Posterior Extensions of the Human Compact Atrioventricular Node

A Neglected Anatomic Feature of Potential Clinical Significance

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Background—Catheter ablation procedures have revived interest in the detailed anatomy of the specialized atrioventricular (AV) septal junctional area. The compact AV node usually is considered to have a blunt posterior end. The objective of this study was to reconstruct the human compact AV node in relation to the landmarks of Koch’s triangle, with emphasis on its posterior extension.

Methods and Results—In 21 hearts obtained from autopsies, the AV nodal septal junctional area was removed en bloc, serially sectioned, and reconstructed. None of the hearts showed a blunt posterior ending of the compact AV node; 13 showed posterior extensions on both the right and left sides; 7 had a rightward posterior extension only; and 1 heart showed a single leftward extension. Hence, a rightward posterior extension was present in 20 of 21 hearts. Furthermore, in 16 of these 20 hearts, the rightward extension continued to the level of the coronary sinus ostium; in 7, the bundle extended beyond this anatomic landmark. The mean length of the right posterior extension was 4.4 ± 2.0 mm; that of the leftward posterior extension was 1.8 ± 0.9 mm. Superimposed onto the slope of the muscular AV septum, viewed from the right atrium, the rightward extension ran close to the tricuspid annulus with the leftward extension positioned superiorly.

Conclusions—The human compact AV node contains rightward and leftward posterior extensions, with the right extension close to the tricuspid annulus. It is tempting to speculate that these extensions are involved in “slow pathway” conduction. (Circulation. 1998;97:188-193.)

Key Words: atrioventricular node ▪ arrhythmia ▪ electrophysiology ▪ reentry ▪ tachycardia ▪ ablation

Catheter ablation procedures, particularly those for AV nodal reentrant tachycardia, have led to a renewed interest in the detailed morphology of the AV node and its atrial inputs. In this context, the precise positioning of the AV node is increasingly important. It is generally acknowledged that the human AV node is located in the triangle of Koch, delineated by the eustachian ridge or sinus septum (which harbors the tendon of Todaro), the membranous septum (as part of the central fibrous body), and the line of attachment of the septal leaflet of the tricuspid valve. The dimensions of Koch’s triangle, however, vary considerably from one individual to another, which is clinically relevant in the case of catheter ablation procedures in this area that are guided largely by anatomic landmarks. In this context, it is of considerable interest that most clinical investigators are accustomed to consider the compact part of the AV node as a structure with a blunt posterior end. This ignores the fact that as early as 1906, Tawara described posterior extensions of the node, an observation endorsed by our own studies in 1975. These posterior extensions have not received much attention since that time, and it seems almost as if they have been forgotten completely. In light of the revived interest in the electrophysiological characteristics of the AV septal junctional area, this could turn out to be a critical omission. For instance, in patients with AV nodal reentrant tachycardia, a “slow pathway” is considered part of the reentrant circuit, and catheter ablation near the coronary sinus (CS) orifice is the most favored and highly successful approach. Thus far, however, no one has shown such a pathway electrophysiologically, and a morphological substrate for a slow pathway has not been traced either. Hence, the question arises whether posterior extensions of the compact AV node could serve as potential candidates.

The present study is based on serial histological sectioning and subsequent reconstruction of the AV septal junctional area in human hearts, obtained from individuals without a history of AV nodal reentrant tachycardia or paroxysmal atrial fibrillation, and has been designed to investigate in detail the posterior extensions of the compact node.

Methods

The present study is based on 21 hearts obtained at autopsy; none of the patients had a history of persistent supraventricular arrhythmia. All hearts were fixed in 4% formalin. Before the microscopic investigations of the AV node, we measured the length of Koch’s triangle by taking the distance between the membranous septum and the widest part of the mouth of the CS. Thereafter, the AV septal junctional area was removed en bloc, which included the anterior part of the os of the
When considered necessary, the adjoining posterior tissue blocks were also taken. The block of tissue containing the full length of Koch’s triangle was then carefully cut into parallel slices of 5-mm thickness each (Fig 1). All slices were embedded and serially sectioned at 10 \( \mu \text{m} \) thickness. Initially, every 20th section was stained with either hematoxylin-eosin or a trichrome stain. This allowed the recognition of the compact AV node and posterior extensions as a first step. Thereafter, the sections in between were mounted and stained to achieve a full reconstruction related to the anatomic landmarks of Koch’s triangle. The actual length of AV nodal tissues was calculated from the microscopic sections, which also allowed a reconstruction of the AV nodal tissues in relation to the length of Koch’s triangle. For each heart, the results are expressed as the mean±SD. Correlations between two parameters were assessed by the method of Spearman.

**Results**

There were 15 male and 6 female hearts, taken from patients ranging in age from 31 to 84 years (mean age, 56.2 years). Heart weights ranged from 380 to 640 g, with a mean weight of 471 g. The mean length of Koch’s triangle was 24.6±4.6 mm. Structural heart disease was found in 5 hearts; old or recurrent myocardial infarction in 3; and aortic stenosis in 2. The major cause of death was lung disease in 9 patients, malignant disease in 5, acquired immunodeficiency syndrome in 3, heart failure in 3, and sepsis in 2.

In each of the 21 hearts, a distinct, compact AV node was found (Fig 2A). As anticipated, the compact node was positioned on the right side of the septal slope of the muscular component of the AV septum. Anteriorly, the compact node was characterized by a complex architecture of interweaving cells. In most instances, a deep stratum was identified, immediately resting on and partially buried into the connective tissue of the central fibrous body, composed of small interlacing cells. In such instances, a superficial stratum was present, although without a distinct delineation from the deep stratum, composed of lattice-like bundles of cells. However, the distinction between deep and superficial layers was not always readily identifiable. The compact node was covered on its right atrial aspect by a transitional cell zone. The posterior extensions described herein were direct continuations of the compact node, whereas a further subdivision into deep and superficial strata was no longer present. In fact, posterior extensions were composed of tightly packed, small cells, occasionally with a marked nodalike

Figure 1. Opened right side of the heart with the membranous septum transilluminated. The block of tissue removed for histological serial sectioning is shown. It contains Koch’s triangle and, for the purpose of this diagram, has been divided into three parallel slices. The area posterior to the os of the coronary sinus (CS), which is the subeustachian pouch (⌘) and represents AV free wall junction, has been removed en bloc, also if considered necessary. The dotted line represents the annular attachments of the septal tricuspid valve leaflet. ER indicates eustachian ridge.

Figure 2. Composite showing the histology of the AV node and its posterior extensions. A, The compact AV node (arrows) resting on the slope of the muscular AV septum. B, A section close to the mouth of the os of the coronary sinus, showing the leftward (L) and rightward extensions (R), both of which are circled. C and D, Higher magnifications of the leftward and rightward extensions (arrows), respectively. Hematoxylin-eosin stains. Bar=1 mm.
arrangement, and were identified as discrete posterior continuations of the compact AV node. In case of a leftward and rightward extension, the site of origin was readily recognized. In case of a sole leftward or rightward extension, the site of origin was identified as the site where the posterior continuation of the compact AV node veered away from the middle part, either to the left or the right. Each of these 21 hearts contained a posterior extension, which originated from the compact AV node (Fig 2B to 2D); none of the hearts had a compact AV node with a blunt posterior ending. The cellular component makeup was like that of the compact node. Of these 21 hearts, 13 showed a posterior extension that was both right and left sided; 7 hearts had a rightward posterior extension only (Fig 3), whereas only 1 heart showed a sole leftward extension (Fig 4). A diagrammatic survey is provided in Fig 5.

Once the compact node and both rightward and leftward extensions were superimposed on the slope of the muscular AV septum, it immediately became obvious that when viewed from the right atrial aspect, the leftward extension was positioned superior to the rightward extension (Fig 6). The latter, moreover, took a course parallel to and closely related to the tricuspid annulus. Except for the case with a leftward extension only, the rightward posterior extension was by far the most prominent in each instance. In fact, in 16 hearts, this extension could be traced all the way underneath the anterior margin of the CS osium. The maximum length of the right posterior extension was 9 mm, with a mean of 4.4±2.0 mm. In contrast, the maximum length of the leftward extension was 4.0 mm, with a mean of 1.8±0.9 mm. It appeared that the mean length of the rightward posterior extension exceeded that of the length of the compact AV node (4.4±2.0 versus 3.7±0.9 mm). Moreover, in 7 of these 16 hearts, the posterior bundle extended even beyond the anterior margin of the os of the CS and continued for some distance in the subeustachian pouch, which represents the posteroinferior right atrial free wall (Fig 7). In these 21 hearts, the length of the compact AV node varied between 2 and 5 mm, with a mean of 3.7±0.9 mm. In the same hearts, the length of Koch’s triangle varied between 15 and 32 mm, with a mean of 24.6±4.6 mm. The length of the compact AV node and posterior extensions showed no relation to the dimensions of Koch’s triangle.

**Discussion**

The human AV node is not characterized by a blunt posterior end as depicted in most electrophysiology texts. In fact, in this series of 21 randomly selected and basically normal hearts, not a single specimen had such a blunt-ended AV node. On the contrary, 20 of the 21 AV nodes had a rightward posterior extension from the compact part of the AV node, 13 of which had an additional leftward extension. The remaining heart showed an AV node with only a leftward posterior extension. These extensions showed similar cellular and architectural characteristics as those of the compact AV node. The latter has been described as a layered structure with superficial and deep strata; others have described an additional intermediate layer. In the posterior extensions, such an additional subdivision was no longer apparent. The cells were small and closely packed, with an interweaving architecture (Fig 2C and 2D) and, occasionally, a distinct nodelike cellular arrangement (Fig 7). The latter situation is very much reminiscent of the nodelike structures described by Anderson et al. Eventually, these extensions faded out; the leftward extension eventually disappeared within the central fibrous body at the site of the mitral valve annulus, whereas the rightward extension gradually disappeared amid atrial myocardium.

It is of considerable interest, moreover, that the AV nodal-bundle axis reached the level of the anterior margin of the os of the CS in 16 of 20 hearts. In addition, in 7 of these hearts,
the axis continued even further into the subeustachian pouch, which basically no longer reflected AV septal junction but rather inferior free wall junction.

Recent reviews of the anatomy of the AV conduction tissues in electrophysiology textbooks fail to mention these extensions, other than a rather vague statement that "nodelike cells are often seen at or near the anulus of the tricuspid, mitral and aortic valves. Some of these . . . may join the regular AV node." It is only in a chapter produced by Anderson and colleagues, referring to previous studies in the 1970s, that posterior extensions are clearly mentioned and shown, albeit without further emphasis on their potential significance. We are unaware, moreover, of any recent original works that encompass the posterior extensions of the compact AV node in humans. Hence, it appears as if these structures have almost been forgotten.

Nevertheless, Tawara’s 1906 epic work “Das Reizleitungs-system des Säugetierherzens” clearly states that a small, parallel-oriented bundle of fibers originates from the node to run posteriorly approximately to the anterior region of the CS, where it connects with the usual atrial fibers. Tawara’s famous plates, composed of meticulously reconstructed drawings of microscopic sections, beautifully illustrate these extensions and actually show that Tawara himself had already noticed that these bundles of fibers diverted rightward and leftward (see his Tafel I Menschherz No. 136 and No. 143).

In our studies in 1975, we confirmed the existence of two distinct contributing segments to the compact node. Both approached the compact node from beneath the mouth of the CS, closely adherent to the fibrous anulus (see Becker and Anderson, Fig 8, page 272), being divergent posteriorly, but...
anteriorly both merged with the half oval of compact nodal cells. They probably represent the deep and superficial nodal segments described by Truex and Smythe.14–16 The observation of a posterior tract in close proximity to the tricuspid annulus raised discussions concerning whether those extensions represented a tract of cells described by James7 and considered to run from the posterior “internodal tract” to the His bundle, thus bypassing the compact AV node. We refuted this concept at the time, and our present extended observations endorse this viewpoint. The extensions always originated from the posterior aspect of the compact AV node and never from the anterior part or from the His bundle.

It is a bit embarrassing that the discussions concerning the possibility of AV nodal bypass tracts at the time took away further interest in the potential significance of these peculiar extensions. It is because of more recent electrophysiological studies in humans, particularly those related to AV nodal reentrant tachycardia, that interest has been revived. It has been shown that part of the reentrant circuit is likely to be a slow pathway. In addition, the area to ablate this slow pathway is considered to be underneath or slightly anterior to and below the anterior margin of the mouth of the CS.7–9 Thus far, however, a true pathway has not been shown, either electrophysiologically or morphologically.

In an experimental study performed in isolated, blood-perfused porcine and canine hearts, designed to identify the origin of double potentials in Koch’s triangle, McGuire and colleagues18 provided evidence that the low-frequency component that followed a large-amplitude, high-frequency component was caused by depolarization of nodal or conduction-type tissue. They suggested that this tissue could represent the anatomic substrate of the slow AV nodal pathway. This concept was endorsed by the fact that the site of their recordings coincided with the site of the slow pathway, as demonstrated by mapping and ablation studies in humans7–9,19–21. It is of considerable interest, therefore, that the compact AV node and its posterior extensions, once superimposed onto the slope of the muscular AV septum within the confines of Koch’s triangle, colocalize with the sites of double potential recordings and with the area of slow pathway ablation. Our anatomic observations suggest, therefore, that the posterior extensions could take part in the reentrant circuit in patients with AV nodal reentrant tachycardia. If so, and if the posterior extensions do represent the anatomic substrate for a slow pathway defined electrophysiologically, the implication is that the slow AV nodal pathway basically fits within the natural variability of an anatomically normal compact AV node.

Developmental Considerations

The observation that posterior extensions of the compact part of the AV node are a regular feature of AV nodal anatomy is also of considerable interest once put into the perspective of AV nodal development. It is presently acknowledged that the AV conduction tissues develop from a “specialized” myocardial ring that, in the early human embryo, encircles the interventricular foramen.22 At this stage (5 weeks’ development), the AV canal is still positioned over the presumptive left ventricle. At subsequent stages (6 to 7 weeks’ development), the AV canal expands toward the right, thus enabling the right atrium to directly contact the developing right ventricle.

During this process, the ring of “specialized” myocardium moves with the rightward expansion of the AV canal and, once fully developed, encircles the right AV junction along the lower rim of the right atrium. The larger part of this right AV ring is considered to disappear, with only the compact part of the AV node and the AV (His) bundle remaining in the normal heart. At the same time, the concept of a single interventricular ring could explain why hearts with abnormal septation or abnormal expansion of the right AV orifice, such as hearts with a straddling tricuspid valve, contain an AV node and bundle in an unusual position. Similarly, the “atriofascicular” tracts that connect the right atrium to the right ventricle, exhibiting Mahaim physiology, may well find their origin in so-called Kent nodes that are considered to represent the remnants of the ring tissue. Indeed, Kent nodes confined within the atrial tissues, which have been shown to be a regular finding in normal hearts,23 may fit the same concept. Along the same lines of thought, one may anticipate that the posterior extensions of the compact part of the AV node, as described in the present study, reflect the remains of the ring tissue alluded to above. Indeed, the node-like structure found in one of our hearts (see Fig 7) closely resembles a Kent node, as previously discussed.11

Our present observations thus appear to be endorsed by the current concepts of AV conduction development.

Study Limitations

An obvious limitation of the present study is that we have not been able to study hearts with documented AV nodal reentrant tachycardias. As far as we are aware, two case reports exist24,25 that provide a histological description of the site of slow pathway ablation, each in a patient with clinically successful ablation. In both instances, the site of ablation was readily identified and localized posterior to the AV node. These observations have been claimed to refute the concept that the AV nodal reentrant circuit is entirely intranodal. This may well be correct, but because the posterior extensions of the AV node are not mentioned, one should not therefore deny the possibility that such extensions could have been part of the slow pathway. Indeed, one may envision that part of the reentrant circuit is formed by the posterior AV nodal approaches and that the “burn” has destroyed the approach rather than the “slow pathway” in the strict sense. In this context, it may be of interest to refer to an electrophysiological study performed in patients enrolled for cardiac transplantation.26 None of the patients reported in that study had AV nodal reentrant tachycardia, but dual AV nodal pathways could be documented in all patients before transplantation. Histological evaluation of these hearts did not reveal any departure from normal, although no further specifications are provided with respect to the posterior extensions as discussed in the present study.

It is our opinion, therefore, that the posterior extensions of the compact AV node in humans could provide part of the reentrant circuit of AV nodal reentrant tachycardia, although we readily admit that we have no definitive proof for such a statement. Nevertheless, it is a tempting concept, particularly because it could take further research into mechanisms underlying AV nodal reentrant tachycardia away from attempts to identify an “abnormal pathway.”
Acknowledgment
During the course of this study, Dr Inoue was a research fellow from the Showa University Hospital, Showa University School of Medicine, Tokyo, Japan.

References
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Circulation. 1998;97:188-193
doi: 10.1161/01.CIR.97.2.188

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

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