LEFT ATRIAL APPENDAGE DOPPLER FLOW PATTERNS : IMPLICATIONS ON THROMBUS FORMATION

A THESIS

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بسم الله الرحمن الرحيم

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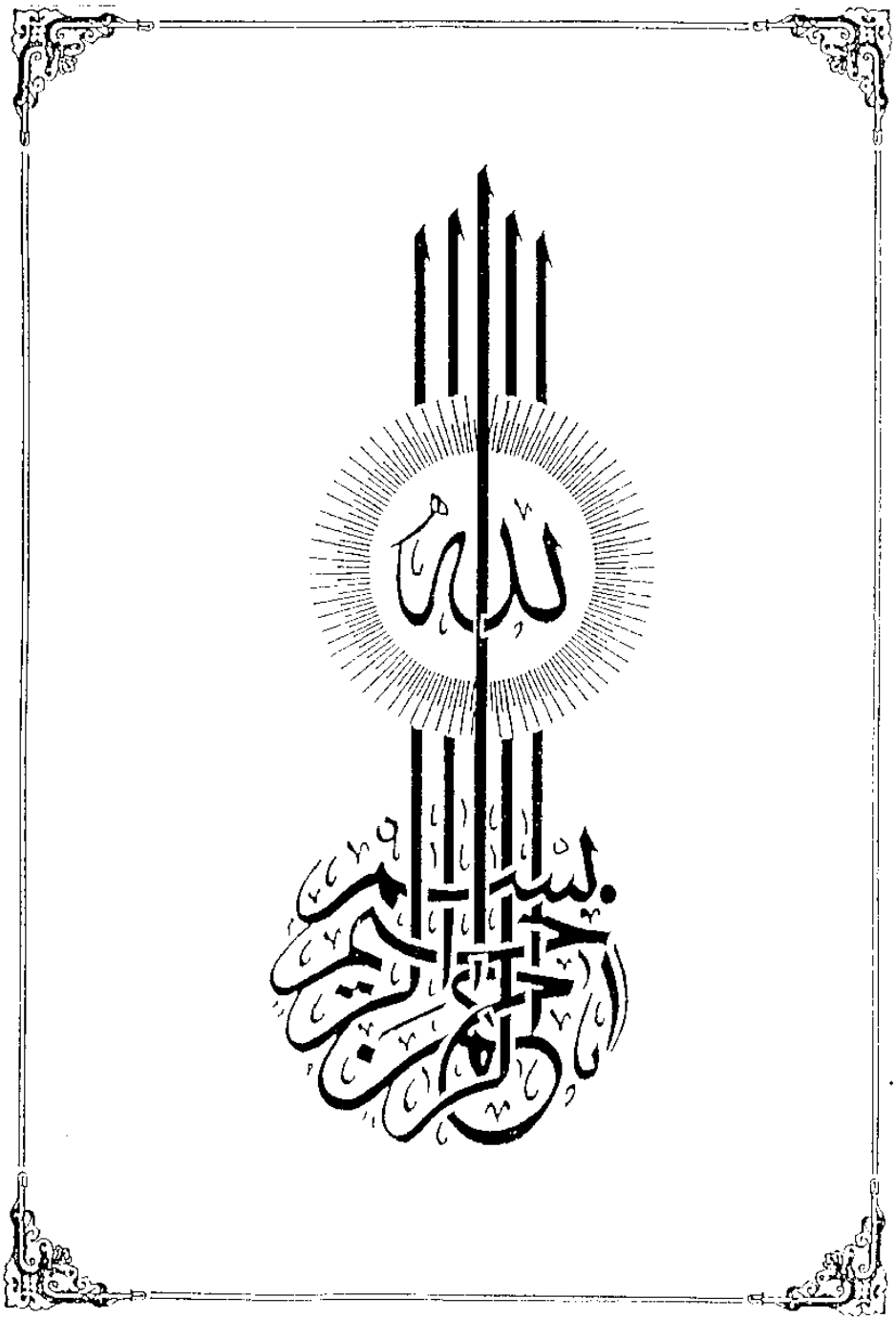

This work
is dedicated to

My Father, My Mother & My Fiance

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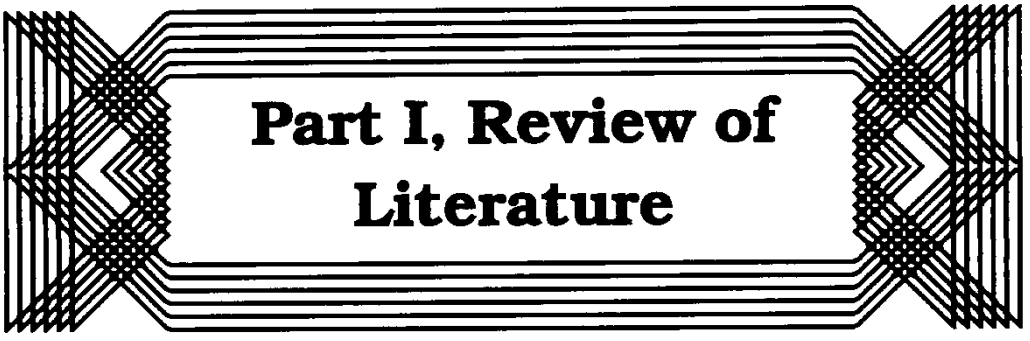

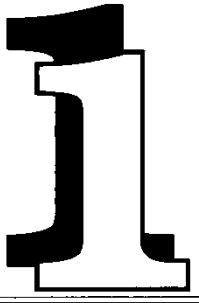

ANATOMY OF THE LEFT ATRIUM AND LEFT ATRIAL APPENDAGE

The left atrium is located superiorly, in the midline and posterior to the other cardiac chambers. It forms the base of the heart, the esophagus abuts directly upon its posterior surface, while the aortic root impinges upon its anterior wall. The right atrium is located to the right and anterior. Left ventricle is to the left, anterior and inferior. Anteriorly, the long, narrow left atrial appendage projects forward and partly overlaps the beginning of the pulmonary trunk.

The left atrium is 3 mm in thickness, slightly thicker than the right atrium (Hurst., 1990). The wall of the atria contains three anatomically distinct layers:

1. **The outer epicardium.**
2. **The muscular myocardium.**
3. **The inner endocardium.**

The epicardium is continuous with the outer covering of the ventricles and is composed of mesothelium, connective tissue, and

some fat. It also contains small nerve branches and the main coronary blood vessels.

The endocardium consists of endothelium and a layer of fibroelastic connective tissue that is somewhat thicker in the atria than in the rest of the heart.

The inner myocardial layer contains many nerve fibers and sensory endings. These are particularly abundant in the areas of the sinus node, the atrioventricular node, and the junction of both the venae cavae and right atrium and the pulmonary veins and the left atrium. In general, that atria have a richer supply of sensory endings than the ventricles. Small ganglion cells occur in the atrial myocardium, and nerve trunks accompany the main branches of the coronary arteries.

The muscular layer of the atrial myocardium may be divided into two groups:

1. **A superficial layer that encircles both atria, and;**
2. **A deep layer that is independent for each chamber.**

These fibers form two muscular loops that encircles the venous inlet into each atrium.

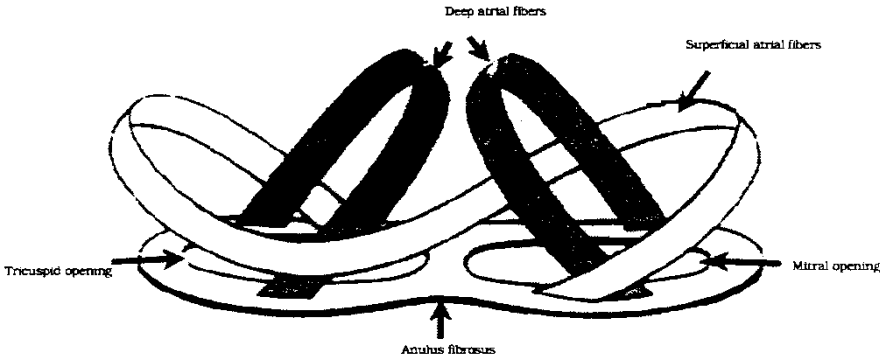

Fig 1: Highly schematic representation of atrial muscle fibers. The superficial fibers encircle both atria; the deep fibers are independent for each chamber. Contraction of these muscle bands tends to propel the atrial contents toward the tricuspid opening on the right side and the mitral opening on the left side.

A number of distinct muscle bundles have been identified in each layer. The superficial muscle fibers fan out over the surface from their origin in the anterior part of the septum and the base of the superior vena cava. Most of these fibers insert into the anulus fibrosus. Contraction of the superficial and deep muscle bands reduces the top-to-bottom and lateral dimensions of the atria. This acts to propel the contents toward the AV ring. A prominent group of superficial fibers, the interatrial band, arises near the base of the superior vena cava and passes on the anterior surface of the atria to the left atrium, where it divides and encircles the left atrial appendage. This band has been suggested as a direct path for conduction of the excitatory process to the left atrium. Other fibers extend to the right from the base of the superior vena cava and encircle the right atrial appendage.

In general, the deep muscle fibers run at right angles to the superficial fibers and make up the major muscular elements of the atria. Anular fibers surround the orifice of the superior vena cava, the coronary sinus, and the pulmonary veins. In addition, muscle bundles extend across the atrial septum to form the musculature of the atrial septum.

Atrial-venous junction:

High-speed angiocardiographic studies show that both the orifice of the superior and inferior venae cavae and the entrance of the pulmonary veins into the left atrium narrow and become elliptical during atrial systole due to contraction of the annular muscle fibers that surround these openings. As a consequence of this dimensional change, the resistance offered to the movement of blood through the caval openings is increased. This increase is usually sufficient to prevent regurgitation from the atria during the brief reversal of the caval-atrial pressure gradient produced by atrial contraction. However, as these vessels remain patent, they will act as a safety valve and permit blood to reflux into the veins if atrial pressure becomes unduly elevated.

On the left side of the heart, the pulmonary vein-left atrial junction tends to collapse if atrial pressure is low. Under these

circumstances, the elliptical cross section is maintained throughout diastole. As a result, a «vascular waterfall» is produced in that back flow of blood is prevented but forward flow is only minimally restricted. This occurs because interference with the normal flow will lead to an elevation in venous pressure and dilatation of the atrial-venous junction. However, a modest increase in left atrial pressure will not have much effect on the collapsed segment of pulmonary vein. Thus, back flow from the atrium under these circumstances will be inhibited by the high-resistance atrial-venous junction (Robert et al., 1985).

The trabecular left atrial appendage is a remnant of the original embryonic left atrium that develops during the third week of gestation. The left atrial cavity develops later and is formed from an outgrowth of the pulmonary veins.

Physical characteristics of the atria:

The cardiac atria serve as storage reservoirs for blood returning to the heart. This function is particularly significant during ventricular systole when the forward movement of blood is stopped by the closed AV valves and blood collects in the atria.

The atria are similar to the venous system in that they are collapsible when partially filled but function as an elastic structure when filled. The capacity of normal adult atria is approximately 160 ml for the right atrium and 140 ml for the left atrium. The left atrium, in addition to being smaller, is also less distensible (Robert et al., 1985).

The function of the left atrial appendage is unknown. Bercher et al (1983) speculated that the atrial appendage fills the space that is created within the pericardial sac during ventricular systole as the ventricles eject blood and decrease in size. The appendage passively fills during ventricular systole and then passively empties during ventricular diastole.

PATHOPHYSIOLOGY OF MITRAL STENOSIS

In normal adults, the cross-sectional area of the mitral valve orifice is 4 to 6 cm^2. When the orifice is reduced to approximately 3 cm^2, which is considered to represent mild mitral stenosis, blood can flow from the left atrium to the left ventricle only if propelled by an abnormal, though small, pressure gradient. When the mitral valve opening is reduced to 1 cm^2, which is considered to represent critical mitral stenosis, a left atrioventricular pressure gradient of approximately 20 mm Hg and therefore, in the presence of a normal left ventricular diastolic pressure, a mean left atrial pressure of approximately 25 mm Hg is required to maintain normal cardiac output at rest. The elevated left atrial pressure in turn raises pulmonary venous and capillary pressures, resulting in exertional dyspnea.

The first bouts of dyspnea in patients with mitral stenosis are usually precipitated by exercise, emotional stress, sexual intercourse, infection or atrial fibrillation, all of which increase the rate of blood