INDICATIONS FOR AORTIC VALVE REPLACEMENT IN PATIENTS WITH AORTIC VALVE INSUFFICIENCY

Essay

TO BE

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By

Sayed Zakaria Mohamed

M.B., B.Ch.

SUPERVISED BY :

Prof. Dr. Hassan Ezz El-Din Attia, M.D.

Prof. of Cardiology

Faculty of Medicine

AIN SHAMS UNIVERSITY

Prof. Dr. Ali Ahmed Ibrahim

Assist. **Prof. of Cardiology**

Faculty of Medicine

AIN SHAMS UNIVERSITY

FACULTY OF MEDICINE

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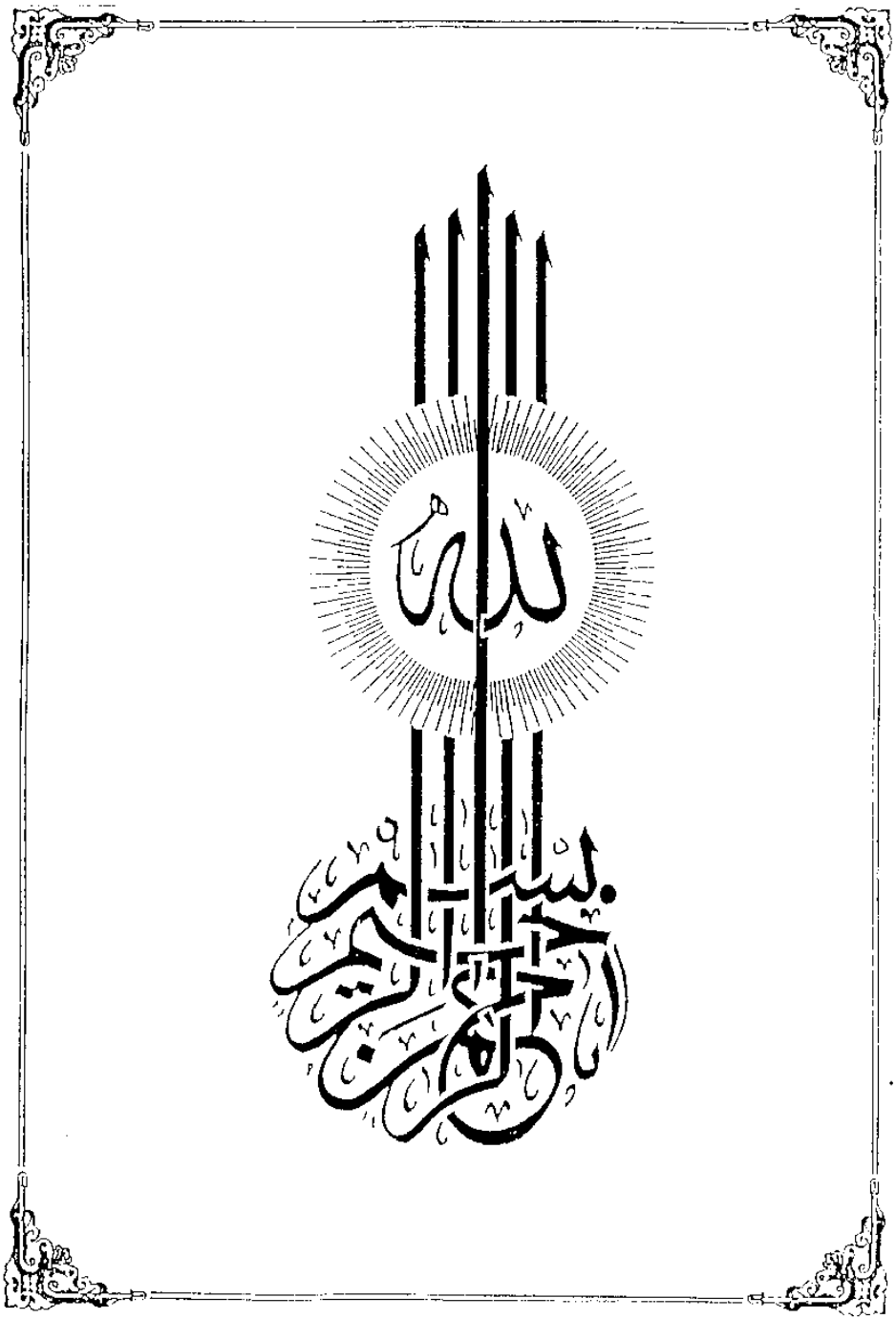

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INTRODUCTION AND AIM OF THE ESSAY

The choice of optimal timing of aortic valve replacement in patients with aortic insufficiency often poses a dilemma for clinicians. Although patients with long-standing aortic insufficiency may tolerate the volume overload state for many years, once cardiac symptoms and evidence of congestive heart failure occur, progression to irreversible left ventricular dysfunction may develop (Osbakken et al., 1981).

If aortic valve replacement is performed at a time when left ventricular function is normal, then the patient may be exposed unnecessarily to the potential hazzards that accompany prosthetic heart valves. However, if surgical correction of the volume overload state is delayed excessively, irreversible cardiac dysfunction may have already occurred and results of the operation may be unsatisfactory.

Several studies have investigated preoperative clinical, haemodynamic and noninvasively derived variables in attempts to identify those patients whose condition will improve after undergoing surgery and those patients who may develop refractory congestive heart failure, however, results of these studies have been conflicting. It is conceivable that information from a single test or

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measurement might prove sufficient to identify an optimal time for surgery, but it is much more likely that serial studies or a combination of tests, or both, would provide the reliable evidence needed before proceeding with surgery in a minimally symptomatic or asymptomatic patient. The aim of this essay is to throw light on these uptodate studies, with a concise review of the literature.

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CHAPTER I

ANATOMY OF THE AORTIC VALVE

The aortic orifice has a circular shape and is about one inch in diameter (Lookhart et al., 1965). Since the plane of the orifice is oblique with the right posterior side lower than the left anterior side. The orifice lies infront and to the right of the left atrioventricular orifice (Warkich et al., 1973), from which it is separated by the anterior cusp of the mitral valve. It is also posteroinferior and slightly to the right of the pulmonary valve (Last, 1973). The portion of the ventricle immediately below the aortic orifice is termed the aortic vestibule and possesses a largely fibrous instead of muscular wall. The ventricular septum appears at the area of the commissure between the right and non coronary cusps (Clark et al., 1980).

The human aortic valve consists of three semilunar cusps which surround the orifice of aorta, with two cusps anterolaterally i.e coronary cusps and the third lies posterior i.e. noncoronary cusp (Hurst, 1978). The cusps are made of relatively inelastic free material. Each consists of duplication of endocardium with little fibrous tissue interposed between the two endothelial layers. The cusps are of approximately equal size and are cupshaped (Fig. 1).

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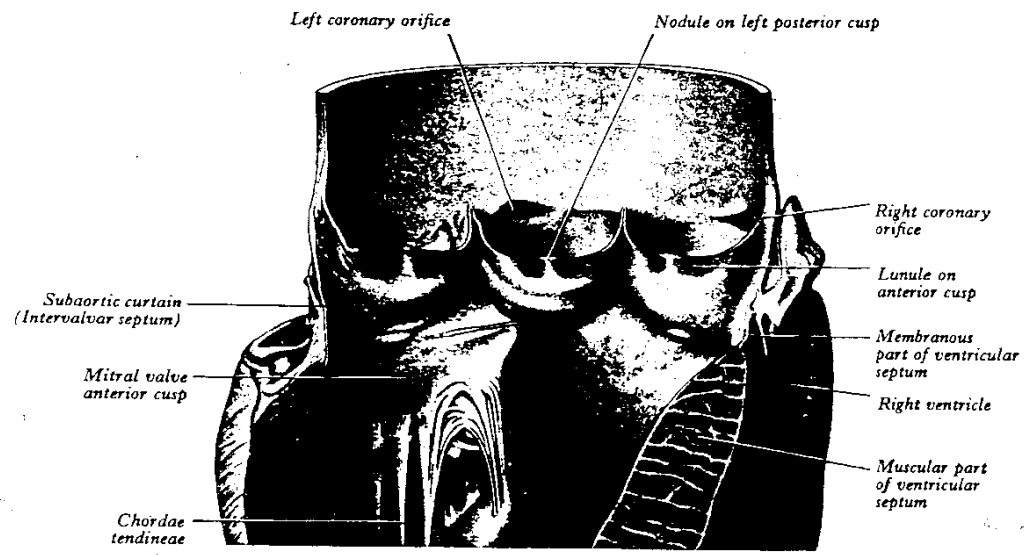

Fig. (1): Aortic orifice and aortic valve cusps. (After Warkwich, 1973).

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The U shaped convex lower edge of each cusp is attached and suspended from the aortic root with the upper free edge projecting into the lumen. Behind each cusp the vessel wall bulges outward forming a pouch like dilatation known as the sinus of valsalva whose distal limits extend to the uppermost attachement of the commissures (Sabiston et al., 1976). The cusps with their corresponding sinuses form pockets facing upwards and preventing backward flow into the ventricle. The free edge of each cusp is concave with a nodular interruption of the center of the leaflet termed the noduli arantii or corpus arantii. From this nodule tendinous fibres radiate through the cusps to its attached margin but are absent from the two narrow crescentic portions which are termed the lunules. These are placed on each side of the nodule and immediately adjoining the free margin (Merklin et al., 1969). The cusps of the aortic valve are thicker and stronger than those of the pulmonary valve. Also, the lunules are more distinct and the nodules are thicker and more prominant.

The coronary arteries arise from the aortic bulb,which is marked externally by an indentation known as the supra-valvular ring, as both main coronary arteries usually arise just below this ring (Oram, 1981) and internally it is corresponding to the upper third of the sinus (Hurst, 1978).

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Functional anatomy of the aortic valve:

Pulse duplicator studies of valvular motion indicate that at the beginning of ejection phase, all three cusps are rapidly and passively thrust upward away from the center of aortic lumen to form a stellate orifice.

A further opening of the valve is dependant on forward flow velocity which as increased, the configuration of the valve orifice changes from stellate to triangular and later to circular in shape. Eddy currents within the sinuses of Valsalva cause a slow wave-like motion of the free edge and billowing of the base of each cusp (McMillian, 1955), thus, preventing occlusion of the coronary ostia and maintaining a posture that allows slight reversal of flow to result in immediate closure without regurgitation (Rushmer, 1961). During ventricular diastole the cusps fall passively into the lumen of the vessel as they support the column of blood above. The noduli arantii meet in the center and contribute to the support of the leaflets. The geometry of the cusps and the strong fibrous tissue support provide excellent approixmations of leaflets and prevent regurgitation.

Surface anatomy of the aortic ring and ascending aorta:

It corresponds to a line 2.5 cm long drawn from the medial end of the left third intercostal space downwards and to the right. Two parallel lines drawn from the

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extermities of this line upward and to the right as far as the right border of the sternal angle outline the ascending aorta (Warkwish and Williams, 1973).

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CHAPTER II

AETIOLOGY AND PATHOLOGY OF AORTIC INSUFFICIENCY

Aortic regurgitation may be caused by:

A. Valvular disease (intrinsic disease of the valve).

B. Aortic root disease.

Patients with aortic regurgitation due to primary valvular disease may show secondary dilatation of the aortic annulus which intensifies the amount of regurgitation (Braunwald, 1980).

Gibson (1978) adds to the broad causes of aortic regurgitation loss of support to aortic valve cusps which occurs in some cases of ventricular septal defect.

A. Incompetence of aortic valve due to intrinsic aortic valve disease:

Rheumatic fever is one of the commonest causes of primary aortic insufficiency (Segal et al., 1956) and (Enghoff,1973).

The aortic valvular incompetence of rheumatic origin may be either pure or associated with varying degree of stenosis. Classically when pure valvular insufficiency is present, the rheumatic process has spared the commissures (Rackely et al., 1982). The cusps show intrinsic change characterised by contracture from scarring causing

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shortening of the cusps in both length and breadth. Because of these changes the surface area of the cusps is inadequate to guard the valvular orifice. So, at the center of the aortic valve orifice there is a triangular defect through which blood regurgitates. In addition, rheumatic endocarditis may cause fusion between adjacent cusps at the conmissures. These cusps involved in the process of adhesion at the commissures are prevented from full lateral excursion during ventricular systole. If the change is sufficient, valvular stenosis may accompany the insufficiency (Eliot et al., 1964).

Roberts et al. (1970) claimed that isolated aortic regurgitation is seldom caused by rheumatic fever. However, DePace et al. (1984) stated that rheumatic valve disease appear to be the most common cause of aortic regurgitation, although there is decline in its incidence.

infective endocarditis may attack a valve previously affected by rheumatic disease, congenitally deformed valve or rarely normal aortic valve (Tompessett and Lubash,1961). The most common endocarditis inducing bacterial agent is streptococcus viridans. Its incidence accounts for 39-50% in most of several clinical series, while staphyloccoci accounts for 12-23%. Infective endocarditis results in aortic regurgitation in several ways. Vegetations may

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