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Valvular Heart Disease



★ Objectives:

1. Know the Clinical presentation of Valvular Heart.
2. Identify the Etiology of Valvular Heart Diseases.
3. Do Clinical assessment of Valvular Heart.
4. Do Laboratory and non invasive assessment of Valvular Heart Diseases.
5. Apply Medical management of Valvular Heart Diseases.
6. Apply Invasive management of Valvular Heart Disease.

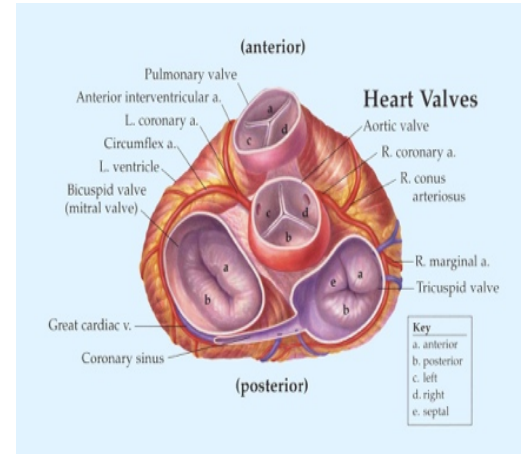
★ Resources Used in This lecture:

Slides, Step-up, Kaplan CK and Davidson's table.

Introduction

The heart is composed of 4 valves:

1. **Tricuspid valve:** Contain 3 cusps (anterior posterior septal /medial). Lies between Right atrium and right ventricle.
2. **Mitral valve:** Contain 2 cusps: (anterior & posterior). Lies Between left atrium and left ventricle.
3. **Pulmonary valve:** The valve is formed of 3 semilunar cusps (2 anterior and one posterior) between right Ventricle and pulmonary artery.
4. **Aortic Valve:** Aortic valve is formed of 3 semilunar cusps which are similar to those of pulmonary but differ in being one anterior and 2 posteriors. Between left ventricle and the aorta.



All of the valves, when functioning normally, act as one-way valves, allowing blood to flow either from one chamber to another, or allowing blood to flow out of the heart, in only one direction. The valves control the flow of blood through the heart by opening and closing during the contractions of the heart.

Heart Sounds:

- Normal Heart sounds

S1: "lub" of "lub-dub" closure of **tricuspid** and **mitral** valves.

S2: "dub" of "lub-dub" closure of **aortic** and **pulmonary** valves.

✓ Gallop rhythms and are heard in both normal and abnormal situations.

S3: "lub-dub-ta" → Indicates heart failure or volume overload. S3 heart sound indicates increased volume of blood within the ventricle from blood rushing in from the atria. Benign in youth, some trained athletes, and pregnancy but if it re-emerges later in life it may signal cardiac problems, such as a failing left ventricle as in dilated congestive heart failure (CHF).

S4: "ta-lub-dub" It is a sign of a pathologic state, usually a failing or hypertrophic left ventricle, as in systemic hypertension, severe valvular aortic stenosis, and hypertrophic cardiomyopathy. The sound occurs just after atrial contraction at the end of diastole and immediately before S1. best heard at the cardiac apex.

Valvular Heart Disease

- **Stenosis:** narrowing of the valve causing obstruction the blood outflow → can't open
- **Regurgitation:** the backward flow of blood through a defective heart valve → can't close
- **Prolapse:** slipping forward or down of an excessive or redundant leaflet

IMPORTANT NOTES

-All valvular heart disease can be congenital in nature.

-**Rheumatic fever** can lead to any form of valve disease, but most commonly **mitral stenosis**.

-**Ischemia** and **hypertension** commonly cause **regurgitation**.

- *General Symptoms :*

SOB and many presentation of *CHF* is present in all forms of valvular disease.

Murmurs are the only specific in terms of presentation.

Lesions on the **right side** of the heart (tricuspid & pulmonic valve) increase loudness with **inhalation**.

Lesions on the **left side** of the heart (mitral and aortic valve) increase loudness with **exhalation**.

- *Diagnostic test:*

1. Best initial test for all forms is echocardiogram.
2. Transoesophageal-echo both sensitive and specific.
3. Catheterization is the the most accurate test.

- *Treatment:*

All forms are associated with fluid over load → give diuretics

Mitral stenosis → dilated with balloon Aortic → surgical removal

Regurgitation → vasodilators (ACE,ARB,snifidipine and Hydralazine) Surgical replacement must be done before heart stretches to much → if it was stretched, surgery will not change anything.

-Tricuspid and pulmonary valves are very rare to happen but if so:

- **Tricuspid valve:**

-Most commonly affected valve from endocarditis by bacterial infection.

-It causes either regurgitation or stenosis.

-Commonly affect young patients (drug abusers or inpatients)

-Carcinoid Heart disease causes thickening of the valve → tricuspid stenosis

- Tricuspid regurgitation usually is caused secondary to Pulmonary HTN.

-Tricuspid valve disease usually found *accidentally*, if you found a disease in TV other than the 2 mentioned previously think of something happening in the heart.

- **Pulmonary valve:**

-Don't occur by its own.

-In rheumatic HD it usually causes pulmonary regurgitation.

-We call it pulmonary incompetence and it causes → graham steell murmur.



Aortic Valve Pathologies

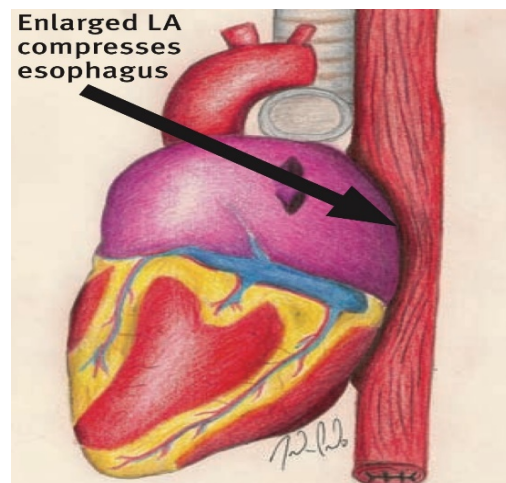
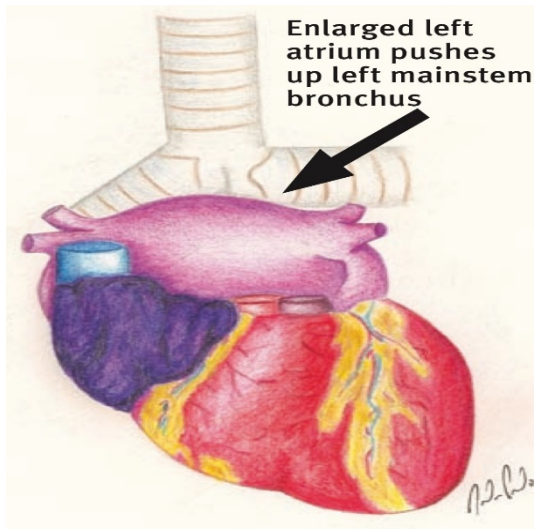
	Aortic Stenosis	Aortic Regurgitation
Etiology	<ul style="list-style-type: none"> -Calcification of a congenitally abnormal bicuspid valve -Calcification of aortic valve in elderly -Rheumatic Fever 	<p>Acute: Infective endocarditis, Trauma, aortic dissection/aneurysm</p> <p>Chronic:</p> <p>Inflammatory (Syphillis, Giant Cell Arteritis, Coll Vasc Dis-Ankylosis Spondylitis, Reiters)</p> <p>Inheritable (Marfans, Osteogenesis Imperfecta)</p>
Patho-physiology	<ol style="list-style-type: none"> 1- Aortic stenosis causes obstruction to LV outflow, which result in LV hypertrophy LVH 2- when aortic valve falls below 0.7 cm² cardiac output fails to increase with exertion causing Angina “normal at rest” 3- with long standing aortic stenosis the LV dilates causing progressive LV dysfunction 4- with severe stenosis LV dilation pulls mitral valve annulus apart causing mitral regurgitation 	<ol style="list-style-type: none"> 1- Inadequate closure of aortic valve leaflets causes leakage of blood to ventricles leading to increased LV end diastolic volume. 2- Left ventricle dilation and hypertrophy occur in response to maintain stroke volume and prevent diastolic pressure from increasing excessively 3- over time compensation fails leading to increased left-sided and pulmonary pressure
Clinical Presentation	<p>Symptoms: Heart failure – worst, due to increased LVEDp Angina - common due to increased demand Syncope with exertion</p>	<p>Symptoms: Dyspnea, orthopnea and PND “due pulmonary congestion”, Angina, Palpitations, Fatigue Cyanosis & shock “acute aortic regurgitation”</p>
	<p>Signs: Harsh Systolic Ejection Murmur – late peaking, S4 gallop (from LVH) Sustained Bifid LV impulse (from LVH) Pulsus Parvus et Tardus (Carotid Impulse)</p>	<p>Signs: Diastolic Decrescendo Blowing Murmur Hyperdynamic LV apical impulse, Bounding Pulses S4, S3 Gallop-advanced AI. pical Rumble – “Austin Flint Murmur”</p>
Diagnoses	<p>ECG: LV hypertrophy, LA enlargement CXR: Calcified aortic valve, enlarged LA/LV Echo: “Best initial” LVH, thickened & immobile aortic valve, dilated aortic roots Cardiac Cath: “most accurate” useful in symptomatic patients before surgery, older</p>	<p>ECG: LVH CXR: LVH and dilated aorta Echo: performed to chronic, stable patients to assess need for surgery Cardiac Cath:, to assess severity of regurgitation and degree of LV dysfunction</p>
Treatment	<p>Medical: has limited role Surgical: Aortic valve replacement “treatment of choice” indicated in symptomatic patients</p>	<p>Asymptomatic: medical therapy Serial echo check ups, salt restriction, diuretics, Vasodilators, afterload reduction “ACEI, arteriodilators”, digoxin Symptomatic: Aortic valve replacement Acute Aortic Regur: medical emergency, perform emergent valve replacement</p>

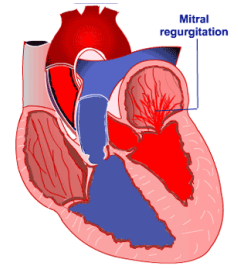
Mitral Stenosis

Etiology	<ul style="list-style-type: none"> - Rheumatic fever (most common) The patient may not recall a history of rheumatic fever. - Mitral annular Ca+ massive (rare) - Congenital (rare) 	
Patho-Physiology	<ul style="list-style-type: none"> - Rheumatic fever: immune-mediated damage to the mitral valve → scarring and narrowing of the mitral valve orifice → back flow of blood to the cardiopulmonary circuit and consequently → Congestion. - Anything that increases flow across the mitral valve (exercise tachycardia and so on) → Exacerbates the pulmonary venous HTN. - Patients are usually asymptomatic until the mitral valve area is reduced to approximately 1.5 cm² 	
Clinical Presentation	Symptoms	Signs
	<p>Congestive heart failure symptoms:</p> <ul style="list-style-type: none"> - <i>Dyspnea, orthopnea, PND.</i> - <i>Palpitations, chest pain.</i> - <i>Pulmonary venous HTN.</i> <p>Unique (MS) symptoms:</p> <ul style="list-style-type: none"> - Dysphagia: the dilated left atrium pressing on the esophagus.(see pic below) - Hoarseness: the dilated left atrium pressing on the laryngeal nerve. - Hemoptysis: the elevated left atrium ruptures the anastomosis of small bronchial veins - Atrial fibrillation. 	<ol style="list-style-type: none"> 1- S2 is followed by an opening snap. 2- Mitral stenosis murmur: (diastolic) <ul style="list-style-type: none"> - The opening snap is followed by a low pitched diastolic rumble and presystolic accentuation. Always remember Diastolic murmur=iuretics 3- Murmur is followed by a loud S1 <ul style="list-style-type: none"> - This can be the most prominent physical finding.
All signs and symptoms will increase with exercise and during pregnancy.		

Mitral Stenosis cont.

Diagnosis	<ul style="list-style-type: none"> - Echo is the best initial. - Catheterization is the most accurate 		
	Echo	ECG	CX-ray
	<ul style="list-style-type: none"> - Left atrial enlargement. - Thick classified mitral valve. - Narrowed fish mouth shaped orifice. - Signs of RVF in advanced disease. 	<ul style="list-style-type: none"> - Atrial rhythm disturbance - Atrial fibrillation - Left atrial hypertrophy 	<ul style="list-style-type: none"> - Assess left atrial enlargement. - Straightening of the left heart border. - Elevation of the left main bronchus. - Second bubble behind the heart.
Treatment	Medical		Surgical
	<ul style="list-style-type: none"> ○ Diuretics: for pulmonary congestion and edema. ○ Beta-blockers: for palpitation and to decrease heart rate. ○ If the patient has A-Fib treat them accordingly (digoxin and warfarin) 		<ul style="list-style-type: none"> ○ Percutaneous balloon valvuloplasty (was invented to keep pregnant women out of the OR) ○ Open commissurotomy (not any more)





Mitral Regurgitation

Mitral regurgitation is quite the opposite of mitral stenosis but surprisingly it has the same complications (CHF symptoms)!! And that due to the cyclic physiology of the heart and the lung.

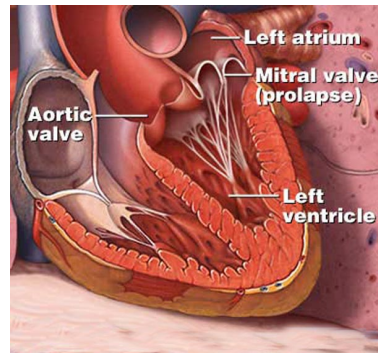
In Mitral Regurgitation the valve is open or leaking when it is supposed to be close during ventricular systole (systolic murmur). So when the left ventricle contracts the blood goes to the aorta and also some flows back to the left atrium. Mitral Regurgitation is associated with any condition that causes left ventricular dilation thus pulling the valve leaflets apart and spates them creating weaker leaking valve.

	Acute	Chronic
Pathophysiology	<ul style="list-style-type: none"> •Abrupt elevation of left atrial pressure in the setting of normal LA size and compliance, causing backflow into pulmonary circulation with resultant pulmonary edema. • Cardiac output decreases because of decreased forward flow, so hypotension and shock can occur. 	<ul style="list-style-type: none"> •Gradual elevation of left atrial pressure in the setting of dilated LA and LV (with increased left atrial compliance) • LV dysfunction occurs due to dilation. • Pulmonary HTN can result from chronic backflow into pulmonary vasculature.
Cause	<ul style="list-style-type: none"> •Endocarditis (most often Staph. Aureus). • Papillary muscle rupture (from infarction) or dysfunction (from ischemia). • Chordae tendineae rupture (usually 2-5 days post MI) 	<ul style="list-style-type: none"> • Mitral valve prolapse (MVP). • Rheumatic fever. • Marfan's syndrome. • Cardiomyopathy.
Prognosis	<p>is associated with much higher mortality</p>	

Mitral Regurgitation cont.

Clinical Presentations	Symptoms		Signs		
	<ul style="list-style-type: none"> - Dyspnea on exertion, PND, orthopnea - Palpitations - Pulmonary edema 		<ul style="list-style-type: none"> - Holosystolic murmur at the apex, which radiates to the back or clavicular area, depending on which leaflet is involved (radiates to the axilla) - AFib is a common finding <p>*Other findings: Diminished S1, widening of S2, S3 gallop; laterally displaced PMI; loud, palpable P2</p> <p>The murmur is worsened with Hand grip Squatting Leg raising</p>		
	Echo		ECG		CX-ray
	<ul style="list-style-type: none"> - Mitral regurgitation. - Dilated LA. - LV decreased. - LV function. 		<ul style="list-style-type: none"> - Left atrial enlargement. - Left ventricular hypertrophy. 		<ul style="list-style-type: none"> - Cardiomegaly. - Dilated LV. - Pulmonary edema.
Treatment	Medical			Surgical	
	<ul style="list-style-type: none"> ○ Preload reduction therapy: vasodilators Start with <i>ACE inhibitors</i> if the patient has a cough go for <i>AR Blockers</i> still can't tolerate go for <i>hydralazine</i> ○ Chronic <i>anticoagulant</i> if A-Fib is present ○ <i>IABP</i> as bridge to surgery for acute MR. 			<ul style="list-style-type: none"> ○ Mitral valve repair or replacement (performed before left ventricular function is too severely compromised) <p>How to assess left ventricular function?</p> <ul style="list-style-type: none"> - EF < 60% → indication of surgery - End Ventricular Systolic Diameter : EVSD > 40mm → indication of surgery 	

Mitral Prolapse



Definition	MVP is defined as the presence of excessive or redundant mitral leaflet tissue due to myxomatous degeneration of mitral valve leaflets and/or chordae tendineae. The redundant leaflet(s) prolapse toward the left atrium in systole, which results in the auscultated click and murmur.
Etiology	MVP is common in patients with genetic connective tissue disorders, such as: Marfan's syndrome, osteogenesis imperfecta, and Ehlers-Danlos syndrome.
Symptoms	<ul style="list-style-type: none"> - Mostly asymptomatic. - TIA - Chest pain - Palpitations
Signs	<ul style="list-style-type: none"> ○ Mid-systolic click or late systolic click ○ Midsystolic rumbling <p>Squatting decreases murmur and click because it increases LV chamber thus delaying the onset of the click and murmur and increases with standing and the Valsalva maneuver.</p>
Diagnosis	Echo is the most useful tool
Treatment	<i>Beta blockers</i> for chest pain
Prognosis	<ul style="list-style-type: none"> ○ Overall → benign condition ○ Sudden death and arrhythmias are rare ○ Associated with anxiety



✓ Aspirin: is used to control TIA
 ✓ Prophylaxes antibacterial therapy is used if associated with MR (Sub-Acute bacterial endocarditis)

MCQ's

1- A seventy-five-year-old patient presents to the ER after a sudden syncopal episode. He is again alert and in retrospect describes occasional substernal chest pressure and shortness of breath on exertion. His lungs have a few bibasilar rales and his blood pressure is 110/80. On cardiac auscultation, the classic finding you expect to hear is

- A. A harsh systolic crescendo-decrescendo murmur heard best at the upper right sternal border
- B. A diastolic decrescendo murmur heard at the mid-left sternal border
- C. A holosystolic murmur heard best at the apex
- D. A mid-systolic click

ANSWER:

The answer is **A**. The classic symptoms of aortic stenosis are exertional dyspnea, angina pectoris, and syncope. Physical findings include a narrow pulse pressure and the systolic murmur as described in answer A (rather than the aortic insufficiency murmur of answer B, the mitral regurgitation murmur of answer C, or the mitral valve prolapse click of answer D).

2- A seventy-two-year-old male comes to the office with intermittent symptoms of dyspnea on exertion, palpitations, and cough, occasionally productive of blood. On cardiac auscultation, a low-pitched diastolic rumbling murmur is faintly heard toward the apex. The origin of the patient's problem probably relates to

- A. Rheumatic fever as a youth
- B. Long-standing hypertension
- C. Silent MI within the past year
- D. Congenital

ANSWER:

The answer is **A**. The history and physical exam findings are consistent with mitral stenosis. A diastolic rumbling, apical murmur is characteristic. An accentuated first heart sound and opening snap may also be present. The etiology of mitral stenosis is usually rheumatic. It is rarely congenital. Two-thirds of patients afflicted are women.

3- In a 30-year-old asymptomatic female with newly diagnosed mitral valve prolapse, which of the following is true?

- A. Echocardiography demonstrates displacement of one or both mitral valve leaflets posteriorly into the left atrium during systole
- B. Migration of the systolic click and systolic murmur toward the first heart sound will occur during squatting
- C. Prophylactic beta-blocker therapy is indicated
- D. Significant mitral regurgitation is likely to occur (>50% chance) sometime in her life
- E. Restriction of exercise is advised to reduce the risk of sudden cardiac death

ANSWERS:

The answer is **A**. The fundamental defect in mitral valve prolapse is an abnormality of the valve's connective tissue with secondary proliferation of myxomatous tissue. The redundant leaflet or leaflets prolapse toward the left atrium in systole, which results in the auscultated click and murmur and characteristic echocardiographic findings. Any maneuver that reduces left ventricular size, such as standing or Valsalva, allows the click and murmur to occur earlier in systole; conversely, those maneuvers that increase left ventricular size, such as squatting and propranolol administration, delay the onset of the click and murmur. While most patients with mitral valve prolapse have a benign prognosis, a small percentage die suddenly. Severe mitral regurgitation is an uncommon complication. Antibiotic prophylaxis to prevent endocarditis is recommended for those with typical auscultatory findings, including a systolic murmur. Beta-blocker therapy is reserved for symptoms, including those related to arrhythmias.