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Cardiac Arrhythmias

From Basic Mechanism to State-of-the-Art Management

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This book is dedicated to my family, Gabriela, David, Victoria, and Peter, for their love and support; to all the current and future professionals of arrhythmias for whom this book was written; Additional thanks to Dr. Lucian Gheorghe, Dr. Vasile Murgu, Dr. Ionel Droc, Dr. Mocanu Iancu, Dr. Vasile Greere, Dr. Daniel Nita, Dr. Adela Cirstea, Dr. Gabriel Cristian. Special thanks are owed to Dr. Blanca Calinescu, Dr. Chantal Trudeau, Dr Jenica Roates, Associate Professor Liviu Chiriac, Professors Alexandru Campeanu, Tiberiu Nanea, and Ion C. Ţintoiu, for their guardianship, friendship and support through the good and bad times. I am eternally grateful to all Editors of this book who actively participated with me to realize this Edition.

Ambrose S. Kibos, MD, PhD

Foreword

The field of cardiac arrhythmias has had a remarkable and light-speed progress in the past two decades. From being the Cinderella of Cardiology and practiced in dark damp basements in many institutions around the world, this field is now at the forefront of every cardiology division. Similarly the progress in understanding the mechanisms, anatomy, diagnosis, and treatment of all different forms of cardiac arrhythmias has been overwhelming in the past decade. The twenty-first-century cardiac electrophysiologist has to a possess a large bulk of knowledge in this field and is confronted with the challenge of staying updated at the millisecond pace in which new developments occur, a fate rarely seen in other subspecialties. For this reason this first edition of *Cardiac Arrhythmias: From Basic Mechanism to State-of-the-Art Management* encompasses one of the most comprehensive updated textbooks available to date. In its four sections this multiauthored international compilation brightly reviews the anatomy of arrhythmias, diagnostic methods, and diseases associated with arrhythmias and treatment of all forms of cardiac arrhythmias with lavishly illustrated and ample discussions on the most modern ablation and mapping techniques available to date. Similarly the field of devices in the management of cardiac arrhythmias is superbly reviewed. This new textbook adds to the field of cardiac arrhythmias a new and fresh perspective and should be useful not only for the trainee and new upcoming electrophysiologist but also for the seasoned one. This book should be on the shelves of all cardiac arrhythmia units around the globe as it is authored by a significant number of the most influential electrophysiologists in the field and presents a comprehensive and practical approach to cardiac arrhythmias.

Carlos A. Morillo, MD, FRCPC, FACC, FHRS, FESC

Preface

The past 50 years have witnessed the growth and evolution of clinical electrophysiology from a field whose initial goals were the understanding of arrhythmia mechanisms to one of significant therapeutic impact. The development and refinement of implantable devices and catheter ablation have made non-pharmacological therapy a treatment of choice for most arrhythmias encountered in clinical practice. The purpose of this book is to provide the "caring electrophysiologist" with an electrophysiologic approach to arrhythmias, which is predicted on the hypothesis that a better understanding of the mechanisms of arrhythmias will lead to more successful and rationally chosen therapy. As such, the techniques suggested to address these issues and specific therapeutic interventions employed represent a personal view on intuition, based on experiences of world renowned scientists. These include among others, Steve Fishberger, Vidal Essebag, Bradley Knight, G André Ng, Mauricio Scanavacca, Cheuk-Man Yu, Ion Ţintoiu and Mark Slevin.

Ambrose S. Kibos, MD, PhD

Letter from the Editors

Cardiac Arrhythmias: From Basic Mechanism to State-of-the-Art Management

The editors of this book had three primary objectives. The first objective was to develop an outline for a comprehensive, modern-era textbook on heart rhythm disorders that properly covered each of the major topics in the field – from basic mechanisms to state-of-the-art clinical management. Careful organization of the chapters and precise wording of each chapter title were important to ensure that critical issues were all addressed and presented in a logical fashion. The second goal was to appropriately select the best possible authors for each chapter. The ideal author for a book chapter is not just an expert in the field with recognized experience and authority, but is also someone who is able to effectively communicate the key teaching points for the topic, has excellent writing skills, and can reliably meet submission deadlines. It was critical that these two objectives were accomplished very early in the planning stages of the book to lay the groundwork for the contributors.

The third objective of the editors was to ensure that the finished product was of the best possible quality. Each chapter was thoroughly reviewed by at least one editor for both content and presentation. This can be a challenging task when the chapters are each written by different authors selected from all over the world, where English may not be the primary language and where books may have variable writing styles and formats. In addition, most of the chapters themselves were a product of collaboration among multiple authors who had to choreograph their work. Fortunately, we had the luxury of reviewing work from an outstanding international group of authors. This made our task enjoyable and educational.

Any multiauthored book that ventures to cover as many topics as are covered in this book will inevitably contain some redundancy. We felt that it was more important to allow each author to fully present his or her topic in a chapter that could stand alone, rather than to try and eliminate all redundant content.

We would like to thank each and every author for their effort and contribution and hope that you enjoy reading this book as much as each of the editors did when putting this collection together.

Chicago, IL, USA — Bradley P. Knight, MD, FACC, FHRS

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Anatomy and Physiology of the Atrioventricular Node: Basic Concepts

1

Ambrose S. Kibos and Blanca F. Calinescu

Abstract

To appreciate the arrangement of the muscular connections between the atrial walls and the compact atrioventricular node, it is necessary to understand their anatomic relationships. Koch described the landmarks to the atrioventricular node that many subsequent investigators have illustrated the conduction axis running horizontally rather than vertically. The cardiac electrical physics is the "soul" of the heart, and hence, understanding of anatomy and physics is critical to unlocking the understanding of electrical workings and facilitates ablation procedures.

Keywords

Dual atrioventricular • Triangle of Koch • Electrocardiographic • Atrial fibrillation • Wave fronts

Introduction

Conduction within and through the critical architecture of the cardiac conduction system long fascinated scholars, beginning with the manifestation of initial electrocardiographic (ECG) recordings of cardiac electrical activity [1]. In 1963, the first published case report involving supraventricular tachycardia in canine was done by Moe et al. [2]. Later, in the following decade, Mendez and Moe [3] demonstrated the same findings in isolated rabbit AV nodal preparations using advanced microelectrode recordings. They demonstrated reentry as well as the collision of impulses from two surgically separated atrial sites, called alpha and beta pathways (later on named fast and slow pathways, respectively), that they could only explain by separate AV nodal inputs meeting in a common pathway. In the later years of the twentieth century, Janse et al. [4], through pivotal studies they conducted, helped to map out the complex electrophysiology of the AV node, correlating it with anatomy. Finally, a critical investigation by de Paes Carvalho and de Almeida [5] established the atrionodal (AN), nodal (N), and nodo-His (NH) regions of AV nodal conduction, with the calcium dependency of the upstroke for the cells in the N region being demonstrated a short time later.

Dual AV Nodal Physiology

Moe et al. [6] first demonstrated that pacing could initiate and terminate supraventricular tachycardias (SVTs) drawing proper conclusions from existing evidence that the electrophysiologic features could only be explained by two or more pathways that are well distinct [7, 8]. For the first time, it was shown that during SVT [9] a ventricular impulse could reach the atrium retrograde without necessarily colliding with the anterograde impulse of the SVT and thus obtaining two ventricular responses from one atrial impulse. During atrial pacing stimulus [10], these two findings occur if two AV nodal pathways are present. Later research in patients after RF catheter ablation [11, 12] showed that elimination of conduction is one of the approaches to the AV node with abolition

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of AVNRT also offered convincing proof of existence of two pathways. It is important to understand why there is duality [13], what is responsible, and why it has developed as a part of many. What about the slow pathway? Sparse distribution of gap junctions resulting in cellular uncoupling [14], uneven allocation of ion channels and autonomic innervation [15], anisotropic conduction leading to tortuous pathways, and Ca^{2+} dependency of the depolarization upstroke of cells in the N region but a probable Na+ contribution in the AN and NH regions all contribute [16–18]. The AV junction is a large area; therefore, the function may not be able to be deduced from structural changes in the AV node and its approaches, since presently it is not known how much of any structure is needed for its proper function. Any disease that may affect the heart has the potential to affect the AV junction including the AV node and its approaches, thereby having the potential for development of arrhythmias [19].

AV Conduction During Atrial Fibrillation

We have learned from anatomists since the early work of Tawara [20] that the histologically specialized tissues of the AV node lie deep within the atrial walls and are overlain with atrial myocardium showing anatomic features favoring anisotropic conduction. RF procedures [21] have been used to tease out different pathways and their electrophysiologic features. It is still clear that the two pathways communicate with each other so that abolition of slow pathway by RF automatically shortens the refractory period of the fast pathway [22]. AV nodal modification, which generally involves slow pathway ablation [23], results in slowing of ventricular rate during atrial tachycardia and atrial fibrillation. Conduction during atrial fibrillation [24], critically important in the overall ablation strategy management, is also crucial to understand mechanistically [25], with collision of impulsion producing summation that regulates AV conduction during atrial fibrillation [26].

Conclusions

Dual pathway electrophysiology is based on separate wave fronts that propagate in functionally, rather than electrically, isolated domains [27]. The envelope of the transitional cells and brief trespass through compact nodal region in the anterior margin of the triangle of Koch constitutes the fast wave-front domain. The deeper inferior/posterior extensions and the compact cell region are the proposed domain of the slow wave front [28]. These domains are not exclusive, so that interaction of the wave fronts can be observed and can produce a variety of complex conduction phenomena. The reciprocating echo beat and AVNRT are just one manifestation of this interaction [29]. Finally, the specialized part of the conduction system is a part of the entire heart, and therefore, any discussion of the conduction system should include the entire heart and vice versa [30]. This is a brief discussion of the AV nodal history, physiology, and connections, and therefore, the interested reader is encouraged to read the original works described in detail elsewhere in this book.

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Anatomy and Physiology of the Atrioventricular Node: What Do We Know Today?

2

Hidekazu Miyazaki

Abstract

Atrioventricular (AV) node consists of a part of the sole pathway of impulse conduction from the atria to the ventricles. It becomes the base of occurrence and maintenance of various arrhythmias involving this region by special electrophysiological characters as follows: The conduction across the node is quite slow, it functions as two separated conductors with property of fast or slow conduction, and it has the automaticity. AV junctional area including the node itself potentially works as the ectopic center of a subsidiary pacemaker when the sinus node as the primary pacemaker fails to control the cardiac rhythm. Muscular bundles consisting of dual AV pathways are not anatomically distinguished but can be functionally differentiated by the difference of conduction property and refractoriness. A fast pathway connects with the center of the node from anterior part of interatrial septum, and a slow pathway does from posteroinferior area to the tricuspid valve. In cells of slow pathway, the occurrence of automaticity is determined and the expression of ion channels is similar to ones in the center of the node. These facts suggest that the dual-pathway physiology of the AV node is not only formed by special electrophysiological property of cells but based on the morphology.

Keywords

Atrioventricular node • Fast pathway • Slow pathway • Dual-pathway physiology • Nodal cell • Automaticity

Abbreviations

AN	Atrio-nodal
AP	Action potential
AV	Atrioventricular
AVNRT	Atrioventricular nodal reentrant tachycardia
CS	Coronary sinus
Cx	Connexin
DAVNP	Dual atrioventricular nodal pathway
FP	Fast pathway
$I_{Ca,L}$	L-type calcium channel current
$I_{Ca,T}$	T-type calcium channel current
I_{K1}	Inward rectifier potassium current
IVC	Inferior vena cava
MV	Mitral valve
NH	Nodo-His
PNE	Posterior nodal extension
RA	Right atrium
RV	Right ventricle
SP	Slow pathway
TV	Tricuspid valve

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Atrioventricular (AV) node is a part of the only normal electrical connection between the atria and the ventricles and plays a role to coordinate heart rate. Since the discovery of the AV node by Tawara [1], it still fascinates many

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investigators in this field and has been enthusiastically studied by pioneers in anatomy, physiology, and cardiology. Highly intense debates have been exchanged on several major themes reflecting duality of the electrophysiological character of the AV node, which is known to be both a conductor of impulses and an oscillator. Characteristics of the AV node with slow conduction and long refractory period cause a delay of electrical impulse transmission from the atria to the ventricles, which promotes the blood repletion to the ventricles but prevents the ventricles from working excessively fast in atrial tachyarrhythmia. The AV node can become the subsidiary center of the pacemaker activity of the heart when the primary pacemaker in the sinus node fails to control the cardiac rhythm as the result of either a depressed automaticity or an impaired conduction. By showing the peculiar properties aforementioned, the AV node plays an extremely important role in pathophysiology which affects the occurrence of various arrhythmias. The ion currents, the channels, and the molecular mechanisms affecting its electrophysiological characteristics have been clarified through the development of molecular biology which involves in many investigations from recording an action potential (AP) of a cell with microelectrodes to an application of the patch-clamps technique to an isolated nodal cell. The elucidation of the structure of the AV node, the peculiarity of the AV conduction at histological and clinical levels, and the mechanism of the occurrence of arrhythmia has made tremendous progress since the introduction of the clinical electrophysiological study including a record of the His bundle electrocardiogram and the catheter ablation. But unsolved problems still remain.

A profound complexity of the AV nodal area has presented a significant challenge to anatomists. Many scientists have disagreed on several major definitions of the components of the AV node, and the lack of a common terminology for the tissues at AV node was recently acknowledged [2]. Due to the lack of consensus on such fundamental issues of anatomy of the AV node, electrophysiologists have to choose between two distinctly different "anatomic" and "potential" approaches during clinical evaluation of AV conduction and therapy of AV nodal reentrant tachycardia (AVNRT) [3]. The "anatomic" approach is based on targeting the anatomic landmarks of a slow pathway (SP), such as the isthmus between the orifice of the coronary sinus (CS) and the tricuspid valve (TV). The "potential" approach is based on targeting a characteristic low-frequency potential being as the signature of a SP [4, 5].

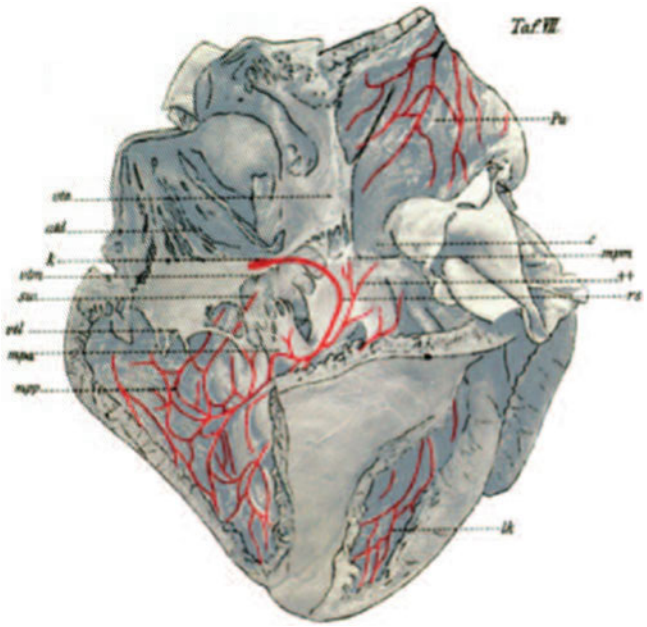

Fig. 2.1 The disposition of the atrioventricular conduction axis. Conduction pathway from the atrioventricular node (*k*) to the bundle of His, the right bundle (*rs*), and then to the right ventricular muscles in a human heart (Reproduced from Tawara [1])

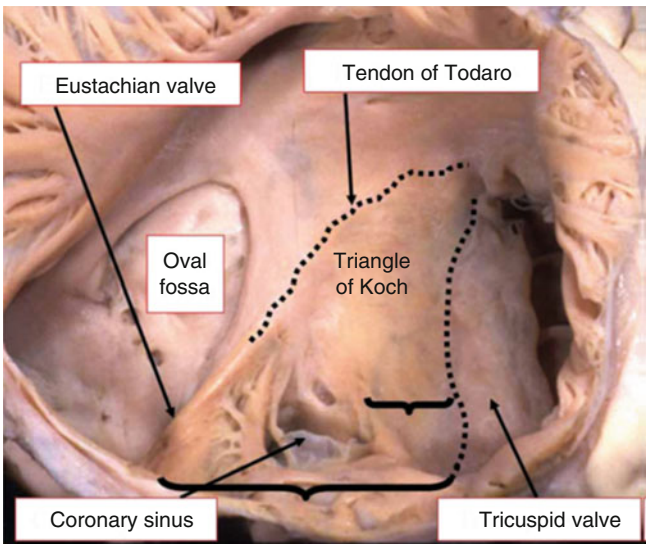

Fig. 2.2 Magnified view of the important posterior wall of the right atrium. Note the location of the triangle of Koch, delimited by the site of the tendon of Todaro, the attachment of the septal leaflet of the tricuspid valve, and the orifice of the coronary sinus. The *small bracket* shows the site of the septal isthmus, whereas the *large bracket* shows the inferior, or cavo-tricuspid, isthmus (Reproduced from Anderson and Cook [13], with permission)

Structure of the AV Node

The AV node, a.k.a Tawara's node, was first discovered and reported by Dr. Tawara in 1906 [1]. His excellent works are not only the morphologic discovery of the nodal tissue but also a suggestion of its functional role as part of "the impulse conduction axis of the heart" through which the atria and the His-Purkinje system are connected (Fig. 2.1). His report was published a few years after Einthoven recorded an electrocardiogram for the first time.

The AV nodal tissue is a flat and fan-shaped mass located in the fibrous triangle at the bottom of the right atrium (RA) and in the upper part of the triangular area first illustrated by Koch [6] (Fig. 2.2). In human its size is roughly 6 mm in

length and 3 mm in width. It connects with atrial muscle bundles through the transitional cells and with the His bundle through the nodal infrastructure. Thus, the node *per se* is not isolated from surrounding tissues, which is also described in the first report of Tawara. The nodal cells which consist of the midsection of the AV nodal area have a small spindle-like shape and form cell bundles, which is similar to sinus nodal cells. But the points slightly different from them are that a bunch of muscle cells runs along the long axis of the AV node and makes a complex network structure by divergence and anastomosis. And it is clearly distinguished from other cardiac muscle tissues (e.g., atrial and ventricular muscles, Purkinje fibers). In fine structure of nodal cells, a transverse tubule does not exist, the connection between cells is simple, and the surface of the gap junction is smaller [7, 8].

The anatomic definition of the AV node has been debated. On the purely morphologic grounds, Anderson's group insists that the term *AV node* can be applied only to morphologically nodal tissue outside of a fibrous collar [9]. Conversely, other investigators suggest the existence of an anterior enclosed and a posterior open part of the node [10] and that the AV node includes all structures contributing to atrial-His interval and being associated with rate-dependent and dual-pathway properties [11]. Billette's definition is most acceptable and we will adhere to his definition of the AV node as a heterogeneous structure, consisting of the compact AV node as well as nodal extensions and approaches, comprising tissues of the triangle of Koch.

Location of the AV Node

As described by Tawara [1], the AV conduction axis is a continuous system of histologically discrete cells which originates from the atrial myocardium and inserts in the ventricular Purkinje cells. The atrial components of this axis constitute the AV node, or the "Tawara's node" (Fig. 2.1). The node, along with its surrounding zones of transitional cells, is positioned at the base of the atrial septum, occupying the upper part of the triangle of Koch (Fig. 2.2). The apex of the triangle is the AV component of the membranous septum (Fig. 2.3) [12, 13]. This fibrous structure is continuous on its left side with the thickened rightward end of the region of fibrous continuity between the leaflets of the aortic and mitral valves (Fig. 2.4). This fibrous thickening, a.k.a. the right fibrous trigone, together with the membranous septum, forms the so-called central fibrous body. When seen from the right side, the structures forming the sides of Koch's triangle insert directly to the membranous septum (Fig. 2.3). The more posterior border is the fibrous strand continuation of the valve of the inferior vena cava (IVC), a.k.a. the tendon of Todaro. The anterior border of the triangle is the line of attachment of the septal leaflet of the TV. This structure crosses the right ventricular (RV) aspect of the membranous septum in most hearts, dividing it into AV and interventricular components. The muscular triangle delimited by these two borders separates the hinge of the TV in the RV from that of the mitral valve in the left ventricle and seems to be a septum. In the strictest sense, however, the area is not truly a septum, since a fibroadipose continuation of the inferior AV groove separates the atrial wall forming the floor of the triangle from the underlying ventricular musculature (Fig. 2.4). The artery supplying the AV node traverses this tissue plane, extending superiorly to the point where the node penetrates the plane of AV insulation to become the bundle of His. The base of the triangle is positioned inferiorly and is occupied by the CS. Adjacent to the orifice of the CS, which is guarded by the Thebesian valve, there is an important isthmus, a.k.a. the septal isthmus. This is bounded by the CS itself posteriorly and the hinge of the TV anteriorly. The musculature of the septal isthmus then continues inferiorly as the vestibule of the TV, while the musculature around the CS orifice itself continues

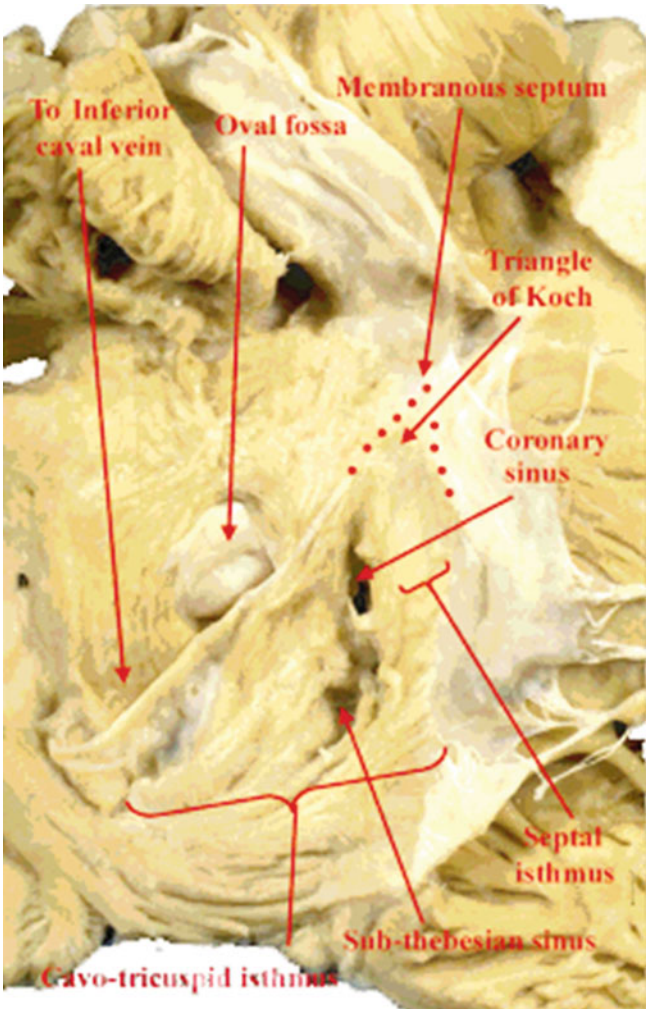

Fig. 2.3 Dissection of the human heart showing the medial wall of the right atrium removed its endocardial lining. This demonstrates the anisotropic orientation of the muscle fibers within the major muscular bundles delineated by the holes in the atrial wall (Reproduced from Anderson et al. [12], with permission)