Complete heart block as a consequence of atrionodal discontinuity

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We have recently studied a case of complete heart block in which there was considerable difficulty in deciding whether it was of congenital or acquired origin. This was because the heart block was first discovered at the age of 2 years in the course of acute diphtheria. Though diphtheritic infections are known to affect the cardiac conduction system, authenticated cases of post-diphtheritic block persisting after the infection are rare, and, furthermore, histopathological study in this case revealed discontinuity between the atrial tissues and the more peripheral parts of the atrioventricular conduction tissues. This has been more commonly observed in congenital cases of complete heart block and it has been postulated on theoretical grounds that this could be the basis for congenital heart block; on the other hand, it has been noted in a single case thought to be of acquired origin. Review of the evidence available failed to allow accurate classification of the case into either congenital or acquired categories. The normal, segmental development of the atrioventricular node, each segment being of different embryological origin, is discussed and the case presented is understandable in the light of this.

The distinction between congenital and acquired heart block, while of considerable clinical importance (Ayers, Boineau, and Spach, 1966), is often difficult to make. This difficulty is illustrated by a recent case we have studied, in which heart block was first observed at the age of 2 years in the course of acute diphtheria. According to the criteria of Yater, Lyon, and McNabb (1933), this history would automatically categorize the case as acquired. However, an unusual type of atrionodal discontinuity was noted on histopathological examination. According to the recent review of congenital heart block by Carter, Blieden, and Edwards (1974), 4 cases of this variety of block were of congenital origin. To these cases should be added the case of Lev, Benjamin, and White (1958). However, in the opinion of the latter authors, a similar arrangement could have resulted from acquired disease, as illustrated in the case reported by Oppenheimer and Oppenheimer (1914). The mechanism for development of this variety of heart block becomes readily understandable on the basis of the concepts of normal nodal architecture and development espoused by Truex and Smythe (1965, 1967), Anderson and Latham (1971), and Anderson and Taylor (1972). Indeed, such block had been predicted on theoretical grounds by Kung and Mobitz (1930) and Mahaim (1931). None the less, it is less readily comprehensible on the basis of nodal structure and formation as propounded by James (1961, 1970). In view of these considerations, we are reviewing the possible morphogenesis of this case and other varieties of heart block in terms of presently demonstrated architecture of the normal atrioventricular node.

Case report

The patient was a girl, born in 1932 after an uneventful pregnancy and normal delivery. No hospital records were available, but she was apparently normal until she contracted scarlet fever at the age of 2 years followed by diphtheria at the age of 2½ years. She was said to have been very ill, and was found to have heart block at the time. In her recovery period she had temporary paralysis of one arm and the face on the same side. A detailed cardiological investigation was not made at this time. She subsequently made a complete recovery and remained well during the rest of her childhood. There was no restriction of her physical activities. At the age of 15 years she complained of episodes of tiredness, vertigo, and breathlessness. She was noted to have a slow pulse and complete heart block was again diagnosed.
From that time she was under periodic medical supervision and though her pulse remained slow (about 40 per minute, range 34 to 54), at no time did she have any symptoms or require any treatment. An electrocardiogram (Fig. 1) showed complete dissociation of atrial and ventricular activity. There was also evidence of partial left bundle-branch heart block but the QRS complexes were of normal duration. However, she led a normal and active life, married, and had three children after uneventful pregnancies. She engaged in full-time employment, by choice rather than necessity, in addition to caring for her home and family without domestic assistance. In October 1971 she returned home from work, apparently quite well. She sat down in her living room and chatted normally with her husband. He left her momentarily, and on return approximately one minute later, she was dead.

**Necropsy findings**

Significant changes were confined to the cardiovascular system. The heart was enlarged (450 g) and showed slight dilatation of the left ventricle with endocardial thickening. No other gross abnormalities were noted. Blocks were removed from various parts of the heart for microscopical examination. The conducting tissues were studied after the method outlined by Davies (1971). Blocks were taken to include the sinus atrial, the atrioventricular node, the bifurcation of the atrioventricular bundle, and both proximal bundle-branches. These blocks were embedded in paraffin and serially sectioned at 5 micron thickness. One section in each 50 cut was mounted and stained with either haematoxylin and eosin, Masson’s trichrome, van Gieson’s, or Orcein elastic techniques.

**Sinuatrial node** This structure was absent. The nodal artery was observed in its usual situation, but it was surrounded by only adipose tissue (Fig. 2A). Small bundles of plain atrial myocardium were dispersed among the adipose tissue, but specialized cells as expected in the node were not observed. There was interstitial fibrosis of the nearby crista terminalis.

**Atrioventricular node** The atrial approaches to the node were normally formed, and became attenuated and separated by fibrous tissue as they approached the fibrous anulus. However, they made no contact with the atrioventricular nodal tissue. The latter was exceedingly hypoplastic, and was represented only by a small bundle of cells orientated parallel to the atrioventricular ring, and closely adherent to the central fibrous body. When this bundle was traced from posterior to anterior, it was found to originate within the fibrous anulus towards its mitral extremity (Fig. 3A); it was particularly poorly formed in this position. As it passed anteriorly it emerged from the fibrous ring, and lay on the right atrial aspect of the central fibrous body (Fig. 3B). It then passed anteriorly and to the left, penetrating the fibrous ring (Fig. 3C). Having pierced the ring, it assumed a position on the crest of the ventricular septum, lying postero-inferior to the membranous septum (Fig. 3D).

Throughout its atrial course, this nodal structure made no contact with atrial myocardium, being separated from the atrial cells by a considerable amount of adipose tissue (Fig. 3A–C). The atrioventricular nodal artery and vein were observed coursing through this adipose tissue, but the intermediate portion of the node was absent (Fig. 3B, C).

**Bifurcation and bundle-branches** Having reached the ventricular septum, the bundle passed forward and expanded rapidly. It bifurcated in the expected site, and gave rise to a well formed right bundle-branch, which passed distally in normal fashion (Fig. 2B). The left bundle-branch was less well formed. Far fewer cells were observed than would be expected in the normal left bundle-branch, and they were widely separated by connective tissue. When traced distally the conducting fibres were observed to be involved in the thick endocardial fibrosis of the left ventricle. This thickening had the typical histological appearance of endocardial fibroelastosis. There was also slight generalized adaptive-type hypertrophy of the myocardium, the innermost fibres being involved in the endocardial fibrosis.

**Relation to normal nodal architecture** In previous presentations it has been suggested that the human atrioventricular node possesses a laminar architecture (Anderson and Latham, 1971; Anderson and Taylor, 1972), being composed of superficial, intermediate and lower nodal segments.

The course of the nodal tissue presently demonstrated is similar to that of the lower nodal segment described in these reports. However, though this concept of nodal architecture bears close comparison with that described by Truex and Smythe (1965), it is by no means widely accepted. For instance, it differs in several important respects from the concept espoused by James (1961). Therefore, in order to illustrate the laminar concept for comparative purposes as presently required, we will describe the architecture of the normal node as it appears from our studies.

**Structure of normal atrioventricular node** The histologically distinguishable specialized cells of the node can be divided into superficial and deep portions, the deep portion itself being composed of the intermediate and lower segments. The superficial segment represents the terminal portions of the atrial myocardium. These terminal fibres, which may also be termed transitional cells, are distinguishable from the working atrial myocardium of the septum by virtue of their smaller size and the fact that they are separated into small bundles by connective tissue septa (Fig. 4 and 5). Such fibres are recognized in three main areas. A posterior group of fibres approaches the nodal area from the region of the coronary sinus. These fibres pass beneath the sinus from the lower extension of the crista terminalis, and run forwards above the sinus from the sinus septum. The other two groups approach from the atrial septum, and occupy the area beneath the fossa ovalis.
FIG. 1 Electrocardiogram from the case reported. Note the complete dissociation of atrial and ventricular activity. The QRS complexes are narrow, indicative of the presence of a high ventricular pacemaker. There is evidence of partial left bundle-branch block.

FIG. 2 Photomicrographs of histological sections from the reported case. Fig. 2A is taken from the expected site of the sinusatrial node. The nodal artery is present, but is surrounded only by adipose tissue and occasional plain atrial fibres. Specialized cells typical of the sinusatrial node were not identified. Fig. 2B is taken through the bifurcation of the atrioventricular bundle. The bundle (AVB) is present astride the muscular septum (SM) and beneath the membranous septum. Note the well-formed right bundle-branch (RBB). The left bundle-branch (LBB) is less well formed, and contains fewer fibres than would be expected in a normal left bundle-branch.
FIG. 3 Photomicrographs taken through the atrioventricular junctional region of the reported case. Fig. 3A is posterior, the figures then run anteriorly to Fig. 3D which is taken just before formation of the atrioventricular bundle. Note that throughout the extent of the junctional area, there is no contact between the atrial myocardium (AM), which shows specialized characteristics in its distal segment, and the poorly formed atrioventricular node (AVN). The nodal fragment is buried posteriorly in the tissue of the fibrous annulus (FA), as seen in Fig. 3A. When traced anteriorly it emerges from the annulus, expanding in size (Fig. 3B and C), and lies in the right atrium. It is separated from the atrial myocardium by adipose tissue (AT), through which courses the artery and vein to the node (AVA and V).
FIG. 4 Photomicrographs of the atrioventricular node of a midterm human foetus processed to demonstrate cholinesterase activity as reported by Anderson and Taylor (1972). Fig. 4A is considerably posterior to Fig. 4B. Note that in both sections of the node, three segments are distinguished by their reaction to cholinesterase. In the posterior section (Fig. 4A), the two deep segments are ChE positive. The more intensely positive segment (LNS) is related to the mitral side of the fibrous annulus (FA), while the other deep segment (INS) is related to the tricuspid valve (TV). The distal segments of atrial myocardium are well innervated, and constitute the atrial segment of the node (ANS). In the anterior section (Fig. 4B), the three segments overlie each other, and an intensely positive lower portion (LNS), a less well-stained intermediate portion (INS), and a well-innervated atrial portion (ANS) are seen. Note that part of the lower segment is beneath the fibrous annulus, astride the muscular septum (arrowed).
FIG. 5 Sections from an atrioventricular node from a 4-year-old child, killed in a drowning accident. In this node the three segments are clearly delineated. The atrial segment (curved arrow) intervenes between the atrial myocardium and the deeper nodal segments. Note that the transition is made below the level of the tendon of Todaro (TT), and that the atrial specialized cells communicate with both the superficial right-sided (SS) and deeper left-sided (DS) segments of the atrial septum. The lower nodal segment (closed arrow) starts posteriorly (Fig. 5A) within the substance of the fibrous annulus, on its mitral side (MV). When followed anteriorly (Fig. 5B to D) it runs rightwards and merges with the intermediate segment (open arrow). The lower segment forms the greater part of the atrioventricular bundle.

Note that in Fig. 5D the lower segment is close to making a direct connexion with the left side of the interventricular system. The intermediate segment (open arrow) starts posteriorly on the tricuspid side of the annulus, and expands anteriorly (Fig. 5C and D) where it contacts the lower segment, forming the connexion between atrial segment and atrioventricular bundle.
The transitional cells pass into the node in two layers, and connect the node with the deep and superficial parts of the atrial septum. Both groups connect with the septal musculature in front of and behind the fossa ovalis, but the deeper group is mainly in contact with the anterior limbus of the foramen, but on the left atrial side of the septum (Fig. 5). These fibres pass directly into the deeper segments of the node.

The superficial transitional cells stream down from the septum throughout the anteroposterior extent of the node. At their point of contact with the atrial fibres they are further subdivided by the presence of the tendon of Todaro, which runs anteroposteriorly through this group of fibres. When traced inferiorly the superficial transitional cells mostly pass over the deeper nodal segments to reach the tricuspid valve base, and form the well-demonstrated superficial segment (Fig. 4 and 5). The fibres do, however, contact the deeper nodal segments via interconnecting transverse strands. We have not observed these fibres contacting the node or bundle once it has become encapsulated by the fibrous ring (Fig. 5). All these atrial nodal, or transitional fibres are distinguished in foetal specimens by their rich innervation (Fig. 4).

The deeper nodal segments can be considered to constitute the 'compact' atrioventricular node. In foetal specimens they are distinguished by their cholinesterase reaction. The lower nodal segment is intensely cholinesterase positive, the intermediate segment less so (Fig. 4). In children the intermediate segments can be traced forwards from the tricuspid annulus side of the node. In contrast, the lower nodal segment is embedded posteriorly in the mitral side of the fibrous annulus (Fig. 5A). When the two segments are traced anteriorly, the intermediate segment is seen to receive the bulk of the posterior nodal input fibres (Fig. 5B). In this situation it may still be separated from the lower nodal segment by a fibrous septum (Fig. 5B and C). Only at the anterior extent of the node may the fibrous tissue disappear, and then the two nodal segments make contact (Fig. 5D). When traced posteriorly the atrioventricular bundle is observed to be directly continuous with the deep node. However, at the stage at which the deep node becomes surrounded by the fibrous body and separated from the atrial tissues, both intermediate and lower segments are usually still recognizable.

We have not observed contact with atrial fibres anterior to this point. Thus in the normal node, the atrial input fibres impinge mainly on the intermediate segment, the most direct input being from the left atrial side of the septum. The intermediate segment in turn contacts the lower nodal segment, and both are directly continuous with the atrioventricular bundle. The point at which the node becomes the bundle is difficult to make on histological criteria. We have considered the perforating bundle to start at the point at which the node becomes surrounded by fibrous tissue and separated from the overlying atrial tissues. It is important to appreciate that the proximal end of this penetrating bundle has nodal histological characteristics. The normal architecture as presently described is illustrated diagrammatically in Fig. 6.

**Discussion**

The histopathological findings in the case of complete heart block described can be correlated with both the recorded electrocardiographic patterns, and with the absence of the intermediate segment noted in the normal node. The complete lack of contact between the atrial myocardium and the poorly formed atrioventricular node is adequate explanation of the atrioventricular dissociation. The well-formed penetrating atrioventricular bundle and bifurcation correlates well with the narrow QRS complexes, themselves indicative of a high ventricular pacemaker. The poor formation of the left bundle-branch is consistent with the partial left bundle-branch pattern of the electrocardiogram.

With regards to correlation with nodal architecture, we believe that the strand of tissue, which extends through the fibrous annulus, lies on its right surface, and then tracks leftward before becoming re-embedded in the fibrous ring, is directly comparable with the lowermost of the three nodal segments.

In the normal node this segment is that which is most cholinesterase positive. By use of this histochemical reaction, it has been shown to veer to the left, closely adherent to the fibrous annulus. Furthermore, in other normal specimens, it has been shown that it may too become re-embedded in the fibrous ring posteriorly (Fig. 4 and 5). Though this part of the node is clearly present in the heart block case, we believe that the intermediate nodal segment is absent. Since this segment has been shown to form the junctional segment between superficial atrial fibres and the deep segment, its absence prevents the atrial fibres contacting the node. We are thus able to correlate our findings with clinical and anatomical evidence. However, it is less easy for us to use this evidence to classify the heart block as either congenital or acquired.

The distinction between acquired and congenital categories of heart block was first introduced by Davis and Stechter (1928). Its clinical usefulness has been endorsed in the recent review of Ayers et al. (1966). These authors have stressed that congenital block usually has a better prognosis, since in most cases narrower QRS complexes, faster ventricular rates, and hence fewer Adams-Stokes attacks are observed than in cases of the acquired variety. For these reasons the clinical opinion in our case was in favour of a congenital origin, preferring to regard the diphtheritic infection as coincidental. The histopathological findings would, at first sight, also favour a congenital origin. As Lev et al. (1958) indicated, only one case of acquired atrionodal discontinuity has been recorded (Oppenheimer and Oppenheimer, 1914). Furthermore, in
most histologically studied cases of chronic complete heart block, the lesions tend to be distal and to affect the bundle-branches (Davies, 1971). In all his considerable experience, Davies considered that he had not observed a case of acquired origin with findings comparable to those presently reported (1974, personal communication). However, most cases of acquired heart block are the result of chronic illness, whereas in our case it is unlikely that the block resulted from chronic illness. It would be helpful if we were able to make a direct comparison with histopathological findings in previous diphtheritic cases. However, histopathological studies of diphtheritic involvement of the conducting tissue are rare. James and Reynolds (1963) described findings in a case which terminated fatally in the acute period with complete heart block. In this case the conducting tissues were widely destroyed together with the cardiac autonomic nerves, but selective destruction of parts of the conducting tissue was not observed. However, Davies (1971) has mentioned the predilection of acute diphtheritic infection for conducting tissues, while Engle (1949) suggested that the toxin might be concentrated in the atrioventricular node and its branches. Such selective concentration may indeed be consistent with diphtheritic infection producing destruction of only the intermediate nodal segment. Furthermore, review of the authenticated cases of permanent post-diphtheritic heart block (following the suggestions of Griffith and Herman (1952) concerning authentication) shows that their history is not too dissimilar.

**FIG. 6** Diagrammatic representation of the nodal architecture demonstrated histologically in Fig. 4 and 5. The top left hand figure shows the AV junctional area viewed from its right aspect, while the lower left hand figure shows a plane view of the junction. The two right hand figures are frontal sections through the plane a-a and b-b indicated in the left hand figures. The areas occupied by the nodal segments are indicated by the hatched areas.

Abbreviations: LNS lower nodal segment, INS intermediate nodal segment, SNS superficial nodal segment, FO foramen ovale, SS sinus septum, CS coronary sinus, TV tricuspid valve, MV mitral valve, TT tendon of Todaro, S2° septum secundum, S1° septum primum, VS ventricular septum, TO tricuspid orifice, MO mitral orifice, pb penetrating bundle, lbb left bundle-branch, rbb right bundle-branch.
from that of the presently reported case. The cases of Sprague and White (1927), Perry (1939), and Slama and Temkine (1966) all had survived for considerable periods with few problems and no Adams-Stokes attacks. They also had narrow QRS complexes and high ventricular rates. It is interesting that, like our case, all were female. In view of the equivocal nature of the evidence reviewed above, we consider that we are still unable to determine whether the block in our case was congenital or post-diphtheritic in origin.

We are able, however, to consider our findings in relation to concepts of both atrioventricular development and the pathogenesis of heart block, the two being closely related. Though the concept of a laminar node differs from the descriptions of nodal architecture provided by James (1961), it is by no means original. When Tawara (1906) first described the atrioventricular node, he indicated that it was difficult to distinguish the node from the bundle, and that the atrial part of the node was itself divided into two components, which can be interpreted in terms of the superficial and deep segments presently described. Furthermore, his illustrations clearly demonstrate the sequestration of what we have termed the 'lower nodal segment'. In a previous publication one of us (R.H.A.) had stated that the description of Tawara was not in keeping with the present findings (Anderson and Taylor, 1972). We are now aware that this opinion represented a misinterpretation of Tawara's study. Subsequent German morphologists continued to describe atrial and ventricular components of the node, and their studies have been reviewed by Schiebler and Doerr (1963). However it is difficult to be sure of the exact disposition of nodal components from their description and illustrations and hence we are unable to compare them directly with our findings. In contrast, as we have indicated, the description of Truex and Smythe (1965) is much more comprehensible and it is readily apparent that the present findings are endorsements of their results and their nodal reconstruction (Truex and Smythe, 1967). They described the three nodal segments presently referred to. They also stated that the specialized atrial fibres extended into the atrial septum as three broad bands indicating that others had described these as junctional fibres. They distinguished the two parts of the deep or compact node, but did not indicate their precise disposition. None the less, we believe our present findings are in close agreement with this previous description. To a certain extent the differences between these findings and those of James (1961) are differences of interpretation and emphasis.

However, James does not describe the segmenta-

tion of the compact node and has subsequently expanded his concept of nodal architecture to incorporate specialized internodal tracts (Sherf and James, 1969). We were unable to distinguish such tracts on either histological or histochemical criteria. Furthermore, like Truex and Smythe (1967), we were unable to find the bypass tracts of 'Purkinje' fibres which he described. In this respect, Merideth and Titus (1968) were also unable to distinguish these fibres, yet they have been widely implicated as a possible substrate for ventricular pre-excitation.

It is significant that the nodal architecture presently described is readily explainable on the concept of nodal development propounded by Anderson and Taylor (1972). They hypothesized that the deep part of the node was derived from atrioventricular canal musculature while the superficial part was developed from sinus horn musculature. We would now slightly amend this hypothesis. We still consider that the deep part is composed of canal musculature, but believe that the lower nodal segment is formed on the mitral aspect of the posterior portion of the intraventricular septum and that the intermediate nodal segment is derived from the tricuspid aspect of the atrioventricular canal tissue. It is now our opinion that the superficial nodal segment is derived from the downgrowing parts of the atrial septum, and only its posterior portion from the sinus horn musculature (Fig. 7A). This concept of nodal development is not only supported by the histological findings presently described, but is also endorsed by findings in other congenitally malformed hearts. The malformation in the hearts of particular significance is such as to prevent correct alignment of the interatrial and posterior interventricular septa, either by virtue of absence of the posterior septum (single ventricle with outlet chamber) or because of atrioventricular discordance (classically corrected transposition). In both these conditions it has been demonstrated that the normal node is not formed, because of the absence of its lower nodal portion. The intermediate and atrial segments of the node form a hypoplastic structure in the interatrial septum (Fig. 7B). Atria and ventricles are connected via an anterior node formed from an expanded segment of the tricuspid annulus of the atrioventricular canal musculature (Anderson, Arnold, and Wilkinson, 1973a; Anderson et al., 1974b). Further evidence relative to the concept is provided by yet another malformed heart. In this specimen with atrioventricular discordance, the atrial segment of the node was formed in the morphologically right atrium, yet the lower nodal segment was formed in relation to the inverted posterior portion of the ventricular septum. The node,
therefore, straddled the central fibrous body with the intermediate segment connecting the other two portions (Anderson, Arnold, and Jones, 1972). While all these cases are readily explainable on the theory presently advanced, they are less well explained if one contends that the node is formed in its entirety from either atrioventricular canal musculature (Mall, 1912) or the left sinus horn (Patten, 1956; James, 1970).

The hypothesis is also readily applicable to the pathogenesis of congenital complete heart block. Lev and his colleagues (Lev, Paul, and Cassels, 1967; Lev et al., 1971; Lev, 1972) have suggested that block results following maldevelopment of the central fibrous body. While this is undoubtedly true, it is also clear that fibrous tissue is more likely to form between two structures of different embryological origin rather than to infiltrate into a homogeneous structure. Thus the atrionodal form of block is consistent with malunion of the atrial and ventricular segments of the node, either as a consequence of formation of fibrous tissue between the segments, or failure of formation of one of the nodal segments (Fig. 7C). Furthermore, heart block caused by separation of the formed node from the bundle (Lev, 1972) is explainable on the basis of a lack of union of the nodal conducting tissue with the distal ventricular conducting tissues (Fig. 7D). There is strong evidence that the ventricular elements are formed in situ rather than by a process of migration from the node (James, 1970; Anderson and Taylor, 1972). Finally, the variety of congenital block which occurs in malformed specimens (Lev, 1972) is also explainable either on the basis of malunion of nodal and ventricular tissues, as exemplified by cases of single ventricle (Anderson et al., 1974a), or as a consequence of pathological change in a malformed conducting system, as shown by study of corrected transposition specimens (Anderson et al., 1974b).

In conclusion, we would also state that the findings presently described have significance outside the realm of heart block. In their review of complete heart block, Ayers et al. (1966) referred to the concept of three nodal zones delineated with electrophysiological techniques in the rabbit heart. A more recent review has suggested an importance for the

**Fig. 7** Diagrammatic illustration of the proposed mode of development of the different nodal segments, and their significance to congenital heart block. Fig. 7A shows the normal mode of development. The segments 1–3 combined to form the node, which in turn joins the ventricular specialized tissue formed in situ (segment 4). Note the position of the anterolateral expansion of the tricuspid ring tissue (segment 2A). The inset A1 illustrates the mode of growth of the atrial segment 1 with the down-growing portions of the interatrial septum (IAS). (IVS-Interventricular septum.) Fig. 7B illustrates the mode of formation of the anterior node in single ventricle from the anterolateral expansion 2A. Owing to the absence of the posterior interventricular septum (post IVS), the posterior node (segments 1 and 2) is unable to contact the ventricular specialized tissues (segment 4). Fig. 7C shows the postulated morphogenesis of atrionodal discontinuity. Owing to the absence of the intermediate segment (segment 2), the atrial segment (1) is unable to contact the lower segment (3), which is, however, in contact with the ventricular tissues (segment 4). The inset (C1) shows the site of the atrionodal discontinuity. Fig. 7D shows the "usual" form of congenital heart block in which the node is normally formed from segments 1, 2, and 3 but fails to contact the ventricular tissues (segment 4), possibly because of overgrowth of the fibrous annulus.
concept of three nodal zones in the study of arrhythmias in the human heart (Krikler, 1974). However, diagrammatic representation of the node in this review must be considered as hypothetical in the extreme. It could be argued that conclusions based on the hypothetical are unacceptable in a scientific context. None the less, recent combined anatomical and electrophysiological studies have shown that the delineated zones in the rabbit heart have their morphological counterparts (Anderson et al., 1973b). It may well be that the nodal segments presently described are the basis for a similar arrangement in the human heart. However, further studies are required to validate this possibility and further to elucidate nodal architecture. We are, therefore, continuing our investigation to this effect.

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