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Review Article

Dual Atrioventricular Nodal Pathways Physiology: A Review of Relevant Anatomy, Electrophysiology, and Electrocardiographic Manifestations

Bhalaghuru Chokkalingam Mani, MD, Behzad B. Pavri, MD

Jefferson Medical College of Thomas Jefferson University, 925 Chestnut Street, Mezzanine, Philadelphia, PA19107

Address for Correspondence: Bhalaghuru Chokkalingam Mani, MD, Department of Medicine, Thomas Jefferson University Hospital, 803 College Building, 1025 Walnut Street, Philadelphia, PA19107. E-mail: bhalaghuru.cm@gmail.com

Abstract:

More than half a century has passed since the concept of dual atrioventricular (AV) nodal pathways physiology was conceived. Dual AV nodal pathways have been shown to be responsible for many clinical arrhythmia syndromes, most notably AV nodal reentrant tachycardia. Although there has been a considerable amount of research on this topic, the subject of dual AV nodal pathways physiology remains heavily debated and discussed. Despite advances in understanding arrhythmia mechanisms and the widespread use of invasive electrophysiologic studies, there is still disagreement on the anatomy and physiology of the AV node that is the basis of discontinuous antegrade AV conduction. The purpose of this paper is to review the concept of dual AV nodal pathways physiology and its varied electrocardiographic manifestations.

Key Words: dual AV nodal, arrhythmias, physiology, manifestations

The term "dual AV nodal pathways" is loosely used by many clinicians when analyzing electrocardiograms and telemetry strips. This term, to the novice physician, might suggest two, distinct anatomic structures related to the atrioventricular (AV) node, but often the true understanding of this entity is either missing or incomplete. The purpose of this manuscript is to review the anatomy, physiology, and electrophysiologic functional characteristics of the human AV node, and demonstrate the variety of electrocardiographic manifestations of what should correctly be described as "dual AV nodal pathways physiology". This review may be most helpful to physicians-in-training – interns, residents, and cardiology or pulmonary fellows; practicing internists with special interest in electrocardiography may also find this of value.

Anatomy of the AV node

The AV node is part of the AV conduction axis. By virtue of its unique property of *decremental conduction*, the AV node delays the impulse arriving from the atria, thereby allowing the ventricles to stay in diastole, providing sufficient ventricular filling time. This property of decremental conduction also allows for protection of the ventricles from very

rapid rates during atrial fibrillation. Though there were numerous researchers who identified the presence of an electrical connection between the atria and the ventricles, the physical existence of the AV node was definitively proven by Tawara. [1,2]

The compact AV node is located at the base of the atrial septum in the triangle of Koch [3] (**Figure 1**). The triangle is visualized on the right side of the endocardial surface of the interatrial septum, and is formed anteriorly by the insertion of the septal leaflet of the tricuspid valve and posteriorly by the fibrous tendon of Todaro [4]. The apex of the triangle is formed by the junction of these two boundaries and the base is formed by the superior lip of the orifice of the coronary sinus [5]. The AV node can be considered to be made up of two zones, transitional and compact. The transitional zone consists of "transitional cells", so called because they are intermediate in morphology and function between the compact nodal cells and the atrial myocytes. This zone of transitional cells envelops the compact AV node, serving as the connection between the surrounding atrial myocardium and the compact node [6].

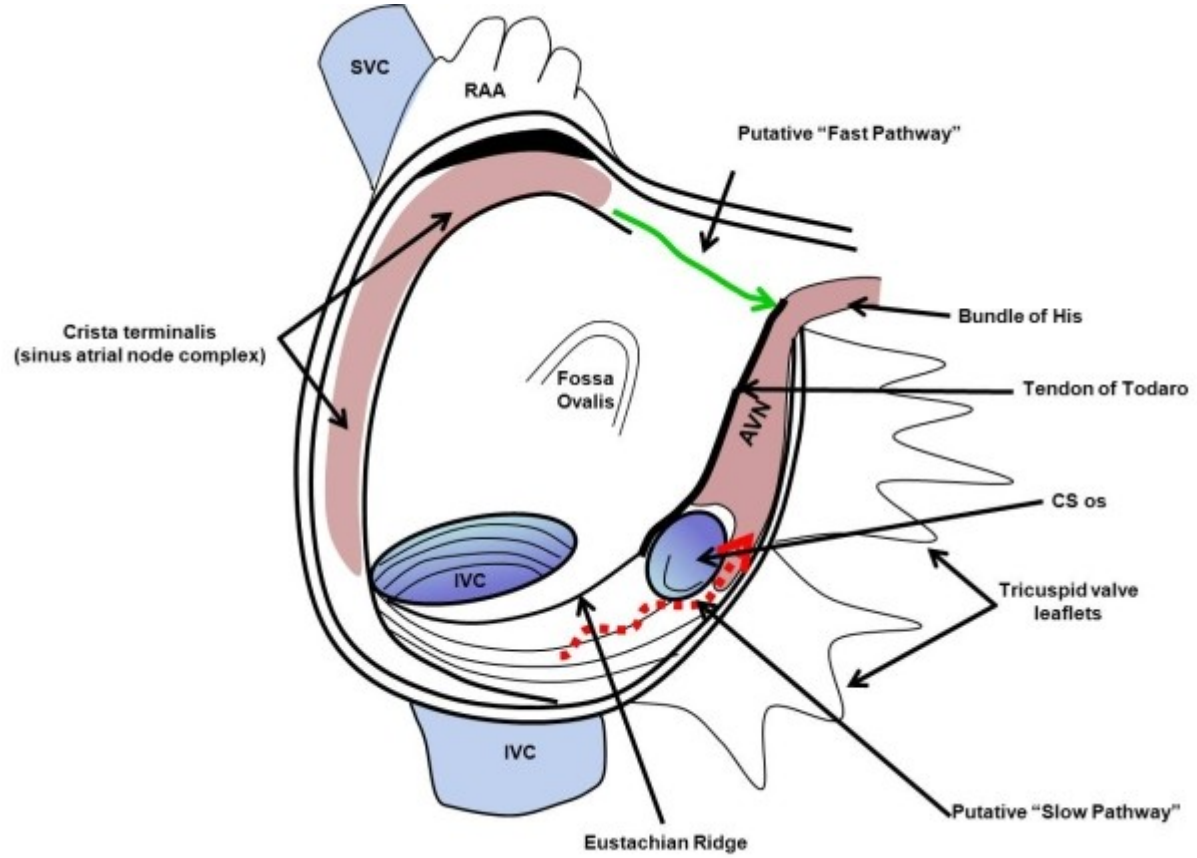

Figure 1: Schematic representation of the interior of the right atrium, as viewed in the right anterior oblique projection. The transmission of impulse from the sinoatrial node over the "fast pathway" (green arrow) and over the "slow pathway" (red dashed arrow) to the AV node is depicted.

The compact zone measures 5-7 mm in length and 3-4 mm in width and is located in the triangle of Koch. The compact AV node gives rise to three posterior extensions: one in the direction of the coronary sinus along the tricuspid annulus (the putative "slow pathway"), a second in the anterior portion of the triangle of Koch near the compact portion of the AV node (the putative "fast pathway"), and the third in the direction of the mitral annulus (the left atrial extension) [7]. The anatomic separation of about 15 mm [8] between the anterior (fast) and posterior (slow) approaches is what allows safe ablation of the putative "slow pathway" for the treatment of AV nodal reentrant tachycardia without the creation of complete heart block.