these rhythms are only rarely associated with typical jumps in recovery curves such as those observed in humans. Moreover, reentrant rhythms in the rabbit AV node are often limited to a few beats. Thus, the PNE may be
a substratum for reentrant rhythms in both species, but patients suffering from AVNRT may have other specific characteristics yet to be elucidated.

The PNE slow potentials were recorded with unipolar electrodes. These electrodes were best suited for signal recognition and very small PNE dimension (Table). It would have been difficult to position multiple electrodes over the PNE. However, unipolar recordings are sensitive to far fields. Indeed, many PNE recordings showed a small early deflection, likely from nearby transitional or atrial tissues. These potentials had a much smaller amplitude and a different timing than the slow PNE potentials (Figures 3 through 6). Moreover, the close correspondence between APs and surface potentials at any given PNE site (Figure 8) supports the PNE origin of the slow potentials. The slow nature of both transmembrane and surface potentials at any given PNE site also supports the PNE origin of the potentials.

A full understanding of the electrical activation of the PNE and other AV nodal structures during reentrant rhythms will obviously require combined functional and mapping studies with a greater resolution than that provided by single or few recordings. Dye mapping may be helpful in this respect.42,64–67 However, the success of PNE mapping may require substantial developments in terms of resolution, functional compatibility, and focusing to overcome problems caused by tridimensional anatomic and functional complexity within a very small area. Additional studies will also be necessary to elucidate the electrophysiological basis of slow conduction and short refractoriness in PNE. The very existence of this proarrhythmic PNE structure in the normal heart also poses an unresolved teleological challenge.

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