INTRODUCTION

Aortic stenosis is a common condition, becoming more prevalent with the ageing population. After the appearance of symptoms, aortic stenosis is associated with a high rate of death, if left untreated, up to 50%, within 2 years of symptom onset. Surgical valve replacement is the only definitive therapeutic strategy, indicated in presence of severe symptomatic disease.

Current conventional surgical techniques consist of partial or complete sternotomy with extracorporeal circulation and cardioplegic cardiac arrest ***(Ren, 2012).***

Despite the benefits of aortic valve replacement, this conventional technique is associated with a high operative mortality in the elderly patients with significant co morbidities, including severe respiratory dysfunction, renal insufficiency, and compromised cardiac function ***(Walther et al., 2007).***

Recent advances nowadays tend to replace all the conventional modalities by recent minimally invasive procedures reducing most of the complications of the conventional techniques.

Transcatheter aortic valve insertion is a new development that potentially offers a number of advantages to patients and healthcare providers. These include the avoidance of sternotomy and cardiopulmonary bypass, and much faster discharge from hospital and return to functional status. Modern design and manufacturing techniques have led to the development of a number of valve prostheses which can be compressed or crimped, reducing their size, and allowing delivery to the heart on a catheter through a vascular sheath ***(Klein et al., 2009).***

This transcatheter procedure is performed via puncture of the left ventricular (LV) apex or percutaneously, via the femoral artery or vein. Patients undergo general anesthesia, intense hemodynamic manipulation, and transesophageal echocardiography (TEE) ***(Billing et al., 2009).***

The role of the anesthetist in such a procedure is very important, as patients are often elderly with multiple co morbidities and organ dysfunction. The hemodynamic consequences of vascular and cardiac access and valve deployment in the beating heart may lead to morbidity and mortality if not adequately managed, and the postoperative course may be complicated. Careful anesthetic management along with meticulous perioperative care should facilitate improvements in outcome ***(Klein et al., 2009).***

AIM OF THE WORK

To discuss the new alternative management of aortic stenosis the transcatheter valve replacement, its approaches, technique and complications, together with the perioperative anesthetic management for such a procedure.

Chapter One

ANATOMICAL AND PATHOLOGICAL CONSIDERATION OF AORTIC VALVE DISEASE

Anatomy of aortic valve:

- ***Aortic root***

The aortic root is the direct continuation of the left ventricular outflow tract. It is located to the right and posterior, relative to the subpulmonary infundibulum, with its posterior margin wedged between the orifice of the mitral valve and the muscular ventricular septum. It extends from the basal attachment of the aortic valvar leaflets within the left ventricle to their peripheral attachment at the level of the sinutubular junction ***(Anderson, 2000).***

Approximately two thirds of the circumference of the lower part of the aortic root is connected to the muscular ventricular septum, with the remaining one third in fibrous continuity with the aortic leaflet of the mitral valve. Its components are the sinuses of Valsalva, the fibrous interleaflet triangles, and the valvar leaflets themselves ***(Piazza et al., 2008)***.

The leaflets have a core of fibrous tissue, with endothelial linings on their arterial and ventricular aspects. Their origin from the supporting left ventricular structures, where the ventricular components give rise to the fibroelastic walls of the aortic valvar sinuses, marks the anatomic ventriculoarterial junction ***(Piazza et al., 2008).***

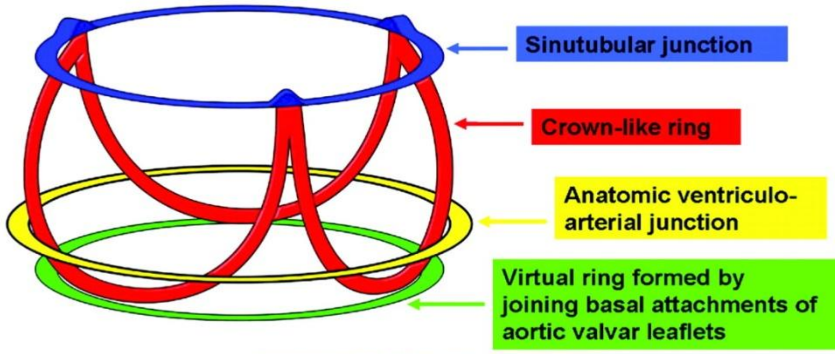
- ***Rings within the aortic root***

There are several rings to be found within the aortic root. The annulus defined by surgeon is a semilunar crown like structure demarcated by the hinges of the leaflets. The aortic root contains at least 3 circular rings and 1 crown like ring ***(Anderson, 2000).***

The valvular leaflets are attached throughout the length of the root. The leaflets take the form of 3-pronged coronet, with the hinges from the supporting ventricular structures forming the crown like ring. The base of the crown is a virtual ring formed by joining the basal attachment points of the leaflets within the left ventricle. This plane represents the inlet from the left ventricular outflow tract into the aortic root ***(Thubrikar et al., 2005).***

The top of the crown is a true ring “the sinutubular junction “demarcated by the sinus ridge and the related sites of attachment of the peripheral zones of apposition between the aortic valve leaflets (figure 1). It forms the outlet of the aortic root into the ascending aorta. The semilunar hinges then cross another true ring "the anatomic ventriculo-arterial junction”. The overall arrangement is well seen when the aortic root is opened subsequent to removal of the valvular leaflets ***(Antunes, 2005).***

A



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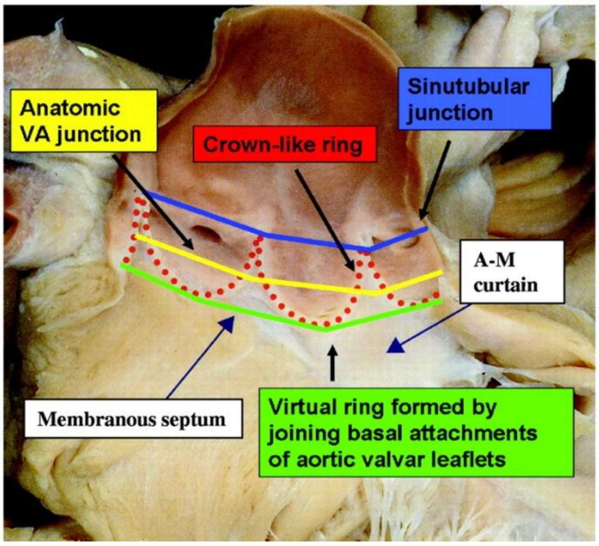

Figure (1): Three-dimensional arrangement of the aortic root ***(Piazza et al., 2008).***

This preparation also shows that the leaflets arise from ventricular muscle only over part of their circumference. The larger part of the noncoronary leaflet of the valve, along with part of the left coronary leaflet, is in fibrous continuity with the aortic or anterior leaflet of the mitral valve, with the ends of this area of fibrous continuity being thickened to form the so-called fibrous trigones ***(Anderson, 2006).***

The normal aortic root has a consistent shape with varying size. Measurements of the human aortic root revealed that the diameter at the level of the sinutubular junction exceeds that at the level of the virtual ring which formed by joining together the basal attachments of the leaflets ***(Antunes, 2005).***

- ***The aortic valve***

The normal aortic valve is trifoliate. It consists of the complex of the three semilunar cusps (figure 2) and there adjacent sinuses of valsalva. Terminology of the sinuses is derived from the respective coronary arteries, i.e., right, left, and non coronary.

Each of the three leaflets of the normal aortic valve has a free margin and a margin where it is attached in semilunar fashion to the aortic root. The maximal height of each leaflet is considerably less than that of its sinus on account of its scoop-shaped free margin ***(Siew yen ho, 2009).***

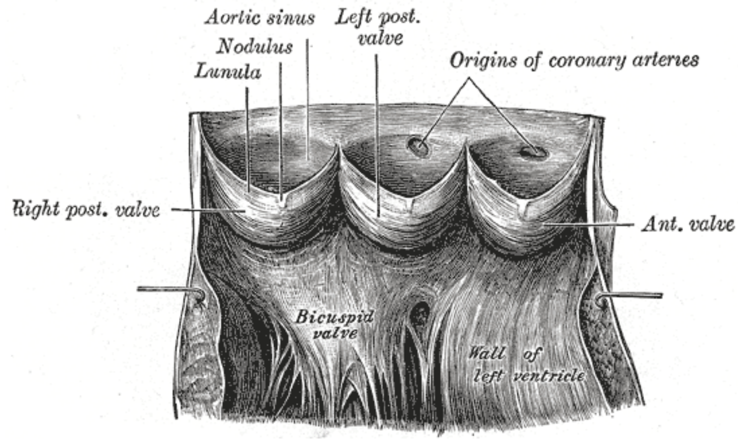

Figure (2): The normal trifoliate aortic valve ***(McAlpine, 1975)***

When the valve opens, the leaflets fall back into their sinuses without the potential of occluding any coronary orifice. The semilunar hingelines of adjacent leaflets meet at the level of the sinutubular junction, forming the commissures. The body of the leaflets are pliable and thin in the young, although its thickness is not uniform. Each leaflet has a somewhat crimpled surface facing the aorta and a smoother surface facing the ventricle ***(McAlpine, 1975).***

The leaflet is slightly thicker towards its free margin. On its ventricular surface, is the zone of apposition, known as the lunule, occupying the full width along the free margin and spanning approximately one-third of the depth of the leaflet. This is where the leaflet meets the adjacent leaflets during valvular closure. At the midportion of the lunule, the ventricular surface is thickened to form the nodule (of Arantius) that extends along 60% of the inferior margin of the lunule ***(McAlpine, 1975).***

With the valve is in closed position, the inferior margin of the lunules meet together, separating blood in the left ventricular cavity from blood in the aorta. Fenestrations in the lunules are common, especially in the elderly, but the valve remains competent because they are above the closure line. Larger fenestrations that extend beyond the zones of apposition, however, can lead to significant valvar regurgitation ***(Siew yen ho, 2009).***

- ***Relationship Between the Left Ventricular Outflow Tract and the Aortic Root***

The left ventricular outflow tract is composed of a muscular component (ie, the muscular ventricular septum) and fibrous component (ie, the area of fibrous continuity between the leaflets of the aortic and mitral valves), with the former being more extensive. The orientation of the outflow tract is known to change with aging. This change in geometry was examined in a series of normal human hearts, comparing findings in individuals aged <20 years with those aged >60 years ***(Ioan Tilea et al., 2013).***

Examination of the angle between the outlet and apical trabecular parts of the ventricular septum showed significant differences. In hearts from individuals aged >60 years, the angle varied between 90 and 120 degrees. In those from individuals aged ≤20 years, the angle varied between 135 and 180 degrees.

In these younger patients, the left ventricular outflow tract represented a more direct and straight extension into the aortic root. This would also seem to explain the additional observation that in all of the elderly hearts, the majority of the circumference of the aortic inlet projected to the right of a line drawn through the outlet part of the muscular ventricular septum.

This means that in elderly patients, the left ventricular outflow tract may not extend in straight fashion into the aortic root but rather may show a rightward “dog” leg ***(Ioan Tilea et al., 2013).***

The presence of a subaortic septal bulge or a hypertrophied septum may create an obstacle to proper seating of the aortic prosthesis within the left ventricular outflow tract, for this reason it has been considered by some to be a relative contraindication to the implantation of a certain type of aortic prostheses ***(Jose et al., 2011).***

- ***Interleaflet Triangles and Their Relationship to the Mitral Valve and Membranous Septum***

As a result of the semilunar attachment of the aortic valvular leaflets, there are 3 triangular extensions of the left ventricular outflow tract that reach to the level of the sinutubular junction. These triangles, however, are formed not of ventricular myocardium but of the thinned fibrous walls of the aorta between the expanded sinuses of Valsalva (figure 3). Their most apical regions represent areas of potential communication with the pericardial space or, in the case of the triangle between the right and left coronary aortic leaflets, with the plane of tissue interposed between the aorta and anteriorly located sleeve-like subpulmonary infundibulum. The 2 interleaflet triangles bordering the noncoronary leaflet are also in fibrous continuity with the fibrous trigones, the mitral valve, and the membranous septum ***(Piazza et al., 2008).***

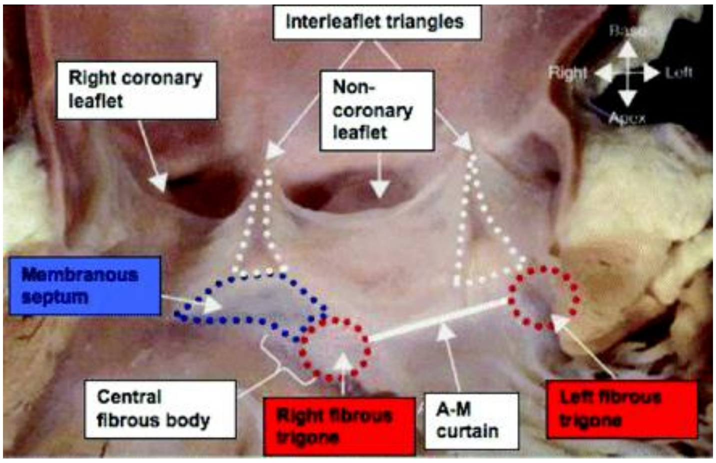

Figure (3): The aortic root opened from the left ventricle shows the fibrous continuities between the interleaflet triangles, the fibrous trigones, and the membranous septum. A-M indicates aortic-mitral ***(Piazza N et al., 2008)***

- ***<u>Relationship Between the Aortic Valve and the Conduction System</u>***

Within the right atrium, the atrioventricular node is located within the triangle of Koch. This important triangle is demarcated by the tendon of Todaro, the attachment of the septal leaflet of the tricuspid valve, and the orifice of the coronary sinus. The atrioventricular node is located just inferior to the apex of the triangle adjacent to the membranous septum, and therefore the atrioventricular node is in fact in close proximity to the subaortic region and membranous septum of the left ventricular outflow tract. That's why pathologies involving the aortic valve can lead to complete heart block or intraventricular conduction abnormalities ***(Anderson, 2006).***

The atrioventricular node continues as the bundle of His, piercing the membranous septum and penetrating to the left through the central fibrous body. On the left side, the conduction axis exits immediately beneath the membranous septum and runs superficially along the crest of the ventricular septum, giving rise to the fascicles of the left bundle branch. When viewed from the left, the bundle is intimately related to the base of the interleaflet triangle separating the noncoronary and right coronary leaflets of the aortic valve, with the superior part of the bundle intimately related to the right coronary aortic leaflet. This has important implications with the potential to induce abnormalities of conduction after percutaneous insertion of a new aortic valve ***(Piazza et al., 2008).***

The atrioventricular node, located in the wall of the right atrium at the apex of the triangle of Koch, is relatively distant from the root. As the conduction axis, penetrates to the left, through the central fibrous body, however, it is positioned at the base of the interleaflet triangle between the non- and right coronary aortic sinuses. Having penetrated through the fibrous plane providing atrioventricular insulation, the bundle then branches on the crest of the muscular ventricular septum, the left bundle branch fanning out on the smooth left ventricular side, while the cord-like right bundle branch penetrates back through the muscular septum, emerging on the septal surface in the environment of the medial papillary muscle ***(Anderson, 2006).***

Aortic valve pathology:

The aortic valve has two functions. By opening, it controls the direction in which blood flows and, by closing, it allows pressure differentials to exist in a closed system. Abnormal valve function produces either pressure overloading caused by restricted opening or volume overloading caused by inadequate closure. Valvular heart disease can be approached on the basis of the pathologic lesion-aortic stenosis or aortic regurgitation-or pathophysiologically, as pressure overloading versus volume overloading ***(Bonow et al., 2006).***

Aortic stenosis

Aortic stenosis refers to obstruction of flow at the level of the aortic valve and does not include the subvalvular and supravalvular forms of this disease. Aortic valve stenosis is usually defined by restricted systolic opening of the valve leaflets, with a mean transvalvular pressure gradient of at least 10 mm Hg ***(Bonow et al., 2006).***

- ***Prevalence and Etiology***

Calcific aortic stenosis and congenital bicuspid aortic valve stenosis account for the overwhelming majority of aortic stenosis cases, followed by less common conditions, such as rheumatic aortic stenosis and congenital aortic stenosis. In older adults, mild thickening, calcification, or both of a trileaflet aortic valve without restricted leaflet motion (i.e., aortic sclerosis) affects about 25% of the population older than 65 years. Calcific aortic stenosis, however, affects approximately 2% to 3% of those older than 75 years. Thus, not all patients with aortic sclerosis go on to develop obstructive aortic valve disease ***(Nonaro; 2010).***

Congenital bicuspid aortic valve stenosis is a major common cause of aortic stenosis; The altered architecture of the bicuspid aortic valve induces turbulent flow with continuous trauma to the leaflets, ultimately resulting in fibrosis, increased rigidity and calcification of the leaflets, and narrowing of the aortic orifice in adulthood ***(Ren, 2013).***

The approximate overall incidence of an anatomic bicuspid aortic valve is 1% to 2% of the population. Of these, about one half will develop aortic stenosis and up to one third will develop aortic regurgitation ***(Nonaro, 2010).***

The main causes of acquired aortic stenosis include degenerative calcification and, less commonly, rheumatic heart disease (table 1). Degenerative calcific aortic stenosis (also called senile calcific aortic stenosis) involves progressive calcification of the leaflet bodies, resulting in limitation of the normal cusp opening during systole. This represents a consequence of long-standing hemodynamic stress on the valve and is currently the most frequent cause of aortic stenosis requiring aortic valve replacement. The calcification may also involve the mitral annulus or extend into the conduction system, resulting in atrioventricular or intraventricular conduction defects ***(Ren, 2013).***

Risk factors for degenerative calcific aortic stenosis include hypertension, hypercholesterolemia, diabetes mellitus, and smoking. The available data suggest that the development and progression of the disease are due to an active disease process at the cellular and molecular level that shows many similarities with atherosclerosis, ranging from endothelial dysfunction to, ultimately, calcification ***(Hughes et al., 2005).***