DIAGNOSIS AND MANAGEMENT OF DIFFERENT TYPES OF HEART BLOCK

ESSAY

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cardiology

BY

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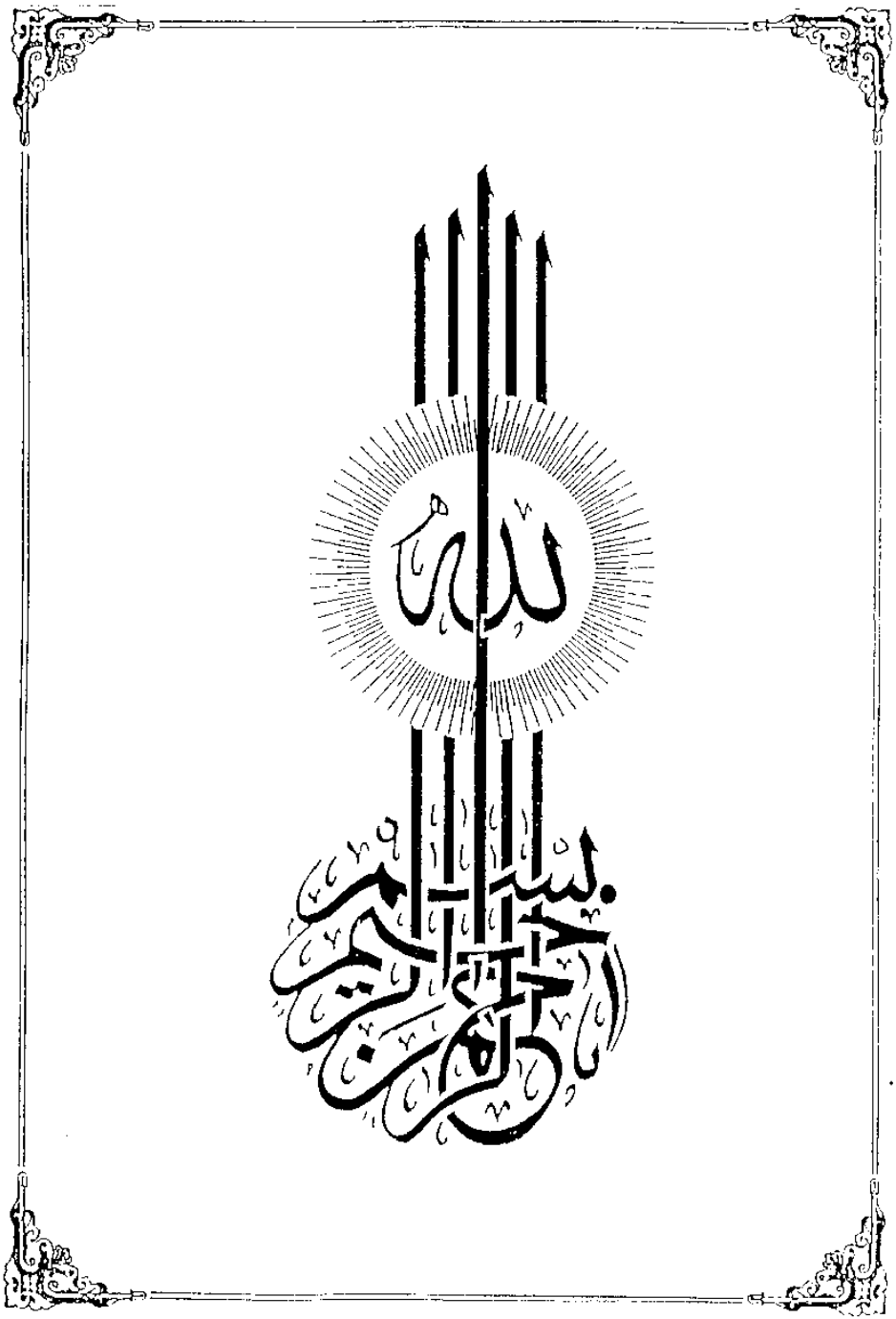

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INTRODUCTION

Until the late nineteenth century, fierce controversy existed between the neurogenic and myogenic theories of cardiac conduction. In 1883, Walter Gaskell mapped myogenic circuits traversed cardiac impulses. Based on his studies, he insisted that specialized strands of tissue bridged the atrium and ventricles. He urged that these be tracked down. In 1893, Wilhelm His, Jr., discovered a muscle bundle uniting the auricular and ventricular septal walls, thus firmly establishing the theory of myogenic conduction. This important anatomic milestone formed the basis for subsequent electrophysiologic studies and His bundle electrocardiography.

In 1958, Alanis and associates recorded His bundle potentials during perfusion by a donor animal. In the following years, several others reported electrogram recording of the conducting tissue. Stuckey & Hoffman and their associated recorded His bundle potentials during open heart surgery in 1959. Isolated reports of catheter recordings of His bundle activity in man were reported by Giraud and associates as early as 1960. In 1968, Scherlag and associates reported the catheter technique for His bundle recordings in the

intact dog. In the same year, Narula, Scherlag, and their associates developed the catheter technique for stable His bundle recordings in man. Since then, the catheter technique has been extensively utilized in numerous laboratories all over the world.

CHAPTER I

Anatomy And Physiology Of The Conduction System

ANATOMY AND PHYSIOLOGY OF THE CONDUCTION SYSTEM

Anatomy

The sinus node and AV node are analogous structures arising at the junction of the right and left superior cardinal veins, respectively, with the sinus venosus (Patten, 1953). Grossly the sinus node lies near the junction of the superior vena cava with the right atrium (Hudson, 1960 and James, 1961).

The sinus node is about 10-20 mm long, lying just beneath the sulcus terminalis. The shape of the node is roughly a flattened ellipse, and it does not have a head or tail as is commonly alleged. Being located a millimeter or less beneath the epicardium, it is heir to all the diseases which afflict the surface, most notable pericarditis (Fig. I.1).

Internally the most intriguing feature of the sinus node is its constant relation to a disproportionately large, centrally located artery. The framework of the sinus node is dense collagen which is closely attached to the entire circumference of the central artery.

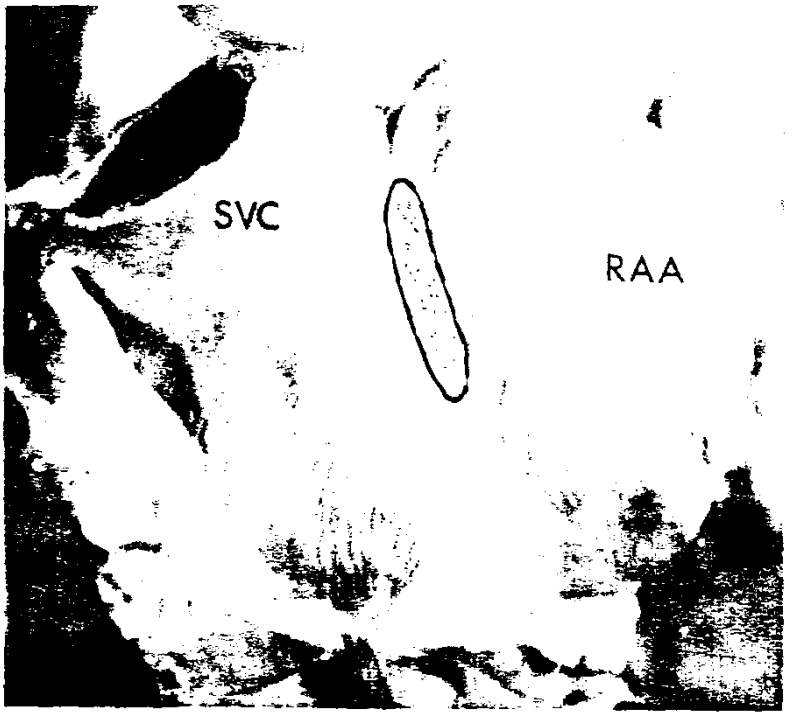

Fig. (I.1): Region of the S-A node, SVC, superior vena cava; RAA, right atrial appendage. S-A node is diagrammatically depicted by broken lines. (Chung, E.K.: Artificial cardiac pacing practical approach, The Williams & Wilkins Company P. 20).

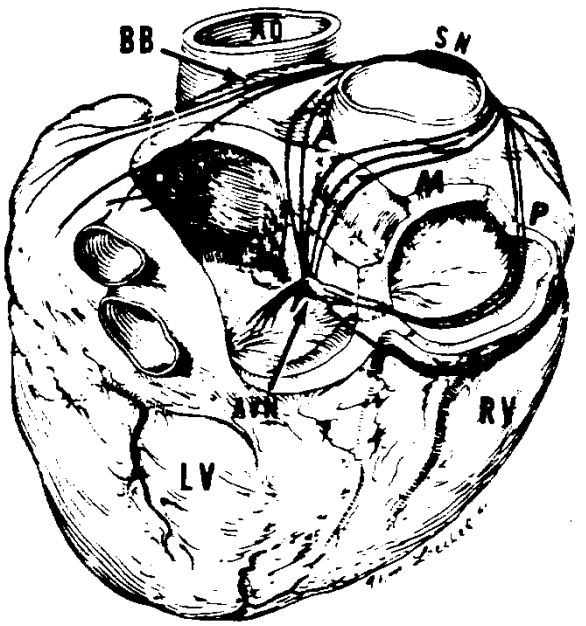

Fig. (I.2): A diagram of the three internodal tracts: A = anterior, M = middle, and P = posterior. The major interatrial tract branches from the anterior internodal tract and courses along with Bachmann's bundle (BB) to the left atrium. SN = sinus node, AVN = atrioventricular node, Ao = aorta, LV = left ventricle, RV = right ventricle. (From James, T.N. and Sherf, L.: Specialized tissues and preferential conduction in the atria of the heart., Am. J. Cardiol. 28: 414, 1971).

Within this collagen lattice, interlacing bundles of fibers also attach to this framwork. These fibers are of smaller diameter and stain paler than ordinary myocardium with almost all dyes. There are numerous nerve endings but no ganglia within the sinus node. At the anterior and posterior margins of the sinus node, however, there are many ganglia. Within the midportion of the sinus node is a variable number of stellate cells which have a large round nucleus and are known as P cells. Sinus node fibers distribute from these cells, which are often near nerve endings. It has been suggested that these primitive appearing cells may be the site of actual pacemaking within the sinus node.

Pathology of the sinus node is closely related to two of its anatomical features: its proximity to the epicardium and its proximity to the centrally located artery. Pericarditis almost invariably involves at least the epicardial surface of the node and often even deeper portions.

Vascular lesions are generally of 2 types: those causing occlusion of the main coronary artery proximal to the sinus node branch, and those involving small

arteries directly. The former vascular lesions are usually associated with acute ventricular myocardial infarction and commonly with atrial arrythmias. The lesions of the small arteries include all the diseases affecting such arteries e.g., lupus erythmatosus and polyarteritis nodosa.

The AV node: (Fig. I.3)

The human AV node is situated just beneath the right atrial endocardium directly above the insertion of the septal leaflet of the tricuspid valve and just anterior to the ostium of the coronary sinus.

The surface it presents toward the right atrium is convex, and the opposite surface (concave) rests on the collagenous base of the mitral annulus. Internally slender fibers of AV node interweave to form a meshwork, but there is much less collagen between fibers than in the SA node. Fibers are slightly thicker and shorter than those of the sinus node but not as thick as those of ordinary myocardium.

Behind the AV node, in the very small area between it and the coronary sinus, there are a number of autonomic ganglia. These are presumably vagal ganglia. In

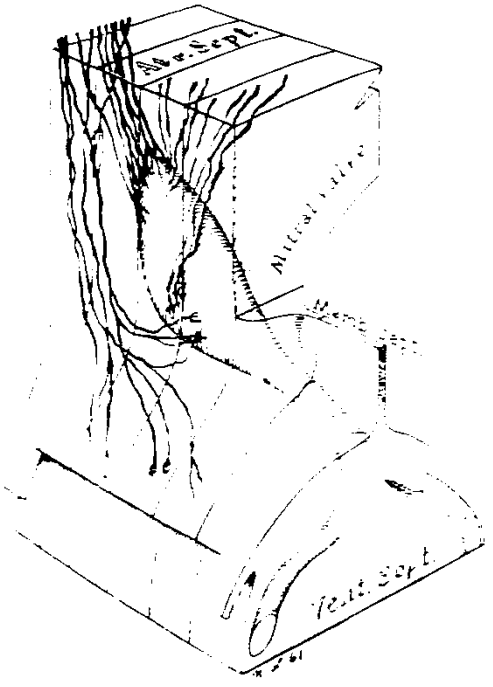

Fig. (I.3): Diagram of the atrioventricular junction, showing the three-dimensional relationships of the atrioventricular node and bundle of His to surrounding structures: atrial septum, mitral valve annulus, and membranous interventricular septum. The right bundle branch extends from the atrioventricular node as a tubular structure under the right septal endocardium, the left bundle branch cascades down the left side of the septum as a sheet of cells (From James, T.N.: Morphology of the human atrioventricular node, with remarks pertinent to its electrophysiology. Am. Heart J. 62:652, 1961).