Origin and Significance of Double Potentials Near the Atrioventricular Node
Correlation of Extracellular Potentials, Intracellular Potentials, and Histology

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Background Atrioventricular junctional (AV nodal) reentrant tachycardia can be cured by catheter ablation of the slow pathway, which is part of the reentrant circuit. Previous work has suggested that extracellular double potentials may help identify the site of the slow pathway, but the origin and significance of these potentials are controversial. The aim of this study was to identify the source of these potentials.

Methods and Results Studies were performed in isolated, blood-perfused porcine (n=8) and canine (n=4) hearts. Several methods were used to identify the origin of potentials: microelectrode recording, extracellular mapping, pacing from multiple sites, and light microscopy. Two types of double potentials, similar to those found in humans, were found in all hearts. LH potentials consisted of a low-frequency deflection followed by a high-frequency deflection during sinus rhythm or anterior septal pacing. HL potentials consisted of a high-frequency deflection followed by a low-frequency deflection. LH potentials were found close to the coronary sinus orifice. They were caused by asynchronous activation of the sinus septum and the region between the coronary sinus orifice and tricuspid annulus. HL double potentials were found along the tissues of origin of these potentials. A secondary aim was to use microelectrodes to examine cellular electrophysiology of the perinodal region in blood-perfused large mammal hearts, since previous studies have been limited to superfused preparations.

Conclusions LH potentials are caused by asynchronous activation of muscle bundles above and below the coronary sinus orifice. Their proximity to the site of the slow pathway is probably serendipity. HL double potentials are caused by asynchronous activation of atrial cells and a band of nodal-type cells close to the tricuspid annulus. The band of nodal-type cells is not part of the compact AV node and may represent the substrate of the slow AV nodal pathway.

Key Words • atrioventricular node • tachycardia • reentry • action potentials • radiofrequency ablation

Atrioventricular junctional reentrant tachycardia (AVJRT) can be cured by catheter ablation of sites near the coronary sinus orifice. This technique damages the slow pathway, part of the reentrant circuit used by this tachycardia. Previous work has suggested that double potentials in extracellular electrograms may help to locate the slow pathway so as to target the delivery of radiofrequency energy. The origin of double potentials is unknown, but it has been suggested that they are caused by depolarization of the slow pathway or its atrial termination. The significance of double potentials, however, is controversial, because slow pathway ablation can be achieved at sites at which the potentials are absent. Moreover, these potentials may be found at sites distant from the slow pathway and in humans and animals without arrhythmias. The aim of this study was to determine the...
signals, we also examined the distribution of action potential morphologies in Koch’s triangle and at several sites around the tricuspid annulus.

Preparation of the Heart

The experimental animals were of either sex and consisted of eight New Yorkshire pigs, 6 to 8 weeks old, weighing 18 to 25 kg, and four mature mongrel dogs weighing 18 to 30 kg. Animals were premedicated with azaperon (12 mg/kg IM) and atropine (500 μg IM), then anesthetized with sodium pentobarbital (35 mg/kg IV) and metomidate (5 mg/kg IV). After endotracheal intubation, animals were ventilated with room air, and heparin (5000 IU IV) was given. Modified Tyrode’s solution (1 L) was infused via a peripheral vein. A midline sternotomy was performed, and a large-bore needle was placed in the cranial caval vein for the collection of 1 to 2 L blood/Tyrode’s solution mixture, which was later used to perfuse the heart. The heart was excised and rinsed in cold Tyrode’s solution, then the aortic root was cannulated and perfused with a Langendorff apparatus as described previously.14 Perfusion was at constant pressure (60 cm H2O). The temperatures of the endocardium and coronary sinus effluent were monitored and maintained at 37.0°C to 37.5°C by warming the perfusate and by partly surrounding the heart in a heating jacket.

A Y-shaped incision was made in the superior vena cava and anterolateral wall of the right atrium to expose Koch’s triangle. Part of the septal leaflet of the tricuspid valve was resected to expose the right bundle branch. Bipolar pacing electrodes were placed on (1) the right atrial endocardium near the anterior limbus of the fossa ovalis, (2) on the endocardium about 5 mm posterior to the coronary sinus orifice near the tricuspid annulus, (3) on the epicardium of the right ventricular outflow tract, and (4) on the left atrial endocardium near the orifice of the right pulmonary veins. Bipolar recording electrodes were placed (1) on the right atrial appendage, (2) overlying the His bundle, and (3) on the proximal right bundle branch. A unipolar electrode was used to explore Koch’s triangle and the region surrounding the coronary sinus orifice for the presence of double potentials. The indifferent pole of this electrode was placed in the right ventricular cavity. Extracellular signals were amplified with a gain of 500 to 1000 by use of a bandpass of 0.01 to 500 Hz and recorded with an eight-channel electrostatic recorder at paper speeds of 200 or 250 mm/s. In addition, signals were digitized (14-bit accuracy) and recorded with an eight-channel DAT recorder (DTR 1801, Biologic).

The Semibipolar Electrode

Low-frequency extracellular signals may be caused by depolarization of tissue that is relatively far from the recording electrode (far-field signals). Alternatively, they may be caused by depolarization of tissue adjacent to the recording electrode, provided that the action potential upstroke of these cells is slow. To differentiate between these two mechanisms, we used a semibipolar electrode as described by Veenstra et al.13 The indifferent pole of this electrode was located in the cavity fluid, approximately 1.5 mm directly above the point at which the active pole contacted the endocardium. The proximity of the poles ensured that there was a high level of common-mode rejection; thus, far-field signals were attenuated but local signals were not. This electrode has the advantage over true bipolar electrodes that electrograms are not affected by the direction of excitation.

Microelectrode Recordings

Recordings were made by standard techniques, with microelectrodes drawn from borosilicate glass with tip resistances of 15 to 30 MΩ. Maximum action potential upstroke velocity (dV/dtmax) was measured with a continuous analog differentiator. Microelectrode recordings were made simultaneously with extracellular recordings within 0.5 mm of the active pole of the extracellular electrode. Since it is not possible to achieve stable microelectrode impalements in blood-perfused large mammal hearts because the vigorous cardiac contraction breaks the microelectrode tip or leads to expalement, 1 to 2 g of DAM was added to the perfusate to dampen cardiac contraction (final concentration, 10 to 15 mmol/L). DAM has marked negative inotropic effects while having little effect on the action potential in concentrations of 10 to 20 mmol/L.16

Effect of DAM

To assess the effect of DAM, the presence and shape of double potentials, the atrio-His and HV intervals (measured in the His bundle electrogram), the atrial effective refractory period, the atrioventricular (AV) node effective and functional refractory periods, and the longest cycle length of atrial pacing causing second-degree AV nodal block (Wenckebach point) were examined before and after the administration of DAM in six hearts. Measurements before and after DAM were compared by the paired t test.

Action Potential Survey

Intracellular recordings were made in two porcine and two canine hearts at 15 to 20 sites in Koch’s triangle. At each site, two or three cells were impaled. Since microelectrode studies indicated the presence of nodal-like cells at sites distant from the compact AV node, recordings were also made in the posterior, lateral, and anterior regions of the tricuspid annulus. In the latter regions, recordings were made directly adjacent to the annulus and at six or seven sites in a line perpendicular to the annulus. Distances of 1 to 2 mm separated the latter sites.

Histological Studies

Histological studies were performed in three porcine hearts. In these hearts, pins were placed at sites where high-frequency deflection followed by a low-frequency deflection (HL) double potentials were recorded. Sections were stained with hematoxylin and eosin and elastic van Gieson stains and examined by light microscopy.

Results

Experimental Preparation

Hearts maintained stable AV and ventriculoatrial (VA) conduction for up to 4 hours after perfusion began. Sinus rhythm was present initially, but after the atriotomy, which transected the sinus node and sinus node artery, sinus rhythm was present in only 25% of hearts. In the remaining hearts, the spontaneous rhythm originated in either the AV junction or the posterior or lateral right atrium. After addition of DAM to the perfusate, it was possible to achieve stable microelectrode impalements in all hearts. Impalements could be maintained for periods as long as 20 minutes. Double potentials were present in the baseline state and remained unchanged after DAM administration. DAM caused no significant change in conduction intervals, refractory periods, or the Wenckebach point (values expressed as mean±SD before versus after administration of DAM): AH interval, 50±10 versus 50±20 milliseconds; HV interval, 32±5 versus 32±5 milliseconds; atrial effective refractory period, 170±30 versus 160±20 milliseconds; AV node functional refractory period, 240±50 versus 220±30 milliseconds; and Wenckebach point, 210±50 versus 190±30 milliseconds. The AV node effective refractory period could not be determined either before or after DAM in five of...
six cases because AV refractoriness was determined by atrial refractoriness.

**Types of Double Potentials**

Two types of double potential were found in all hearts. The first type (HL) consisted of a high-frequency component followed by a low-frequency component (Fig 1A, 1B, and 1C). This potential was similar to that described by Haissaguerre et al. in patients with AVJRT. The two deflections were separated by an isoelectric baseline in some cases but not in others. The second type of double potential (LH) consisted of a relatively low-amplitude, low-frequency component followed by a large-amplitude, high-frequency component, similar to that described by Jackman et al. (Fig 1D, 1E, and 1F).

**Distribution of Double Potentials**

The distribution of the two types of double potentials was similar in all hearts (Fig 2). HL potentials were found in the region posterior to the AV node, just above the tricuspid annulus, extending to the region between the coronary sinus orifice and tricuspid annulus. The low-frequency component of HL potentials was largest close to the tricuspid annulus. LH potentials were found posterior and inferior to the coronary sinus orifice and in the proximal coronary sinus. There was some overlap in the regions covered by the two types of potential. In this overlap zone, both types of low-frequency potential were present in addition to the large high-frequency potential, so that the electrogram had three components.

**Characteristics of HL Double Potentials**

The two deflections of HL potentials were relatively widely separated in the anterior and middle of Koch's triangle, but moving posteriorly, the interval between the two decreased. Posterior to the coronary sinus orifice, the two deflections frequently coincided, and it became difficult to discern the low-frequency deflection, since it became hidden by the larger high-frequency component. Surprisingly, the semibipolar recordings indicated that the larger high-frequency component was "far-field" and that the smaller low-frequency component was caused by local excitation. The far-field signal did not coincide with depolarization of nearby endocardial sites, which suggested that it was caused by depolarization of deeper layers (see below).

Facing the anterior limbus did not significantly alter HL double potentials compared with sinus rhythm. Pacing the atria at other sites caused small changes in relative timing of the two components, but in almost all cases, the high-frequency component preceded the low-frequency component. During ventricular pacing, the sequence of the two components was reversed in only two cases (17%). Rapid atrial pacing caused little change in the high-frequency component but markedly decreased amplitude and frequency of the low-frequency component. At rapid rates, the low-frequency component commonly disintegrated, as described by Haissaguerre et al.1

**Origin of HL Double Potentials**

HL double potentials were caused by asynchronous depolarization of superficial and deep cell layers near
high-frequency component coincided with depolarization of atrial cells (or transitional cells with characteristics close to the atrial end of the spectrum) in the deeper layers. These cells had triangular action potentials with relatively rapid upstrokes and high resting membrane potentials (dV/dt_{max} of upstroke, 60 to 300 V/s; resting membrane potential, more negative than −75 mV). Rapid pacing caused minor decreases in dV/dt_{max} in these cells (Fig 5).

The intrinsic deflection of the slow component in the extracellular electrogram coincided with depolarization of superficial cells (cells close to the right atrial endocardium). These cells had action potentials similar to those of nodal cells (dV/dt_{max}, 5 to 25 V/s; resting membrane potential approximately −65 mV). Stimulation of these cells at progressively faster rates caused marked slowing of the action potential upstroke, notching of the upstroke, and finally Wenckebach-type responses (Figs 5 and 6). Despite the fact that many of these cells had characteristics of AV nodal cells, a continuous band of these cells could be found close to the tricuspid annulus around the entire AV ring (see below). Moreover, they clearly did not participate in AV or VA conduction because (1) during rapid atrial pacing, Wenckebach-type block and aborted action potentials were found in some of these cells in the presence of 1:1 AV conduction (Fig 6); and (2) during ventricular pacing, the slow component of the HL potential and depolarization of these cells occurred after excitation of atrial cells in all but two cases (17%). Thus, these cells could not have participated in VA conduction.

**Characteristics of LH Double Potentials**

Rapid atrial pacing had little effect on either component of LH potentials. Pacing of the anterior limbus did not significantly alter LH double potentials compared with sinus rhythm. However, pacing of the left atrium or the region between the coronary sinus orifice and tricuspid annulus caused large differences in the relative timing of the two components (Fig 7). Pacing at the site of double potentials caused reversal of the sequence of the two components so that the high-frequency component preceded the low-frequency component. Pacing of the sinus septum increased the interval between the two components.

**Origin of LH Double Potentials**

Recordings using microelectrodes and semibipolar electrodes indicated that LH double potentials were caused by asynchronous depolarization of two large muscle bundles above and below the coronary sinus orifice. The large high-frequency component was caused by local activation in the region between the coronary sinus orifice and tricuspid annulus. The smaller, low-frequency component was a far-field signal caused by depolarization of the sinus septum, the region above the coronary sinus orifice, lying between the latter and the orifice of the inferior vena cava. Evidence for this was as follows.

1. At the site of LH potentials, recordings from the semibipolar electrode indicated that the high-frequency component was caused by local activation but that the low-frequency component was a far-field signal. The low-frequency component always coincided with the extracellular signal recorded from the sinus septum. This relation was not due to chance,
Fig 5. Tracings showing effect of rapid atrial pacing on high- followed by low-frequency deflection (HL) double potentials and simultaneously recorded action potentials. Upper channel recording from His bundle (HB). Middle channel recording from unipolar exploring electrode (EE) showing HL double potential. Lower channel action potential recorded by microelectrode (ME) at same site as double potential. A, Depolarization of an atrial-type cell coincides with the high-frequency component of the double potential (dashed line). Pacing at increasing rates has relatively little effect on the action potential or the high-frequency component of the double potential. B, Depolarization of a nodal-type cell coincides with the low-frequency component of the double potential (dashed line). Pacing at increased rates leads to marked slowing of the action potential upstroke and decreased size of the action potential. There is a concomitant decrease in size and slowing of the downstroke of the low-frequency component of the double potential. A indicates atrial deflection; H, His bundle deflection; V, ventricular deflection; and CL, cycle length.

since it held true when pacing was performed from different sites, which altered the interval between the two components (Fig 7).

2. Microelectrode studies confirmed that the high-frequency component coincided with depolarization of cells directly adjacent to the extracellular electrode and
that the low-frequency component coincided with depolarization of cells in the sinus septum (Fig 8). These cells had characteristics of atrial cells or transitional cells close to the atrial end of the spectrum. Action potentials were triangular, with relatively rapid upstrokes and moderately high resting membrane potentials (dV/dt max, 60 to 300 V/s; resting membrane potential, more negative than −75 mV).

3. Pacing from the sinus septum increased the interval between the two components of the double potential, whereas pacing near the site of the double potential caused the sequence of the two components to be reversed, so that the high-frequency preceded the low-frequency component.

**Distribution of Action Potential Types**

Cells more than 15 mm from the tricuspid annulus exhibited action potentials typical of atrial cells. Action potentials were triangular, the resting membrane potential was typically more negative than −85 mV, and the upstroke velocity was rapid (typically >120 V/s). Action potentials in cells of the compact AV node had slower upstrokes (5 to 25 V/s) and more positive resting membrane potentials (approximately −65 mV). The contour of these action potentials was rounded rather than triangular. Between these two extremes, the transitional cells exhibited a smooth continuum of characteristics (Figs 9 and 10). However, a superficial ring of nodal-type cells was present around the entire tricuspid annulus. A zone of transitional cells separated these cells from the atrial cells in the body of the atrium and from atrial-type cells in deeper layers. Thus, the direction of the gradient of change was perpendicular to the tricuspid annulus rather than perpendicular to the atrium–AV nodal axis (Figs 9 through 11).

**Histology**

Sections from the sites of double potentials showed that individual cells in the superficial subendocardial layers had the morphological characteristics of transitional or nodal cells but that the cells were not arranged in an interweaving fashion as is found in the compact AV node (Figs 12 and 13). These cells were smaller than working atrial myocardial cells, with less marked striations, and were frequently separated from one another by fine strands of connective tissue. No boundary or morphological characteristic distinguished the superficial cells with nodal-type action potentials (which caused the low-frequency component of HL double potentials) from the deeper layers (which caused the high-frequency component).

**Discussion**

The findings of this study suggest that the spatial relation between LH double potentials, similar to those described by Jackman et al., and the slow pathway is probably a fortunate accident. LH potentials are caused by asynchronous activation of two large muscle bundles separated by the mouth of the coronary sinus. There is no doubt that these potentials can be used to guide radiofrequency ablation of the slow pathway, but LH potentials appear to be a marker for the region between the coronary sinus orifice and the tricuspid annulus, where the slow pathway is frequently found, rather than specific for the slow pathway itself. This conclusion is supported by a previous study that used multipoint plaque electrodes and was performed in humans with AVRT.

In that study, LH-type potentials were found at the point of earliest atrial excitation during retrograde slow pathway conduction in four of five cases but were also found up to 16 mm from that site. Moreover, the largest LH double potential was found at the site of the slow pathway in only two of five cases. That study also found that the low-frequency component of the LH double electrogram coincided with local activation in the sinus septum and thus was likely to be a far-field signal.
The finding that the low-frequency component of the HL double potential is caused by depolarization of nodal- or conduction-type tissue suggests the possibility that this tissue is the anatomic substrate of the slow AV nodal pathway. Premature stimulation of these cells caused marked slowing of the action potential upstroke, and this would be expected to decrease conduction velocity. If other inputs to the AV node were rendered refractory by a premature stimulus, conduction via this band of nodal-type cells would explain the long conduction intervals observed during slow pathway conduction. Moreover, the site of these cells is consistent with the site of the slow pathway, as demonstrated by mapping and ablation studies.\textsuperscript{1-3,5,17,18} In the mapping study of McGuire et al.,\textsuperscript{5} HL-type potentials were found at the point of earliest atrial activation during retrograde slow pathway conduction in five of five cases, but these potentials were also found up to 17 mm from that site. This is not surprising, since the cells of origin are distributed along the entire tricuspid annulus and gradually merge with transitional cells that extend for more than 1 cm above the annulus. If the low-frequency component of HL potentials is truly caused by depolarization of the slow pathway, it is possible that some feature of the double electrogram, such as amplitude of the low-frequency component, may be a more specific marker for the pathway. This question can only be answered by examining the characteristics of the electrogram at the site of successful slow pathway ablation. We emphasize, however, that we found no direct evidence that the nodal-type cells are the substrate of the slow pathway; they could also be "dead-end" pathways.\textsuperscript{19}

Fig 8. Tracings showing that local depolarization of the sinus septum coincides with the low-frequency component of low-followed by high-frequency deflection double potentials. Upper channel is unipolar exploring electrode (EE) recording from site inferior to coronary sinus orifice (marked "1" in inset). Lower channel is microelectrode (ME) recording from sinus septum (marked "2" in inset). The low-frequency deflection coincides with action potential upstroke in sinus septum (dashed line). The artifact (arrow) near the apex of the action potential is an extracellular atrial deflection recorded by the indifferent electrode. A indicates atrial deflection; V, ventricular deflection; FO, fossa ovalis; TT, Todaro's tendon; CS, coronary sinus; and TVA, tricuspid valve annulus.

Fig 9. Action potentials recorded from the endocardium of Koch's triangle in a mature dog. Inset shows sites of recording. Note the nodal-type action potentials recorded near the tricuspid annulus. As the recording site moves away from the annulus, action potentials become more "atrial" in type. Note the changes in resting membrane potential, action potential amplitude, and overshoot. Small artifacts near the foot and apex of action potentials are caused by "contamination" by extracellular signals recorded by indifferent electrode. Action potential upstroke velocities were 9, 25, 60, 100, 150, and 230 V/s, respectively. CS indicates coronary sinus; TT, Todaro's tendon; TVA, tricuspid valve annulus; and CFB, central fibrous body.

Fig 10. Diagram showing electrophysiological characteristics of endocardial cells in Koch's triangle in porcine heart. Values shown at site of recording. A, dV/dt of action potential upstroke. B, Action potential amplitude. C, Resting membrane potential. Note that the direction of change from atrial-type cell to nodal-type cell is toward the tricuspid annulus rather than toward the AV node.
It is uncertain why the H and L components of the HL potential occur almost simultaneously in the region inferoposterior to the coronary sinus orifice but progressively separate when the recording site is moved anterosuperiorly along the tricuspid annulus. We speculate that the layers giving rise to the H and L potentials are well coupled in the posterior part of Koch's triangle but not well coupled in the anterosuperior region, close to the AV node. There does not appear to be an anatomic barrier between the two layers. A previous study from this laboratory used a multipoint plaque electrode to map activation in this region during sinus rhythm. Activation of H and L potentials was mapped separately. In that study, excitation approached Koch's triangle from the anterior septum and swept posteriorly, giving rise to the H potential, then appeared to enter the "L" layer and changed direction to pass anteriorly toward the AV node.

The fact that these nodal-type cells may be found in animals without AVJRT or electrophysiological evidence of dual AV nodal pathways suggests that the substrate for dual pathways may be present in normal animals. Presumably dual pathways are not demonstrable in most animals, however, because a relatively short refractory period in the fast pathway favors conduction over the latter pathway so that slow pathway conduction is not observed. Thus, the functional defect in humans with AVJRT may be an abnormally long refractory period of the fast pathway.

A Ring of Nodal-Type Cells Around the Tricuspid Annulus

The present study presents electrophysiological evidence for a ring of nodal-type cells around the tricuspid annulus. Previous studies have suggested that a strip of AN or transitional cells may be present posterior to the node, and "dead-end pathways" of nodal cells have been demonstrated within Koch's triangle. Nodal-type cells have been found in the tricuspid valve but have not been demonstrated around the tricuspid ring. The existence of this ring of conduction tissue is supported by anatomic studies. This specialized conduction tissue is probably a remnant of AV canal tissue demonstrated in embryonic hearts. Clearly, this tissue remains present into adult life, since in the present study it was found in mature dog hearts.

Experimental Preparation

This study reports for the first time the use of microelectrode recordings in the perinodal region of blood-perfused large-mammal hearts. Microelectrodes cannot ordinarily be used to record from the perinodal region in blood-perfused hearts because the vigorous contraction breaks the fragile tip of the microelectrode or leads to expalement from the cell. Thus, previous studies have been limited to superfused preparations.
Most studies have used rabbit hearts that have been stretched and pinned to reduce contraction. These preparations have been superfused with crystalline solutions rather than perfused with blood. Superfusion allows only the superficial cell layers to survive; this reduces the force of contraction but may lead to artifacts because the deeper hypoxic cells interact with the surviving superficial cells. The use of DAM to dampen contractions allowed us to use blood perfusion and avoid the limitations of the superfusion technique.

**Limitations of This Study**

Since the double potentials of the present study were found in experimental animals rather than in humans with AVJRT, it might be argued that they are not equivalent to those described by Haissaguerre et al or by Jackman et al. There is considerable evidence, however, that these potentials are equivalent. The location, the relative size and timing of the two components, and the effect of pacing from various sites were similar. The low-frequency component of the HL potential decreased in size and with increasingly rapid pacing, as does the "slow potential" described by Haissaguerre. Moreover, "slow potentials" were also found in humans without AVJRT.

The present study used unipolar rather than bipolar recording, as was used by Haissaguerre et al and Jackman et al. This may have caused differences in the extracellular waveforms of double potentials in the present study compared with those found previously. We used unipolar recording because our aim was to localize the tissues of origin of double potentials. With unipolar recording, the intrinsic deflection of the extracellular electrogram coincides with the depolarization of tissue directly beneath the electrode. If bipolar electrodes had been used, activation could only have been localized to an indeterminate point between the two poles of the electrode. Our filter settings also differed from those used in the previous studies. We used a wider bandpass to lessen the chance of artifacts produced by filtering. Yet despite these differences in recording technique, the appearance and behavior of the double potentials in the present study are remarkably similar to those found in previous studies.

It is also possible that the ring of nodal-type cells around the tricuspid annulus is an artifact caused by regional ischemia or regional hypothermia. This is unlikely, however, since the distribution of these cells was similar in all hearts and "normal" atrial and nodal action potentials were found at appropriate sites nearby. Moreover, we regularly checked the endocardial temperature at these sites and found it to be 37.0°C to 37.5°C. It is also unlikely that the nodal-type action potentials were caused by perfusion of DAM, since normal atrial action potentials were found at other sites and there is evidence that concentrations of 10 to 15 mmol/L do not markedly affect action potential morphology.

**Conclusions**

LH potentials are caused by asynchronous activation of muscle bundles above and below the coronary sinus orifice. Their proximity to the site of the slow pathway is
probably a fortunate coincidence. HL double potentials are caused by asynchronous activation of atrial cells and a band of nodal-type cells close to the tricuspid annulus. The band of nodal cells is not part of the compact AV node and may represent the substrate of the slow "AV nodal" pathway.

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