Mitral valve
atrioventricular valve located between the left atrium and left ventricle

Mitral Valve Prolapse

Mitral valve prolapse is a deformity that usually produces no symptoms. Rarely, it progresses and can result in sudden death. This condition occurs in up to 2.5% of the general population and more frequently in women than in men.

- In mitral valve prolapse, a portion of one or both mitral valve leaflets balloons back into the atrium during systole. Rarely, ballooning stretches the leaflet to the point that the valve does not remain closed during systole. Blood then regurgitates from the left ventricle back into the left atrium. About 15% of patients who develop murmurs eventually experience heart enlargement, atrial fibrillation, pulmonary hypertension, or heart failure.

- sx: fatigue, shortness of breath, lightheadedness, dizziness, syncope, palpitations, chest pain, or anxiety.

Mitral regurgitation

Mitral regurgitation involves blood flowing back from the left ventricle into the left atrium during systole. Often, edges of mitral valve leaflets do not close completely during systole because leaflets and chordae tendineae have thickened and fibrosed, resulting in their contraction. The most common causes of mitral valve regurgitation in developed countries are degenerative changes of the mitral valve (e.g., mitral valve prolapse) and ischemia of the left ventricle. The most common cause in developing countries is rheumatic heart disease and its sequelae.

Ssx: Chronic mitral regurgitation is often asymptomatic, but acute mitral regurgitation (e.g., resulting from a myocardial infarction) usually manifests as severe congestive heart failure. Dyspnea, fatigue, and weakness are the most common symptoms. Palpitations, shortness of breath on exertion, and cough from pulmonary congestion also occur.

Mitral Stenosis

Mitral stenosis is an obstruction to blood flowing from the left atrium (blood stasis) into the left ventricle. It most often is caused by rheumatic endocarditis, which progressively thickens mitral valve leaflets and chordae tendineae. Leaflets often fuse together. Eventually, the mitral valve orifice narrows and progressively obstructs blood flow into the ventricle.

Ssx:
- dyspnea on exertion (DOE) as a result of pulmonary venous hypertension. Symptoms usually develop after the valve opening is reduced by one third to one half its usual size. hoarsness may develop A-FIB

- Patients may experience progressive fatigue and decreased exercise tolerance as a result of low cardiac output.

- An enlarged left atrium may create pressure on the left bronchial tree, resulting in a dry cough or wheezing. Patients may expectorate blood (i.e., hemoptysis) or experience palpitations, orthopnea, paroxysmal nocturnal dyspnea (PND), and repeated respiratory infections.

- As a result of increased blood volume and pressure, the atrium dilates, hypertrophies, and becomes electrically unstable (patients experience atrial dysrhythmias).
Aortic Regurgitation

Aortic regurgitation is flow of blood back into the left ventricle from the aorta during diastole. It may be caused by inflammatory lesions that deform aortic valve leaflets or dilation of the aorta, preventing complete closure of the aortic valve. Blood from the aorta returns to the left ventricle during diastole, in addition to blood normally delivered by the left atrium.

S/sx: Aortic insufficiency develops without symptoms in most patients. Some patients are aware of a forceful heartbeat, especially in the head or neck. Marked arterial pulsations visible or palpable at carotid or temporal arteries may be present as a result of increased force and volume of blood ejected from a hypertrophied left ventricle. Exertional dyspnea and fatigue follow. Signs and symptoms of progressive left ventricular failure include breathing difficulties.

Aortic Stenosis

Aortic valve stenosis is narrowing of the orifice between the left ventricle and aorta. In adults, stenosis often is a result of degenerative calcifications. Calcifications may be caused by proliferative and inflammatory changes that occur in response to years of normal mechanical stress, similar to changes that occur in atherosclerotic arterial disease. Diabetes, hypercholesterolemia, hypertension, and low levels of high-density lipoprotein cholesterol may be risk factors for degenerative changes of the valve. Congenital leaflet malformations or an abnormal number of leaflets (i.e., one or two rather than three) may be involved. Rheumatic endocarditis may cause adhesions or fusion of the commissures and valve ring, stiffening of the cusps, and calcific nodules on the cusps.

S/sx: Many patients with aortic stenosis are asymptomatic. Have
- exertional dyspnea, caused by increased pulmonary venous pressure due to left ventricular failure.
- Orthopnea, PND, and pulmonary edema also may occur.
- dizziness and syncope.
- Angina pectoris is a frequent symptom; it results from increased oxygen demand of the hypertrophied left ventricle with decreased blood supply due to decreased blood flow into the coronary arteries and decreased time in diastole for myocardial perfusion.
- Blood pressure is usually normal but may be low. Pulse pressure may be low (30 mm Hg or less) because of diminished
**VALVULOPLASTY**

Repair, rather than replacement, of a cardiac valve is referred to as valvuloplasty. In general, valves that undergo valvuloplasty function longer than prosthetic valve replacements and patients do not require continuous anticoagulation.

- The type of valvuloplasty depends on the cause and type of valve dysfunction.
- Mechanical is more durable and used for younger patients.
- Mechanical valves do not deteriorate or become infected as easily as tissue valves.
- The most common valvuloplasty procedure is commissurotomy. Each valve has leaflets; the site where the leaflets meet is called the commissure. Leaflets may adhere to one another and close the commissure (i.e., stenosis). Less common.

**Mechanical valve replacement Indications:** patients with renal failure, hypercalcemia, endocarditis or sepsis, who require valve replacement

**Mechanical complication:** thromboemboli and long term use of required anticoagulants

**Tissue valve replacement:** less likely to form a thromboemboli, and long term anticoagulation is not needed and require replacement more frequently.

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**Valvular tissue replacements and how to get them**

Bio prosthetic valves:
- porcine valve (pig)
- bovine valve (cow)
- homografts (human cadavers)

Risk of clot formation is small therefore long term anticoagulation may not be needed.
**Cardiomyopathy**

A subacute chronic disorder of the heart muscle. Treatment is palliative not curative and the client needs to deal with a shortened life span and lifestyle changes.

***sodium is the major electrolyte I loved with cardiomyopathy R/T fluid overload from HF***

**Dilated cardiomyopathy**: fibrosis of myocardium and endocardium. Dilated chambers, mural wall thrombi prevalent.

**Nonobstructed**: hypertrophy of the walls, hypertrophied septum, small chamber chamber size.

**Obstructed**: obstruction of left ventricle outflow tract associated with the hypertrophied septum and mitral valve incompetence.

**Restrictive cardiomyopathy**: mimics constrictive pericarditis, fibrosed walls cannot expand or contract with chambers narrowed, emboli, stiff ventricular walls.

**Endocarditis**

**Rheumatic Endocarditis**

Inflammation of the inner lining of the heart and valves and specifically the mitral valve. Acute rheumatic fever, which occurs most often in school age children, may develop after an episode of group A beta-hemolytic streptococcal pharyngitis. Rheumatic endocarditis is a unique infective endocarditis syndrome.

**Infective Endocarditis**

Infective endocarditis is a microbial infection of the endothelial surface of the heart. It usually develops in people with prosthetic heart valves, cardiac devices (e.g., pacemaker), or structural cardiac defects (e.g., valve disorders, HCM). It is more common in older people. Primary presenting symptoms of infective endocarditis are fever and a heart murmur. Fever may be intermittent or absent, especially in patients who are receiving antibiotics or corticosteroids, in those who are older, and in those who have heart failure or renal failure. A heart murmur may be absent initially but develops in almost all patients. Murmurs that worsen over time indicate progressive damage from vegetations or perforation of a valve or rupture of chordae tendineae.

**Pericarditis**

Pericarditis refers to an inflammation of the pericardium, which is the membranous sac enveloping the heart. It may be a primary illness, or it may develop during various medical and surgical disorders.

Pericarditis may be asymptomatic. The most characteristic symptom of pericarditis is chest pain, although pain also may be located beneath the clavicle, in the neck, or in the left trapezius (scapula) region. Pain or discomfort usually remains fairly constant, but it may worsen with deep inspiration and when lying down or turning. The most characteristic clinical manifestation of pericarditis is a creaky or scratchy friction rub heard most clearly at the left lower sternal border. Other signs may include a mild fever, increased WBC count, anemia, and an elevated ESR or C-reactive protein level. Patients may have a nonproductive cough or hiccups. Dyspnea and other signs and
symptoms of heart failure may occur as a result of pericardial compression due to constrictive pericarditis or cardiac tamponade. The heart rate may increase to maintain cardiac output.

Myocarditis
Myocarditis, an inflammatory process involving the myocardium, can cause heart dilation, thrombi on the heart wall (mural thrombi), infiltration of circulating blood cells around the coronary vessels and between the muscle fibers, and degeneration of the muscle fibers themselves.

The symptoms of acute myocarditis depend on the type of infection, the degree of myocardial damage, and the capacity of the myocardium to recover. Patients may be asymptomatic, with an infection that resolves on its own