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Surgical Valve Replacement (Bioprosthetic vs. Mechanical)

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Additional information is available at the end of the chapter

1. Introduction

The aortic valve separates the left ventricular outflow tract from the aorta. It is a tricuspid valve consisting of three semilunar cusps and the aortic valve annulus. The aortic valve annulus is a collagenous structure lying at the level of the junction of the aortic valve and the ventricular septum, which is the nadir of the aortic valve complex. This area is also referred to as the aortic ring and serves to provide structural support to the aortic valve complex. The annulus is shaped like a crown and extends to the level of the aortic sinuses. It attaches to the aortic media distally and the membranous and muscular ventricular septum proximally and anteriorly. There are 3 aortic valve cusps, each half-moon shaped or semilunar in appearance. A small dilatation of the proximal aorta is associated with each cusp; collectively, these are referred to as the sinuses of Valsalva or aortic sinuses, named after the Italian anatomist Antonio Valsalva. Their association with the respective coronary ostia identifies them: left, right, and posterior (or noncoronary).[1]

Aortic stenosis (AS) is one of the most common diseases of the aortic valve. The most common causes of AS are degenerative calcification, bicuspid aortic valve and rheumatic etiology. Age – related degenerative calcific AS is currently the most common cause of AS in adults and most frequent reason for aortic valve replacement (AVR).[2] That atherosclerosis is a cause of AS is derived primarily from five pieces of evidence: 1) that patients with familial homozygous hyperlipidemia usually develop calcific deposits on the aortic aspects of their aortic valve cusps at a very young age, usually by the teenage years (These individuals have serum total cholesterol levels >800 mg/dl from the time of birth.); 2) that progression of AS can be slowed by lowering total and low-density lipoprotein cholesterol levels by statins; 3) that patients >65 years of age with AS involving a three-cuspid aortic valve (unassociated with mitral valve

disease) usually have extensive atherosclerosis involving the major epicardial coronary arteries and usually other systemic arterial systems; 4) that serum total cholesterol levels and concomitant coronary bypass grafting tend to be higher in patients with AS involving three-cuspid aortic valves than in patients of similar age and sex without AS or with congenitally bicuspid aortic valves; and 5) that histologic study of three-cuspid stenotic aortic valve demonstrates features similar to those in atherosclerotic plaques.[2] Rare causes of aortic stenosis include obstructive, infective endocarditis, Paget's disease, renal failure, drug induced, familial hypercholesterolemia, systemic lupus erythematosus, irradiation, and ochronosis.[3] As the valves stenosis, valvular abnormality produces turbulent flow, which traumatizes the leaflets and eventually leads to progressive cell proliferation, extracellular matrix production, and calcification of the valve. It is degenerative process that leads to proliferative and inflammatory changes that leading to calcification of the aortic valve. Progressive calcification leads to immobilization of the cusps.[3]

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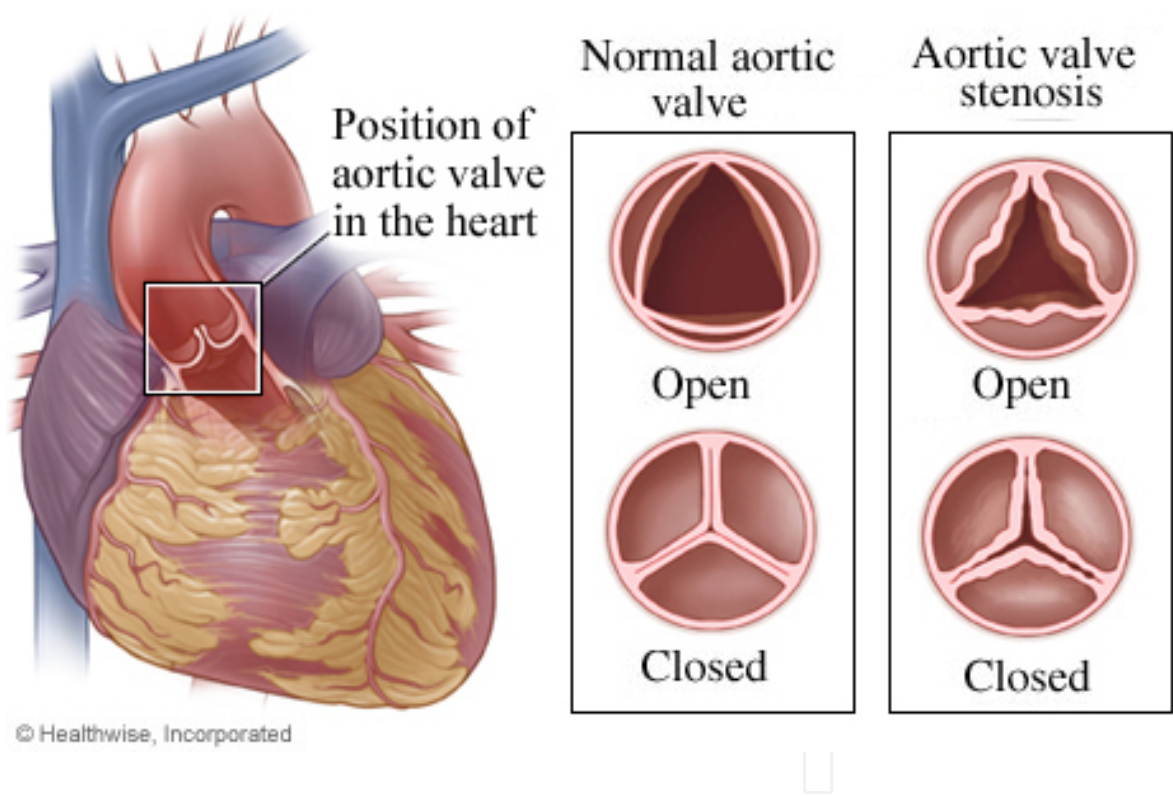
Figure 1. Normal aortic valve and stenotic aortic valve

The pathophysiology of valvular aortic stenosis is one of progressive obstruction and the resultant compensatory changes. With increasing left ventricular outflow tract obstruction, there is pressure hypertrophy of the left ventricle. Left ventricular cavity size and systolic function is initially maintained, as the increase in left ventricular wall thickness acts as a compensatory mechanism to normalize wall stress. The development of pressure hypertrophy is initially a beneficial adaptation. However, this hypertrophy may result in reduced coronary flow reserve and oxygen supply–demand mismatch. These hypertrophied hearts are also more

sensitive to diffuse subendocardial ischemic injury, which may result in both systolic and diastolic dysfunction. As the obstruction progresses to a critical level, the high afterload "overwhelms" the left ventricle and systolic function begins to decrease. With continued severe afterload excess, myocyte degeneration and fibrosis occurs and produces irreversible left ventricular systolic dysfunction. In these patients, both the high afterload and the intrinsic myocardial disease significantly increase wall stress and a vicious cycle of deterioration in ventricular function ensues.[3]

The evaluation of aortic stenosis is based upon the history, the physical examination, and a comprehensive echocardiography. For most patients, two-dimensional echocardiography readily identifies the calcified stenotic aortic valve, and Doppler echocardiography reliably estimates the severity of aortic stenosis in the majority of patients. Many patients with aortic stenosis will remain asymptomatic for decades. The diagnosis of aortic stenosis is usually made in the asymptomatic patient on the basis of a systolic murmur on auscultation and confirmed by echocardiography. Symptoms, when they occur, usually consist of one or more of the classic triad of exertional dyspnea, angina, and syncope. Following symptom onset, there is a high mortality rate with an average survival of 2–3 years. The development of symptoms therefore is a critical point in the natural history of patients with aortic stenosis. Sudden death rarely is the initial manifestation of severe aortic stenosis, occurring at a rate of less than 1% per year in asymptomatic patients.[3] Two-dimensional and Doppler echocardiography is the imaging modality of choice for the diagnosis and quantification of aortic stenosis. Short-axis images from two-dimensional echocardiography demonstrate the number of aortic cusps and the degree of cusp fusion or restricted cusp opening in valvular aortic stenosis. Two dimensional echocardiography is also useful for determining the status of the left ventricle and the degree of hypertrophy. Left atrial enlargement indicates concomitant diastolic dysfunction. The normal area of the adult aortic valve is 3.0 to 4.0 cm^2. Reduction of the normal area usually does not produce symptoms until the valve reaches one-fourth of its normal dimension.[4] The graduation of AS is given in Table 1.

Severity	Aortic jet velocity (m per second)	Mean gradient (mm Hg)	Aortic valve area (cm^2)
Normal	< 2.5	-	3 to 4
Mild	2.5 to 2.9	< 25	1.5 to 2
Moderate	3 to 4	25 to 40	1 to 1.5
Severe	> 4	> 40	< 1

Table 1. Classifications of Aortic Stenosis Severity[4]

There is no effective medical therapy for AS. AVR is the only effective treatment for severe aortic stenosis in adults. Following AVR for AS, one can expect resolution of symptoms, left ventricular hypertrophy (LVH) regression, and improved left ventricular (LV) systolic function secondary to reduced afterload. Importantly, postoperative survival is similar to age-

matched controls after AVR for AS when performed prior to the development of LV dysfunction or congestive heart failure (CHF). Similarly, incomplete regression of LVH after AVR has been associated with adverse outcomes such as reduced long-term survival. Contrary to the immediate improvement in systolic performance, diastolic dysfunction may persist for several more years after AVR. In fact, Gjertsson et al. recently evaluated diastolic dysfunction in AS and found that the proportion of patients with moderate-to-severe diastolic dysfunction actually increased with time after AVR despite normalization of LV mass and appropriate adjustments for senile diastolic dysfunction. Finally, AVR is associated with improved quality of life scores, particularly among the elderly, and has been found to be similar to age-matched individuals without heart disease. [3,5,6]

The American College of Cardiology (ACC) and the American Heart Association (ACH) have jointly developed guidelines in which they published indications for AVR:

a. Definite indications:

- Patients who have severe AS and presented with one or more of its classical symptoms (angina, syncope, heart failure, etc.)

- Patients who have severe AS and required coronary artery bypass surgery, surgery on the aorta or other heart valves

- Patients who have severe AS and left ventricle systolic dysfunction (ejection fraction less than 50 %)

b. Possible indications:

- Patients who have moderate AS and required coronary artery bypass surgery, surgery on the aorta or other heart valves

- Asymptomatic patients with severe AS with abnormal exercise test, or an increase in transaortic gradient during exercise, or left ventricle systolic dysfunction (ejection fraction less than 50 %), or left ventricular dilatation, or significantly elevated left ventricular diastolic pressure. [7]

The European Society of Cardiology (ESC) has also developed guidelines in which they published indications for AVR (Table 2).[8] They strongly recommended early AVR in all symptomatic patients with severe AS.

Management of asymptomatic patients requires careful weighing of benefits against risks. Early elective surgery at these patients can only be recommended in selected patients, at low operative risk. This could be the case in:

- The rare asymptomatic patients with depressed LV function not due to another cause

- Those with echocardiographic predictors of poor outcome suggested by the combination of a markedly calcified valve with a rapid increase in peak aortic velocity of ≥ 0.3 m/s per year

- If the exercise test is abnormal, particularly if it shows symptom development, which is a strong indication for surgery in physically active patients.

- However, on the other hand, breathlessness on exercise may be difficult to interpret in patients with only low physical activity, particularly the elderly, making decisionmaking more difficult. There is no strict age limit for performance of exercise testing and it is reasonable to propose it in patients > 70 years old who are still highly active.[8]

Patients with severe AS and any symptoms	IB
Patients with severe AS undergoing coronary artery bypass surgery, surgery of the ascending aorta, or on another valve	IC
Asymptomatic patients with severe AS and systolic LV dysfunction (LVEF < 50%) unless due to other cause	IC
Asymptomatic patients with severe AS and abnormal exercise test showing symptoms on exercise	IC
Asymptomatic patients with severe AS and abnormal exercise test showing fall in blood pressure below baseline	IIaC
Patients with moderate AS undergoing coronary artery bypass surgery, surgery of the ascending aorta or another valve	IIaC
Asymptomatic patients with severe AS and moderate-to-severe valve calcification, and a rate of peak velocity progression ≥ 0.3 m/s per year	IIaC
AS with low gradient (< 40 mmHg) and LV dysfunction with contractile reserve	IIaC
Asymptomatic patients with severe AS and abnormal exercise test showing complex ventricular arrhythmias	IIbC
Asymptomatic patients with severe AS and excessive LV hypertrophy (≥ 15 mm) unless this is due to hypertension	IIbC
AS with low gradient (< 40 mmHg) and LV dysfunction without contractile reserve	IIbC

Table 2. Indications for AVR in AS

In last 50 years, the varieties of prostheses that have become available for use are numerous. An ideal aortic prosthesis would be simple to implant, widely available, possess long-term durability, would have no intrinsic thrombogenicity, would not have a predilection foe endocarditis and would have no residual transvalvular pressure gradient. Such a valve does not currently exist. Currently available options include mechanical valves, stented biological valves, stentless biological valves, allograft valves and pulmonary auto-

graft valves. Commonly in us are mechanical and biological prostheses.[9] When selecting between mechanical and biologic heart valves, the surgeon and patient must balance the risks and benefits of each choice.

2. Mechanical prostheses

Charles Hufnagel in 1952. Used aortic valve ball and cage prosthesis heterotopically in the descending aorta to treat aortic insufficiency. The first aortic valve replacement with an intra cardiac mechanical prosthesis, which led to long terms survivors, was performed in 1960. Mechanical valves are classified according to their structure as caged-ball, single-tilting-disk or bileaflet-tilting-disk valves. The Starr-Edwards caged-ball valve has been available since the 1960's and comprises a silastic ball, which rests on the sewing ring when closed and moves forward into the cage when the valve opens. The single-disk valves, for example, the Bjork-Shiley prosthesis and the Medtronic-Hall prosthesis, contain a disk that tilts between two struts of the orifice housing. The most popular of the mechanical valves at present are the bileaflet valves, of which the St. Jude Medical valve and the Carbomedics valve are widely implanted. Both these devices are implanted within the aortic annulus. The two semi-circular leaflets of the bileaflet valve are connected to the housing by a butterfly hinge mechanism and swing apart during opening of the valve creating three outflow tracts, one central and two peripheral respectively. In contrast to the configuration of the latter, the Carbomedics Top Hat (Sulzer Carbomedics, Austin, TX) bileaflet aortic valve that was introduced in 1993 has a unique supra-annular design with all its components incorporated within the aortic sinuses.[10,11]

Mechanical valves are made from carbon, Teflon, Dacron, titanium and polyester and are very durable. The current designs for the aortic and mitral positions include ball-and-cage valves, single tilting disc prostheses, and bileaflet prostheses. Bileaflet mechanical valves are the standard in current practice, with the St. Jude Medical (St. Jude Medical, Inc., St. Paul, MN) prosthesis the modern prototype, having been first implanted in 1977. Most of these valves are constructed using carbon strengthened with silicon carbide additives. Other examples of bileaflet mechanical valves include those manufactured by CarboMedics (Austin, TX); Advancing the Standard Medical (ATS, Minneapolis, MN); Medtronic, Inc. (Minneapolis, MN); and Medical Carbon Research Institute, LLC (MCRI, Austin, TX). [10,11,12] The On-XR mechanical valve (MCRI) was introduced in Europe in 1996 and differs from other bileaflet mechanical valves in that it is made from pure pyrolytic carbon. The PROACT (Prospective Randomized On-X R Valve Anticoagulation Trial) study is an FDA-approved multicenter trial, sponsored by MCRI, currently enrolling patients to determine whether or not defined patient groups receiving AVR (low versus high risk for TE events) with the On-X R _ valve may be safely maintained on lower doses of warfarin or, for patients in the lowrisk aortic valve arm, on antiplatelet drugs (aspirin plus clopidogrel) alone compared with standard anticoagulation regimens. No single mechanical valve has shown superior patient outcomes, and all demonstrate extremely low rates of structural valve deterioration, the major advantage of mechanical valves.[3]
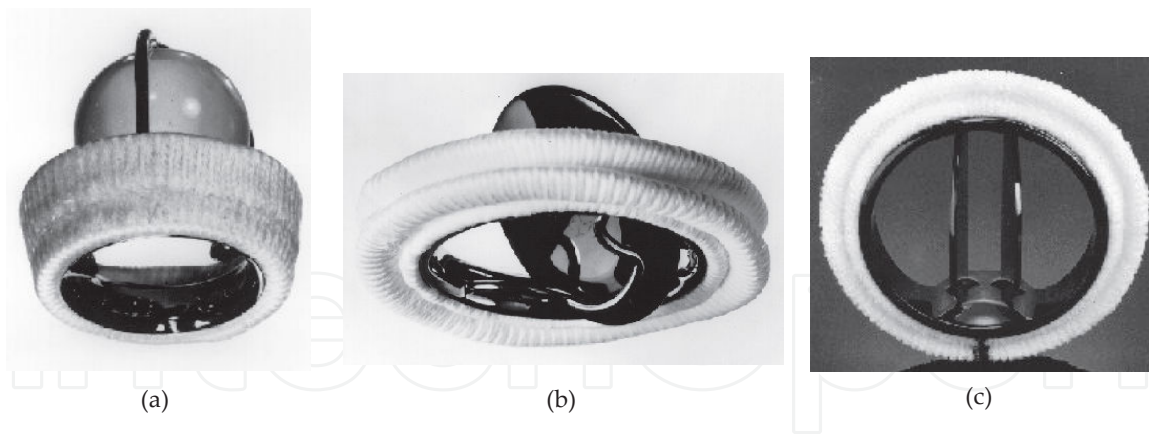
Figure 2. Mechanical valves: a) Starr-Edwards cage-ball valve, b) Bjork-Shiley mono-leaflet valve and c) St. Jude Medical bileaflet valve

3. Biological prosthesis

The biological prostheses include a wide variety of devices. Included within this broad category are the bioprostheses, a term which is used for valves with non-viable tissue of biological origin. The bioprostheses include the heterografts, composed of porcine (actual valves of a pig) or bovine tissue (pericardium of a cow) and the allografts, which are preserved human aortic valves. The initial bioprostheses were mounted on stents to which the leaflets and sewing ring were attached but subsequently stentless valves, which are sewn in free hand, have been developed.[13] Stented bioprosthetic valves, which incorporate a semi-rigid external support structure for the valve leaflets, represent the majority of tissue valves implanted in clinical practice. The external support provides accurate valve mounting, improving ease of implantation. Two types of stented bioprosthetic valves are currently available in the United States: porcine aortic valves, which incorporate chemically stabilized porcine valve leaflets mounted on a stented structure or frame, and bovine pericardial valves. The leaflets of the latter valve type are constructed from bovine pericardium and subsequently mounted on a stented frame. Available porcine valves include the Medtronic Mosaic valve (Medtronic Inc., Minneapolis, MN), the St. Jude Medical Biocor and Biocor Supra valves (St. Jude Medical, Inc., St. Paul, MN), and the Carbomedics Mitroflow valve (Carbomedics, Inc., Austin, TX). Bovine pericardial valves include the Carpentier–Edwards (C–E) Perimount (Edward Lifesciences, Irvine, CA) and the CE Perimount Magna valves as well as the Sorin Soprano (Sorin Group, Saluggia, Italy) valves. At present, based on the best available data, no one bioprosthetic valve appears superior with regard to patient outcomes and none requires systemic anticoagulation with warfarin, which is their major advantage. Their major disadvantage is the incidence of structural valve deterioration and subsequent need for reoperation, although the lifespan of the latest generation of tissue valves is unknown. Recent evidence also suggests that stentless biological valves may have better coronary flow reserve compared to stented valves. Additionally, compared with stented bovine pericardial valves, stentless valves have been associated with increased transvalvular EOA and decreased pressure gradients

during extended follow-up. However, as seen in other studies, LV mass regression after stentless valve implantation was not different from stented aortic bioprostheses.[3,14]
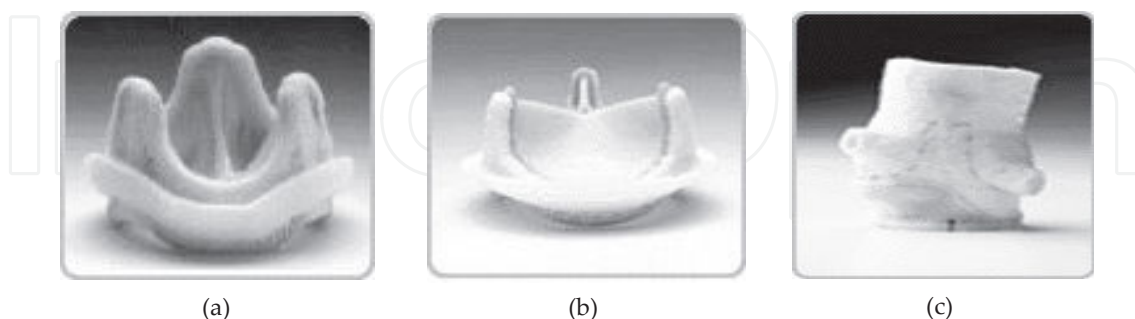
Figure 3. Biological prosthesis: a) stented porcine bioprosthesis, b) pericardial bovine bioprosthesis and c) stentless porcine bioprosthesis

4. Outcomes after aortic valve replacement

The Ad Hoc Liaison Committee for Standardizing Definitions of Prosthetic Heart Valve Morbidity of the American Association of Thoracic Surgery and the Society of Thoracic Surgeons published guidelines during years, which are now widely used in reporting outcomes after valve surgery. They presented a list of developing specific valve-related events during patients remaining lifetime. These valve-related events are:

1.   Structural valvular deterioration

2.   Nonstructural dysfunction

3.   Valve thrombosis

4.   Embolism

5.   Valvular endocarditis and

6.   Bleeding events.[7, 15]

Structural valvular deterioration - Any change in function or deterioration (a decrease of one New York Heart Association functional class or more) of an operated valve due to an intrinsic abnormality, which causes stenosis or regurgitation. Changes intrinsic to the valve include wear, fracture, poppet escape, calcification, leaflet tear stent creep and su-ture line disruption of the components of the operated valve. The definition excludes changes due to infection or thrombosis. *Mechanical prostheses* are extremely resistant to ma-terial fatigue or structural valve deterioration. This is characteristic of mechanical valves

whether they are aortic or mitral valves.[16] Because of the long-term durability of mechanical prostheses a valve replacement rate is less than 2% over 25 years. The most common reasons for reimplantation are pre- and postoperative endocarditis, paravalvular leak and valve thrombosis.[17] *Bioprosthetic valves* are not as durable, have a shorter lifespan and are more susceptible to calcification than human and mechanical valves.[13] Bioprostheses have a significantly higher rate of reoperation due to structural valve deterioration. In large series, freedom from reoperation is > 90% at 10 years, but < 70 % at 15 years. [17,18,19] There is an important predisposition for premature bioprosthetic structural valve deterioration in younger patients, especially those under the age of 40 years.[17]

Nonstructural dysfunction - Any abnormality that is not intrinsic to the valve per se, which causes stenosis or regurgitation. Examples for this include entrapment of pannus, tissue or suture; paravalvular leak; inappropriate sizing or positioning; residual leak and clinically important hemolytic anemia. This definition also excludes changes due to infection and thrombosis. Subvalvular pannus formation is rare with *mechanical bileaflet valves*.[20] Panus overgrowth and prosthetic structural degeneration interfering with normal valve opening and closure may cause hemolysis severe enough for reoperation. Paravalvular leak is an operative complication and it is related to operative technique and to endocarditis.[21]

Valve thrombosis – Any thrombus, in the absence of infection, which is attached to or near an operated valve that occludes part of the blood flow path or that interferes with function of the valve. The incidence of prosthesis thrombosis is < 0.2 % per year and it occurs more often in *mechanical prostheses*.[22] It is most commonly due to inadequate anticoagulation or noncompliance. Freedom from valve thrombosis at 20 years is > 97 %.[23,24]

Embolism – Any embolic event that occurs, in the absence of infection, after the immediate peri-operative period. This could be either a neurologic or peripheral embolic event. A neurologic event includes any new, temporary or permanent focal or global neurologic deficit. A peripheral embolic event is due to an embolus that produces symptoms from obstruction of a peripheral (non-cerebral) artery. The incidence of thromboembolic events between bioprostheses and mechanical prostheses are the same.[25] This is a continuous risk factor that is present through the life of patients with *mechanical valve prosthesis*, so they must maintain therapeutic anticoagulant levels. The embolic risk is highest in the first few months, before the ring and valve components have fully endothelialized.[26] Acceptable thromboembolic rates range between 0.8 and 2.3 % per patient-year.[21,22,25] 50 % of these events are neurologic, 40 % are transient and 10 % are peripheral.[21]

Valvular endocarditis – Any infection involving an operated valve diagnosed by customary clinical criteria. It is rare case with prophylactic antibiotics. Around 60 % of events occur early and are associated with staphylococci. The mortality for this event is high. Freedom from endocarditis with mechanical prosthesis is 97 to 98 % at 20 to 25 years. A number of studies have reported a higher incidence of valvular endocarditis after *mechanical valve* replacement in comparison with the biologic valve replacement during the initial few months after implantation. *Bioprostheses* are less susceptible to early infection, which is often restricted to the leaflets, making cure with antibiotics more likely but increasing the chances of late failure due to degeneration of the cusps.[27,28,29]

Bleeding event – Formerly classified as anti-coagulant hemorrhage, a bleeding event is an episode of major internal or external bleeding that causes death, hospitalization, and permanent injury or requires transfusion. This definition applies to all patients, irrespective of anti-coagulation status. *Mechanical valves* are durability but anticoagulation is key of long-term success. International Normalized Ratio (INR) is the standard to which anticoagulation levels should be targeted. Level of INR should be individual for each person. Complications occur during fluctuations in the INR and less during steady-state levels, be they high or low.[30,31] When levels of INR increase, bleeding episodes become more common, and when levels of INR decrease thromboembolic episodes become more common. Some studies showed that around 40 % of the bleeding episodes occurred in the first year after surgery, when levels of INR are more likely to fluctuate. Many studies suggested that in the early postoperative period slowly raise the level of INR to therapeutic levels is needed, to prevent bleeding events. [21,32,33] According to ACC and ACH after mechanical AVR, the goal of antithrombotic therapy is usually to achieve an INR of 2.5 to 3.5 for the first 3 months after surgery and 2.0 to 3.0 beyond that time. At that level of anticoagulation, the risk of significant hemorrhage appears to be 1% to 2% per year.[7] Low-dose aspirin is also indicated in addition to warfarin to result in a lower incidence of thromboembolic event, with a low possibility for bleeding.[34] Older patients are at higher risk for thromboembolic event because of the greater number of risk factors that accumulate with aging.[30] Anticoagulation-related hemorrhage (ARH) is the most common valve-related event. More often it will occur during fluctuations in INR, which happens most often early after valve replacement.[21,22] The most common places for ARH are gastrointestinal tract and central nervous system.[21] Acceptable ARH rates range from 1.0 to 2.5% per patient-year in long term reports.[21,22,25,35] It is very dangerous complication, because mortality more often occurs in relation to bleeding events than in relation to thromboembolic events.[21]

Operative mortality – Operative mortality is defined as all-cause mortality within 30 days of operation. According to the Society of Thoracic Surgeons mortality for isolated AVR is 4.3% and for AVR with concomitant coronary artery disease is 8%.[36] Many factors have been associated with an increased risk of operative mortality in isolated AVR. Some of these risk factors are age, female gender, diabetes, renal failure, and emergency status, previous operation, advanced preoperative NYHA class, lower cardiac index, concomitant coronary artery bypass grafting and longer aortic crossclamp and cardiopulmonary bypass time respectively.[37] In the absence of major comorbidities and preserved ejection fraction, isolated AVR can be performed with an expected mortality of less than 2%.[38]

Several studies have evaluated independent risk factors for operative mortality after AVR. Five variables predictive of increased mortality risk after AVR are common to each of these analyses: preoperative renal failure, urgency of AVR, preoperative heart failure, presence of CAD or recent MI, and redo cardiac operation. Other factors independently associated with operative mortality from the individual studies include preoperative atrial fibrillation, active endocarditis, preoperative stroke, advanced age, lower body surface area, multiple valve procedures, and hypertension. [39, 40]

5. Factors affecting long-term outcome after AVR

- Demographic
  - Older age
  - Male sex
- Clinical
  - Higher pre-operative NYHA functional class
  - Pre-operative atrial fibrillation and non-sinus rhythm
  - Pure aortic regurgitation
  - Hypertension
  - Diabetes mellitus
  - Renal failure
- Surgery-related
  - Longer cardiopulmonary bypass time
- Morphological
  - Previous myocardial infarction
  - Left ventricular structure and functional abnormality
  - Previous aortic valve surgery
  - Coronary artery disease (CAD)

Older patients have a lot of comorbiditis and they are at higher risk for valve-related events. Atrial fibrillation is one of the risk factor for thromboembolism, because of that INR levels must be higher (INR 2.5 to 3.5) than regular.[30,34] The majority of patients undergoing AVR have other cardiac lesions, most commonly CAD, and more complex pathology has been associated with increased risk. Combined myocardial revascularization and AVR increases cross-clamp time and has the potential to increase perioperative myocardial infarction and early postoperative mortality compared with patients undergoing isolated AVR.[7] In addition to severity of CAD and AS, the multivariate factors for late postoperative mortality include low ejection fraction, severity of LV dysfunction, age greater than 70 years (especially in women), and presence of NYHA functional class IV symptoms.[36]

6. Patient selection

Propter selection of patients for valve replacement can bring us excellent long-term results, long-term survival and low incidence of valve-related complications.

In some studies of patients followed over longer time frames, freedom from all valve-related events and freedom from reoperation were improved in patients with mechanical valve prostheses as compared to patients with biological prostheses. [9,16,25] Key of long-term success of mechanical valve prostheses is anticoagulation. Patients that are inconsistent, noncompliant or incapable of managing medications are not good candidates for long-term chronic anticoagulation.[39,41] Also patients with higher levels of education and those from geographic areas with a good medical infrastructure have better compliance with necessary medications and anticoagulant monitoring.[31]

Many centers used bioprosthetic valves for patients who are older than 70 year, based on data by Akins.[42] In patients younger than 60 years of age, the best solution would be implantation of mechanical valves, based on prosthesis durability and they have low-risk for valve-related events.[21] In decade between 60 and 70 years of age, other factors have to be taken into account.[7] According to some studies, patients over 65 years at the time of surgery should receive a biologic valve. Patients under the age of 60 should have a mechanical prosthesis to minimize the risk of structural failure requiring repeat AVR in an octogenarian. Patients between 60 and 65 represent the group in whom there is still considerable debate regarding prosthesis selection. Those patients who have comorbidities such as severe CAD may be less likely to outlive their prosthesis and should receive a biologic valve. A detailed discussion of these risks and benefits of prosthesis selection should occur with all patients and their families prior to entering the operating room. [3,7,22,24,25,37,38]

In the early follow-up period, anticoagulation – related hemorrhage is the most common unwanted event for mechanical valve prostheses. Over the first 10 years of follow-up there is a higher incidence of valve-related events in patients with mechanical prostheses as opposed to those with biologic valves.[32] However, in the next 10 to 20 years after AVR, the incidence of valve failure and valve-related complications are much higher at biologic prostheses than those with mechanical valve prostheses. Some series showed that the time to biologic valve failure was only 7.6 years.[43] This failure rate will increase over time. However, freedom from valve-related events is more strongly influenced by pre-existing comorbidities than the presence of mechanical prostheses.[21], [22, 25, 31]

The elderly patient with severe aortic stenosis poses a therapeutic challenge. In considering elderly patients for aortic valve replacement, important factors include the presence of symptoms, physiologic age, patient expectations, anticipated future activities, and comorbidity. The operation itself carries a higher risk than in younger patients. Extensive calcification of the aorta and annulus as well as fragile tissue presents significant technical difficulties for the surgeon. In addition, particularly in women, the aortic root and annulus may be small and require concomitant enlargement to accommodate the valve prosthesis. Furthermore, protruding arch atheroma occurs in one-fifth of patients > 65 years of age and significantly increases the risk of stroke and mortality during cardiac surgery. Major postoperative complications, nevertheless, remain high, with the incidence of permanent stroke between 4 and 6%. Rehabilitation can also be a problem, as elderly patients take longer to recover from surgery. Survival has clearly improved in these elderly patients with severe symptomatic aortic stenosis who undergo aortic valve replacement. Survival is 80–85% at 1 year and 60–

70% at 5 years, which is similar to an age- and sex-matched population without aortic valve disease. Most patients report improved functional capacity and quality of life, with more than 90% of patients feeling better after surgery.[3]

A major deterrent to mechanical valve replacement in the younger patient is the impact of long-term anticoagulation. Mechanical valves are, however, more ideal for younger patients due to their excellent durability characteristics. Most importantly, younger patients (i.e., patients under the age of 50 years) are a low-risk subset for valve related events. These individuals have very few risk factors for TE, and thus anticoagulation can be run at the lower end of the therapeutic target range, decreasing the incidence of anticoagulant-related hemorrhage without altering the incidence of TE. In fact, many infants and children have been managed with only aspirin with quite good long term results. While this is not recommended in patients older than infancy, it is a feasible alternative. A recent study in patients under 50 years of age followed 254 patients for up to 20 years and found an exceedingly low rate of valve related events, an exceptional long-term overall survival of nearly 88%, and event-free survival probability of 92% at 19 years.[3,44,45]

Patients with an absolute requirement for long-term anticoagulation such as atrial fibrillation, previous thromboembolic events, hypercoagulable state, severe LVD, another mechanical heart valve in place, or intracardiac thrombus, should receive a mechanical valve regardless of age. Patients in whom anticoagulation with warfarin is contraindicated, such as women of child-bearing age wishing to become pregnant, patients with other bleeding disorders, or those who refuse anticoagulation should receive a bioprosthesis. There is growing interest in using mechanical prostheses in women of child-bearing age and providing anticoagulation with subcutaneous low-molecular weight heparin injections. Patients with end-stage renal failure were previously believed to have significantly elevated risk for early bioprosthetic structural valve deterioration. However, increased anticoagulation- related complications are also more likely in this group, and the current ACC/AHA guidelines do not recommend routine use of mechanical prostheses in these patients.[3,7,8,9,10]

The decision between bioprosthetic and mechanical valve should be made by the patient with educated input regarding the pros and cons of each option from the patient's physicians. Today surgeons implant bioprosthetic valves in younger patients who wish to avoid anticoagulation due to lifestyle concerns (e.g. young, active individual, desire to become pregnant, etc.), although surgeons generally will guide patients toward a mechanical option at the time of redo-AVR if their life expectancy exceeds 10–15 years at that time.[3]

7. Operative technique

Aortic valve replacement is most frequently done through a median sternotomy, meaning the incision is made by cutting through the sternum. Once the pericardium has been opened, the patient is put on a cardiopulmonary bypass machine. This machine takes over the task of breathing for the patient and pumping their blood around while the surgeon replaces the heart valve.

Once the patient is on bypass, a cut is made in the aorta and a crossclamp applied. The surgeon then removes the patient`s diseased aortic valve and a mechanical or biological valve is put in its place. Once the valve is in place and aorta has been closed, the patient is taken off the heart-lung machine. Transesophageal echocardiogram can be used to verify that the new valve is functioning property. Pacing wires are usually put in place, so that the heart can be manually paced should any complications arise after surgery. Drainage tubes are also inserted to drain fluids from the chest and pericardium following surgery. These are usually removed within 36-48 hours while the pacing wires are generally left in place until right before the patient is discharged from the hospital.

8. Patient-prosthesis mismatch

Prosthesis-patient mismatch (PPM) is that a smaller than expected effective orifice area (IEOA) in relation to the patient's body surface area (BSA) will result in higher transvalvar gradients. It is condition that occurs when the valve area of a prosthetic valve is less than the area of that patient's normal valve.[46] Several authors suggest that prosthesis-patient mismatch occurs at an IEOA of 0.85 cm^2/m^2.[46,47] Transvalvular gradients begin to rise substantially at IEOAs below this value, and these elevated gradients potentially cause increased left ventricular work that prevents adequate regression of left ventricular hypertrophy. Several factors including age, body mass index (BMI), and pre-operative status of left ventricular function may potentially influence the effect of PPM on post-operative outcomes.[46] PPM is associated with a significant reduction in cardiac index during the postoperative course. The incidence of congestive heart failure was significantly higher in patients with PPM.[48] Several studies reported that early mortality is significantly increased in patients with PPM.[47, 48, 49, 50]

The projected indexed EOA should be systematically calculated at the time of the operation to estimate the risk of PPM. PPM can be avoided by using a simple strategy at the time of operation. Pibarot suggested that surgeon first calculate the patient's BSA from his or her weight and height. Than multiply BSA by 0.85 cm^2/m^2, the result being the minimum EOA that the prosthesis to be implanted should have to avoid PPM, and than choose the prosthesis and the reference values for the different types and sizes of prosthesis.[46, 47]

Due to concerns over PPM, stentless bioprosthetic valves, which generally have a larger EOA sizefor- size compared with mechanical or stented bioprosthetic valves, have been increasingly utilized for AVR. In initial evaluation, stentless valves had better hemodynamics and improved survival rates relative to stented biological or mechanical valves and were more durable than stented biological valves. Stentless valves may be preferred in patients with a small aortic root, and arguments have been made that wider utilization of stentless valves may minimize PPM. Stentless valves also appear to have better hemodynamic profiles than stented valves during exercise testing. Technical reasons for not implanting stentless valves include extensive aortic root calcification, coronary ostia opposed by 180, presence of the two coronary ostia in close proximity, or unusual disproportion between the sinotubular junction and the aortic annulus. Whereas stented valves allow

perfect valve mounting within the aortic annulus, thus reducing the risk of implanting an incompetent valve, postoperative AR and limited durability remain a concern with the free-hand stentless valve insertion technique. This issue may be circumvented with full aortic root replacement using a stentless porcine root.[3.49,50]

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References

[1] Malouf, J. F, Edwards, W. D, Tajik, A. J, & Seward, J. Functional anatomy of the heart. In: Fuster V, O'Rourke RA, Walsh RA, Poole-Wilson P, eds. Hurst's The Heart. 12th ed. New York, NY: McGraw-Hill Companies, Inc; (2008).

[2] Otto, C. M, Lind, B. K, Kitzman, D. W, et al. Association of aortic valve sclerosis with cardiovascular mortality and morbidity in the elderly. N Eng J Med (1999).

[3] Andrew WangThomas S. Bashore. Valvular Heart Disease. Humana Press, a part of Springer Science Business Media, LLC (2009).

[4] Gjertsson, P, Caidahl, K, Farasati, M, et al. Preopeartive moderate to severe diastolic dysfunction: A novel Doppler echocardiographic long-term prognosis factor in pa-tients with severe aortic stenosis. J Thorac Cardiovasc Surg (2005).

[5] Connolly, H. M, Oh, J. K, Orszulak, T. A, et al. Aortic valve replacement for aortic stenosis with severe left ventricular dysfunction: prognostic indicators. Circulation (1997). , 95, 2395-400.

[6] Gjertsson, P, Caidahl, K, & Bech-hanssen, O. Left ventricular diastolic dysfunction late after aortic valve replacement in patients with aortic stenosis. Am J Cardiol (2005). , 96, 722-7.

[7] Bonow, R. O, Carabello, B. A, & Chatterjee, K. de Leon AC Jr, Faxon DP, Freed MD, Gaasch WH, Lytle BW, Nishimura RA, O'Gara PT, O'Rourke RA, Otto CM, Shah PM, Shanewise JS. ACC/AHA 2006 guidelines for the management of patients with valv-ular heart disease: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing Committee to Develop

Guidelines for the Management of Patients With Valvular Heart Disease). Circulation. (2006). ee231. DOI:CIRCULATIONAHA.106.176857, 84.

[8] The European Society of Cardiology Guidelines on the management of valvular heart disease European Heart Journal (2007.

[9] Gott, V. L. Alejo DE: Mechanical heart valves: 50 years of evolution. Ann Thorac Surg (2003). S2230

[10] Bloomfield, P. Choice of heart valve prosthesis. Heart. (2002). , 87, 583-9.

[11] Dewall, R. A, Qasim, N, & Carr, L. Evolution of Mechanical Heart valves. Ann Thorac Surg. (2000). , 69, 1612-21.

[12] Bonow, R. O, Carabello, B. A, Chatterjee, K. C, De Leon, J. R, Faxon, A. C, Freed, D. P, Shah, M. D, & Acc, P. M. AHA 2006 guidelines for the management of patients with valvular heart disease. Journal of the American College of Cardiology, 48(3), 1-148.doi:10.1016/j.jacc.2006.05.021

[13] YangThang, "Mechanical Versus Bioprosthetic Valve Replacement in Valvular Heart Disease: A Systematic Review" ((2011). School of Physician Assistant Studies. Paper 240.http://commons.pacificu.edu/pa/240

[14] Tsialtas, D, Bolognesi, R, Beghi, C, et al. Stented versus stentless bioprostheses in aortic valve stenosis: effect on left ventricular remodeling. Heart Surg Forum (2007). E, 205-10.

[15] Golubovic, M, Mihajlovic, B, Kovacevic, P, Cemerlic-adjic, N, Pavlovic, K, Velicki, L, & Susak, S. Postoperativne neletalne komplikacije posle operacije na otvorenom srcu. Vojnosanitetski pregled (2012). , 69(1), 27-31.

[16] Stamenko, S. Susak. Mitralna regurgitacija- kardiohirurski aspekti dijagnostike i terapije. Mediterran Publishing, Biblioteka Academica, knjiga 15, Novi Sad (2010).

[17] Emery, R. W, Arom, K. V, Krogh, C. C, et al. Reoperative valve replacement with the St.Jude Medical valve prosthesis: long-term follow up. J Am Coll Cardiol (2004). A

[18] Desai, N. D, Merin, O, Cohen, G. N, et al. Long-term results of aortic valve replacement with the St.Jude Toronto stentless porcine valve. Ann Thorac Surg (2004).

[19] Grunkemeier, G. L, Jamieson, W. R, & Miller, D. C. Starr A: Actuarial versus actual risk of porcine structural valve deterioration. J Thorac Cardiovasc Surg (1994).

[20] Vongpatanasin, W, & Hills, L. D. Lange RA: Prosthetic heart valve. N Eng J Med (1996).

[21] Emery, R. W, Krogh, C. C, Arom, D. V, et al. The ST. Jude Medical cardiac valve prosthesis: A year experience with single valve replacement. Ann Thorac Surg (2005). , 25.

[22] Ikonomidis, J. S, Kratz, J. M, Crumbley, A. J, et al. Twenty-year experience with the St.Jude Medical mechanical valve prosthesis. J Thorac Cardiovasc Surg (2003).

[23] Lengyel, M. Vandor L: The role of thrombolysis in the management of left-sided prosthetic valve thrombosis: a study of 85 cases diagnosed by transesophageal echocardiography. J Heart Valve Dis (2001).

[24] Durrleman, N, Pellerin, M, Bouchard, D, et al. Prosthetic valve thrombosis: twenty-year experience at the Montreal Heart Institute. J Thorac Cardiovasc Surg (2004).

[25] Khan, S. S, Trento, A, Derobertis, M, et al. Twenty-year comparison of tissue and mechanical valve replacement. J Thorac Cardiovasc Surg (2001).

[26] Heras, M, Chesebro, J. H, Fuster, V, et al. High risk of thromboemboli early after bioprosthetic cardiac valve replacement. J Am Coll Cardiol (1995).

[27] Mahesh, B, Angelini, G, Caputo, M, Jin, X. Y, & Bryan, A. (2005). Prosthetic valve endocarditis. Ann. Thorac. Surg., 0003-4975, 80(3), 1151-1158.

[28] Velicki, L, Susak, S, Cemerlic-adjic, N, & Redzek, A. Aortic valve endocarditis, Chapter in: Ying-Fu Chen and Chwan-Yau Luo- Aortic valve, InTech Publishing (2011).

[29] Velicki, L, Susak, S, & Srdanovic, I. Kovacevic M; Infective endocarditis of native aortic valve: destruction of leaflet with an aorto-cavitary fistula to the right ventricle, Chirurgia (2010). , 23(6), 261-266.

[30] Koertke, H, Minami, K, Boethig, D, et al. INR self-management permits lower anticoagulation levels after mechanical heart valve replacement. Circulation (2003). Suppl II):II-75.

[31] Butchart, E. G, Ionescu, A, Payne, N, et al. A new scoring system to determine thromboembolic risk after heart valve replacement. Circulation (2003). Suppl II):II-68.

[32] Kumar, D, & Elefteriades, J. Ezekowitz MD: Anticoagulation in patients with prosthetic heart valves. Cardiac Surg Today (2004).

[33] Koo, S, Kucher, N, Nguyen, P. L, et al. The effect of excessive anticoagulation on mortality and morbidity in hospitalized patients with anticoagulant-related major hemorrhage. Arch Intern Med (2004).

[34] Massel, D. Little SH: Risk and benefits of adding antiplatelet therapy to warfarin among patients with prosthetic heart valves: a metaanalysis. J Am Coll Cardiol (2001).

[35] Lund, O, Nielsen, S. L, Arildsen, H, et al. Standard aortic St. Jude valve at 18 years: performance, profile and determinants of outcome. Ann Thorac Surg (2000).

[36] Society of Thoracic Surgeons National Cardiac Surgery DatabaseAvailable at: http://www.sts.org/documents/pdf/STS-ExecutiveSummaryFall2005.pdf. November (2005).

[37] Edwards, F. H, Peterson, E. D, Coombs, L. P, et al. Prediction of operative mortality after valve replacement surgery. J Am Coll Cardiol (2001).

[38] David TE: Surgery of the aortic valve. (1999). *Curr Probl Surg*.

[39] Rankin, J. S, Hammill, B. G, Ferguson, T. B, et al. Determinants of operative mortality in valvular heart surgery. J Thorac Cardiovasc Surg (2006). , 131, 547-57.

[40] Kuduvalli, M, Grayson, A. D, Au, J, et al. A multi-centre additive and logistic risk model for in-hospital mortality following aortic valve replacement. Eur J Cardiothorac Surg (2007). , 31, 607-13.

[41] Butchart, E. G, Payne, N, Li, H, et al. Better anticoagulation control improves survival after valve replacement. J Thorac Cardiovasc Surg (2002).

[42] Akins, C. W, Buckley, M. J, Daggett, W. M, et al. Risk of reoperative valve replacement for failed mitral and aortic bioprostheses. Ann Thorac Surg (1998).

[43] Potter, D. D, & Sundt, T. M. rd, Zehr KJ, et al: Operative risk of reoperative aortic valve replacement. J Thorac Cardiovasc Surg (2005).

[44] Cabalka, A. K, & Emery, R. W. Petersen RJ: Long-term follow-up of the St. Jude Medical prosthesis in pediatric patients. Ann Thorac Surg (1995). S618

[45] Emery, R. W, Erickson, C. A, Arom, K. V, et al. Replacement of the aortic valve in patients under 50 years old with the St. Jude Medical prosthesis. Ann Thorac Surg (2003).

[46] Dumesnil, J. G. Pibarot P: Prosthesis-patient mismatch and clinical outcomes: the evidence continues to accumulate. J Thorac Cardiovasc Surg (2006).

[47] Pibarot, P, & Dumesnil, J. G. Prosthesis-patient mismatch: definition, clinical impact, and prevention. Heart. (2006). , 92, 1022-9.

[48] Milano, A D, De Carlo, M, Mecozzi, G, et al. Clinical outcome in patients with 19-mm and 21-mm St. Jude aortic prostheses: comparison at long-term follow-up, Ann Thorac Surg (2002). , 7337-43.

[49] Rao, V, Jamieson, W, & Ivanov, E. J. et al Prosthesis-patient mismatch affects survival following aortic valve replacement. Circulation (2000). IIIIII9.III9, 5.

[50] Blais, C, Dumesnil, J G, Baillot, R, et al. Impact of prosthesis-patient mismatch on short-term mortality after aortic valve replacement. Circulation (2003). , 108983-988.