LECTURE

Aortic Valve Replacement

Michael J. Hollingsed, BS, PA-C, CCP

West Virginia University
Department of Surgery
Morgantown, WV

Indications for aortic valve replacement can be summarized into two disease processes, moderate to severe aortic stenosis (with or without symptoms) and moderate to severe aortic insufficiency. Fortunately, standards of care differ with each physician and so indications for aortic valve replacement are not clearly defined. To understand how the surgeon or cardiologist decides when to recommend replacement of an aortic valve, a brief review is presented.

History

The first open heart operation was by Rehn, who repaired a stab wound to the heart in 1896 (1). Soon afterward physicians were aware of the significance of the role cardiac valves played and, in 1914, Tuffier described six experimental aortic and pulmonary valvulotomies via digital dilation (2). In the 1920's efforts were limited to wounds of the heart (except for constrictive pericarditis) until 1929 when Cutler and Beck performed one of the first mitral stenosis procedures (3). World War II again limited concern to cardiac injuries and removal of intracardiac foreign bodies, but, in 1952 Bailey and associates performed a transventricular dilatation with a mechanical dilator to separate fused commissaries for aortic stenosis (4). Gibbon developed the pump-oxygenator in 1954 (5) which expanded the treatment for aortic valve disease by introducing accurate valvulotomies. That wasn't enough, however, and numerous trials of excision of the diseased leaflet and replacement with a prosthetic leaflet followed. From 1960 to 1961 suboptimal results for prosthetic replacement were experienced by Bahnson (6), Harken (7), McGoon (8) and Lillehei (9) until Dr. Starr's original ball valve became clinically successful in 1960 (10). Definitive treatment for aortic valve disease was on the horizon.

Aortic Valve Anatomy (11)

The aortic valve is located in the root of the aorta in a dilated area called the sinus of valsalva at the outflow of the left ventricle into the main systemic artery. The ostia of the right and left main coronary arteries, which supply blood to the myocardium, lie in the aortic sinus of valsalva. The aorta has three semilunar cusps. The cusps of the aortic valve are the right coronary cusp, left coronary cusp and the posterior or noncoronary cusp. The aortic valve differs in structure from the other valves. The leaflets are thinner, have no chordae tendinal, and form cusps which, when the valve is closed, are similar in appearance to a half moon. For this reason it is called a semilunar valve. The valve is composed of dense connective tissue covered on either side with endocardium. The connective tissue lends strength to the valve leaflets and the endocardial tissue serves an antithrombogenic function. The function of the valve is to permit flow in one direction. Blood flowing out of the ventricle during contraction forces the semilunar cusps to collapse against the wall of the sinus of valsalva. At the end of contraction, blood begins to back flow from the aorta into the left ventricle but, instead of filling the ventricle, it fills the cusps causing the edges to meet and the valve to close. The shape of the sinus of valsalva helps to direct the blood so that it does fill and open the cusps instead of passing the collapsed leaflets and re-entering the left ventricle. A centrally placed nodule of dense connective tissue is located on the free margin of each valve and on each side of the nodule is a thinned out area, the lunule. The lunule are composed of two layers of epithelium back to back with a small amount of connective tissue between. After systole, the thin flexible lunule make contact with those adjacent cusps, simultaneously, the hard nodules come together to plug the center preventing backflow.

Etiology Classifications

Etiologic classifications of aortic valve disease can be separated by either disease process or the hemodynamic end point of aortic disease - stenosis or regurgitation. As the practitioner often sees the result of the disease, a brief review of the causes of aortic stenosis and regurgitation is highlighted.

Rheumatic Valvular Aortic Stenosis

Rheumatic fever accounts for 30 - 50% of aortic valve disease - depending on the study quoted. Characterized by pancarditis and specific involvement of the heart valves, it begins with inflammation and swelling of the annulus, extends through the cusps and then causes a valvulitis (12). The cusps become thickened and edematous with capillary vascularization and cellular infiltration (lymphocytes, occasional polymorhonuclear

Address correspondence to: Michael J. Hollingsed, West Virginia University, School of Medicine, Health Sciences Center, Department of Surgery, Morgantown, WV 26505, (304) 293-3311

leukocytes) occurring. Verrucae (wart-like vegetations of hyaline eosinophilic material resulting from the swollen, degenerated collagen and disintegrated cells) appear near the valvular surface. The inflammation produces exudate which causes thickening and deformity of the valve and commissural fusion between adjacent cusps (12). Adherence begins at the less mobile portions of the cusp where they attach to the aortic wall. Cusps become thickened and fixed into position and ultimately fuse into a solid ring. This causes a small, triangular and irregular aperture in the center of the valve which causes not only a stenosis, but insufficiency. Occasional inordinate fusion of the right and left cusps create an apparent bicuspid valve. Calcification is usually more pronounced in the anterior cusps (12).

Subacute Bacterial Endocarditis

Acute bacterial endocarditis (from a previous bout of rheumatic fever) usually causes aortic regurgitation. One of the predisposing causes for the aortic valve to become dysfunctional is for it to be originally abnormal or bicuspid. This is usually seen at catheterization or at the time of surgery. Aggravating bacterial causes is exemplified by the classic drug addict. Very few of these individuals have normal architecture of the valve (13). The bacterial process erodes the valve with anuerysmal and/or perforation occurring with secondary aortic insufficiency (13). The growth of vegetations can cause regurgitation. One of the unique occurrence with bacterial endocarditis is the nontraumatic rupture. One rarely finds ruptures in syphilitic aortitis or in rheumatic/atheromatous valves. Precipitous death can occur when a leaflet is perforated (abrupt change in the diastolic murmur) and progressive congestive heart failure occurs. The most common cause of death occurs from heart failure due to valve destruction (13, 14, 15). Emboli from vegetative/mycotic anuerysms can also lodge in multiple sites including the abdomen and cause a septicemia and eventual death.

Two-dimensional echocardiography can describe the movement of the anterior cusps, measure vegetations and indicate whether the valve annulas is normal or involved with abscess formation. This diagnostic tool is extremely useful to the surgeon for planning the operation.

Until recently, a six-week course of antibiotics was believed to be the treatment of choice prior to aortic valve replacement (12). During this interval, patients died from heart failure. Currently, a more aggressive short course of antibiotics prior to surgery has improved overall results (13, 15). Even when the patient has become afebrile and has had appropriate antibiotics, microorganisms are seen within the substance of the excised valve material. Therefore, prolonged use of antibiotics is still recommended (13).

Syphilis

Syphilis is infrequently seen in the United States. Typically it begins in the aorta around the vas vasorium with initial perivascular cellular infiltration which compromises nutrient vessels thereby destroying muscular and elastic layers of the tunica media as well as roughening the intima (12).

Valve dysfunction occurs when the syphilitic process affects the aortic root. Dilatation of the aortic ring and widening of the commissures occurs, the cusps may evert, retract, stiffen and shorten with resultant aortic insufficiency.

As with any process which affects the layer of the vascular tree, the coronary ostea may be affected and narrowed with coronary insufficiency occurring. To place syphilitic valvular disease in perspective, less than 5% of autopsied aortic regurgitant patients have syphilitic valvular disease (16).

Traumatic Aortic Valve Disease

Traumatic Aortic Valve Disease (TAVD) is the most common of all traumatic valvular lesions, though aortic regurgitation occurs infrequently (12). Although the aortic valve is injured more often than any other valve, the only other injuries that produce aortic regurgitation (other than penetrating injuries) are those produced by unusually strenuous effort or direct trauma (blow to the chest wall, fall from a tree) (12,17).

The injury occurs when the ventricles are in early diastole and the aortic valve is under maximal tension. Usually a single cusp is torn from its commissural attachment. Traumatic regurgitation can be caused by a penetrating wound which will usually result in a linear opening in the cusp. Occasionally more than one cusp may be involved.

In many of the cases of traumatic aortic regurgitation, the aortic valve had some underlying disease (sub-bacterial endocarditis, syphilis) though TAVD can occur in a normal valve. The signs and symptoms are usually classical and spontaneous; abrupt chest pain, musical diastolic murmur, signs of free aortic regurgitation, and progressive heart failure.

Marfan's Syndrome

The presentation of Marfan's does not need to be discussed here for diagnostic purposes. However, few would miss the classic features; tall/slim physique, elongated arms and legs, long tapering fingers, hyperextensibility of the joints, pectus deformity of the thorax, subluxation of the lens of the eye with predisposition to ultimate blindness and a high arched palate. Cardiovascular anomalies restricted to the aorta are aortic regurgitation, ascending aortic arch aneurysm and predisposition to aortic dissection.

Marfan's causes a medial necrosis from myxoid degeneration of the medial layer of the aorta (contains stellate cells surrounded by basophilic ground substance in the loose myxomatous stroma (12,18)). Cystic medial necrosis, per se, is not pathognomonic of Marfan's syndrome - rather it is a degenerative change in the aortic wall and can occur in otherwise normal individuals more than 40 years old (12). Incomplete Marfan's ("forme fruste") is manifested by aortic regurgitation and ascending aortic dissection occurring with medial necrosis, yet none of the other classical stigmata appear.

The cardiovascular surgeon usually sees two manifestations of Marfan's Syndrome with the end result of aortic regurgitation. One is progressive aneurysmal dilatation of the ascending aorta secondary to cystic medical necrosis with gradual annular

dilatation and secondary aortic regurgitation. The second is a dissecting aneurysm and hematoma of the ascending aorta which results in acute aortic regurgitation (12).

Corrective procedures include creation of a bicuspid valve with excision of the non-coronary leaflet and replacement of the ascending aorta with a woven dacron graft (18). Unfortunately, there is diseased tissue left to propagate. Currently, a more aggressive technique includes suturing a tubular prosthetic graft directly to prosthetic valvular rim before the prosthetic valve is inserted in the normal subcoronary position. The right and left coronaries are then sutured into the buttonholes of the tubular graft previously made by the surgeon.

Calcific Aortic Valvular Disease

Of the two classes of calcific aortic valvular disease, congenital bicuspid valve is the most common. Initially the bicuspid valve may have no dysfunction or a small amount of regurgitation. The incidence and degree of calcification increases with age. A large percentage of patients are symptomatic by age 20. At age 30 virtually all exhibit dysfunction (12). Bicuspid valve can also be associated with a PDA and coarctation (12). Pathological configuration of a congenital deformed valve may be obscured by subsequent calcific changes, atheromatous plaques or secondary subacute bacterial endocarditis (SBE). In severe disease it may be difficult to distinguish between a congenital bicuspid valve and one secondary from rheumatic fever although usually one is able to find rheumatic stigmata. Usual treatment consists of valvulotomy in infancy (which becomes calcified later in life)(12) and finally aortic valve replacement in adulthood.

The secondary cause of calcific aortic valvular disease is idiopathic aortic stenosis. Rheumatic fever has been presumed to cause a majority of cases though there has been some doubt in its frequency in patients over 50 (12). It is unlikely that rheumatic fever would first produce a murmur after middle age, unless the patient had a definite history of rheumatic fever in adult life. Idiopathic aortic stenosis can be secondary to aging, degeneration and atherosclerosis. In evaluating calcific aortic disease, careful attention to calcification adjacent to the aortic cusps, sinus of Valsalva, anterior leaflet of the mitral valve, bundle of His, mitral annulus and coronaries should be investigated.

Aortic Stenosis

Aortic stenosis (AS) is defined by the Joint Committee of Hospital Standards (1986) as a gradient greater than 50 mmHg (torr) across the aortic valve during cardiac catheterization. Usually the clinician first sees the patient with the manifestations of aortic stenosis; syncope, angina and congestive heart failure.

Clinical Presentation of Aortic Stenosis

Some of the signs and symptoms of aortic stenosis rarely show until 10 to 20 years after the stenotic lesion appears. Occasional mild dyspnea on exertion (DOE) may be present. When pulmonary congestion progresses to decompensation, a fatal outcome will happen within months. The turning points in aortic stenosis are: developments of angina pectoris, syncope and/or left ventricular failure. Any singular entity of or combination of these symptoms spell a gloomy outcome (12,13). Sudden death accounts for 20% of aortic stenosis fatalities. Syncope is usually found in one-third of patients. The AS murmur (systolic, diamond shaped ejection murmur) is heard best at the aortic area to the right of the sternum.

Roentgenogram and Electrocardiogram in Aortic Stenosis—

The presence of calcification of the aortic valve on chest roentgenogram is significant. Left ventricular dilatation occurs late in the disease course.

The electrocardiogram may demonstrate left ventricular hypertrophy, but may be normal. Occasionally a heart block, bundle branch block, or an intraventricular conduction defect may be associated with calcific AS from extension of calcium into the fibrous septum.

Catheterization in Aortic Stenosis

Cardiac catheterization is diagnostic if the systolic aortic to left ventricular pressure gradient is equal to or greater than 50 mmHg. Combined with a normal cardiac output this is moderate or severe AS. The left ventricular pressure (both systolic and diastolic) are elevated as well as left atrial pressure as seen with Swan-Ganz measurement of pulmonary capillary wedge (PCW) pressure.

The aortic valve area (normal AV area = 2.6 - 3.5cm^2) can be calculated from the Gorlin formula:

$$\text{Area} = \frac{\text{cardiac output}}{44.5 \times \text{syst fill time} \times \text{syst aortic-ventric gradient}}$$

Mild AS	=	0.8 - 1.0cm$_2$
Moderate AS	=	0.5 - 0.7cm$_2$ gradient 50mmHg
Critical AS	=	<0.5 w/gradient >50mmHg

Always check mitral valve integrity especially with mitral regurgitation and associated coronary artery disease.

Pathophysiology of Aortic Stenosis

Aortic stenosis can be easily understood by reviewing pathophysiologic changes. Always remember that AS is a outflow obstruction = left ventricular (LV) pressure overload and essentially fixed cardiac output.

Pathologic changes occur at three anatomical levels. Supravalvular obstructions are usually due to abnormal supravalvular membranes. Valvular obstructions interestingly occur at three different age groups with onset of symptoms at different times in those age groups. Congenital bicuspid valves cause calcific changes early in life (symptoms typically occur at 40 - 50 years), presumably secondary to turbulence. Rheumatic heart disease, the most common cause of aortic valve disease, causes fusion of the commissures, thickened leaflets and a murmur by age 30. However, symptoms may not occur until age 50 - 60. Senile, calcific or degenerative AS causes accumulation

of calcium in the pockets of the aortic cusps and usually leave the free edge of the aortic leaflet free and normal. Symptoms usually appear at 60 - 70 years age. Subvalvular changes are usually hypertrophic obstructive cardiomyopathies or subvalvular membranes. Other pathological markers seen in AS include classic (enormous) concentric hypertrophy of the LV muscle without significant dilatation of the LV chamber. This can cause decreased LV compliance from a thickened myocardial wall and relative subendocardial with a concurrent high myocardial oxygen demand.

Some of the pathophysiological changes that occur are very relevant to diagnostic and future therapeutic approaches. Do not be fooled into thinking that the stenosis may be minor in chronic AS. If the measured gradient is low on cardiac catheterization, and no regurgitation is present, elevation of the left ventricular end diastolic pressure (LVEDP) is often a sign of early LV failure. In fact, the aortic valve gradient may actually fall as myocardial contractility is no longer able to sustain the high intraventricular pressure. One of the final points to remember is that cardiac output may remain normal with mild elevation of LVEDP and left atrial pressure (LAP) until failure occurs.

Pre-operative Management in Aortic Stenosis

Remembering all the pathophysiological nuances that occur in AS, several management points appear. The clinician must remember to maintain the patient's mean arterial pressure (MAP). Assure that the patient is full (by PCW) - but not overfilled and allowed to slip in failure. Avoid inotropic stimulation unless the patient falls below baseline hemodynamic measurements. Avoid bradycardia; low stroke volume requires a higher heart rate to maintain coronary perfusion. Avoid tachycardia; a faster ejection will result in increased oxygen demand and therefore increased aortic gradient. Finally, deterioration can occur if atrial 'kick' is lost so the health care practitioner must be prepared to cardiovert if sinus rhythm is lost and hemodynamics fall.

For the surgical assistant, observations may reveal a greatly enlarged heart, and, when heart failure occurs, dilatation of the LV chamber develops as well as dilatation of the right atrium and right ventricle. A "jet" lesion produced by a narrow orifice allowing blood to strike the aortic wall may result in local thickening and fibrosis in the ascending aorta (12). In long standing severe AS significant poststenotic dilatation of the proximal ascending aorta usually occurs.

Aortic Regurgitation

Aortic regurgitation (AR), as defined by the Joint Committee on the Accreditation of Hospitals, is diagnosed "if cardiac catheterization reveals marked regurgitation on aortic injection." However, there are numerous aspects to this pathological condition.

Hemodynamics of Aortic Regurgitation

The dynamic effects of AR are directly proportional to the size of the orifice, amount of regurgitation and the amount of pressure between the ascending aorta and left ventricle in early diastole. The magnitude of reflux into the LV can be clinically assessed by the intensity of the regurgitant murmur, degree of LV thrust and chest xray size of the LV. Keep in mind that CHF occurs in the terminal stages of AR.

One helpful aspect of tachycardia in AR is that it shortens the diastolic phase and reduces volume return.

Symptoms of Aortic Regurgitation

Usually the patient is symptom free for 10 years or has had a history of minor symptoms. Abrupt onset of CHF can present and, if untreated with surgical intervention, death usually occurs in five years. Occasional precordial chest pains occur in the early stages as well as palpitations. Progressive symptoms of dyspnea on exertion (DOE), angina and peripheral vasomotor phenomena with severe sweating and intolerance to heat suggest progression of disease.

Physical Findings in Aortic Regurgitation

There are many physical findings found in AR, all related to the patient's hyperkinetic circulatory status. As a result of the increased volume ejected during systole and rapid run-off in diastole, many classical signs are present for the watchful clinician. The wide pulse pressure and initial bounding of the pulse has been called "water-hammer pulses" and Corrigan pulse - where the pulse collapses rapidly. The water hammer pulse can be felt easily in the carotids where the double peaked carotid pulse can be felt in severe AR and combined AR and AS. Quincke's pulse can be seen by pressing on the nail bed and watching for capillary pulsation.

The blood pressure in AR is also diagnostic. The description of systemic blood pressure in the leg greater than the arm by 40 mmHg is Hill's sign. A deceased diastolic blood pressure and increased systolic blood pressure are also suggestive of AR. In auscultating the femoral arteries you can occasionally hear a "pistol shot" sound.

Murmurs in aortic valve disease are the most described in the literature. The murmur has been describe as a high pitched, early decrescendo diastolic murmur heard best to the left of the sternum in the second intercostal space, especially on expiration with the patient leaning forward. The S4 (atrial) gallop is soft and must be listened for. Occasionally, you will hear the Austin-Flint murmur (diastolic rumble) at the apex. Durozier's murmur/sign is the diastolic murmur while auscultating the femoral arteries with pressure ("fro" portion of the "to and fro" murmur).

Murmurs which radiate down the left sternal border suggest AR secondary to damaged leaflets. If the murmur radiates down the right sternal border, AR may be secondary to dilated root anatomy. An S3 may also be heard.

The only sign in acute AR may be the soft, blowing murmur at the left sternal border and a loud S3. When the hemodynamic alterations of acute AR respond to the sudden backflow into a poorly compliant LV, the volume overload markedly increases the LV diastolic pressure (and volume) which results in increased pulmonary capillary wedge pressure (PCWP),

secondary pulmonary hypertension and resultant pulmonary edema. Chest X-rays are unremarkable except what was noted previously. EKG's usually exhibit a strain pattern secondary to LV dilatation and hypertrophy; occasional LA enlargement may be seen.

As AR progresses, the advanced AR patient will exhibit angina (seen in 50% of patients when aortic diastolic pressure <50 mmHg). LV failure gradually occurs with progression of disease to left heart failure and right heart failure.

Roentgenogram in Aortic Regurgitation

The chest X-ray reveals an enlarged left ventricle contour which may be evidenced by a boot shaped heart. Calcification of the aortic valve and wall may suggest syphilitic aortitis. In acute AR you may only see pulmonary edema changes. In long standing AR, the coronaries can appear larger than normal and are usually free of disease.

Echocardiography in Aortic Regurgitation

Echocardiography is one of the most diagnostic and descriptive examinations for AR. With this non-invasive procedure you can determine LV size (especially dilatation), whether there are any vegetations (from bacterial endocarditis), if there are any torn cusps and if the mitral valve vibrates in diastole suggesting early closure of the mitral mechanism which is present in acute and severe cases of AR.

Catheterization in Aortic Regurgitation

Differential changes between acute and chronic AR has been described by Mann. In acute aortic regurgitation, the patient exhibits a slow heart rate, small left ventricular end diastolic volume (LVEDV), and a small end systolic and total systolic stroke volume. Chronic aortic regurgitation demonstrates an increased aortic systole and pulse pressure and amplification of the peak systolic pressure in the peripheral arteries, especially in the femoral and popliteal arteries.

Classification of aortic regurgitation can also be estimated by the amount of regurgitant dye volume into the left ventricle when the dye is injected under pressure. 1+ aortic regurgitation is when the regurgitant dye clears with the first heart beat. In 2+ regurgitation (moderate) there is increased dye reflux into the left ventricle in diastole which faintly opacifies the left ventricle. 3+ aortic regurgitation (moderately severe) finds the left ventricle and aorta equally opacified with dye. Severe or 4+ aortic regurgitation is exemplified by dense opacification of the left ventricle on the first systolic beat and the left ventricle is more opacified than the aorta.

The regurgitant fraction (RF) calculation is also used to grade regurgitant lesions. The formula is:

$$RF = \frac{RSV \times 10}{TSV}$$

RF = regurgitant fraction
RSV = regurgitant systolic volume
TSV = total systolic volume

Using this formula, derived numbers for the scientific clinician cannot be used to accurately determine the degree of regurgitation. If the regurgitant fraction (RF) is equal to or less than 20% then mild AR is considered to exist. If the RF is 20-40%, this would be considered moderate AR. RF equal to 40-60% is moderate severe AR, and RF greater than 60% is severe AR.

Other catheterization values to look for include the pressure gradient. Usually there is none across the aortic valve in pure regurgitation, but the diastolic pressure in the ascending aorta will be collapsing and may approximate LVEDP. Compensated AR may have a normal LVEDP. If the LVEDP is greater than 25 mmHg, suspect congestive heart failure. In aortic root angiography you can determine the degree of AR and check for dissection. The LV angiogram will demonstrate LV function and regurgitant fraction. If angina is present always check the coronaries for luminal obstructions. The prognosis is good if the LVESV is less than 30 ml/M_2, but poor if greater than 90 ml/M_2.

Pathology of Aortic Regurgitation

Pathologic changes in AR can be summarized by three categories. In Category I the valve annulus is normal, suggesting an acute process, perforation, fenestration, thickening and/or shrinkage of the leaflets and possible prolapse of a floppy leaflet all which prevent adequate central coaptation of the valvular leaflets. Category II is where the valve annulus is dilated suggesting a chronic process. Here the leaflets are frequently pliable, thick and without microscopic evidence of gross disease. Again, inappropriate leaflet coaptation is the problem. Category III is rheumatic valvulitis. Here the commissures are fused and calcification may be present. Usually a combination of stenosis and regurgitation are present.

The final pathologic picture is when stroke volume (SV) and workload in AR has progressed to dilatation and hypertrophy of the LV. Sometimes the ventricle enlarges to the degree where it resembles an ox heart or "cor bovinum."

Pathophysiology of Aortic Regurgitation

In chronic AR, rheumatic heart disease accounts for a majority of patients. Other causes would include: bacterial endocarditis; syphilis (syphilitic aortitis causes aortic root dilatation); congenital lesions (bicuspid valve, ventricular septal defect causes the aortic leaflet to be pulled down, hypoplastic aorta and supravalvular/membranous AS which can cause regurgitant effects); Marfan's Syndrome, which results in a cystic medionecrosis and has been described earlier in this paper; rheumatic arthritis; Reiter's Syndrome; aortic valve sclerosis; dissecting aortic aneurysm; ankylosing spondylitis; and finally, hypertension, which increases afterload and therefore regurgitation.

The health care provider must remember the pathophysiological changes that are occurring to plan his/her therapeutic approach and management. First, left ventricular overload will cause eventual left ventricle hypertrophy and dilatation. Second, low aortic diastolic pressure causes low

coronary blood flow. The last point to remember is that there is increased LV output and work. Therefore, "pearls" of management would be to avoid hypertension which increases regurgitation. By decreasing peripheral vascular resistance, you decrease aortic regurgitation which would promote beneficial results. Avoid bradycardia which causes decreased diastolic pressure during regurgitation, increased intraventricular volume and pressure, which results in decreased coronary blood flow. The last "pearl" is to keep the patient 'full' by carefully monitoring the fluid status, but don't overfill.

Indications for Surgery in Aortic Regurgitation

JCAH standards (1986) leave the cardiologists and surgeons to determine appropriate treatment with their recommendation for surgery "if cardiac catheterization reveals marked regurgitation on aortic injection." Most surgeons would recommend operation in the absence of complicating features. The surgeon will also temper his timing for an operation with the knowledge that most patients with moderate AR can lead a normal lifestyle for years with no changes in symptoms. Surgery would be hastened with the onset of symptoms especially if there is progressive increase in heart size on serial chest X-rays. Significant reduction of functional capacity despite digitalis and diuretic therapy would also cause concern and recommendation for surgery.

Several other signs would prompt surgical intervention. Severe regurgitation, diastolic pressure less than 50 mmHg with angina, or clinical left ventricular failure or right and left heart failure are also signs of failing capacity of the heart. Finally, surgery would be prompted if regurgitation is secondary to bacterial endocarditis or annular dilatation which becomes progressive.

In a study done by Segal and Associates of 100 cases of AR that were left untreated, death resulted approximately 6.5 years after development of symptoms and after diagnosis. Approximately 75% of patients with moderately severe AR will survive 5 years and only 50% will survive 10 years. More importantly, without surgery death occurs approximately 5 years after angina appears and within 2 years after CHF (20).

One of the hesitating factors for surgery is a decrease in the cardiac index or marked increase in LVEDP. These factors signify a poor surgical candidate with poor prognostic outcome.

In view of the current low mortality rate after AVR and the advancement of improved prosthetic valves, surgery should be proposed for the young person with normal heart size who is unable to perform physically demanding work. Most clinicians would recommend surgical intervention if onset of symptoms appear. Also, surgery would be recommended if there was a progressive increase of cardiac size on serial chest roentgenograms.

Delay in surgical management could result in decreasing cardiac index or a marked increase in LVEDP, signifying ventricular failure. In these patients, surgical intervention has a poor prognosis.

Preoperative Evaluation

Patients with symptomatic AS, AR or mixed aortic valve lesions require a critical assessment of the cardiovascular, pulmonary, neurologic and renal functions prior to surgery. Pulmonary function tests (PFT's), renal function tests (RFT's), chest X-rays (CXR's), electrocardiogram (EKG) and two-dimensional echocardiography should be performed and reviewed before invasive cardiac catheterization is proposed. Patients suspected of a surgically repairable aortic valve should have cardiac catheterization done to examine the valve and amount of regurgitation or stenosis, examination of intracardiac/ventricular cavities and coronary artery patency.

Preoperatively, the patient must be counselled and encouraged to stop smoking. Many surgeons will refuse to operate if the patient continues to smoke over one pack per day or exhibits no desire to change his or her life-style in light of the disease process they are undergoing. Smoking has been proven to predispose the patient to atelectasis postoperative. Also, simply by stopping smoking a few days prior to operation will diminish the circulating carbaminohemoglobin and therefore increase the oxygen carrying capacity of the blood in the critical postoperative period.

Obviously, carious teeth should be removed prior to AVR to prevent bacteriological seeding of the prosthesis. Any subsequent dental procedure should be cleared with the patient's health care provider to prevent valvular dysfunction from bacteriological contamination.

Pre-operative physical therapy teaching as well as respiratory therapy's explanation of postoperative care for the lungs is beneficial to the patient. It also serves as a anti-anxiety session for the patient to be actively involved with his care. The patient should also shower with a bacteriostatic soap and receive prophylactic antibiotics prior to surgery.

Stabilization of cardiac medications and cessation of digitalis and diuretic therapy 24 to 36 hours prior to surgery are recommended, but under intense review by anesthesia clinicians. Some may recommend cessation of antiarrhythmic prescriptions.

Pre-operative laboratory studies would include, but not limited to, complete blood count with platelet count, prothrombin time, partial thrombin time, fibrinogen, complete blood chemistry (especially liver, renal and cardiac studies), electrolytes, glucose, serology studies, sickle cell evaluation for blacks, complete urinalysis with a clean catch culture and type and cross for blood and products.

Obviously all aspirin products and prescriptions with aspirin in them should be deleted prior to surgery. Some surgeons prefer a pre-operative dose of antiobiotics; you should check with the attending surgeon for his/her preference. One last note regarding pre-operative care stressed by the nursing staff: emotional preparation. The patient should be evaluated by all concerned in preparation for the operation. The attending surgeon should be available to answer any final questions brought up and have a firm understanding of the psychological profile of the patient. Any hint of instability must be reviewed and brought to the attention of the attending.

If there is a suspicion of an aortic aneurysm, a CT should be performed to delineate the pathology.

Summary

Symptomatic aortic valvular disease carries a poor prognosis unless surgical therapy is carried out. Prompt replacement of the diseased native valve with a prostheses is the currently accepted mode of treatment and can be performed with an acceptable operative morbidity and mortality. The prognosis of patients with symptomatic valvular disease is clearly enhanced with AVR.

Asymptomatic patients present more of a management problem. The clinician must be aware that a prosthetic valve is inherently associated with some morbidity and does not function exactly as a native valve. The fact that a "perfect" prosthesis does not exist mandates very careful selection of asymptomatic patients for AVR. One must maximize the time that the patient lives with the diseased native valve, but on the other hand, one must ensure that valve replacement is carried out before irreversible myocardial changes have occurred. This is particularly challenging in patients with aortic insufficiency, where considerable cardiac deterioration may occur with only subtle clinical manifestations.

Although many patients have good hemodynamic improvement, the major goal of valve replacement is to prevent further cardiac decompensation. Both mechanical and biologic prostheses are capable of functioning in this capacity, but there are advantages and disadvantages associated with each type of valve. Generally, the mechanical valve is more durable and can be expected to require replacement at the rate of only 1 - 3% per patient year. Unfortunately, the mechanical prostheses require the use of coumadin therapy to minimize the incidence of thromboembolic complications. Coumadin itself can be the source of considerable morbidity and must be monitored quite closely, especially in the elderly population. On the other hand, bioprostheses do not require long-term anticoagulation. The major drawback to the use of these valves is their limited durability. It appears that approximately 20% of patients with bioprostheses will require reoperation within 10 years of the initial procedure.

Clearly, the decision to recommend valve replacement requires a sound understanding of the pathophysiology of aortic stenosis and insufficiency. Likewise, the choice of the type of valve to be used must be based on a firm appreciation of the characteristics of mechanical and biologic prostheses.

With this knowledge in hand, it will be appropriate to explore the technical aspects of valve replacement in the next presentation.

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Suggested Reading Support Material

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