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Review

Mahaim, Kent and abnormal atrioventricular conduction

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1. Introduction

In the normal heart, atrioventricular conduction occurs across the specialized axis comprising the atrioventricular node (with its zones of transitional cells), the bundle of His, and the ventricular conduction pathways. This axis, for conduction to be normal, must be the solitary muscular connection between the atria and the ventricles. Should there be additional muscular connections across the fibrofatty tissue plane separating the atrial from the ventricular muscle masses, then the scene is set for the abnormal rhythm known as ventricular pre-excitation. Electrophysiologists have long recognised that there are different patterns of pre-excitation, and controversy has raged concerning their anatomic substrates. There is now little doubt that classical Wolff–Parkinson–White pre-excitation is produced by muscular strands which cross the plane of atrioventricular insulation at some point outside the specialized conduction axis. There is, however, still no consensus concerning the anatomic substrates, and, perhaps more importantly, the nomenclature of so-called "Mahaim" conduction. In this overview, therefore, we have summarized the historical background to this controversy, presenting examples of the anatomic structures we believe to be responsible for the different electrocardiographic patterns.

2. Historical considerations

Although there had been occasional reports [1], and some illustrations, of what we now recognize as specialized areas of cardiac muscle adjacent to the atrioventricular junctions [2], the first detailed accounts describing a specialized atrioventricular conduction axis appeared in the year 1893 [3,4]. These accounts have themselves subsequently proved to be the source of much controversy. While Wilhelm His Jr described a solitary muscular connection extending from the atrial to the ventricular septum [3], Stanley Kent claimed to have demonstrated multiple pathways around the anatomic junctions. Furthermore, Kent believed the multiple pathways he thought he had discovered were responsible for normal atrioventricular conduction [4]. Subsequent to these reports in 1893, nonetheless, even the most experienced anatomist found it difficult to confirm the existence of the purported atrioventricular conduction axis. Thus, Arthur Keith, the leading cardiac anatomist of the time, who shortly thereafter discovered the sinus node [5], describes in his autobiography [6] how he was asked by Sir James Mackenzie to look into the structure of the muscular bundle claimed by His to be the conduit for atrioventricular conduction. Keith admitted to extreme scepticism concerning the existence of such a structure, and had gone so far as to write a letter to *The Lancet* denying its existence. Fortunately for Keith, prior to the publication of his letter, he was given by Mackenzie a copy of a newly published monograph [7]. This monograph was the work of Sunao Tawara, a Japanese working in the laboratory of Aschoff, the latter soon to become one of the most distinguished German pathologists. As Keith states in his autobiography [6], the descriptions in the book of Tawara [7] proved sufficiently detailed and accurate to permit Keith to find again and again the specialized axis for conduction. Tawara had shown how the muscular axis

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Table 1
Nomenclature of European Study Group, 1975

Anatomic substrate	Electrocardiographic manifestation
Accessory atrioventricular connections	
● working myocardium	Classical WPW syndrome
● specialized myocardium	
Nodoventricular and fasciculo-ventricular connections	"Mahaim" physiology
Atriofascicular connections and enhanced atrioventricular conduction	Lown–Ganong–Levine syndrome

extended from transitional atrial cells through the atrioventricular node and bundle of His to the insulated ventricular conduction fibres. In the words of Keith, the work of Tawara ushered in a new epoch in our understanding of the action of the heart.

Yet, brilliant as was the monograph of Tawara [7], not all were convinced of its accuracy. Kent, for example, continued to claim priority for his own mistaken belief that multiple substrates for atrioventricular conduction were to be found in all normal hearts [8–11]. Nor did Tawara's findings entirely complete our knowledge of the nature of the axis for normal atrioventricular conduction. In the 1940's, Ivan Mahaim, with his co-worker Winston, described multiple muscular connections in the normal heart which joined the atrioventricular bundle to the crest of the muscular ventricular septum [12,13]. When discovered, there was no obvious physiological role for these so-called paraspecific fibres. But, with the explosion of electrophysiologic investigations in the late 1960's and early 1970's, the anatomic studies of Mahaim and Winston [12,13], along with those of Kent, attracted renewed interest. As long ago as 1914, the physiologist Mines had argued that the multiple muscular atrioventricular connections proposed by Kent could be the substrate for re-entry tachycardia [14]. When it was eventually shown that the Wolff–Parkinson–White syndrome was indeed due to accessory atrioventricular muscular connections [15], it became the vogue to describe these connections as "Kent bundles", despite the fact that Kent had never, in any of his writings, given any accurate description of the multiple muscular connections he claimed crossed the atrioventricular grooves. Then, when electrophysiological studies showed that there were several variants of ventricular pre-excitation [16,17], it was argued that some of these electrocardiographic patterns could be explained on the basis of anomalous conduction through the paraspecific fibres described by Mahaim and Winston [12,13]. These were the accepted theories when the study group of the European Society of Cardiology presented a nomenclature of the anatomic substrates for pre-excitation (Table 1). In this concept, the paraspecific fibres, known to be ubiquitous in adult human hearts, albeit developed to varying degree, were divided into nodo-ventricular and fasciculo-ventricular fibres [18]. At the same time, the European study group argued that the occasional fibres found extending from the atrial myocardium into the atrioventricular bundle, described by Brechenmacher as atrio-Hisian tracts [19], could appropriately be labelled as atriofascicular fibres [18]. These fibres were held to be responsible for the so-called Lown–Ganong–Levine variant of pre-excitation [20], this

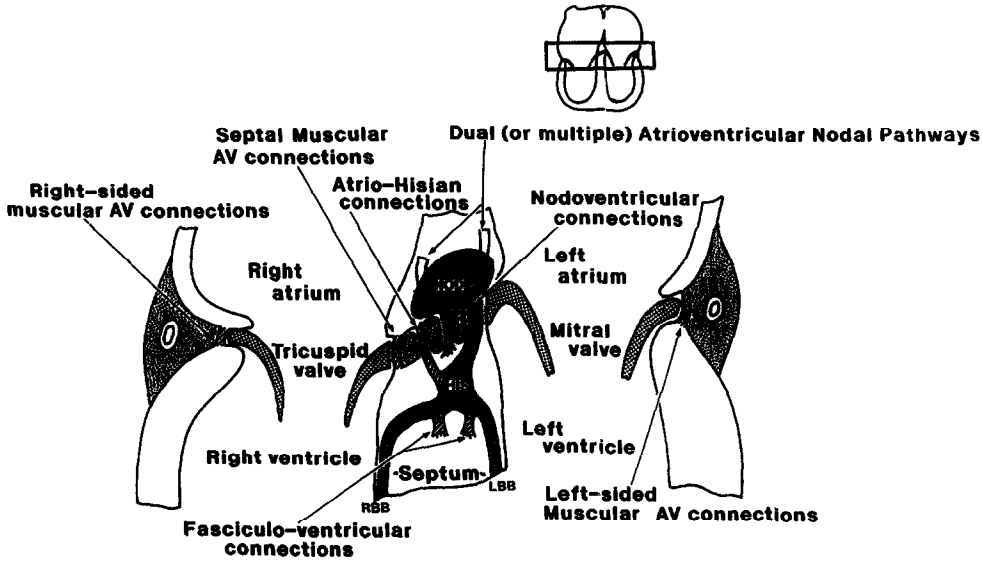

Fig. 1. This idealized diagram is a cross-section across the atrioventricular junctions as shown in the inset, and illustrates conceptually the potential substrates for by-passing the usual delay produced within the specialized atrioventricular junctional area. RBB, LBB — right and left bundle branch.

abnormal rhythm being characterised by a short PR interval but a normal QRS complex, since the impulse was distributed in the ventricles via the normal ventricular conduction axis. The European group also proposed roles for the substrate of Wolff–Parkinson–White syndrome, and for accelerated conduction through the atrioventricular node (Fig. 1).

All seemed well with these explanations until Gillette and colleagues [21] described another variant of pre-excitation in which the anomalous muscular atrioventricular connection was shown to have the property of decremental conduction. Interest in this pathway was kindled by the group from London, Ontario, who removed the pathway at surgery and showed it to be composed of histologically specialized myocardial tissue [22,23]. Subsequent studies from the Canadian group confirmed that this pathway was frequently the substrate for the electrical pattern recognized as "Mahaim conduction" [23]. Thus, in most cases, rather than nodo- or fasciculo-ventricular fibres being the pathway for this type of pre-excitation, the abnormal rhythm was held to be due to an anomalous connection which originated in atrial myocardium and inserted into or close to the right bundle branch. Apparently unaware of the previous use of the term by the European study group to describe atrio-Hisian conduction, Leitch and colleagues suggested that this specialized accessory atrioventricular connection could be termed an "atriofascicular" tract [24]. It so happens that the proximal components of such a tract had already been discovered and illustrated by Becker and his colleagues [25], who pointed to the marked similarity of this specialized atrial structure to the multiple atrial nodes illustrated previously by Kent [9].

Confusion, therefore, still reigns concerning the anatomic substrates for the variants of ventricular pre-excitation. In the paragraphs to follow, we will discuss the electrocardiographic manifestations of these various types of pre-excitation, show the anatomic substrates believed to produce the abnormal rhythms, and propose a modification of nomenclature to remove the paradoxes highlighted above.

3. Accessory muscular atrioventricular connections

All now agree that these are the structures responsible for the majority of cases of Wolff–Parkinson–White syndrome. Although they are sometimes said to pass through holes in the fibrous atrioventricular rings [26], we are unaware of any morphological evidence which substantiates this concept. The connections now known to produce pre-excitation [25] are slender bundles of myocardial fibres which skirt the fibrous rings, running through the fibrofatty tissues of the atrioventricular groove so as to join the atrial and ventricular muscle masses. The first such connection demonstrated anatomically was described by Wolferth, Wood and Geckeler, and was right-sided [15]. The best histological description, nonetheless, was provided by Öhnell for a connection which was left-sided, and which was reconstructed in addition to being demonstrated histologically [27]. Since then, many left-sided connections have been described, virtually all of them being similar morphologically to the original bundle illustrated by Öhnell (Fig. 2). Far fewer right-sided or septal connections have been discovered anatomically, but it is known from clinical studies that such connections can be found at any point around the atrioventricular junctions, and that they can be multiple. In this respect, confusion can result from the suggestion made by clinicians that the septal area of the atrioventricular junction has "anterior" and "posterior" components. Anatomically (Fig. 3), the so-called "anterior septum" is part of the right parietal atrioventricular junction, specifically the area related to the supraventricular crest of the right ventricle. The purported posterior septal area is beneath the floor of the coronary sinus, and is superior to the diverging muscular walls of the right and left ventricles at the crux of the heart. An appreciation of the proper arrangement of these components of the atrioventricular junctions becomes increasingly important for those performing catheter ablation. The true septal component of the atrioventricular junction, with its muscular and membranous components, is relatively small. It corresponds to the area occupied by the triangle of Koch (Fig. 4).

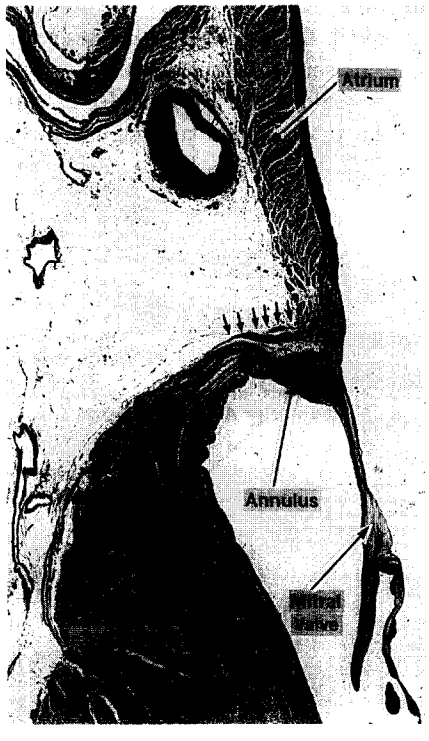

Fig. 2. This histological section shows the typical arrangement of a left-sided muscular accessory atrioventricular connection (arrows). Trichrome stain.

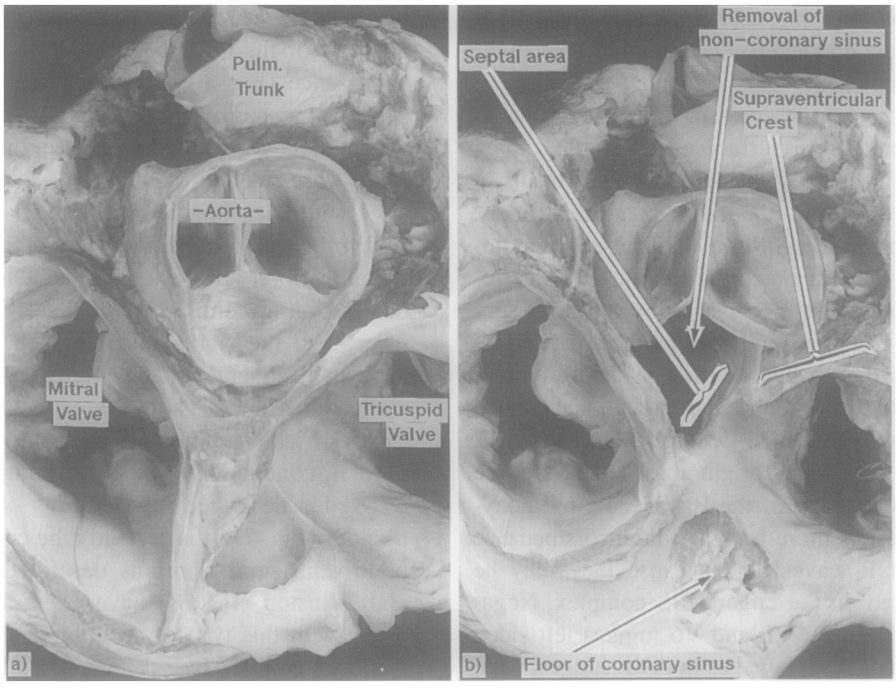

Fig. 3. These dissections, of the atrioventricular junctions seen from above, show that only a small part forms a true septal region. This is shown in (b), prepared by removing the non-coronary aortic sinus. The intact junctions are shown in (a).

The electrocardiogram associated with muscular atrioventricular accessory connections is characterised by a short PR interval (< 120 ms in adults) and a wide QRS complex (> 110 ms in adults). The degree of pre-excitation is variable depending on the atrioventricular nodal conduction time. Any manoeuvre which slows conduction across the atrioventricular node, such as atrial pacing or administration of adenosine, increases pre-excitation. Most tachycardias resulting from these connections involve antegrade conduction over the normal conduction axis, and

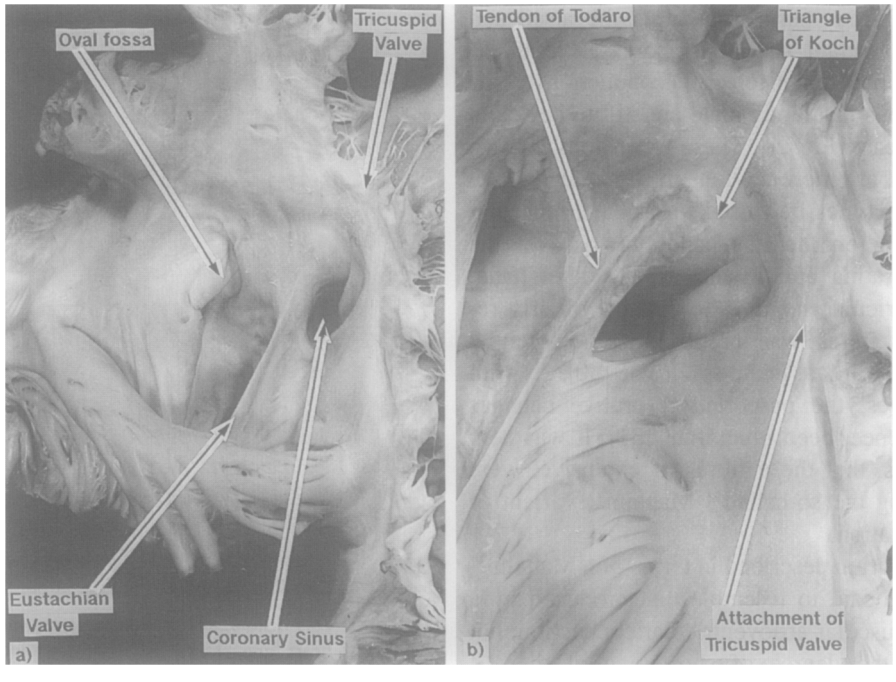

Fig. 4. These preparations show the right atrial structures (a) related to the atrioventricular septum, with further dissection (b) revealing the tendon of Todaro and the landmarks to the triangle of Koch.

retrograde conduction over the accessory connections. This produces a tachycardia characterised by normal configuration of the QRS complexes.

The ventricular insertion of accessory muscular connections may be predicted from surface electrocardiography. Connections which insert into the left free wall result in predominant delta waves in the right precordial leads. Those which insert into the right free wall cause a predominant delta wave in the left precordial leads. The electrocardiogram of pathways close to the atrioventricular septum is much more complex, and less predictive. Certain pathways which insert into the ventricular septum somewhat remote from the atrioventricular groove have been noted to have markedly negative delta waves in leads II, III and AVF [28]. The atrial origin may sometimes be predicted by the morphology of the P wave during tachycardia, but this process is more fraught with errors because of the small size of P waves, and because they may be partially superimposed on the larger QRS complex. Negative P waves in lead I, and AVL and V6 suggest left-sided atrial origins, while negative P waves in AVF suggest right posterior pathways. Right anterior and right anterior paraseptal pathways result in a normal P wave axis during supraventricular tachycardia.

4. Nodo-ventricular and fasciculo-ventricular fibres

Anatomically, these are the muscular structures found in the normal heart which were described initially by Mahaim and Winston [12,13]. Such connections between the underside of the conduction axis and the crest of the ventricular septum are ubiquitous in the fetal heart [29]. In postnatal life, the fibrous tissue separating the atrial and ventricular muscular septal structures increases in thickness and volume, but extensions of conduction tissue permeating the fibrous insulating plate are still to be found in virtually every heart (Fig. 5). These extensions, known also as archipelagos, are more obvious in some hearts than others, and can sometimes be traced to produce muscular connections between, on the one hand, the base of the node and the penetrating bundle and, on the other hand, the underlying ventricular septum. So called "orderly resorption" of these fibres was proposed as a potential mechanism for sudden infant death syndrome [30]. As with the fibres themselves, the purported resorptive changes have, in our experience, been ubiquitous [31]. It was held initially, nonetheless, that these fibres, if extensive, were capable of producing the so-called "Mahaim" variant of ventricular pre-excitation.

The electrocardiogram described in terms of "Mahaim" physiology has been said to resemble that produced by a regular accessory muscular atrioventricular connection, but to be different in the degree of shortening of the PR interval and the degree of pre-excitation. Thus, a normal PR interval with a small delta wave during sinus rhythm was said to be characteristic, with additionally the morphology of the QRS complex in lead I having a rounded appearance (Fig. 6). Presumed Mahaim fibres were also said to occur only on the right side of the septum, thus giving delta waves predominant in the left precordial leads. Many cases of this Mahaim-type tachycardia were pre-excited with a left bundle branch block tachycardia. This was said to be due to antegrade conduction over the presumed Mahaim fibre, and retrograde conduction over the normal conduction system. Many of these fibres were thought to insert into the right bundle branch, despite the fact that such an arrangement had never been demonstrated anatomically. Dual nodal pathways, with the presumed atrioventricular Mahaim fibres arising from one pathway, and the normal conduction system forming the other pathway, were also described on the basis of electrocardiographic tracings (Fig. 7). It is certainly the case that well-structured nodo-ventricular fibres as described initially by Mahaim and Winston (Fig. 8) have been discovered in patients dying with this type of arrhythmia [32,33], but the picture has been clouded by subsequent findings (see below).

5. Atriofascicular fibres

As originally described by the study group of the European Cardiac Society, these fibres, along with acceler-

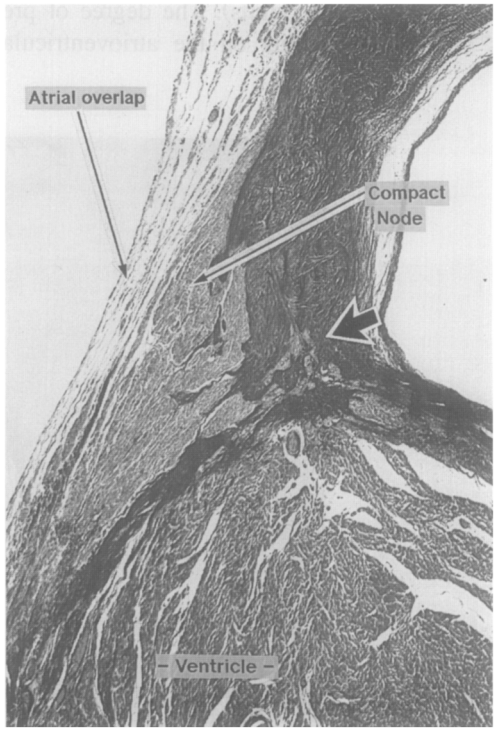

Fig. 5. This histological section is across the atrioventricular node of a normal heart, and shows the extensions into the central fibrous body (arrowed) described as paraspecific fibres by Mahaim and Winston. These are ubiquitous findings, but usually peter out in the fibrous tissue. Trichrome stain.

ated atrioventricular nodal conduction, were held to be responsible for the variant of pre-excitation characterized by a short PR interval but a normal QRS complex. A case with this electrocardiographic pattern had been studied by Brechenmacher [19]. He discovered an anomalous muscular connection which pierced the fibrous collar insulating the penetrating component of the atrioventricular conduction axis (the bundle of His). Histologic characterisation of these fibres, however, is complicated by the marked variability known to exist in normal hearts relative to the mode of entry of the conduction axis into the central fibrous body. For this reason, the European study group suggested that fibres be designated as "atriofascicular" only when they could be shown with certainty to have penetrated the fibrous collar which had already insulated the His bundle from the atrial musculature.

Such connections, which arise in the atrial muscle and insert into the bundle of His, produce an electrocardiogram with a short PR interval and a normal QRS complex. Atrial stimulation fails to result in the normal prolongation of atrioventricular conduction until the refractory period of the atrio-Hisian connection is reached. Supraventricular tachycardia involves antegrade conduction over the atrioventricular node and retrograde conduction over the accessory connection, much like typical atrioventricular nodal re-entry. It is not possible to distinguish with certainty enhanced atrioventricular nodal conduction from that occurring across an atrio-Hisian connection.

The understanding of the anatomy of atriofascicular fibres, nonetheless, has now been further confused by the description of parietal right-sided muscular atrioventricular connections under this title. As we will see, this description has some anatomic justification, but use of the term "atriofascicular" to describe two different anatomic substrates must hold the potential for subsequent confusion.

6. Nodes of Kent

As we described in our introduction, Stanley Kent initially claimed to have described multiple muscular pathways crossing the atrioventricular junctions in the normal

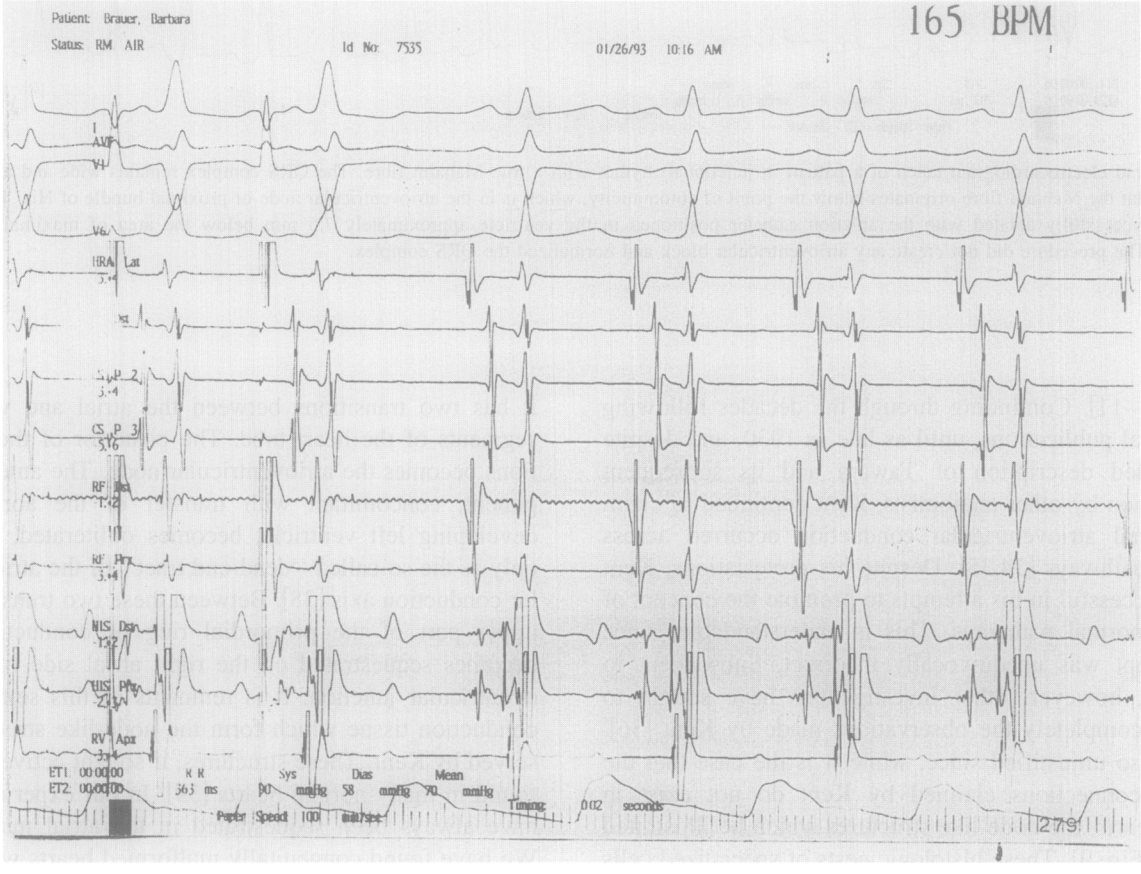

Fig. 6. Surface leads 1, AVF, V1 and V6 recorded simultaneously with high right atrial, coronary sinus, radiofrequency catheter, His bundle and right ventricular apex electrograms. There is a subtle delta wave during sinus rhythm with absence of Q wave in V6. After the first sinus beat, a premature atrial stimulus is given into the high right atrium, resulting in slight prolongation of the P–R interval together with enhancement of the delta wave. On the His bundle tracing, the His bundle depolarization, which has just preceded the QRS, merges into the QRS during the premature stimulation. This is an example of anomalous conduction through a right anterior atrioventricular node-like structure, with the conduction pathway into or near the distal right bundle branch. This pathway was successfully ablated by applying radiofrequency energy to the anterior superior rim of the tricuspid valve annulus with resultant normalization of the QRS complex and prevention of further wide QRS tachycardia.

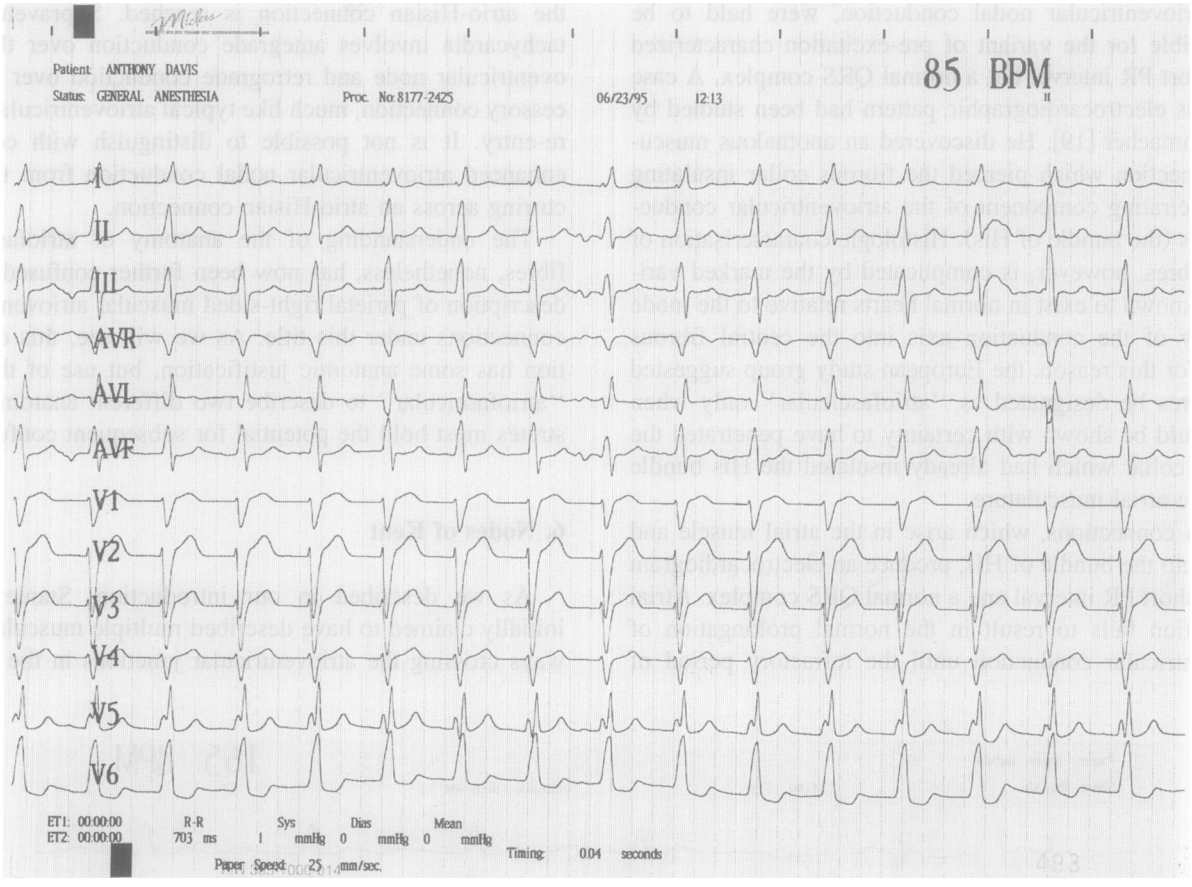

Fig. 7. 12-lead electrocardiogram taken of a patient in junctional rhythm with a true Mahaim fibre. The QRS complex remains wide and a delta wave, indicating that the Mahaim fibre originates below the point of automaticity, which is in the atrioventricular node or proximal bundle of His. This Mahaim fibre was successfully ablated with the ablation catheter positioned in the ventricle approximately 7.5 mm below the area of maximal His bundle deflection. The procedure did not create any atrioventricular block and normalized the QRS complex.

heart [4,8–11]. Continuing through the decades following his original publications, until as late as 1930, and despite the detailed description of Tawara and its subsequent confirmation by other anatomists, Kent continued to claim that normal atrioventricular conduction occurred across multiple pathways [34,35]. Despite his protestations, Kent was unsuccessful in his attempts to promote the concept of multiple normal pathways. This is understandable, since the concept was unequivocally incorrect. Subsequent to that time, however, other investigations have sought to discredit completely the observations made by Kent [36]. This is also unjustified since, while it is the case that the multiple connections claimed by Kent do not exist in normal hearts, the node-like structures which he illustrated do exist (Fig. 9). These histologic nests of specialized cells are remnants of an entire ring of histopathologically specialized tissue which encircles the right atrioventricular junction in the fetal heart [37]. This ring initially surrounds the junction of the inlet and outlet components of the ventricular loop of the developing heart. Part of this ring is not only interventricular, but also atrioventricular. As such, it has two transitions between the atrial and ventricular segments of the heart tube. The posterior of these transitions becomes the atrioventricular node. The anterior component, concomitant with transfer of the aorta to the developing left ventricle, becomes obliterated, persisting only as the so-called ''dead-end tract'' of the atrioventricular conduction axis [38]. Between these two transitions, the upper part of the primordial ring of conduction tissue becomes sequestrated on the right atrial side of the atrioventricular junction. It is remnants of this semicircle of conduction tissue which form the node-like structures observed by Kent. These structures, if sought actively, can be found in many normal hearts [39]. In our experience, they have always been sequestrated in the atrial musculature. We have found congenitally malformed hearts where remnants of the primordial ring of conduction tissue persist as atrioventricular nodes. The exemplar of this arrangement is seen with straddling of the tricuspid valve, where the atrioventricular node can be found at various points around the atrioventricular junction dependant upon where the ventricular septum makes contact with the junction [40].

Anomalous nodes, presumably formed from the primordial ring, are also found in double inlet left ventricle [41] and in congenitally corrected transposition [42]. Intriguingly, dual atrioventricular conduction systems can be found in some

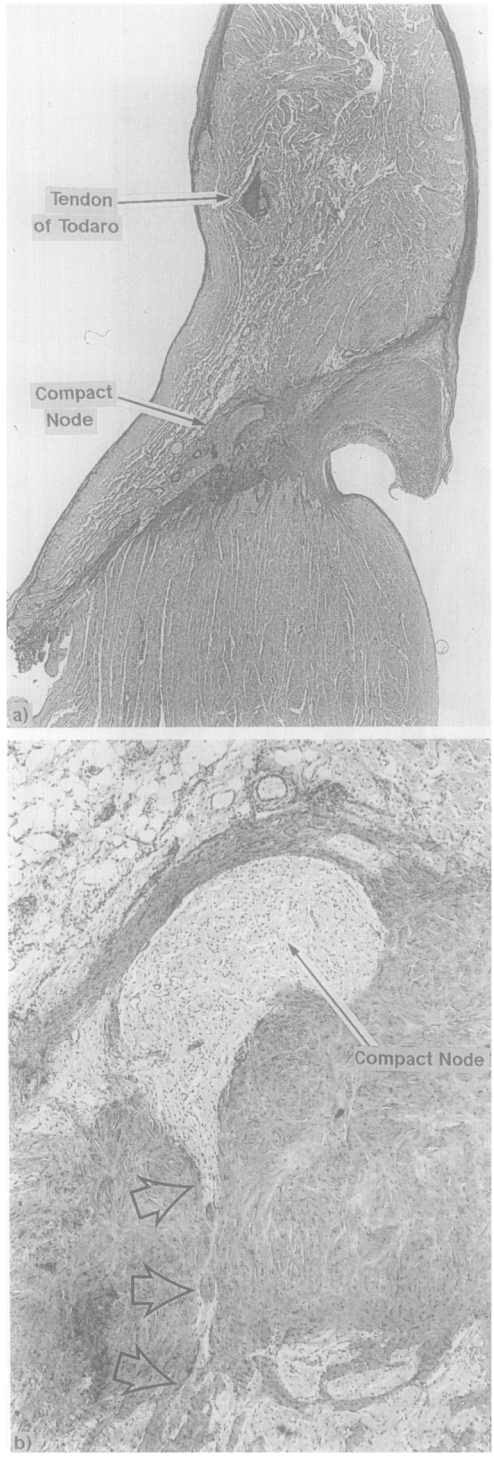

Fig. 8. This pathway (arrowed in b), shown at low (a) and higher (b) magnification, is a persistent paraspecific fibre as initially described by Mahaim and Winston. The patient involved was previously described by Gmeiner and his colleagues [33]. Elastic–van Gieson stain. The nodal tissue is pale in comparison to the dark fibrous tissue in (b).

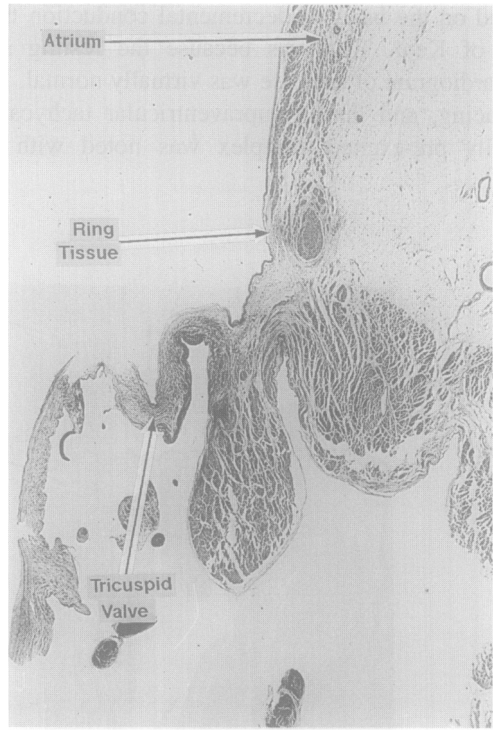

Fig. 9. This section across the tricuspid valve shows a typical segment of atrioventricular ring tissue sequestrated within the atrial myocardium. Trichrome stain.

of these hearts [43], and such a dual system has been observed in one heart known to have shown ventricular pre-excitation [25].

7. Specialized bundles originating in nodes of Kent

As discussed above, although Kent was mistaken in his belief that there were multiple pathways for atrioventricular conduction in the normal heart, this erroneous concept did permit Mines correctly to predict the potential for atrioventricular re-entry [14]. Because of this, when muscular connections were subsequently shown to be the substrate for Wolff–Parkinson–White syndrome, it became conventional to describe them as "Kent bundles". This was despite the fact that, histologically, the accessory muscular connections bore no anatomic resemblance to the structures described by Kent. But, in one heart from a patient known to have ventricular pre-excitation, Becker and colleagues did discover a node of Kent which gave rise to an accessory muscular atrioventricular bundle (Fig. 10). The patient also had Ebstein's malformation, and was shown additionally to possess a typical left-sided muscular atrioventricular connection. At the time, the significance of the specialized connection was not appreciated. As already discussed, Gillette and colleagues [21] subsequently observed a case with the so-called "Mahaim" variant of ventricular pre-excitation which, they argued, could be

explained on the basis of decremental conduction through a node of Kent. This was because the resting surface electrocardiogram of the case was virtually normal. During atrial pacing, and during supraventricular tachycardia, a maximally pre-excited complex was noted with a left bundle branch block pattern. The left bundle branch block pattern was unusually rounded in leads I, AVL and V6. The His bundle potential was seen to occur just before the QRS even during tachycardia. Conduction through the connection was decremental. A surgical incision at the

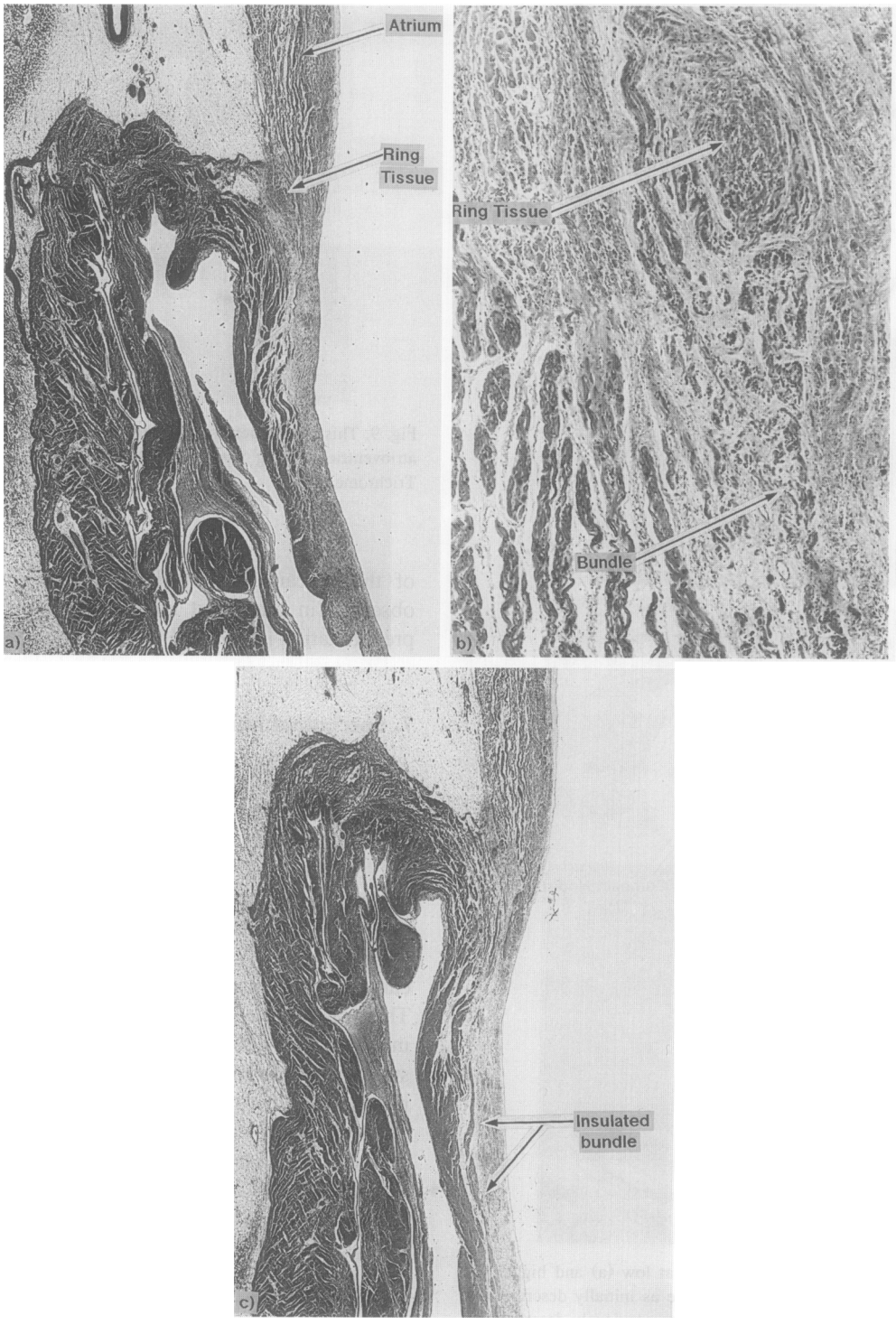

Fig. 10. This series of sections shows a specialized connection originating from a segment of atrioventricular ring tissue. The connection is seen passing into the leaflet of the tricuspid valve. Details of the patient are given in the previous publication of Becker and his associates [25]. Trichrome stain.

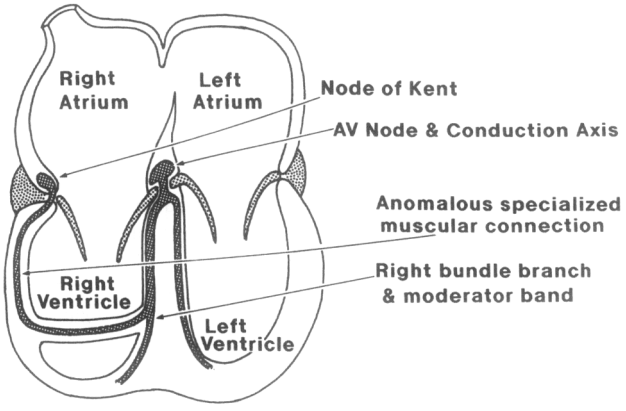

Fig. 11. A potential pathway to account for the so-called "atriofascicular" variant of Mahaim physiology.

atrial side of the right parietal atrioventricular junction abolished both pre-excitation and further episodes of tachycardia. It was postulated that all of these findings could be explained by a connection from right atrium to the distal right bundle branch with an atrioventricular node-like structure on the atrial insertion (Fig. 11). This is exactly the arrangement previously illustrated by Becker et al. [25].

Despite this, it remained the vogue to describe Mahaim physiology on the basis of nodo- and fasciculo-ventricular fibres until, as also discussed above, the group from London, Ontario [23], echoing the earlier experience of Gillette and his colleagues [21], confirmed that the substrate for this conduction occupied a right-sided and parietal position. When removed at surgery, the tissue was shown to have histologic characteristics reminiscent of the atrioventricular node [22]. The group from London, Ontario, hypothesized that the bundles within the ventricle joined up with the right bundle branch and, with some justification, labelled them as "atriofascicular fibres" [24]. As already suggested, nonetheless, this usage can produce confusion with the atrio-Hisian tract, which is unequivocally atriofascicular and had already been labelled in this fashion by the European study group [18].

8. A revised nomenclature for the anatomic substrates of pre-excitation

The advances in electrophysiological knowledge made since 1976 now indicate the need to modify the terminology proposed by the European Study Group on Pre-excitation (Table 1). There can now be no doubt that the classical Wolff–Parkinson–White variant of pre-excitation is produced by accessory muscular atrioventricular connections which, anatomically, bear no resemblance to the structures initially illustrated by Kent. These muscular connections can be situated in left-sided, right-sided or septal locations.

The so-called "Mahaim" physiology can potentially be produced by two substrates. Probably the commonest pathway is to be found at the acute margin of the right atrioventricular junction, but can exist anywhere round the tricuspid orifice. This pathway is histologically specialized, originating in a node of Kent, and inserts either into or in close proximity to the right bundle branch. This structure can be termed a specialized muscular accessory connection, as already proposed in the 1975 nomenclature. The nodo-ventricular and fasciculo-ventricular fibres described by Mahaim and Winston [12,13] can still produce "Mahaim" physiology, but are best accounted for with the descriptive names which are entirely accurate.

What of the cause of the Lown–Ganong–Levine variant of pre-excitation? Because of the confusion now produced by usage of "atriofascicular" to describe the specialized

Table 2
Proposed nomenclature for substrates of ventricular preexcitation

Anatomic substrate	Electrocardiographic findings
Working muscular accessory atrioventricular connection	
● Left-sided parietal	Classical WPW syndrome
● Posterior paraseptal	
● Septal	
● Right anteroparaseptal	
● Right parietal	
Specialized muscular accessory atrioventricular connection	
● Originating in atrial nodes of Kent	● Delta wave with normal PR interval
	● Decremental conduction with programmed stimulation
	● Parietal pathway potentials
Nodoventricular and fasciculo-ventricular fibres	● Delta wave with normal PR
	● Minimal decremental conduction
	● No pathway potentials
Atrio-Hisian fibres	● Short PR — normal QRS
	● Maximal A–H < 100 ms
Unknown anatomic substrate	● Enhanced atrioventricular conduction
	● Short PR — normal QRS
	● Maximal A–H < 100 ms

muscular accessory atrioventricular connections, it is probably best to describe the anatomic substrate for short PR-normal QRS pre-excitation as an atrio-Hisian tract. This then leaves the question of multiple pathways through the atrioventricular node. The sites of ablation used to destroy the so-called "slow" and "fast" pathways show that, without question, parts of these pathways involve the atrial approaches to the atrioventricular node. Findings from cases studied anatomically subsequent to programmed electrical stimulation suggest that the morphologic potential for such dual pathways is ubiquitous [44]. Modifications in terms of anisotropic fibre orientation, differential innervation, or variability in cell-to-cell coupling may underscore the variation in physiologic behaviour. All in all, therefore, the nomenclature proposed by the European Study Group has withstood well the passage of time. With only very limited modifications (Table 2), it sets the scene for understanding the mechanics of arrhythmias as we approach the 21st century.

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