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Atrioventricular Node : Presence of New Functionally and Anatomically Distinct AV Pathway

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I Introduction

The AV node has mystified generations of investigators over the last century and continues today to be at the epicenter of debates among anatomists, experimentalists, and electrophysiologists. Historically, the AV nodo has been defined by classical histological methods; however, with recent studies, a more precise characterization of structure is becoming attainable. Dual pathway electrophysiology, one of the hallmarks of the human AV junction, has been widely investigated over the last century [1]. However, the presence of new functionally and anatomically distinct AV pathway was described [2].

The AV node is a part of electrical control system of the heart that coordinates heart rate. It electrically connects atrial and ventricular chambers. The AV node is an area of specialized tissue between the atria and the ventricles of the heart, specifically in the postero-inferior region of the interatrial septum near the opening of the coronary sinus, which conducts the normal electrical impulse from the atria to the ventricles. It is located at the center of Koch's Triangle - a triangle enclosed by the septal leaflet of the tricuspid valve, valve, the coronary sinus, and the membraneous part of the interatrial septum [3]. The AV node receives two inputs from the atria: posteriorly, via the crista terminalis, and anteriorly, via the interatrial septum. AV conduction during normal cardiac rhythm occurs through two different pathways : the first pathway has a slow conduction velocity but shorter refractory period and the second pathway has a faster conduction velocity but longer refractory period [4]. Atrioventricular node reentry is typically induced with anterograde block over the fast pathway and conduction over the slow pathway, with subsequent retrograde conduction over the fast pathway.

II AV Nodal Reentrant Tachycardia

Mendez and Moe (1966) proposed that atrio-nodal connection utilizes alpha and beta pathways that connect together in the distal node [5]. Anatomical and functional studies of the AV node have demonstrated the presence of two distinct input pathways, providing the substrate for clinically important AV nodal reentrant tachy-arrhythmias. In patients with dual AV nodal pathways, a normally timed sinus impulse will conduct through the AV node via beta pathway, since the beta conducts more rapidly than the alpha pathway. However a premature atrial impulse can arrive at the AV node at such a time that the beta pathway is still refractory from the previous normal beat, but the alpha pathway is no longer refractory. This early impulse will then traverse the alpha pathway, reaching the His bundle after a prolonged conduction time through the AV node this AV nodal conduction delay is manifested by a prolonged PR interval on the surface ECG. If the beta pathway recovers by the time the impulse reaches the distal portion of the alpha pathway, the impulse may conduct retrogradely up the beta pathway (producing an atrial echo beat). If this retrograde impulse is then able to reenter the alpha pathway, a continuously circulating impulse can be established within the AV node (Figure 1) [6].

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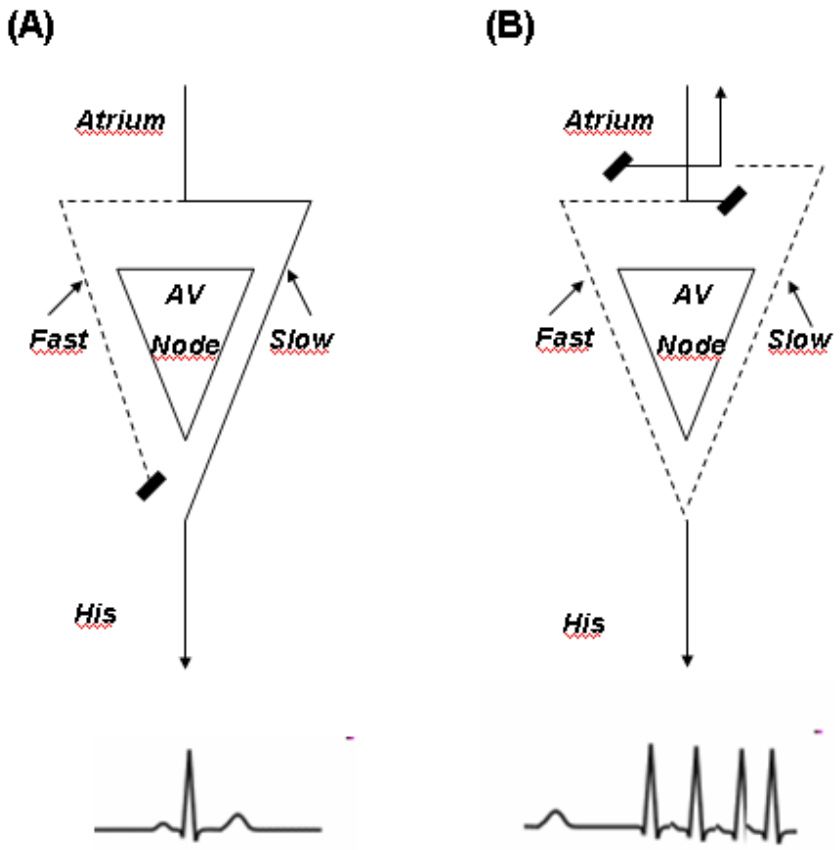

Figure 1. Dual nodal physiology. The atrium, AV node, and His bundle are shown schematically. The AV node is longitudinally dissociated into two pathways, slow and fast, with different functional properties. In each panel of this diagram, solid lines denote excitation in the AV node, which is manifest on the surface electrocardiogram, while dotted lines denote conduction, which is concealed and not apparent on the surface electrocardiogram. (A) During sinus rhythm the impulse from the atrium conducts down both pathways. However, only conduction over the fast pathway is manifest on the surface ECG, producing a normal PR interval. (B) A more premature atrial impulse blocks in the fast pathway, conducting with increased delay in the slow pathway. The impulse conducts retrogradely up the fast pathway producing a single atrial echo. Retrograde conduction occurs over de fast pathway and reentry occurs, producing a sustained tachycardia. The impulse conducts over the slow pathway to the His bundle and ventricles, producing prolonged PR interval on the surface ECG.

Despite the impressive amount of information concerning AV nodal conduction, the role of the AV node in AV block is poorly understood [7]. Electrical stimulation of the vagus nerve in rat induced significant bradycardia, third degree AV block and the P wave appearance was negative in leads II in ECG, suggesting atrial reentry. Morphine injections induce a positive P wave appearance through one inhibitory action on AV nodal reentry [2]. This study sought to describe electrophysiologic characteristic and possible anatomic site of atrial retrograde slow AV node pathways.

III AV Block and Atrial Reentry

Despite the impressive amount of information concerning AV nodal conduction, the role of the AV node in bradycardia is poorly understood. In particular, the spatial and temporal pattern of the AV node engagement from the atria has not been sufficiently studied.

The dromotropic effects of the vagus on atrioventricular conduction are classic and have been studied for a long time. Vagal cardioinhibition is exerted through a reduction not only in the heart rate but also in the rate of propagation of the cardiac action potential and in myocardial contractility. The parasympathetic nervous system innervates the heart through two cervical vagal branches. The right vagal branch mainly influences the heart rate by the modulation of the rhythmogenesis of the sinoatrial node. The left branch predominantly influences the conduction properties of the AV node. Under both types of vagal stimulation protocols, AV blockade appeared most frequently when the left-sided nerves were stimulated first [8].

The unilateral stimulation of the distal segment of left vagus nerve in rats anesthetized with Equitesin®, decreases heart rate and induced significant bradycardia associated with a third degree AV block and negative P wave appearance, consistent with retrograde AV conduction (Figure 2A). The morphine injection induces the appearance of a positive P wave through one inhibitory action on AV nodal reentry (Figure 2B). In the anaesthetized rat, morphine decreased the bradycardia. Presynaptic modulation of neurotransmitter in the regulation of cardiac function was reported by Kosterlitz and Taylor (1959) who demonstrated that morphine reduces the cardiac slowing produced by vagal stimulation [9]. All suggest the presence of a new functionally and anatomically distinct AV pathway [2].

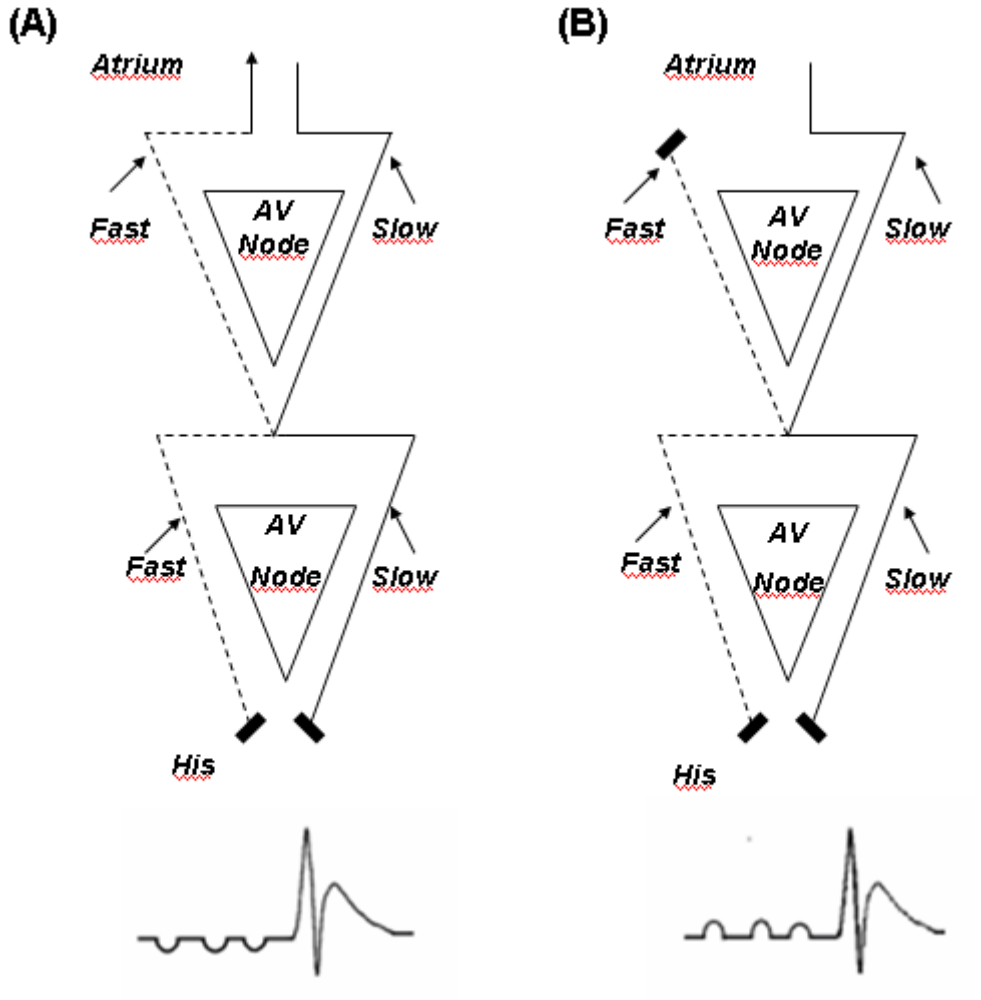

Figure 2. Atrioventricular block and atrial reentry. The figure (A) represents the third degree AV block and negative P wave, suggesting atrial reentry, after electrical stimulation of the vagus. At (B), the P wave wave inversion after treatment of animals with morphine.

The reentry circuits consist of two pathways around the central obstacle: a fast pathway with long refractory period and a slow pathway with short refractory period. During treatment of morphine, the activation signal will travel through both pathways and conflict within the slow pathway. In this case the reentry circuit is equivalent to the fast pathway alone. However, if a premature stimulus enters the circuits before the fast pathway finish its refractory; it will be blocked in the fast pathway and go along the slow pathway, causing significant delay (Figure 2B). If the fast pathway finishes its refractory when the stimulus arrives the circuit exit, the activation wave will go retrogradely along the fast pathway, causing an atrial reentry, during unilateral stimulation of the distal segment of left vagus (Figure 1A).

Spontaneous activity of the mammalian heart is generated in the sino-atrial node. Pacemaker activity is generated in sino-atrial node cells but the regulation by the autonomous nervous system is still a matter of debate. A number of different ion channels and signaling events have been implicated in pacemaker activity [10]. In order to better understand the need and

the operation of the pacemaker, we provide some background. The sino-atrial node potential could also be important for preservation of pacemaker activity in order to generate reentrant atrial. There is a ventricular pacemaker which takes over as the main pacemaker if the AV node fails. After the depolarization of the ventricles, a transient period follows where no further ionic current can be flow through the myocardium. This is known as the refractory period. A recharging (depolarization) of the ventricular myocardium to its resting electrical potential and the heart is then ready to repeat the cycle. The changes in P wave morphology probably result from changes in the sequence of atrial muscle activation arising from a change in the exit site from the SA node. The stimulation of vagus nerve and morphine injection induced a third degree AV block. Third-degree AV block with a ventricular pacemaker is characterized by the complete dissociation of the P waves and the QRS complexes. Under these conditions, there is no PR interval (since the P wave didn't cause the QRS complex). The atria and the ventricles are functioning entirely independently of each other (Figure 2B). The stimulation of vagus nerve induced a significant bradycardia and biphasic the P wave, consistent with retrograde AV conduction (Figure 2A).

Despite the need for a structural and functional correlation in the AV node, the anatomical existence of the substrate for dual pathway electrophysiology is not necessarily indicative of the existence of functional reentry, although required to maintain the reentry circuit. It is apparent that both structural and functional compartmentalization has to exist within the AV node, as revealed by stimulation of vagus nerve and the functional data obtained from ECG. From these data, two distinct pathways or "compartments" are becoming apparent within the AV node, as suggested by stimulation of vagus nerve, that appear to be continuous with the atrial bundle and display distinct conduction properties. With the elucidation of basic elements of both structure and function via investigation, new opportunities are becoming apparent in utilizing the unique properties of AV node for pursuing novel applications relevant to electrophysiology of heart.

IV. Conclusions

Electrical stimulation of the left vagus induced AV block and negative P wave, when morphine was injected the P wave appearance positive through one inhibitory action on AV nodal reentry; suggesting the presence of new functionally and anatomically distinct AV pathway. The proposed system is available as a free and open source platform to the research community.

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