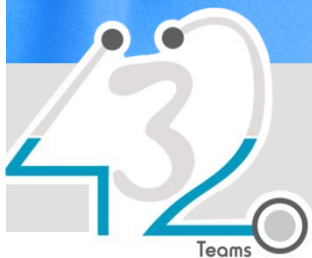


# MEDICINE

432 Team

3

## Valvular Heart Disease



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COLOR GUIDE: • Females' Notes • Males' Notes • Important • Additional

# Objectives

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1. Recognize common VHD
2. Recognize common causes of VHD
3. Know pathophysiology of different VHD
4. Diagnosis of VHD
5. Know management of common VHD

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## General Notes:

**Heart Sounds:** A summary and additional info can be found in our physiology team’s archive

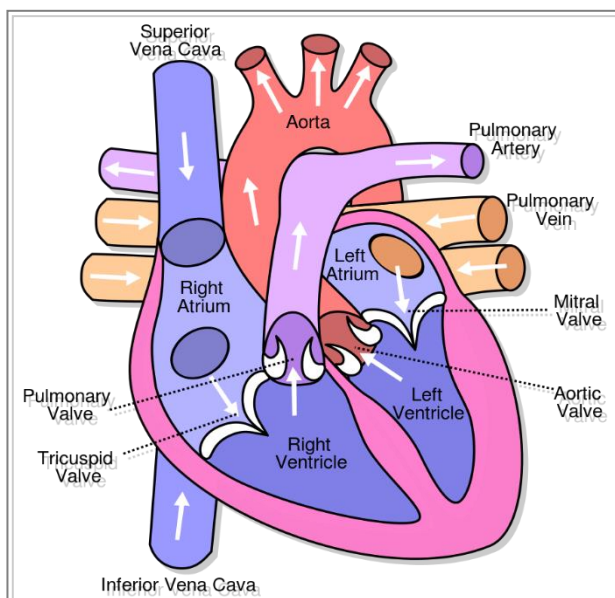
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Due to the lectures being very vague on tricuspid and pulmonary valve disease, all of their information are taken from valvular diseases chapters from Davidson’s & Kumar’s. On examination (valvular disease):

- Abnormal look (*mitral facies*)
- Abnormal pulse (Atrial fibrillation)
- Abnormal JVP
- Apex beat abnormality
- Sternal or parasternal heave
- Thrill (vibratory sensations caused by the heart and felt on the body surface. Always associated with murmurs)
- *Abnormal heart sound*
- **MURMURS:** Systolic or Diastolic .

### Investigations (valvular disease):

- ECG
- CXR
- Echo cardiology: M mode, 2D, 3D, 4D, TEE, Doppler
- 24 hours monitor for heart rhythm
- MRI
- Cardiac catheterization



The heart has 4 valves, each of which can be affected by either stenosis or regurgitation.

### Review :

<b>Systole</b>	AV/PV – opens
S1-S2	MV/TV – closes
<b>Diastole</b>	AV/PV – closes
S2-S1	MV/TV – opens
<b>Systole</b>	AV/PV – opens-----Aortic Stenosis
S1-S2	MV/TV – closes-----Mitral Regurg
<b>Diastole</b>	AV/PV – closes-----Aortic Regurg
S2-S1	MV/TV – opens-----Mitral Stenosis

**Regurg/ Insuff** – leaking (*backflow*) of blood across a *closed* valve

**Stenosis** – Obstruction of (*forward*) flow across an *opened* valve

Normal area for the Aortic valve is 2-3 cm<sup>2</sup>

Critical narrowing < 0.7 cm<sup>2</sup>

Normal area for the Mitral valve is 4-6 cm<sup>2</sup>

Critical narrowing < 1 cm<sup>2</sup>

Aortic Regurgitation AR is the same as Aortic Insufficiency AI

Valve lesions from Pressure overload: AS & MS

Valve lesions from Volume overload: MR & AR

## A) MITRAL VALVE:

### Normal Anatomy & Physiology:

A bicuspid valve with a typical area of 4-6 cm<sup>2</sup> positioned between the left atrium & ventricle. The opening is surrounded by a fibrous ring known as the mitral valve annulus. The cusps are prevented from prolapsing into the left atrium by the action of tendons attached to the posterior surface of the valve, chordae tendineae, which are attached to the cusps on one end the papillary muscles on the other.

During diastole the mitral valve opens as a result of increased pressure from the left atrium as it fills with blood (preloading). Opening allows passive blood flow into the left ventricle. Diastole ends with atrial contraction, which ejects the final 20% of blood that is transferred from the left atrium to the left ventricle (end diastolic volume). Mitral valve closes at the end of atrial contraction to prevent a reversal of blood flow.

### 1- Mitral Stenosis: < more common in women!

**Causes:** Usually a result of rheumatic heart disease (secondary to rheumatic fever by  $\beta$ -hemolytic streptococcus). Other causes include:

- Congenital Mitral Stenosis <rare>
- Systemic Lupus Erythematosus
- Rheumatoid Arthritis
- Atrial Myxoma
- Malignant Carcinoid
- Bacterial Endocarditis
- Lutembacher's syndrome (Acquired Mitral stenosis + ASD)

**Pathophysiology:** Progressive *fibrosis, calcification or fusion of the valves restricts blood flow* to the left ventricle leading to a *rise in left atrial pressure* which in turn causes *left atrial dilation and hypertrophy*. Consequently, *pulmonary venous and arterial pressure also increase resulting in congestion (cause of exertion dyspnea)* in addition to right heart pressure. Progressive dilation commonly causes atrial fibrillation.



A stenotic mitral valve. Notice the thickening of the leaflets & atrial hypertrophy

### Signs & Symptoms:

Symptoms usually start when the valve's surface area is less than 2cm<sup>2</sup>. The hallmark sign is dyspnea on exertion.

#### **Note:**

Gradual rise in atrial pressure results in pulmonary hypertension which protects the patient from pulmonary edema

The increased left atrial pressure causes pulmonary hypertension & atrial fibrillation. Other signs include:

- Fatigue
- Orthopnea & Paroxysmal nocturnal dyspnea
- Mitral facies
- Pulmonary edema (develops when there's a sudden increase in flow rate across a markedly narrowed mitral orifice)
- Palpitations (due to arrhythmias)
- Hemoptysis (due to rupture of thin dilated bronchial veins)
- Peripheral edema
- productive cough of blood tinged or frothy sputum



Rosy cheeks while the rest of the face has a blue-ish tinge due to cyanosis. A sign of mitral stenosis

### Changes caused by mitral stenosis:

- Cusps thicken
- Commissures fuse together
- Chordae tendinae thicken & shorten
- Calcium deposition

### Possible complications:

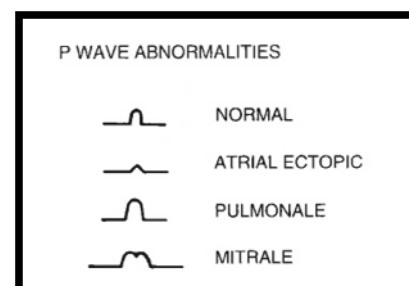
- **Atrial fibrillation** < result in irregular pulse
- Lung congestion
- Blood clots with systemic embolization
- Pulmonary hypertension
- Congestive heart failure

### Examination findings:

- Palpitations (Tapping impulse is felt parasternally on the left side)
- Right ventricular lift
- **Auscultation:**
  - **Loud S1** though it doesn't occur in calcific mitral stenosis
  - **Loud P2**
  - **Opening snap** (all three due to pliable valve)
  - **Mid-Diastolic rumbling murmur**
  - No S3

#### Note:

The second heart sound has two components, A2 & P2.



## Investigations:

- **X-ray:** enlarged left atrium
- **ECG:** bifid P-wave with atrial delay
- **Echo 2D/ Doppler: TEST OF CHOICE**
  - Transthoracic: determine atrial & ventricular size
  - Transesophageal: detect presence of thrombus. Also used to consider surgical or percutaneous intervention
  - Doppler: estimate pulmonary artery pressure

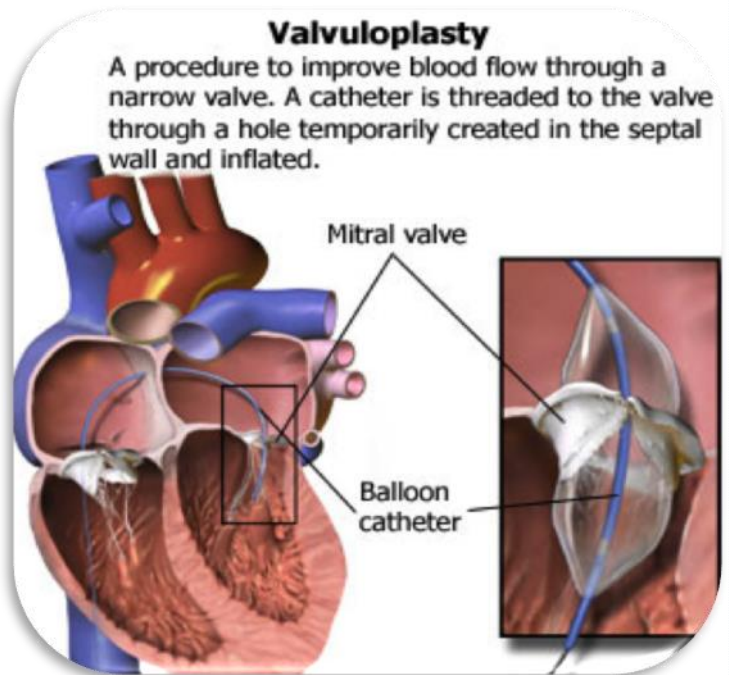
## Management:

- **Medical:**
  - Diuretics < for the congestion
  - Digitalis, Beta blocker and ca channel blockers < AF to control rate
  - Antiarrhythmics < for the AF
  - Anticoagulants < for the AF and clots
  - Antibiotics
- **Surgical:**
  - Trans septal balloon valvotomy (Valvuloplasty) “Ideal for Patients with pliable valves where there is little involvement of the subvalvular apparatus and pt with minimal mitral regurgitation”.
  - Closed valvotomy “ pt with mobile, non calcified, non regurgitant mitral valve “
  - Open valvotomy “ preferred to closed valvotomy , bypass is required “
  - Mitral valve replacement
  - Percutaneous transvenous mitral commissurotomy (ptmc)
  - Surgical commissurotomy
  - Mitral valve replacement “ when mitral regurgitation is present and it valve is badly diseased or calcified “

## 2- Mitral Regurgitation:

**Causes:** the most common causes of are mitral valve prolapse (see page 9), rheumatic heart disease50%. Other causes include:

- *Ischemic heart diseases*
- Cardiomyopathy (dilated , hypertrophic)



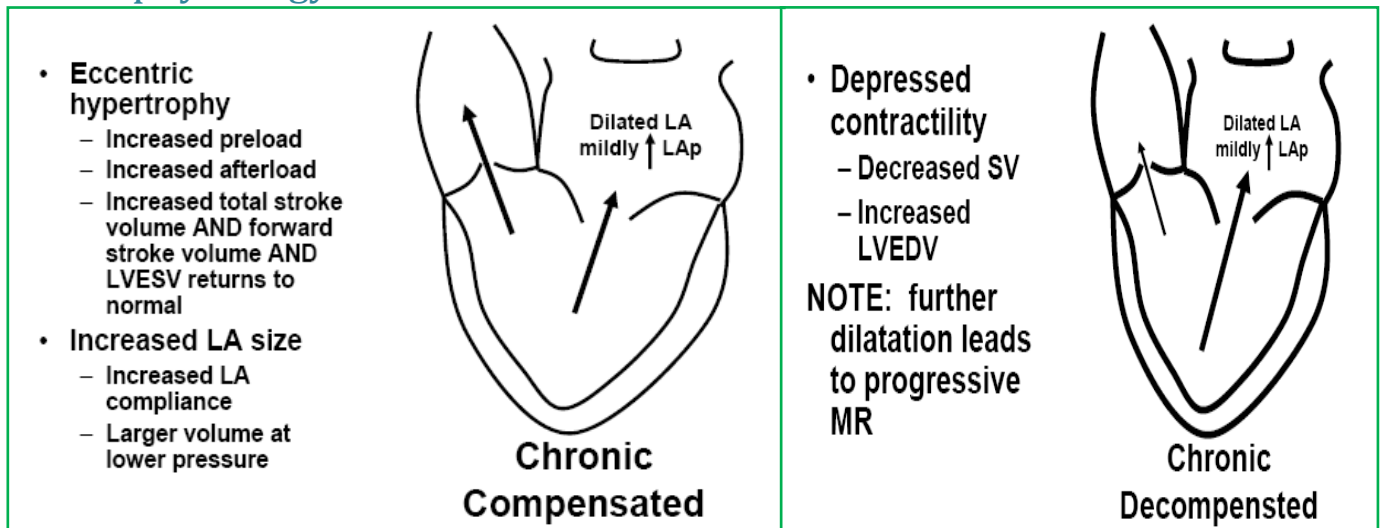
### Note:

~~~~~  
**Marfan's Syndrome:**  
*Genetic connective tissue disorder. Symptoms include skeletal, cardiovascular & visual abnormalities.*  
 ~~~~~



- *Hypertensive heart disease*
- *Infective endocarditis*
- Myocarditis
- Connective tissue disorders (SLE)
- Collagen abnormalities (*Marfan's syndrome*)
- Drugs: Centrally acting appetite suppressant eg: fenfluramine. Dopamine agonist eg: cabergoline.

## Pathophysiology:



- *Atypical chest pain* is the most common symptom
- *Palpitations* may be experienced because of the abnormal ventricular contraction or because of the atrial and ventricular arrhythmias
- *Fatigue*
- *Orthopnea & Paroxysmal nocturnal dyspnea*
- Sudden cardiac death due to fatal ventricular arrhythmias is a very rare but recognized complication
- Auscultation:
  - The most common sign is a *mid-systolic click*
  - Soft first heart sound and split S2
  - Late systolic murmur due to some regurgitation
  - Laterally displaced (forceful) diffuse apex beat and a systolic thrill
  - *Pansystolic murmur*

- *Prominent third heart sound "S3 Gallop"*

## Examination:

- **Acute Regurgitation:**
  - Rales (lung crackles due to pulmonary congestion)
  - Sitting upright
  - Murmurs
- **Chronic:**
  - Diffuse, tapping apical impulse
  - Pulmonary findings
  - **S3**, may be palpable

## Investigations:

- **X-Ray:** left atrial & ventricular enlargement, valve calcification
- **ECG:** Atrial delay (bifid P-wave), ventricular hypertrophy (tall R-waves)
- **Cardiac Catheterization:** prominent left atrial pressure
- **Echo 2D/ Doppler: TEST OF CHOICE**

**Management:** - If the pt is asymptomatic you manage by follow up and prophylaxis against endocarditis. If it progresses then surgical intervention is recommended if it got more advanced or if surgery is contraindicated then we manage with medication.

- **Medication**
  - Beta blockers to relieve atypical chest pain & palpitations
  - Anticoagulants to prevent thromboembolism
  - Vasodilators : ACE inhibitors
  - Diuretics
- **Surgical :**  
Progressive cardiac enlargement indicates early surgical intervention by valve replacement or repair

**\*Mitral Valve Prolapse (MVP):** Also known as a *floppy valve (Barlow's syndrome)*, caused by abnormally large valve leaflets, annulus or chordae or abnormal papillary contractions. Also associated with:

- Myxomatous valve degeneration
- Thyrotoxicosis
- Rheumatic heart disease
- Ischemic heart disease
- Marfan's Syndrome

**\*Signs & Symptoms of MVP:**

- Atypical chest pain "left sub-mammary stabbing pain"
- Palpitations
- *Mid systolic click* is the most common sign (caused by the sudden valve prolapse & tensing of the chordae during systole)
- High risk of thromboembolism
- The most common sign is a mid-systolic click.
- Produced by the sudden prolapse of the valve and the tensing of the chordae tendineae that occurs during systole.
- A late systolic murmur owing to some regurgitation

**\*Treatment of MVP:**

- Beta-blockade is effective for the treatment of the atypical chest pain and palpitations.
- Mitral valve prolapse associated with significant mitral regurgitation and atrial fibrillation, anticoagulation is advised to prevent thromboembolism.
- Mitral valve prolapse associated with severe mitral regurgitation has a risk of sudden cardiac death.

## B) AORTIC VALVE

### Normal Anatomy & Physiology:

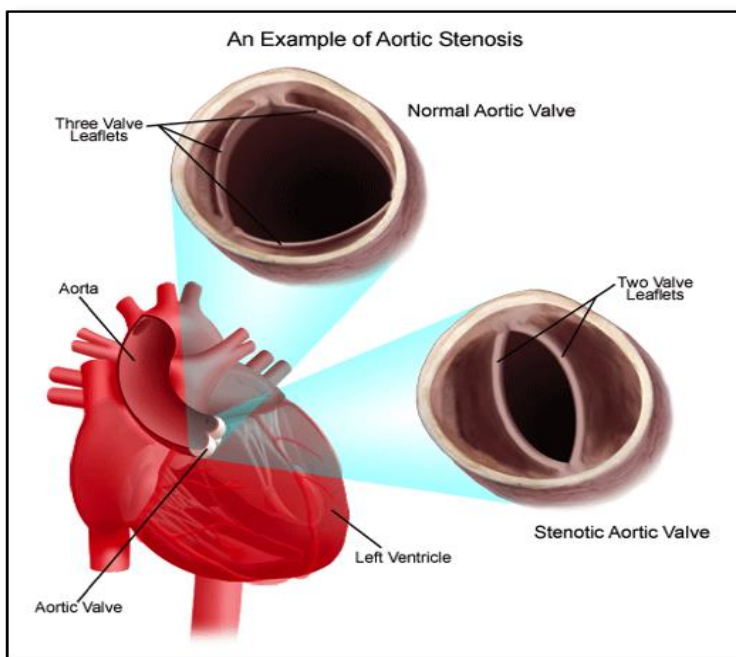
It has three semi-lunar cusps valve that lies between the left ventricle and the ascending aorta. Its opening is supported by a fibrous ring called the aortic annulus.

During ventricular systole, when the pressure rises above the aortic pressure, the valve opens, thereby allowing blood to flow into the aorta. When ventricular pressure falls, the valve closes due to the aortic pressure.

# 1- Aortic Stenosis:

## Causes:

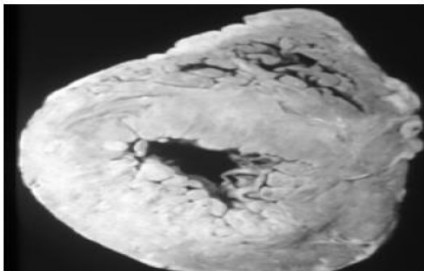
- **Infants and children (*congenital defects*):**
  - Congenital aortic stenosis “Develops progressively due to the turbulence of the blood flow”.
  - Congenital supra-avalvular aortic stenosis
  - Congenital subvalvular aortic stenosis
- **Young adults to middle aged:**
  - Calcification & fibrosis of a congenitally bicuspid aortic valve
  - Rheumatic aortic stenosis
- **Middle aged to elderly:**
  - Degenerative aortic stenosis
  - Rheumatic aortic stenosis
  - Calcification of a congenitally bicuspid aortic valve
- **Calcific Aortic Valvular Disease:**
  - *Commonest cause*
  - Elderly
  - Inflammatory process
  - Risks include elevated LDL, hypertension, diabetes & smoking
- **Bicuspid aortic valve:**
  - *Commonest form of congenital heart disease*
  - Associated with: aortic coarctation, root dilation & aortic dissection
- **Rheumatic stenosis:**
  - Progressive fusion, thickening and calcification of the aortic valve
  - Aortic valve is involved in 30-40% of rheumatic heart diseases
- **Other causes of valvular stenosis:**
  - Chronic kidney disease
  - Paget’s disease of bone
  - Radiation exposure
  - Familial hypercholesteremia (homozygous)
- **Obstruction to left ventricular emptying:**
  - **Supra-valvular stenosis:** congenital fibrous diaphragm above the aortic valve
  - **Sub-valvular stenosis:** congenital fibrous diaphragm below the aortic valve
  - **Hypertrophic cardiomyopathy:** septal muscle hypertrophy obstructing left ventricular outflow



Pathophysiology:

**Ventricular Compensation**

- **Concentric hypertrophy**
  - Reduces wall stress
  - Reduces ventricular compliance
  - LVEDp increases
  - LAp increases



Symptoms:

Patients are usually asymptomatic until the aortic orifice is reduced to one third of its normal size. However, they deteriorate rapidly once symptoms appear. Death may occur within 3 years without surgical intervention.

- **Mild to moderate stenosis:** Asymptomatic
- **Moderate to severe:**
  - **Angina** : imbalance between the supply and demand
    - Elevated LVEDp decreases perfusion pressure
    - Myocardial hypertrophy increases demand
  - **Syncope on exertion:** Inability to increase cardiac output and meet reduced SVR demands
  - **Congestive heart failure** : Elevated LVEDp = Elevated LAp = Pulmonary venous congestion

**Note:**  
 Median survival rate :  
 Angina : 5 years  
 Syncope : 3 years  
 CHF : 2 years

## Examination:

- Slow rising carotid pulse
- Pulsus Parvus et Tardus: on palpation the pulse is weak & late
- Systolic thrill on palpation
- Narrow pulse pressure
- **Auscultation:**
  - Prominent S4
  - Soft S2
  - Systolic ejection murmur
  - Lung crackles due to pulmonary congestion
  - Sustained Bifid LV impulse “LVH”

### Note:

.....  
 Aortic stenosis can be diagnosed only by auscultating the carotid which is characterized by LOW VOLUME & SLOW RISING PULSE  
 .....

## Investigations:

- **X-Ray:**
  - A relatively small heart with dilated ascending aorta due to turbulent blood flow above the stenosed valve (post-stenotic dilation)
  - Calcified aortic valve
- **ECG:**
  - LV hypertrophy
  - LA delay
  - LV ‘strain pattern’ (depressed ST segment & T wave inversion in leads facing the LV)
  - Ventricular arrhythmias may be recorded
- **Echo 2D/ Doppler: TEST OF CHOICE**
  - Calcified, thickened & immobile valve cusps
  - LV hypertrophy
  - Assesses the severity
  - Detect regurgitations
- **Cardiac catheterization:**
  - helpful, confirmatory, needed if the pt is older – look at the coronaries
  - Identify associated CAD
  - Measure gradient between LV & aorta
- **CMR & CT:** assessing aorta for aneurysm, dilation, dissection & coarctation. Rarely necessary

**Management:** Medication for treating the symptoms not the cause!

- **Asymptomatic:** Patients should be under regular review for assessment of symptoms and echocardiography
- **Symptomatic:** Aortic valve replacement, either Bio-prosthetic or Mechanical AVR

## 2- Aortic Regurgitation:

### Causes:

- **Acute:**
  - Acute rheumatic fever
  - Infective endocarditis
  - Dissection of the aorta
  - Ruptured sinus of Valsalva aneurysm
  - Failure of prosthetic heart valve
- **Chronic:**
  - Rheumatic heart disease
  - Syphilis Arthritides
  - Reiter's syndrome
  - Ankylosing spondylitis
  - Rheumatoid arthritis
  - Hypertension (severe)
  - Bicuspid aortic valve
  - Aortic endocarditis
  - Marfan's syndrome
  - Osteogenesis imperfecta

### Pathophysiology:

- **Widened pulse pressure**
  - Stroke volume increased (high SBP)
  - Regurgitant volume increased (low DBP)
- **Imbalance between myocardial supply and demand**
  - Decreased DBP = decreased perfusion pressure = decreased supply
  - Increased LV size (and thus wall stress) = increased demand

### Symptoms:

Patients are **mostly asymptomatic**. Significant symptoms occur later in the disease stage and might not develop until left ventricular failure occurs. **Sometimes the first noticeable symptom in paroxysmal nocturnal dyspnea.**

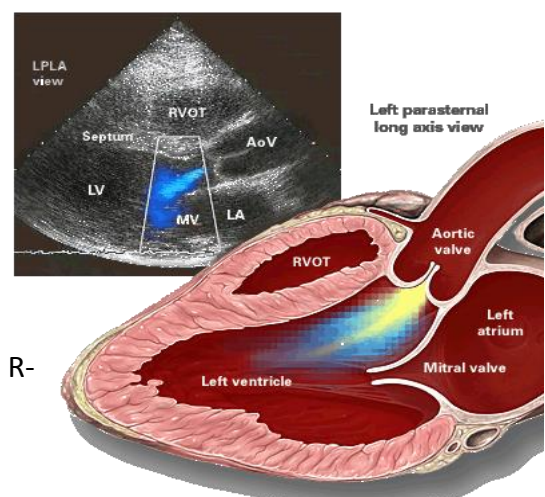
- Pulmonary venous congestion : Dyspnea on exertion
- Inadequate cardiac output : fatigue and diminished exercise tolerance

## Examination:

- **Blood pressure:** **Wide pulse pressure**
- **Pulse:** Sinus rhythm, hyper-dynamic LV apical impulse, **collapsing** & bounding pulses
- **Apex beat:** **Displaced, diffuse, forceful**
- **Murmurs:**
  - **High pitched early diastolic** decrescendo blowing murmur
  - **Ejection systolic** at base and into neck
  - **Mid-diastolic rumble at apex** (**Austin flint murmurs**) this is due to vibration of the anterior leaflet of the mitral valve as it is buffeted simultaneously by the blood jets from the LA and Aorta.
  - S3 & S4 Gallop
- **Other signs:**
  - **Pulsus Bisferiens:** on palpation of the pulse, a double peak per cardiac cycle can be appreciated, also known as *biphasic pulse*
  - **Quincke's:** Capillary pulsation in the nail beds
  - **De Musset's:** head nodding with each heart beat
  - **Duroziez's:** murmur occurring when the femoral artery is auscultated and pressure is applied distally (positive, severe aortic regurgitation)
  - **Pistol shot femorals:** sharp bangs with each heart beat heard on auscultation over the femoral artery

## Investigations:

- **X-ray:**
  - **LV enlargement**
  - Ascending Aorta dilation
  - Calcified aorta (syphilis)
  - **Calcified valves** (valvular disease)
- **ECG:** evidence of **ventricular hypertrophy** (Tall R-wave, deep inverted T-waves, deep S-wave)
- **Echo 2D/ Doppler: TEST OF CHOICE**
  - Dilated LV
  - Hyperdynamic LV
  - **Doppler detects regurgitation**
  - Fluttering anterior mitral leaflet
- **Cardiac Catheterization:** required to assess CAD in elderly before surgery
  - Aortic regurgitation
  - Dilated LV
  - Dilated aortic root





## Management:

If there is an underlying cause (syphilis, aortitis, endocarditis, etc.), don't forget to treat it

- **Asymptomatic:**
  - Routine checkups with echo
  - Beta blockers
  - Vasdilator: Nifedipine & ACE inhibitors
  - Diuretics
  - Antibiotic prophylaxis for endocarditis
  - Surgery is indicated when:
    - LV ejection fraction is  $\leq 50\%$
    - LV dilation (end diastolic dimension  $>70\text{mm}$  or systolic dimension  $>50\text{mm}$ )
- **Symptomatic:**
  - Valve replacement surgery

### Note:

~~~~~  
 Tissue valves are preferred in elderly and when anticoagulants must be avoided, but they are contraindicated in children and young adults because of the rapid calcification and degeneration of the valves.  
 ~~~~~

The females' doctor said it is not important to know much about "Tricuspid and Pulmonary" valves diseases!  
 The following **ORANGE** notes are taken for Davidson & Kumar books!!!

## C) TRICUSPID VALVE

### Normal Anatomy:

Lies between the right atrium & ventricle. Made up of the 3 valve leaflets, the annulus, the supporting chordae tendineae, and the papillary muscles.

### 1- Tricuspid Stenosis:

#### Causes:

Usually due to rheumatic heart disease, but also frequently associated with mitral and aortic valve diseases. It is also seen in **carcinoid syndrome and Endocarditis in IV drug abusers**. Note: rarely alone. Usually occurs in conjunction with other valve lesions

#### Pathophysiology:

Tricuspid stenosis results in a compensatory increase in atrial pressure to maintain normal cardiac output. This may cause systemic venous congestion resulting in hepatomegaly, ascites & edema

## Symptoms:

Most symptoms are related to the associated left sided valve lesions. Right sided symptoms include: abdominal pain (hepatomegaly), swelling (Ascites), & dyspnea

## Examination:

- Hepatomegaly
- Pre-systolic pulsation felt over the liver
- Edema
- Auscultation:
  - Mid diastolic rumble
  - Tricuspid opening snap

## Investigations:

- **X-ray:** may see prominent right atrial bulge
- **ECG:** tall p-wave
- **Echo:** thickened and immobile valve

## Treatment:

Managed with diuretics and salt restriction. However, valve replacement is usually necessary.

## 2- Tricuspid Regurgitation: The commonest right-side disease

### Causes:

**Secondary to other diseases (functional regurgitation) usually occurs in cases of atrial dilation: cor pulmonale, myocardial infarction & pulmonary hypertension. Primary, affecting the valve (organic regurgitation), may occur in rheumatic heart disease, endocarditis, carcinoid syndrome & congenital anomalies such as Ebstein's anomaly**

### Symptoms:

- High right atrial pressure
- High systemic venous pressure
- Right side failure signs:
  - Ascites
  - Hepatomegaly
  - Edema

### Examination:

- Liver is palpable, right ventricle impulse felt on left sternal edge
- Pan-systolic murmur is heard, best on left sternal edge
- Atrial fibrillation is common

## Investigations:

- **Echo:** thick valve and dilated left ventricle

## Management:

Secondary regurgitation usually disappears with managing the underlying cause. Primary regurgitation patients with normal pulmonary artery pressure can tolerate it well. However, *severe regurgitation requires valve replacement surgery. Worth to note that a lot of patients do eventually require surgery.*

**Right-sided valvular lesions change in intensity with inspiration**

## D) PULMONARY VALVE:

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### Normal Anatomy:

Three cusped, semi-lunar valve between the right ventricle and pulmonary artery.

### 1- Pulmonary Stenosis:

#### Cause:

**Usually due to a congenital defect (pediatric).** But can occur (rarely) due to rheumatic disease or carcinoid syndrome. *Multiple congenital pulmonary arterial stenosis are usually due to rubella infection during pregnancy.* May be valvular, subvalvular or supralvalvular.

#### Symptoms:

- Mild cases may be asymptomatic
- Right ventricular hypertrophy due to flow obstruction, which in turn causes right atrial hypertrophy
- Fatigue
- Syncope
- Right sided heart failure signs

#### Examination:

- Harsh mid-systolic ejection murmur, usually associated with thrill
- If moderately severe, prominent S4 and jugular venous pressure are present

#### Investigations:

- **X-ray:** Prominent pulmonary artery (post stenotic dilation)
- **Doppler echocardiogram is the investigation of choice**
- **ECG:** signs of right atrial and right ventricle hypertrophy

**Treatment:**

- Mild to moderate cases usually do not progress or require treatment
- Severe stenosis requires pulmonary valvotomy

**2- Pulmonary regurgitation:**

Most common lesion of the pulmonary valve. Usually associated with pulmonary hypertension. Shows decrecendo diastolic murmur. *Usually has no symptoms, treatment is rarely necessary*

**IMPORTANT NOTES FROM EXTERNAL RESOURCES**

Notes	
Davidson's	p616-625
Kumar & Clark's	p740-750

**SUMMARY**

	Signs	Finding
<b>Mitral Stenosis</b>	Hemoptysis Fatigue Orthopnea & PND Mitral facies	Loud S1 Loud P2  Mid-Diastolic rumbling murmur  Opening snap
<b>Mitral Regurgitation</b>	Atypical chest pain Palpitations Fatigue  Orthopnea & Paroxysmal nocturnal dyspnea	Pansystolic murmur  S3 Gallop  Rales mid-systolic click
<b>Aortic Stenosis</b>	Angina Syncope on exertion Congestive heart failure	Slow rising carotid pulse & radiate to the carotids Systolic ejection murmur  Prominent S4
<b>Aortic Regurgitation</b>	Dyspnea on exertion Fatigue	Mid-diastolic rumble at apex "radiate to the apex " High pitched early diastolic decrescendo S3 & S4 Gallop

## Questions

- 1) Which of the following is *not* a finding of mitral stenosis?
  - a. Prominent S3
  - b. Loud P2
  - c. Right ventricular lift
  - d. Diastolic rumble
  
- 2) Chronic mitral regurgitation causes \_\_\_\_\_, while acute regurgitation results in \_\_\_\_\_?
  - a. Increased atrial pressure - atrial dilation
  - b. Pulmonary edema - little increase in atrial pressure
  - c. Little pressure increase - atrial dilation
  - d. atrial dilation – increased atrial pressure
  
- 3) Aortic valve stenosis shows all of the following except?
  - a. Loud S2
  - b. Prominent S4
  - c. Systolic ejection
  - d. Systolic thrill
  
- 4) How is the age helpful in determining the cause of AS?  
Patient who is younger than 30 “congenital”  
30-70 “rheumatic or bicuspid”  
Elder than 70 “calcification”
  
- 5) What are the common non-auscultatory findings in MS?  
Malar flush, elevated JVP, peripheral cyanosis and all the signs increased with pregnancy and exercise.
  
- 6) What are the common physical findings in chronic severe MR?  
Loud holo-systolic murmur, S3 gallop, rales, edema, liver distention
  
- 7) Which is the most common VHD ( Valvular heart disease):
  - a. Mitral stenosis
  - b. Mitral regurgitation
  - c. Aortic stenosis
  - d. Tricuspid regurgitation

8) A 52-year-old man attended the out-patient clinic for review having been lost to follow-up for several years. He had a past history of aortic valve endocarditis 10 years earlier, which had been successfully treated with antibiotics. He was asymptomatic but his family doctor had noted a murmur on routine examination. On examination his pulse was 60 beats per minute and his blood pressure was 155/55mmHg. Auscultation revealed systolic and diastolic murmurs at the left sternal border. There was no clinical evidence of heart failure. An echocardiogram revealed a left ventricular end-systolic dimension of 55mm, an end-diastolic dimension of 65mm, a left ventricular ejection fraction of 53% and an aortic root diameter 46mm. The aortic valve was bicuspid with severe regurgitation and a peak trans-valvular pressure gradient of 30 mmHg. What is the most appropriate treatment at this stage?

- a. aortic valve and root replacement
- b. aortic valve replacement
- c. bisoprolol
- d. no intervention required
- e. ramipril

9) A 28-year-old man with Down's syndrome was seen in the Emergency Department complaining of breathlessness. On examination he had pitting oedema of his ankles and clubbing of his fingers and toes. The JVP was elevated to +7cm. He had a parasternal heave, a widely split second heart sound and a grade 2 systolic murmur loudest in the pulmonary area and at the left sternal border. A transthoracic echocardiogram suggested an atrio-ventricular septal defect but imaging of the right heart was difficult and it was not possible to obtain an estimate of the right heart pressures.

What physical sign is most suggestive of pulmonary vascular disease?

- a. clubbing
- b. elevated JVP
- c. fixed splitting of second heart sound
- d. parasternal heave
- e. peripheral oedema

Source of the last

2Q:<http://www.secardiologia.es/files/institucional/Documento%20cómo%20p reparar%20ESC%20EXAM.pdf>

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**Answers:**

1st Question: a

2nd Question: d

3rd Question: a

7<sup>th</sup> Question: c

8<sup>th</sup> Question: E

9<sup>th</sup> Question: A