IMAGING OF THE AORTIC VALVE

THESIS

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BY

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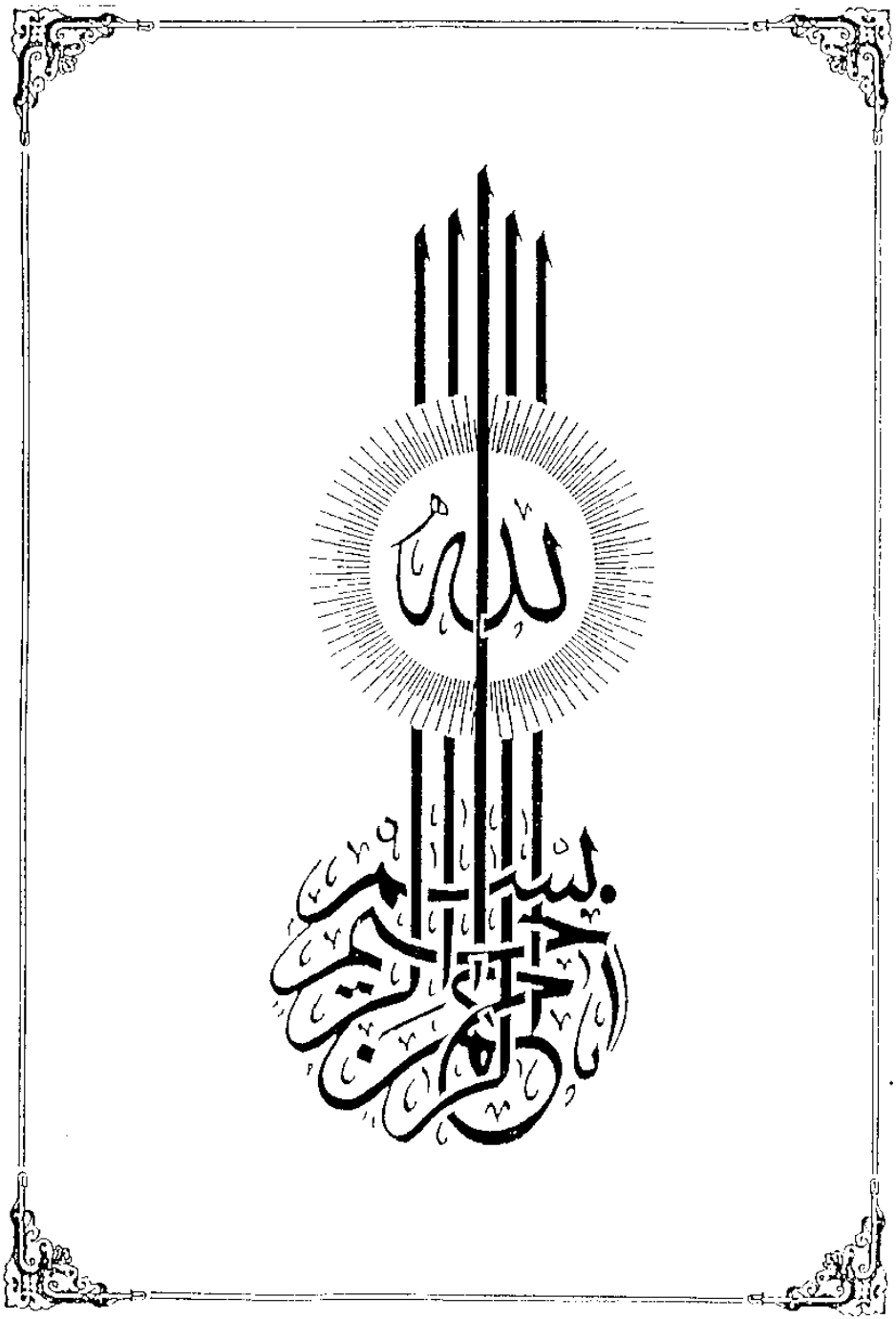

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Introduction

Decisions regarding the management of patients with aortic valve disease are fundamentally related to the presence or absence of cardiac symptoms and to the presence or absence of left ventricular dysfunction. Much of stimulus for the use of noninvasive diagnostic techniques in patients with aortic valve disease has resulted from limitations in the assessment of left ventricular function from the clinical history and physical examination, the ECG and the chest X-ray. Assessment by these clinical tools may not always be easy or accurate.

Hence, the need for objective reliable imaging modalities to know whether the patient has a normal or abnormal valve because of the potential need for prophylactic treatment for endocarditis; to determine left ventricular size and function to help estimate the severity of the problem and to select the proper time for aortic valve replacement.

This work aims at discussing the different and recent reliable imaging modalities in diagnosis and estimation of the severity of aortic valve disease.

Anatomy of the Aortic Valve

The aortic valve is situated at the junction of the left ventricular outflow tract and the ascending aorta.

Developmentally, the aortic valve represents the aortic part of the truncus arteriosus *(Grant, 1962)*

The aortic valve is normally tricuspid and is composed of three basic components: a fibrous skeleton, delicate cusps and sinuses of Valsalva *(James et al., 1982)*. These components form the three cuplike structures that constitute the entire valve mechanism *(Fig. 1)*.

The crownlike skeleton of the valve is a composite of three U-shaped solid collagenous structures and is in fibrous continuity with the anterior leaflet of the mitral valve and with the membranous septum.

The three semilunar delicate fibrous cusps are of approximately equal size and are referred to as right, left and non-coronary (posterior) cusps. From the aortic aspect, each cusp appears concave. The cusps are attached by their convex margins to the wall of the aorta at its junction with the left ventricle. At the centre of the free margin of each cusp, there is a tiny fibrocartilagenous nodule known as the corpus Arantii *(Hudson, 1965)*. These nodules fill the small central gap between the cusps in the diastole. On either side of each nodulus is an extremely thin crescent-shaped portion of the cusp termed the lunula. The lunulae are occasionally fenestrated near the commissures *(James et al., 1982)*.

During ventricular systole, the cusps are passively thrust upward away from the centre of the aortic lumen. During ventricular diastole, the cusps fall passively into the lumen of the aorta as they support the column of blood above. The noduli Arantii meet in the centre and contribute to the support of the leaflets. The geometry of the cusps and the strong fibrous tissue support provide excellent approximations of the leaflets and prevent regurgitation of blood *(James et al., 1982)*.

The aortic sinuses (sinuses of Valsalva) are dilated pockets of the aortic root that form the outer component of the three cuplike closing structures of the aortic valve. The coronary arteries arise in the upper third of two of the sinuses. The origins of the coronary arteries are the basis of the nomenclature for the sinuses and cusps. The ostia of the right and left coronary arteries identify the right and left sinuses and cusps. The sinus and cusp without an associated artery are termed non-coronary (posterior) *(James et al., 1982)*.

The aortic orifice is a circular aperture infront and to the right of the mitral valve orifice, from which it is separated by the anterior cusp of the mitral valve. The aortic valve ring usually lies below that of the pulmonary valve and its diameter is about 2.5cm. *(Hudson, 1965)*.

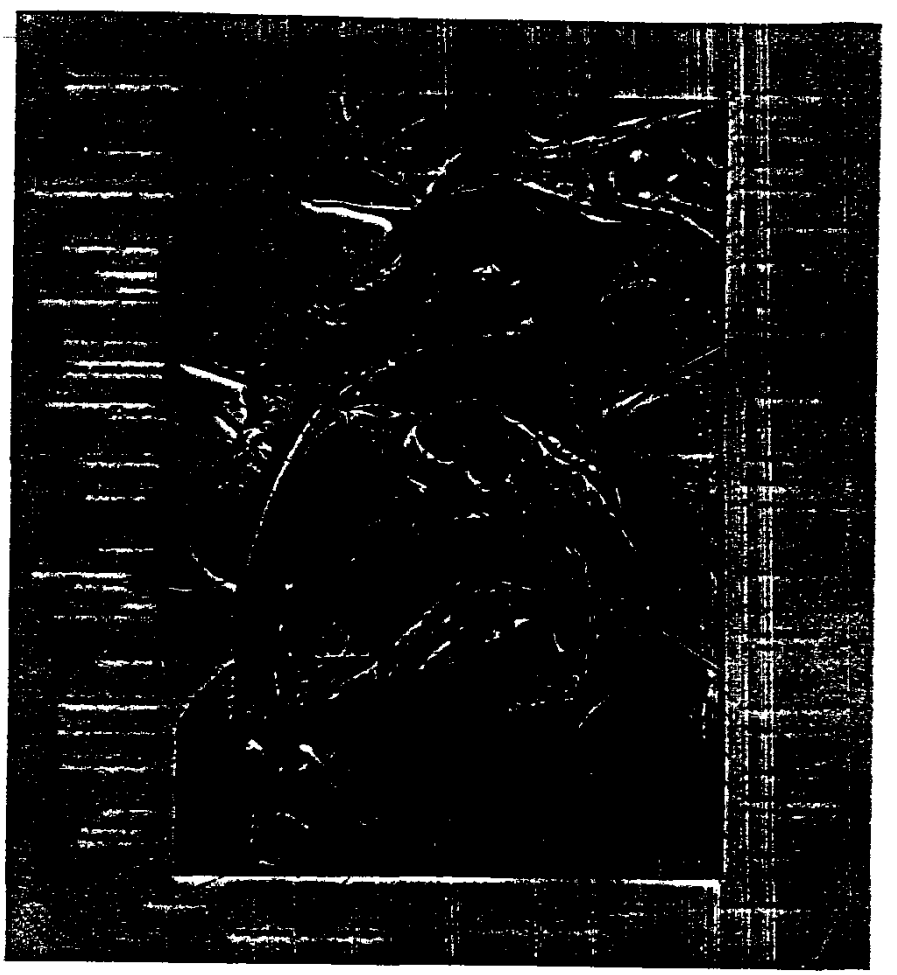

Fig. 1: View into the left ventricle and the origin of aorta with its semilunar valve *(from: Pernkopf, 1980).*

Goor, Lillehei and Edwards, 1969, described the relations of the aortic cusps. The non-coronary cusp is related to the right atrium, interatrial septum and left atrium; the left coronary cusp to the exterior and the pulmonary trunk; and right coronary cusp lies opposite the crista supraventricularis. Some rotational variations in the aortic valve may occur so that, either the left commissure or the left cusp is related to the atrial septum.

The aortic valve is somewhat tilted, so that, parts of the right and left cusps are sunk deeper into the heart than the part arising from the anterior mitral leaflet. Consequently, the ascending aorta is not perpendicular to the base of the ventricles but shows an angle of about 120 . Since the mitral valve is also slightly tilted, the angle formed between the axes of these two valves is approximately 40 *(Du Plessis and Marchand, 1964)*. Occasionally, in older age, the angle that the aortic axis forms with the base of the heart may reach 160 *(Goor et al., 1969)*.

Pathology of the Aortic Valve

I. AORTIC STENOSIS

Obstruction to left ventricular outflow tract is localized most commonly at the aortic valve (valvular). However, obstruction may occur above the valve (supra-valvular), or below the valve (discrete subvalvular) or may be caused by hypertrophic obstructive cardiomyopathy.

Valvular aortic stenosis without accompaning mitral valve disease is more common in men, and very rarely occurs on a rheumatic basis but, instead, is usually either congenital or degenerative in origin *(Roberts, Perloff and Costantina, 1971)*.

A- VALVULAR AORTIC STENOSIS

* Aetiology of Valvular Aortic Stenosis :

The condition of the aortic valve stenosis is known to occur by two mechanisms: congenital and acquired *(Folger, 1980)*.

I. Congenital Aortic Valve Stenosis

Congenital aortic stenosis represents 3-5% of all congenital heart diseases. Valvular aortic stenosis is the most frequent form of congenital left ventricular outflow tract obstruction. It occurs much more frequently in males than in females, with the sex ratio approximately 4:1 *(Mody and Mody, 1975)*.

The congenital unicomissural aortic valve is inherently stenotic and the unicuspid aortic valve is the only type of valvular stenosis at the time of birth *(Edwards, 1961)*. It produces severe obstruction in infancy and is the most frequent malformation found in fatal valvular aortic stenosis in children under the age of one year *(Moller, et al., 1966)*.

Congenital bicuspid aortic valves are not usually stenotic at birth, but many of them subsequently become stenotic and then are examples of acquired stenosis of a congenitally malformed valve *(Roberts, 1970)*. In contrast to this view, *Folger, 1980* postulated that congenital valvular aortic stenosis is nearly always the consequence of bicuspidization of the valve at birth.

Rarely, the aortic valve has three fused cusps with a congenitally stenotic central orifice, it then resembles the simple dome-like valve seen in some patients with pulmonic stenosis *(Roberts, 1970 a)*.

Approximately 20% of children with congenital valvular aortic stenosis have other cardiovascular malformations, the more common of which are coarctation of the aorta and patent ductus arteriosus, and all three of these lesions may coexist. Other less common malformations are ventricular septal defect and pulmonic stenosis *(Friedman, 1984)*.

II. Aquired Valvular Aortic Stenosis

Acquired valvular aortic stenosis may be a result of:

A) Rheumatic stenosis

This type is known also as the fibrous type of aortic stenosis and appears to be a direct result of rheumatic endocarditis. Such valves are frequently associated with rheumatic disease of the mitral valve and not infrequently with rheumatic disease of the tricuspid valve as well *(Edwards, 1965)*.

B) Idiopathic calcific stenosis

The calcific type of valvular aortic stenosis is seen almost universly in valves that lack the usual three cusps. This type is the commonest variety of acquired valvular aortic stenosis and occurs predominantly in men aged 60-70 years *(Pomerance, 1972)*.

The main theories that have been considered for the aetiology of calcific stenosis are:

1. Degenerative (senile) calcific stenosis (Fig. 8).

This common cause of aortic stenosis in adults appears to result from years of normal mechanical stress on the valve.

2. Atherosclerotic

Severe atherosclerosis involves the aorta and other major arteries; this form of aortic stenosis occurs most frequently in patients with severe hypercholestrolemia and is observed in children with homozygous type II hyperlipoproteinemia *(Braunwald, 1984)*. In this theory, *Cohen (1980)* thinks the calcific valvular stenosis is due to senile sclerosis thought to be a "wear-and-tear" degeneration of the aortic valve cusps.

3. Bacterial endocarditis

When bacterial endocarditis becomes healed, calcific foci at the sites of previous vegitations and valvular inflammation occurs *(Edwards, 1962)*.

4. Rheumatic endocarditis

A rheumatic background for calcific valvular aortic stenosis is supported by a history of acute rheumatic fever given by the patient and a minor mitral valve changes indicative of antecedent rheumatic endocarditis such as vascularization of the anterior mitral leaflet and/or mild commissural fusion *(Edwards, 1962)*.

C) Rare types

1. Rheumatoid involvement of the valve is a rare cause of aortic stenosis and results in nodular thickening of the valve leaflets and involvement of the proximal part of the aorta *(Braunwald, 1984)*.

2. Ochronosis is another rare cause of aortic stenosis *(Braunwald, 1984)*.

3. Uncommonly, aortic stenosis may result from the presence of a congenitally papillary mass or flap of endocardial tissue that obstructs an otherwise normal valve *(Rackley, et al., 1982)*.

4. Extensive thrombosis at the valve site has been a cause of aortic stenosis in lupus erythematosus *(Rackley et al., 1982)*.

* Pathology of Valvular Aortic Stenosis

I. Unicuspid Valve *(Fig. 2)*

It is a congenital type of aortic valves which has one cusp and one commisure *(Fig. 3)*. Attaching to the aortic wall at one region, the single cusp runs towards the opposite wall of the aorta, but fails to join the aortic wall in that position. Instead, the cusp turns on itself and makes a second attachement with the aortic wall near the first attachement to create the single commissure characteristic of this valve *(Edwards, 1965)*. He added that being deformed as it is, the valve bears some resemblance to the pulmonary valve in so-called dome-shaped congenital stenosis. This type of valves is intrinsically stenotic and in some instants, incompetence of the valve may be manifested additionally.

II. Bicuspid Valve

This type of valves may have a congenital or an acquired background as a result of rheumatic endocarditis.

Congenitally bicuspid valves may be stenotic with commissural fusion at birth, but more commonly they are not responsible for serious narrowing of the aortic orifice during childhood *(Emanuel et al., 1978)*, their abnormal architecture induces turbulent flow, which traumatizes the leaflets and ultimately leads to fibrosis, increased rigidity and calcification of the leaflets *(Fig. 4)* and narrowing of the aortic orifice *(Braunwald et al., 1963)*. Classically, the cusps are oriented anteriorly and posteriorly, the anterior or conjoined cusp being the larger, and from its sinus the two coronary arteries arise *(Fig. 5) (Waller et al., 1973)*. Infective endocarditis may develop on a congenitally bicuspid valve, which then becomes regurgitant. Rarely, a congenitally bicuspid valve is purely regurgitant in the absence of antecendent infection *(Fig. 6) (Friedman, 1984)*. Dissecting aneurysm of the aorta may complicate the bicuspid aortic valve whether or not it is associated with calcific stenosis and/or coarctation of the aorta *(Edwards, Leaf, and Edwards, 1978)*.