Ultrastructure of the Human Atrioventricular Node

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SUMMARY
Ultrastructure of the A-V node was studied in two human hearts. Fine details of intranodal cellular organization were then resynthesized on the basis of light microscopic examinations of over 250 human hearts. There are four different types of cells and no anatomic syncytium in the human A-V node: (1) a simple rounded cell identical to P cells of the sinus node, (2) a slender transitional cell which is by far the most numerous type in the A-V node, (3) Purkinje cells, and (4) ordinary working myocardium; both of the latter are almost exclusively at the nodal margins. The possible significance of these ultrastructural features relative to certain functions of the A-V node, such as pacemaking and delay of conduction, is discussed.

Additional Indexing Words:
P cells  Transitional cells  Electron microscopy
Purkinje cells

In the mammalian heart the A-V (atrophicventricular) node is a small structure of critical importance to normal cardiac function. With every normal cardiac cycle there is a slight delay (approximately 0.04 sec) in A-V transmission which is thought to occur in or near the atrioventricular junction. If the normal pacemaker of the heart (the sinus node) fails, one of the most efficient alternate pacemakers is believed to be in or near the A-V node. For the fullest understanding of how the A-V node works, one must include detailed knowledge of its anatomy. Certain structural features of the A-V node can only be studied with the electron microscope. These features include the membranes of individual cells, intercellular junctional relations, and the nature and the distribution of intracellular organelles. The potential importance of a clearer understanding of these anatomic features applies especially to interpretation of microelectrode electrophysiological studies, but a re-synthesis of the ultrastructural details based on light microscopic findings will help clarify function at the more complex multicellular level in the A-V node.

Methods
The two principal problems in studying the ultrastructure of the human A-V node lie in finding this small structure and in the necessity of using postmortem material. How we have coped with these two problems is described in detail in a previous report on the comparative ultrastructure of the human and canine sinus nodes. The procedures are briefly as follows: Specimens to be prepared for ultra-thin sectioning are usually about 1-mm cubes, which means any area to be studied in this fashion must either be a large mass of relatively homogeneous tissue (for example, liver or skeletal muscle) in which any millimeter cube is suitable, or if it is a special small region, one must be particularly familiar with its gross and microscopic anatomy. Based on several years of experience with microscopic studies of human2 and subhuman3-5 A-V nodes, we were confident of finding the proper
structure. A number of additional check procedures to assure identity of the final specimen were found satisfactory for the sinus node and applied again here. From light microscopic examination of sections prepared as alternate 1-mm slices of A-V node obtained between the ones from which ultra-thin specimens were prepared, and from similar examination of the entire remaining tissue after the A-V nodal area had been carefully cut out and removed for electron microscopy, it was determined that all tissue studied for ultrastructural detail was from within the A-V node or less than 1 mm from its atrial margin (fig. 1). Of all the 1-mm cubes examined, about 90% were from within the A-V node. None was from the region of His bundle. To minimize postmortem changes, the human specimens were obtained and fixed within 3 hours after death. What postmortem changes inevitably occur were clearly interpretable as such, based on previous studies by others as well as our own experience.

Specimens from two hearts of patients dying with noncardiac disease formed the principal basis of this study. One was a 2-year-old child and the other a 38-year-old adult. Comparative observations were made on one canine A-V node obtained and fixed within 5 minutes after death. Specimens (1 mm³) were immersed immediately in cold phosphate-buffered 6.5% glutaraldehyde; after 2 hours they were transferred to 0.2 M sucrose. After several hours in sucrose they were post-fixed for 2 additional hours in osmium. Following dehydration in a graded alcohol series, they were placed in propylene oxide for embedding in Araldite. Sections were cut on a Porter-Blum ultratome and stained with uranyl acetate and lead hydroxide. Photographs were made with several different RCA electron microscopes, and then enlarged to the desired final magnifications as indicated.

Over 250 human A-V nodes have been studied in this laboratory with light microscopy, and comparative observations have been made between human and animal (dog, rabbit and steer) hearts. On the basis of this experience and reexamination of appropriate A-V nodal areas after completion of the electron microscopy, the ultrastructural topography of the A-V node was assessed.

**Results**

**Individual Cellularity of the A-V Node**

The A-V node is distinctly composed of individual cells, and there is no anatomic syncytium. Although cellular boundaries are obvious with electron microscopic examination, they are sufficiently fine so that their existence is easily missed on light microscopic study (figs. 2 and 3), even when sought retrospectively. The nature of the cellular boundaries and intercellular organization will be discussed after each of these features is separately described. The human and canine A-V nodes were so similar that they will not be discussed separately.

**Types of Cells in the A-V Node**

Unlike the cells of the ventricular myocardium, which are of relatively uniform appearance, there are at least four different types of cells in the A-V node. (1) The simplest of these is a rounded or ovoid cell with a relatively smooth surface (figs. 4 to 6). It contains few sarcosomes and myofibrils, both of which are randomly distributed within the cell. In all essential features these simple cells in the A-V node are identical to the P cells of the sinus node, which have been postulated as the sites of pacemaker impulse formation. They have a poorly developed sarcoplasmic reticulum, but there is active pinocytosis at their surface. Although they tend to occur in small groups or clusters, they are also frequently found interposed between transitional cells, but never in direct contact with working myocardial cells. (2) The second type of cell in the A-V node is slender and elongated, resembling miniature working myocardial cells except for a simpler internal organization (figs. 7 to 10). It is by far the most numerous cell type found in the A-V node. Since its internal features are intermediate in complexity between those of P cells and ordinary working myocardial cells, it resembles the transitional cells of the sinus node and will be referred to by that name. The transitional cells join end-to-end and end-to-side to form an interweaving network which comprises the bulk of the A-V node. The continuity of this network is periodically interrupted by single or small groups of P cells. Transitional cells in various sites make contact with all four cell types found in and near the A-V node. In view of the variable complexity of their internal organization, they are the most heterogeneous type of cell in the A-V node (as in the
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sinus node), including some which are nearly as simple as P cells and others which are nearly as complex and intricately organized as working cells. (3) The third cell type encountered resembles in all respects ordinary working myocardium. It is seen only at the atrionodal margin and not within the A-V node proper. Its junctions are with transitional cells, other working myocardial cells, and Purkinje cells, but never with P cells. (4) The fourth cell type is identical to characteristic Purkinje cells, exhibiting relatively few myofibrils which are slender and are arrayed in orderly rows parallel to the long axis of the cell (fig. 11). There is usually a large clear zone about the nucleus. Some of these cells in and around the A-V node are densely packed with sarcosomes, while others which are otherwise of the same appearance have remarkably few sarcosomes. The sarcosome-rich and sarcosome-poor contrast was also observed in some transitional cells. Other details of the internal organization of these and the other three types of cells will be discussed under specific intracellular features.

Membranes

All the cells of the A-V node have a two-layered external membrane, consisting of a distinct electron-dense internal plasma membrane and a less sharply defined and slightly thicker external basement membrane. The appearance of both limiting membranes is identical to that of cells in the sinus node and myocardium generally. While the external membranes of transitional cells, working cells, and Purkinje cells are usually scalloped, with the indentations registered in line with the Z bands of myofibrils, the borders of P cells are usually gentle curves without indentations. Clustered P cells were found less frequently in the A-V node than in the sinus node, but when clustered they sometimes share a single basement membrane which envelopes the group. Two or more transitional cells may also be bridged by a mutual basement membrane. When the basement membrane bridges adjacent cells, there is usually an intercellular indentation at the junctional surface and profiles of interrupted junction are sometimes seen continuing inward at deeper levels (fig. 9).

Intermembrane distances between cells of the A-V node are similar to those of the sinus node. When cells are covered with both membranes, their distance apart is extremely variable and may be considerable, as can be appreciated from light microscopic examination (figs. 2 and 3). Between cells there are collagen, numerous unmyelinated nerve endings, and capillaries. No nerve endings were found to terminate directly on any cell surface in the A-V node, a situation identical to that in the sinus node and in contrast to the specialized nerve terminals on the cell surface in skeletal muscle. There is more intercellular collagen in the adult's A-V node than in the child's, but in both there is much less collagen than in the sinus node. When plasma membranes are apposed without an accompanying basement membrane, the intercellular distance appears constant at about 100 Angstrom units. No tight junctions were found between cells of the A-V node. The usual thickness of the plasma membrane itself was about 80 Angstrom units and that of the basement membrane about 120.

Junctions between working cells and Purkinje cells were through typical intercalated discs. Between transitional cells, and between them and either working or Purkinje cells, there were usually intercalated discs but of rather simple structure (figs. 9 and 10). There were no fully developed intercalated discs between transitional cells and P cells nor between P cells. Between P cells the principal form of junction was plasma membrane to plasma membrane apposition, with a few scattered desmosome-like thickenings in apparent random placement (figs. 4, 5, and 12). Between P cells and transitional cells there were usually more frequent desmosomes on the apposed plasma membranes. Desmosomes were of constant thickness, measuring about 1,000 Angstrom units across both components taken perpendicular to the long axis of the plasma membrane; parallel
to the long axis of the plasma membrane the desmosomes measured one to several thousand Angströms. Since the desmosomes appeared to represent thickening of the plasma membrane, the intermembrane distance was narrowed at these points, although a lucent intermembrane gap of 60 or 80 Angströms was still present.

Sarcosomes

Ultrastructure of the sarcosomes (mitochondria) in our study can only be interpreted within certain limitations because these were postmortem specimens. As would be anticipated, there was postmortem swelling and distortion of many sarcosomes. However, their total number and intracellular distribution, as well as their relationship to myofibrils and other organelles, were still clearly apparent. In the P cells the sarcosomes were sparse in number and randomly distributed without specific relationship to the myofibrils; thus their appearance resembled that of the sarcosomes in the P cells of the sinus node. As myofibrils increased in number and in parallel array in increasingly complex transitional cells, an associated increase in the intracellular organization of sarcosomes was observed, so that in the most complex transitional cells the sarcosomes were lined up in a sandwich array between parallel rows of myofibrils, just as they are in working myocardial cells. In Purkinje cells and transitional cells which were sarcosome-rich, they were packed throughout the cell; in sarcosome-poor cells the sarcosomes were arrayed (in sparse number) in lines between the similarly sparse myofibrils.

One special type of sarcosome (fig. 13) was observed only in P cells or, more rarely, in a few Purkinje cells or transitional cells. It was never present in working myocardial cells. The internal structure of these sarcosomes was simple, with relatively few cristae mitochondriales and little intercristal matrix, and its cross-sectional appearance was often in the form of a rosette with one outer and another inner circle joined by interposed cristae. Although we cannot exclude the possibility that this appearance was due to postmortem change, the following evidence suggests it is not. No such sarcosomes were found in working cells, although a large number obtained and fixed in exactly the same fashion were examined in the study. The almost exclusive presence of this special sarcosome in P cells resembles the findings in the sinus node, in which specimens obtained in the dog were fixed in vivo with glutaraldehyde perfusion of the sinus node directly through its nutrient artery. Even if this finding in P cells of the A-V node is a postmortem artifact, it is clearly one which is found almost exclusively in P cells, which leads us to believe those sarcosomes were initially different in vivo.

Myofibrils

The only unusual feature of A-V nodal myofibrils was their small size and variable distribution in some cells. P cells contained relatively few myofibrils which were small (containing few myofilaments) and randomly oriented rather than in parallel array. Myofibrils of the P cells very rarely inserted at the external membrane, although the boundaries of one or a few myofilaments could not always be defined with certainty. In transitional cells the myofibrils became increasingly arrayed in parallel lines along the long axis of the cells, resembling those of working myocardial cells, and the number of filaments per myofibril increased to a volume comparable to that of myofibrils in working cells. In the Purkinje cells the myofibrils were regularly fewer in number than in working cells, although the cross-sectional diameter of Purkinje cells was usually greater, and each myofibril contained relatively few myofilaments. The pale appearance of Purkinje cells (due largely to sparse and thin myofibrils) and their perinuclear clear zone are the distinctive feature on light microscopic examination which is now more precisely definable from the ultrastructural details.
Sarcoplasmic Reticulum

As with interpretation of the sarcosomes, one must consider possible postmortem artifactual distortion of the sarcoplasmic reticulum. However, previous studies\(^6\) have suggested that such distortion is minimal in myocardial cells for many hours after death. The P cells of the human A-V node contained relatively little sarcoplasmic reticulum, as is also true in the human sinus node.\(^4\) Pinocytotic vesicles were numerous along the plasma membrane of P cells both on the external surface and along intercellular apposed plasma membranes. Transitional cells contained increasingly well-developed sarcoplasmic reticulum, as well as elements of transverse tubular system in register along the Z bands (fig. 14). The sarcoplasmic reticulum of working myocardial cells and Purkinje cells was fully developed. Golgi apparatus was not identified in cells of the A-V node, although this may in part be due to limitations of postmortem study. Both smooth and rough sarcoplasmic reticulum was seen. Large empty vesicles were present in the cytoplasm of some of all four A-V nodal cell types, probably representing dilated cisternae or sinks of sarcoplasmic reticulum.

Nuclei

In P cells the nucleus occupies a disproportionately large percentage of the cross-sectional area of the cell. Nuclear chromatin was extensively clumped as would be anticipated in postmortem examinations. Occasional nucleoli were observed. The nuclear membranes were similar to those of the external cell surface, containing a thinner but more distinct inner layer and thicker more diffuse outer layer. Nuclei of the P cells were generally round or ovoid, while those of the other three cell types tended to be elongated in the long axis of the cell. All nuclei were centrally located within the cells.

Other Organelles

Perhaps the most striking feature of P cells is their "empty" cytoplasm, with few organelles. No centrioles were found in P cells of the A-V node, although a few were found in the sinus node. Lysosomes (figs. 8 and 11), which were at least as frequently seen in A-V nodal cells as in the sinus node, varied a great deal in size and internal contents and tended to occur in small intracellular groups. Lysosomes were rarely seen in P cells. Various other structures with single or multiple membranes, concentrically arranged, were observed, but their purpose and function are unclear. Except for scattered small areas of cytoplasmic granularity which may have represented glycogen, none was found in this postmortem study.

Synthesis of Ultrastructural Anatomy of the A-V Node Based on Light Microscopic Experience

One problem in interpreting the fine structure of the A-V node is inherent in electron microscopic study of any biological unit or organ with heterogeneous cells having variable groupings and organization. With current technology the specimens for study are so small and their precise origin and orientation in the original tissue so difficult to ascertain that one is left with a formidable puzzle. In the present investigation we believe that accumulated previous experience with light microscopy of the A-V node, plus a retrospective reexamination of pertinent light microscopic features after ultrastructural aspects were available, permits an accurate assortment of fine structural details as they appear at the atrionodal margins and at various depths within the node proper. By knowing where cells with various fine structural features occur in the A-V node, one may then form a broad synthesis of its ultrastructural anatomy.

From a combined interpretation of its light and electron microscopic appearance, there is no doubt that the bulk of the A-V node is composed of interweaving slender transitional cells. Scattered within this woven network are P cells occurring alone and in small groups. The frequency of P cells is greatest in the deepest portions of the node even though occasional ones are seen at its margins. More characteristically at the atrionodal margins, however, is a junction principally between Purkinje cells (and fewer
cells of ordinary working myocardium) and the slender transitional cells of the A-V node. This appearance is obvious on light microscopy. Some transitional cells may extend as one or more slender fibers for distances of a centimeter from the nodal margin into the atrial septum. As one examines within the A-V node, an occasional Purkinje cell may be seen at the superficial layers but none deeper. There are no working myocardial cells inside the A-V node. The area of His bundle, where other cells with Purkinje characteristics may be anticipated to be present, was intentionally avoided in the present electron microscopic study. Sarcolemma-poor Purkinje cells are probably the more characteristic ones observed with light microscopy and have been found in abundance at the atrial margin of the A-V node among the bypass fibers. Sarcolemma-rich Purkinje cells probably represent a type which on light microscopy appears as intermediate between typical Purkinje cells (light microscopically) and working cells of the atrial myocardium.

Even on reexamination of the light microscopic preparations of the A-V node it is extremely difficult to define cellular borders and boundaries, except for intercalated discs in the larger cells at the A-V nodal margins. This must be due to the paucity of intercalated discs and similarly dense structures among transitional cells and P cells. When the plasma membrane and basement membrane contain no specialized thickenings, they are simply too faintly stained on nearly all light microscopic preparations to be visible. It is no surprise, therefore, that anatomists for so long erroneously considered cells of the A-V node as comprising an anatomic syncytium.

Discussion

Three electrophysiological functions of the A-V node deserve detailed consideration relative to its ultrastructural anatomy. These are pacemaking, atrioventricular conduction delay, and triage of sinus signals. Each of these functions has notably controversial aspects which are familiar to electrophysiologists. Certain functions overlap, such as atrioventricular conduction delay and triage of supraventricular signals. Nevertheless, these and other qualifying points can be considered most clearly if discussed in context with the three functions indicated.

If the P cells of the sinus node are indeed the site of impulse formation, as the correlative electrophysiological and ultrastructural studies of Trautwein and Uchizono suggest, then the presence of similar cells in the A-V node is certainly compatible with potential pacemaking function by the A-V node. Such potential may or may not operate under normal (or perhaps even abnormal) circumstances in vivo, but at least the anatomic observation supports the likelihood. Under normal circumstances it seems likely that pacemaking by P cells in the A-V node may be regularly precluded by arrival of a conducted sinus impulse prior to their own action potential having reached threshold level to fire. Pacemaking activity, detectable under special conditions in NH or H region in electrophysiological experiments, may be due to P cells present in what is anatomically defined as the deepest portion of the A-V node.

A-V nodal pacemaking activity is rarely if ever a normal function, however, while slight delay of atrioventricular conduction occurs with every normal sinus beat. Several ultrastructural features of the A-V node alone or together may account for this delay. From electrophysiological studies it is known that in both the sinus node and the A-V node conduction is relatively slow and at about the same speed of 0.05 m/sec. One ultrastructural feature of both nodes is the presence of P cells, which have very few specialized intercellular junctions. P cells interrupting the continuity of transitional cells at various levels within the substance of the A-V node may serve a "braking" function.

Current evidence indicates that the intercalated disc or certain components of it, such as areas of tight junctions, serve as points of low resistance in myocardial intercellular
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In neither the present study nor those reported by others have any tight junctions been observed within either the sinus node or the A-V node. Transitional cells in the A-V node usually exhibited simple junctions through scattered desmosomes. They are also of small diameter, an anatomic feature previously suggested as a basis for A-V conduction delay. We may also consider the well-developed sarcoplasmic reticulum of the transitional cells. The relatively more frequent "holes" in the external surface of these cells, where the sarcoplasmic reticulum communicates with the extracellular space, may represent potential current leaks into the interior of the cells at the expense of efficiency of longitudinal conduction.

In a recent review we have discussed the triage function of the A-V node, which consists of sorting and filtering the number and sequential order of supraventricular impulses. The same mechanism which is responsible for the normal delay in atrioventricular conduction undoubtedly accounts for much of the triage function, for example, in determining the maximal number of beats allowed to pass during atrial fibrillation. However, one of the mysteries in investigative electrocardiography has long been why the ventricular response during atrial fibrillation should be irregular if the delay function of the A-V node was a stable and consistent factor. Based on its light microscopic and ultrastructural appearance, there are two anatomic factors which may help account for this variable period of delay for succeeding cardiac cycles in atrial fibrillation: (1) several available input sites for the A-V node at its atrial margin, and (2) the inhomogeneity of its internal structure. The variable possible input sites for the many atrial signals which are inevitably arriving at the nodal margin during atrial fibrillation mean the node must become depolarized in several different fashions, depending on which point on its surface is stimulated earliest and whether this same point has recovered its excitability following a previous stimulus. Even if the interior of the node were perfectly homogeneous (which it is not), a signal arriving at the node through fibers of the bypass region would enter the nodal margin at a point beyond which there was simply less A-V nodal tissue enroute to the His bundle than there is if a signal had arrived at the nodal crest. However, since the interior of the node is distinctly not homogeneous, whether a signal traversed the node with relatively more or less speed also depends on how many nonspecialized junctions it must pass between cells.

Although classic electrocardiographic concepts hold that there is no longitudinal dissociation or separation of impulse conduction in the His bundle and that it always conducts on an all or none principle similar to a single solid wire, we have recently reviewed other evidence that is against this concept. Such evidence includes among other things the longitudinal array of cells in the His bundle, the collagen septa normally present between these cells, and the electrophysiological significance of unspecialized lateral contact (or no lateral contact) between His bundle cells but the abundance of specialized longitudinal contact (intercalated discs). If it is conceded that longitudinal dissociation of impulse conduction in the His bundle may be a normal physiological phenomenon, then the form of excitation front leaving the A-V node to enter the His bundle becomes a factor of critical importance. The ultimate delivery of the stimulus from the His bundle to the ventricles and the form of the QRS complex would depend directly on the shape of the front arriving at the His bundle, and this in turn would depend at an earlier stage on the filtering or triage function of the A-V node. For the triage function of the A-V node, the inhomogeneity of its component cells must be a major factor determining not only the speed of A-V nodal conduction but also the shape of the depolarizing wave front.
References


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Figure 1
A light micrograph of normal human A-V node is shown to illustrate the region (enclosed by the black line rectangle) removed for electron microscopy. Magnification in this and subsequent illustrations is indicated by a black bar and indicated measurement. MV and TV are mitral and tricuspid valves, and IAS is interatrial septum. Goldner trichrome stain.
Figure 2

This light micrograph demonstrates the typical interweaving and interconnection of slender fibers characteristic of the light microscopic appearance of the human A-V node. Although most fiber junctions are by slender cells end-to-end or end-to-side, at some junctions different cells are seen which are better defined in figure 3 and in the electron micrographs as P cells. Goldner trichrome stain.
In these two light micrographs compare the similarity of P cells from the sinus node (SN) and A-V node (AVN) in man. Although the cell membranes limiting the P cells are roughly indicated by the arrows, the intercellular junctions cannot be accurately defined with light microscopy even though they are obvious with the electron microscope. Joining each P cell shown are a number of slender transitional cells. Note the sparsity and random distribution of myofibrils in the P cells, and the relatively large nucleus. See figures 4 to 8 for ultrastructural details of P cells.
Figure 4

This low magnification electron micrograph of a human A-V node demonstrates the interrelationship of P cells (P) and transitional cells (T). Only two representative cells of each type are labeled although others are present. The random distribution of sarcosomes and myofibrils and the sparsity of both are obvious in the P cells. By contrast, the myofibrils of T cells are in an orderly array of parallel rows. Other details are described in the text. At the right lower margin a simple junction between two P cells is indicated by the black arrows; the junction consists of mainly plasma membrane in apposition to plasma membrane with some small desmosomes present only at the lower end.
Figure 5

This electron micrograph of human A-V nodal P cells demonstrates some of their junctional relationships. At the lower corner of cell A there is a dense convolution of desmosomes representing a protrusion of another cell into this one, only the tip of the protrusion having been cut in this section. Between cells B and C there is an intermembrane gap for the upper half of their border, but the lower half is direct plasma membrane to plasma membrane apposition, with two small desmosomes indicated by the arrow. Again note the totally random distribution of myofibrils and sarcosomes.
Figure 6

In this electron micrograph the rounded border and simple internal structure of a human A-V nodal P cell are shown. The presence of Z bands in most of the myofibrils indicates this is not simply a fortuitous cross section of a cell at the nuclear level, but that the three dimensional shape of P cells must be spheroidal.
Figure 7

This light micrograph is from the margin of a human A-V node along the area of bypass fibers. Typical Purkinje cells of the bypass area are seen in the upper half, and typical slender A-V nodal fibers comprise the lower half of the picture. Near the middle of the picture some slender A-V nodal cells are seen joining typical Purkinje cells. This figure serves as an orientation for the fine structure of Purkinje and transitional cells and their functions shown in figures 8 to 10.
Figure 8

In this electron micrograph the size and internal features of a transitional cell (A) and Purkinje cell (B) of the human A-V node are demonstrated. The external margins of each cell are indicated by arrows. The myofibrils of this transitional cell are orderly and parallel and contain abundant myofilaments. Those of the Purkinje cell are also parallel, but the myofibrils contain only few filaments. The italic N overlies nucleus, and L indicates one of several lysosomes.
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Figure 9

Branching and junctions of a Purkinje cell and transitional cells of the A-V node are shown in this electron micrograph. Cell A is a transitional cell with its diameter indicated by the arrow with small a; it joins cell B through a simple intercalated disc indicated by a broad arrow. There is a similar disc in the left upper corner at one margin of cell C, which is a branched Purkinje cell. The largest diameter of cell C is indicated by the longest arrow with a small c, and diameters of its two divisions indicated by the two other arrows with small c's. Intercellular spaces between cells A and C and between divisions of C are indicated by asterisks. Continuations of an intracellular cleft cut in several profiles are indicated by the narrow arrows with IC.
Some Purkinje cells taper at their ends, as shown in this electron micrograph of human A-V node. Cell A tapers at the left lower corner to join another cell through several simple desmosomes. Cell B tapers at its upper end to join cell A through a more complex arrangement of desmosomes into a simple form of intercalated disc.
The central portion of a Purkinje cell is shown in this electron micrograph, with a small clear area around the nucleus (N). The largest of several lysosomes is indicated with L. Note the thin myofibrils containing few myofilaments. Part of a transitional cell is seen in the left upper corner and another Purkinje cell in the right lower corner.
Two sarcosome-rich transitional cells (A and C) have an interposed P cell (B) in this electron micrograph of human A-V node. The irregular outline of the P cell here is unusual and probably represents artifactual distortion. Its junction with cell A is outlined by the two small open arrows, which indicate single desmosomes, and the group of desmosomes indicated by the broad black arrow. Two areas of intercellular clefts are indicated by the asterisks. A less distinct junction between the P cell and another sarcosome-rich transitional cell is visible at the lower right margin of the illustration.
Figure 13

Five transitional cells (A-E) with very simple internal features are shown in this electron micrograph of human A-V node. Boundaries of cell D at its narrow and broader margins are indicated by the open arrows. The special sarcosomes found in some cells of the A-V node are indicated by the thin arrows with Sa; a number of other unlabeled similar sarcosomes can be seen.
The sarcoplasmic reticulum and transverse tubular system of a Purkinje cell are shown in this electron micrograph of human A-V node. The two layers of external membrane are labeled BM (basement membrane) and PM (plasma membrane). Lines of intracellular tubules are cut in profile along the Z bands of the myofibrils, three lines of which are roughly indicated by the broad black arrows. Collagen fibrils are seen outside the cell at the top. Postmortem distortion of the sarcosomes is present.
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