Cardiovascular system

Index

Mitral stenosis (MS)

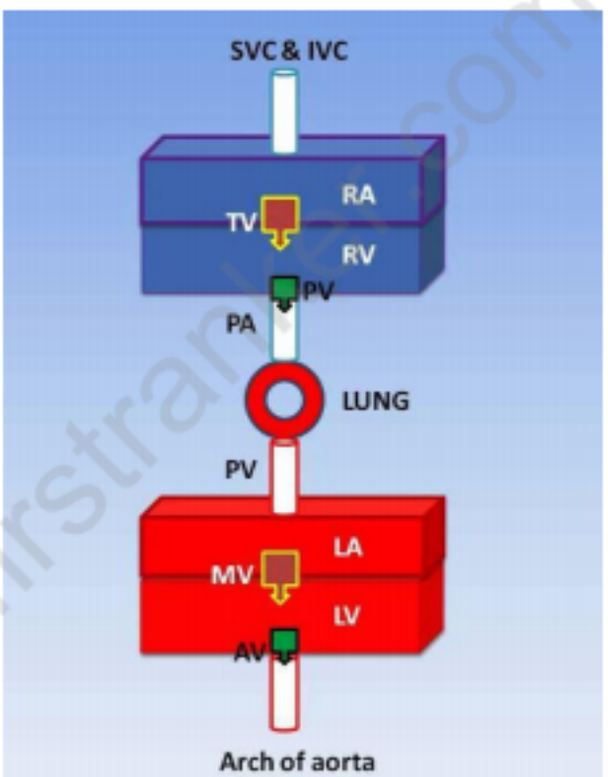
MS is defined as incomplete opening of mitral valve.

Normal orifice of mitral valve: 2-4 sq.cm.

Causes of mitral stenosis:

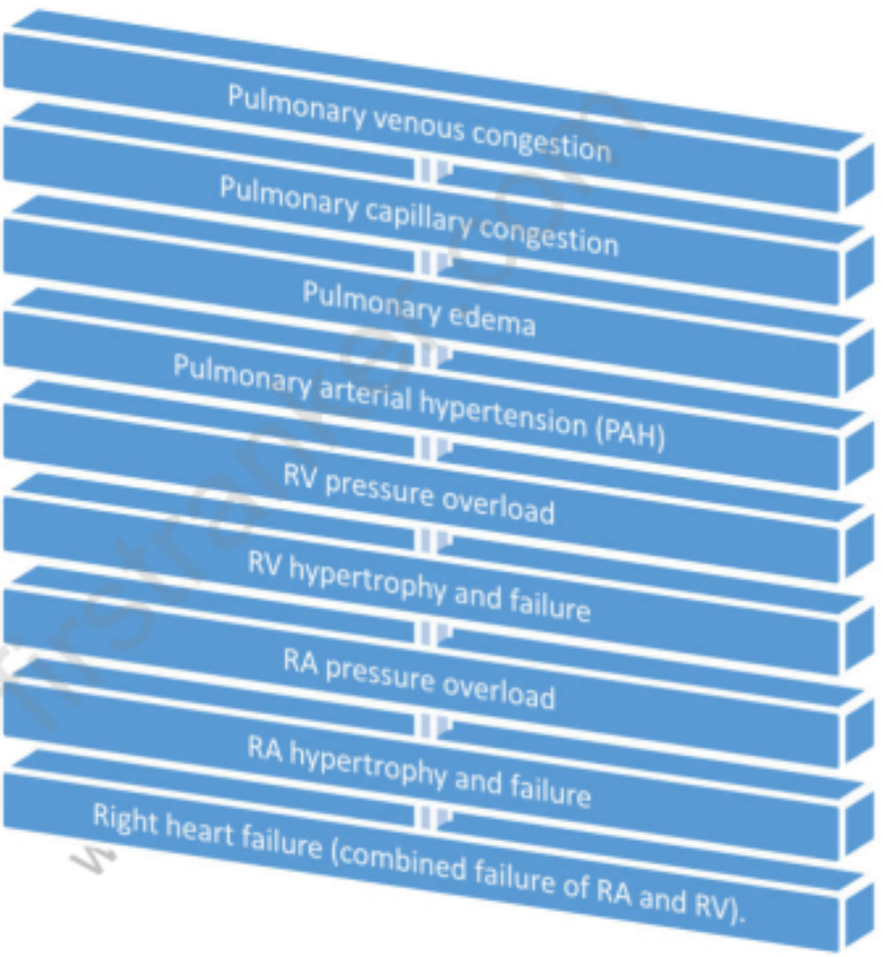
1. The most common cause of MS in India is chronic rheumatic heart disease.
2. Other rare causes include:
   a. Congenital MS,
   b. Hurler's syndrome.

Pathophysiology of MS:



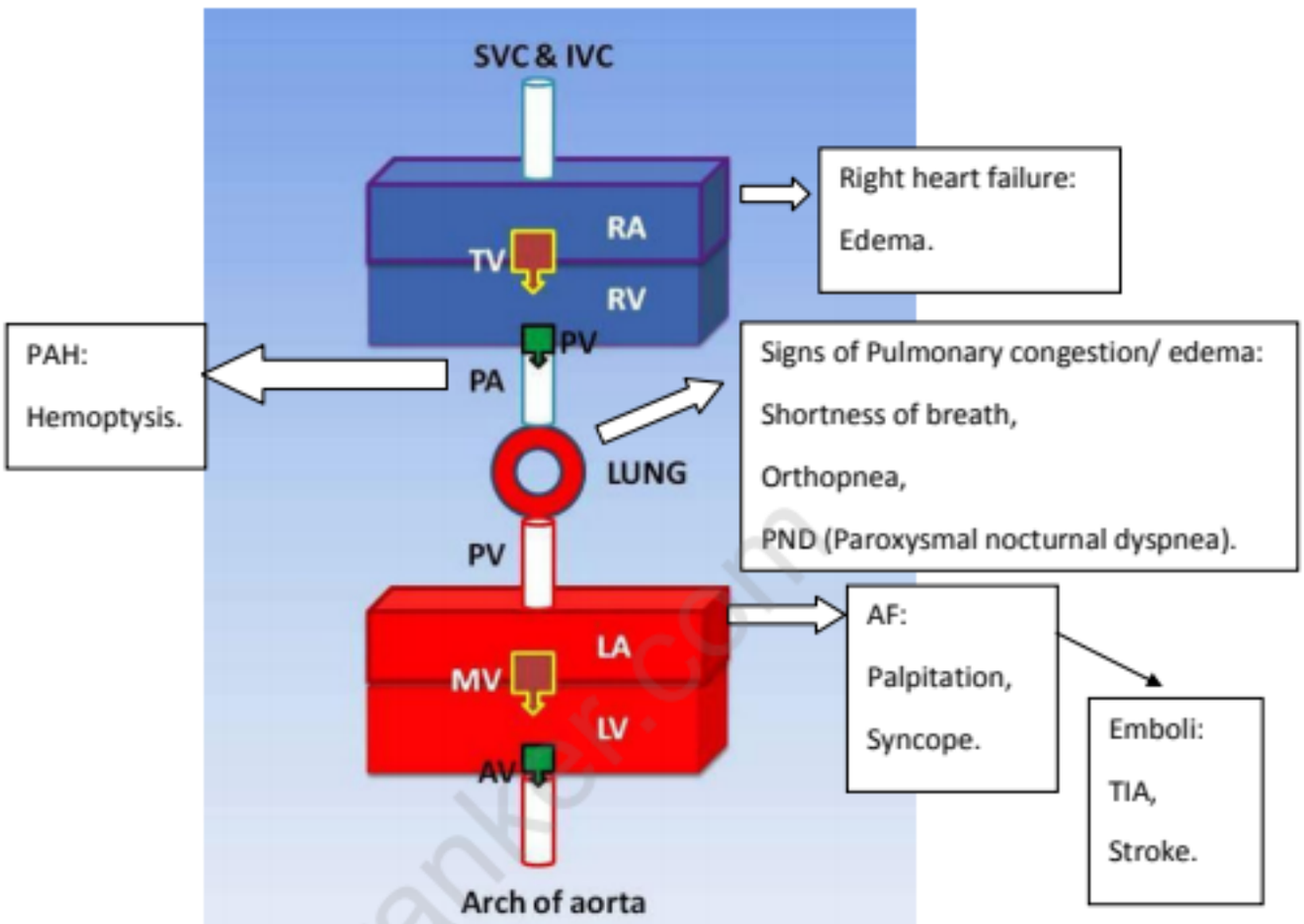
- As the mitral valve is calcified, fibrosed, deformed and stenosed in MS; it opens incompletely; which results in increased LA pressure.
- This chronically increased LA pressure eventually results in LA dilation and hypertrophy.
- As the LA is dilated, the fibres of LA are stretched and scarred; which results in irregular spontaneous impulse generation. This may eventually lead to ***atrial fibrillation (AF)***. The symptoms of AF are palpitation and syncope.

- In a patient with AF, the regular impulses produced by the sinus node for a normal heartbeat are overwhelmed by rapid electrical discharges produced in the LA. So, although the electrical impulses of AF occur at a high rate, most of them do not result in a heartbeat. This results in stasis of blood and formation of clot in LA, which may eventually give rise to emboli and stroke.
- As the LA can't go further dilation/ hypertrophy and fails to maintain such a high pressure, it eventually fails. So, a LA failure results.
- The backward effect of LA failure are as follows (serially) [see the picture above]:



- It should be noted that LV does not undergo a pressure overload, so it usually doesn't undergo dilation/ hypertrophy.
- If PAH occurs, due to increased pressure on pulmonary artery, there is formation of anastomotic channels between pulmonary artery and bronchial artery. These bronchopulmonary anastomotic collaterals are weak and often rupture; giving rise to ***hemoptysis***.

Signs and symptoms of MS at a glance



Some rare symptoms of MS:

- Hoarseness of voice/ Ortner's syndrome (due to recurrent laryngeal nerve compression/ palsy by a dilated LA).
- Dysphagia (due to compression of oesophagus by a dilated LA).

Signs:

a. Raised jugular venous pressure (due to RH failure).
b. Edema (due to RH failure).
c. Pulse: Completely irregular pulse (if AF is present).
d. Heart rate: Tachycardia.

Examination of Mitral Area

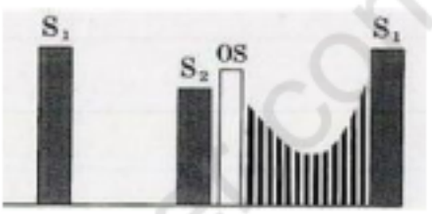
- Apical impulse:
  a. Site: It is normally located in the left 5th intercostal space, about 1 cm. medial to the mid-clavicular line. It is shifted to outwards in late MS (due to RVH).
  b. Character: The apical impulse is **tapping** in character. It signifies accentuated 1st heart sound (S1).
  c. Thrill: Thrill is defined as a palpable murmur produced due to turbulent blood flow, which causes vibration and becomes palpable. Usually the thrill of MS, if present, is diastolic. So it does not coincide with carotid pulse.
  d. Heart sound:
     The S1 is loud, S2 is usually normal.
     Mechanism:
     Mitral valve closes when the ventricular pressure curve crosses above the LA mean pressure. In MS, the LA mean pressure is highly raised above normal; so the ***pressure gradient*** between LA and LV pressure becomes high; so MV closes loudly.
     The S1 may be soft; when the valves are damaged severely and the valve cusps become immobile.
  e. Murmur:
     - Timing: Mid-diastolic.
     - Character: Rumbling.
     - Associated with pre-systolic accentuation best heard over mitral area. May be absent in AF.
       Mechanism: It is produced by LA contraction at the 2nd rapid filling phase of ventricular diastole (which is heard as just before the next ventricular systole).
     - Best heard on left lateral position of the patient.
     - Intensity of murmur can be accentuated by asking the patient to exercise; this murmur is also better heard in left lateral position.

Examination of Pulmonary area

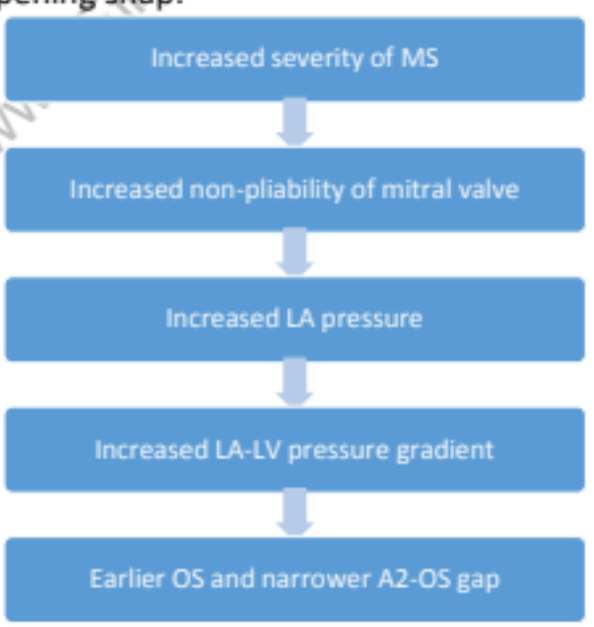
Findings are positive if MS is associated with PAH.

The following findings may be present:

1. Accentuated P2; a palpable P2 may also be present.
   Mechanism:
   The intensity of P2 is dependent on the velocity of blood coursing back towards the right ventricle after ventricular contraction and the suddenness in which this motion is arrested by the closing valve. In patients with PAH, the diastolic pressure within the pulmonary artery is high and therefore the velocity of blood moving toward the tricuspid valve is increased, resulting in an accentuated P2.
2. Murmur:
   It is a mid-systolic murmur (because of the abnormal turbulent blood flow through the normal pulmonary valve just after the isovolumetric contraction phase of ventricular systole).
3. Opening snap (OS):



   It is an abnormal sound produced due to sudden halting of the diastolic descent of mitral valve cusps.
   Mechanism:
   OS results when the valve leaflets are fused, usually from scar tissue, and cannot open fully. Blood attempting to empty from the atrium impacts a *partially opened valve*, creating a sharp sound reflected back toward the cardiac base, at the beginning of mid diastole.
   Significance of opening snap:



4. Other rare findings:
   a. Graham steel murmur (an early diastolic murmur due to functional PR secondary to pulmonary ring dilation caused by long standing PAH) may be found.
   b. Pan-systolic murmur (due to functional TR secondary to tricuspid ring dilation caused by right ventricular dilation) may be found.

Investigation

1. Blood test: Hb/ TC/ DC/ CRP or ESR.
2. Renal function: Urea creatinine Na+ K+.
3. CXR: Not helpful in diagnosis, but may show mitralisation of heart shadow.
4. ECG: May show AF.
5. Echocardiogram:
   a. Confirms the diagnosis.
   b. Confirm presence of rheumatic valvular heart disease.
   c. Can assess severity of MS.
   d. Visualize all cardiac chambers and assess cardiac function.
   e. Will pick up LA clot, if present.

Treatment

It comprises of:

1. Medical management (treatment of complications).
2. Interventional treatment.

Medical Management

Right heart failure (RHF)

1. Diet: Salt and fluid restriction.
2. Diuretics.
3. Daily (regular) monitoring of body weight.

AF

Rhythm control is not considered in most patients of MS with AF, as they have permanent AF which, if cardioverted, will flip back into AF again. So, rate control using the following anti-arrhythmic drugs is preferred:

A. Amiodarone & Anticoagulation therapy (to prevent LA clot formation).
B. Beta blockers.

C. Calcium channel blockers (CCB): Verapamil/ Diltiazem.
D. Digoxin.

Acute pulmonary edema

M. Morphine.

N: Nitrate.

O: Oxygen.

D: Diuretic.

Chronic Left heart failure (LHF)

A. ACE inhibitor/ ARB.
B. Beta blocker.
C. CCB.
D. Diuretic.

Hemoptysis

Pulmonary artery embolization.

Interventional treatment

- The procedure of choice is mitral valve replacement.
- If not feasible/ affordable, then balloon valvoplasty/ valvotomy can be done.

Complications

1. Atrial fibrillation +/ emboli formation.
2. Acute pulmonary edema.
3. Pulmonary arterial hypertension (PAH).
4. Hemoptysis.
5. Right heart failure (RHF).
6. Infective endocarditis (IE).

The causes of mid-diastolic murmur over mitral area

1. Valvular MS.
2. Functional MS: Due to abnormal amount of blood, flowing through a normal mitral orifice.
   Ex.:
   MR,

AR,
VSD.

3. Transient valvulitis of the mitral valve during an episode of acute rheumatic fever (causing transient MS).

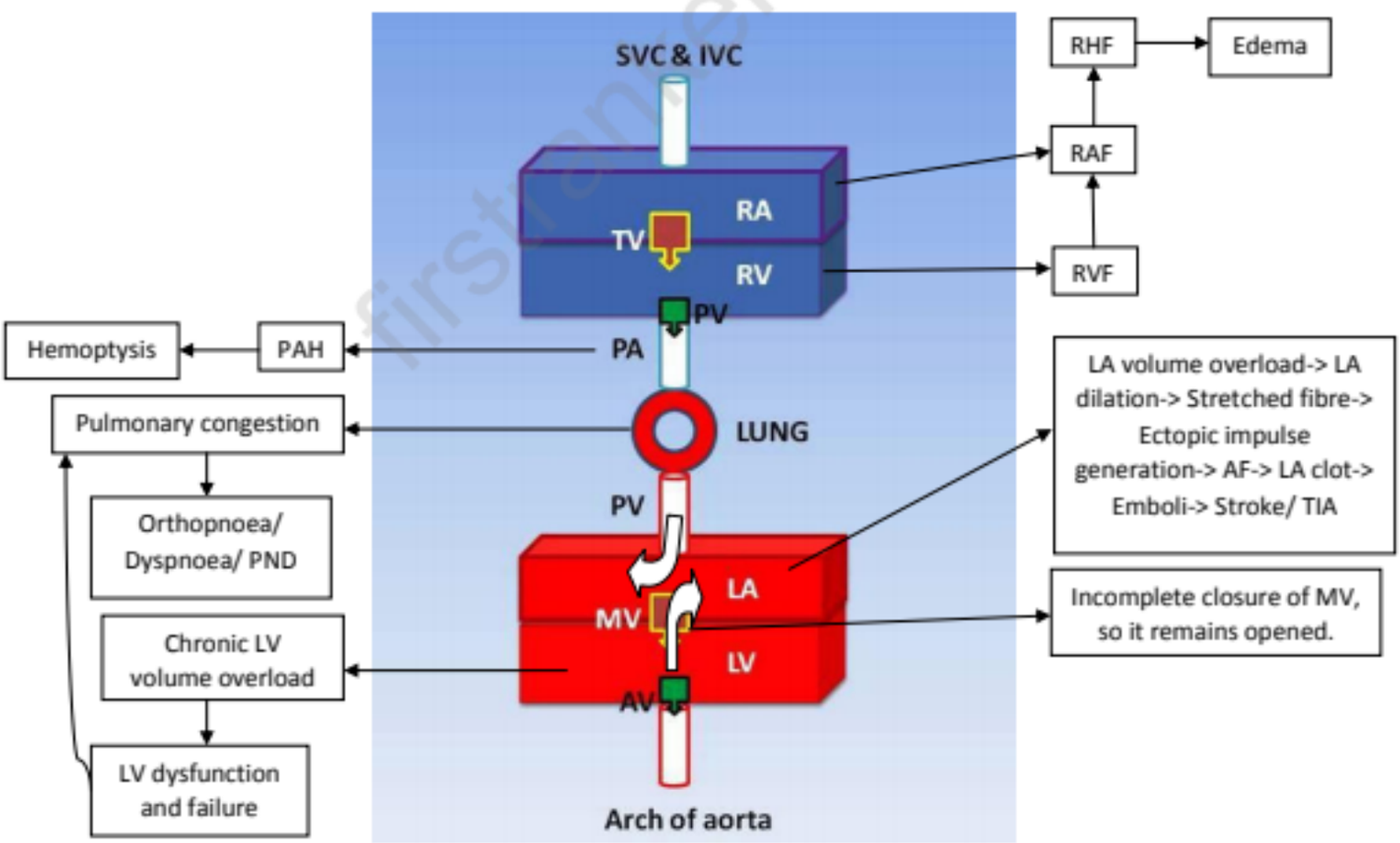
MITRAL REGURGITATION (MR)

MR is defined as incomplete closure of the mitral valve.

Causes:

1. Valve defect:
   a. Valvular heart disease.
   b. Infective endocarditis.
   c. Senile degeneration of mitral valve.
2. Chordi tendon defect:
   AMI.
3. Papillary muscle defect:
   AMI.
4. Mitral valve ring/ annulus defect:
   Functional MR (any condition causing LV dilation, may cause stretching of mitral valve ring).

Pathophysiology:



Symptoms of MR:

1. Due to acute pulmonary edema:
   a. Shortness of breath.
   b. Orthopnoea.
   c. PND.
2. Due to Atrial fibrillation:
   a. Palpitation.
   b. Syncope.
3. Due to Right heart failure:
   Swelling.
4. Due to PAH:
   Hemoptysis.

Signs of MR:

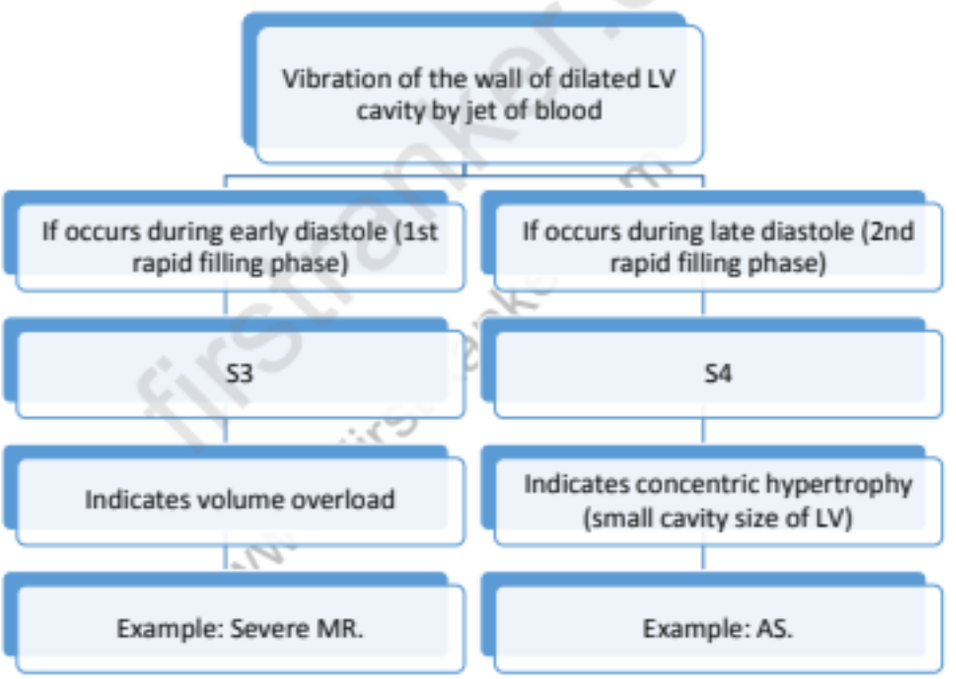
1. Due to RHF:
   a. JVP↑.
   b. Edema.
2. Due to AF:
   a. Tachycardia,
   b. Completely irregular pulse.
3. Due to LV dilation:
   Downwards and outwards shifting of apical impulse.

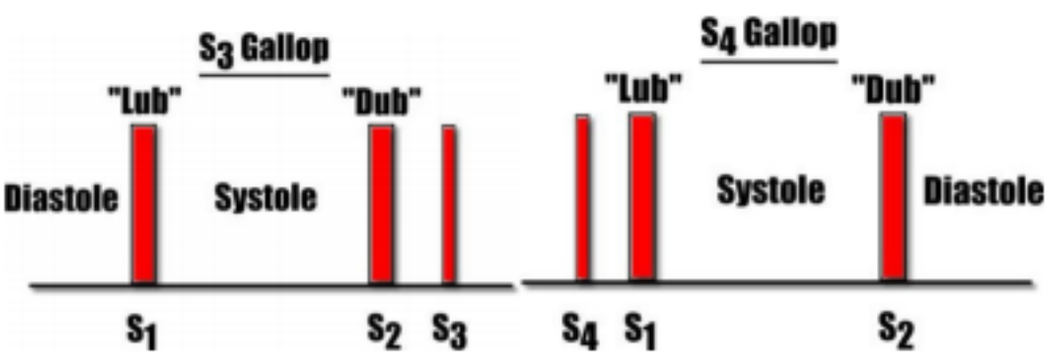
Examination of mitral area

- Forceful ill sustained apical impulse.
  Mechanism:
  Forceful to counteract the volume overload and ill sustained due to narrowing of LV systolic ejection time (as there are 2 outlets of blood during systole of LV).
- Thrill over mitral area may be present, **systolic** in timing.
- S1 is muffled and soft.
  Mechanism:
  Although S1 is due to a combination of mitral and tricuspid valve closure, the mitral valve is the louder aspect. Because the valve closure in mitral regurgitation is incomplete, S1 may be noticeably quieter.

- Murmurs:
  a. Murmur due to MR:
     It is a pan-systolic soft blowing murmur over mitral area, best heard when patient is on left-lateral position. The murmur usually propagates towards the axilla (due to the posteriorly faced right atrium).
  b. Associated murmur:
     A mid-diastolic murmur due to a functional MS may be found.
- Special sounds:
  S3:
  It is an abnormal diastolic sound occurring due to sudden vibration of the wall of dilated LV cavity during the 1st rapid filling phase. It signifies LV dysfunction/ failure. When present in a patient of MR, S3 signifies significant/ severe MR.

[Note:





Investigation

1. Blood: Complete blood count/ TC/ DC/ CRP or ESR.
2. Urine: Urea-creatinine Na+ K+.
3. CXR:
   It may show cardiomegaly or pulmonary edema.
4. ECG: It may show LVH.
5. Echocardiogram:
   a. Confirms the diagnosis.
   b. Confirm presence of rheumatic valvular heart disease.
   c. Can assess severity of MR.
   d. Visualize all cardiac chambers and assess cardiac function.
   e. Will pick up a LA clot, if present.

Treatment

It is divided into 2 groups:

a. Medical management (treatment of complications).
b. Interventional treatment.

Medical management

Acute pulmonary edema

M. Morphine.

N: Nitrate.

O: Oxygen.

D: Diuretic.

AF

Rhythm control is not considered in most patients of MS with AF, as they have permanent AF which, if cardioverted, will flip back into AF again. So, rate control using the following anti-arrhythmic drugs is preferred:

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D. Digoxin.

Right heart failure (RHF)

1. Diet: Salt and fluid restriction.
2. Diuretics.
3. Daily (regular) monitoring of body weight.

Long term treatment of heart failure

A. ACE inhibitor/ ARB.
B. Beta blocker.
C. CCB.
D. Diuretic.

Complications of MR

1. LHF: May be acute or chronic.
2. AF.
3. PAH.
4. RHF.
5. IE.

Causes of pan-systolic murmur over mitral area

Produced at mitral area:

1. Valvular MR.
2. Functional MR.

Not produced in mitral area but transmitted to mitral area:

3. TR.
4. VSD.

Propagation of pan-systolic murmur in MR

1. *Axilla*: As the direction of turbulent blood flow is from LV to LA and the LA is usually located posteriorly in the body, therefore the sound propagates from mitral area towards the axilla near which LA is situated.
2. *Aortic area*: It occurs when the regurgitant stream hits that point of LA wall that is adjacent to the aorta. It is seen when predominantly the *posterior cusp of mitral valve is affected*.

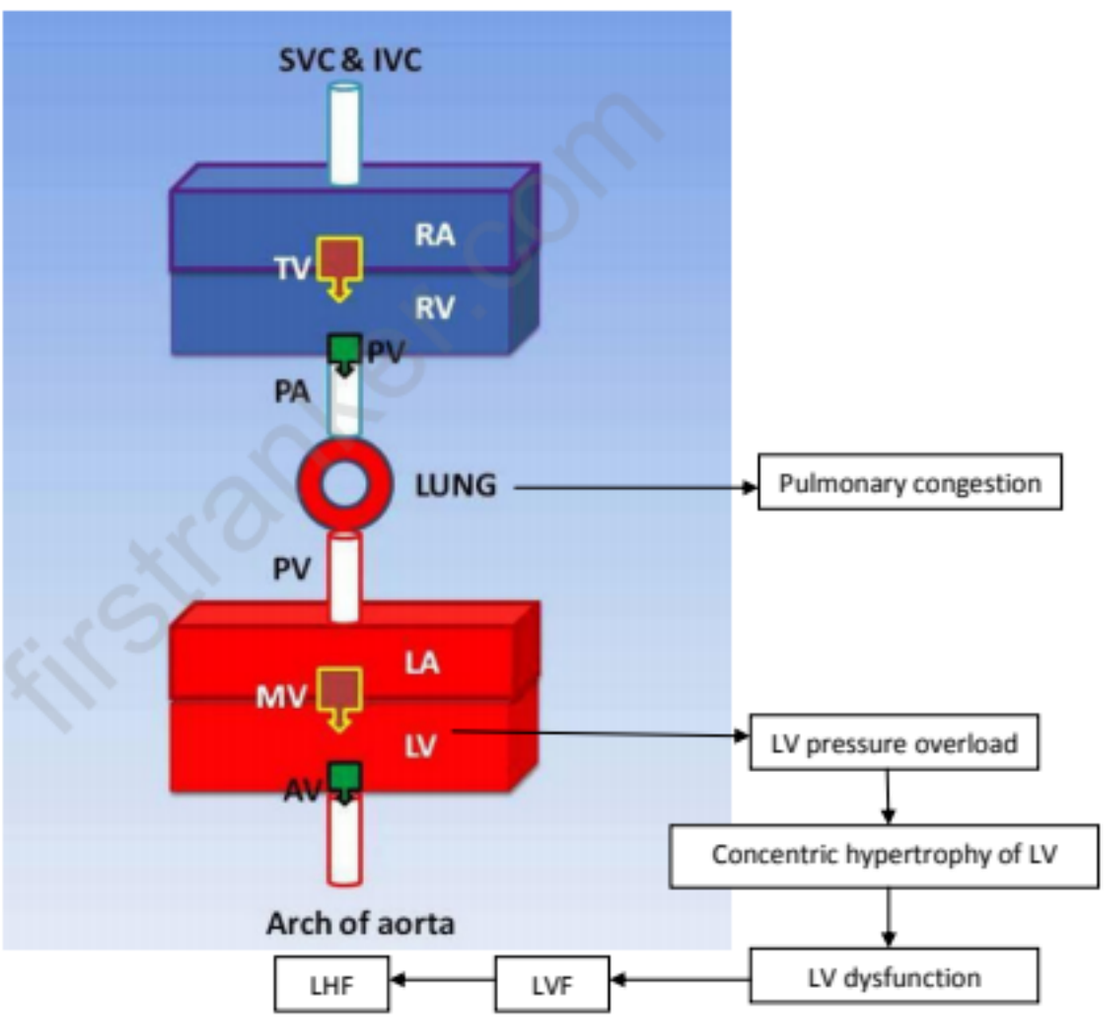
AORTIC STENOSIS (AS)

It is defined as incomplete opening of aortic valve.

Etiology:

A. Age related degeneration/ atherosclerotic change of valve.
B. Bicuspid aortic valve (usually seen in young person).
C. Calcific degeneration of aortic valve (also called aortic sclerosis).
D. Deformity (rare, arising from chronic rheumatic valvular heart disease).

Hemodynamics:



The forward effect of AS is important because initially cardiac output (CO) is maintained but eventually it falls, resulting in low volume pulse and low BP.

A sudden fall of CO may result in syncope or even sudden death.

Note: As coronary artery atherosclerosis is the primary pathology behind AS, so it may precipitate in angina.

Symptoms:

1. Syncope: Due to a sudden fall of CO below a critical level due to severe narrowing of aortic valve orifice.
2. Angina:
   The factors responsible for angina in AS are:
   a. Co-existing coronary artery atherosclerotic disease.
   b. Increased demand of hypertrophic myocardium.
   c. Squeezing of small coronary perforators during systole due to forceful contraction of LV.
3. Shortness of breath/ Orthopnoea/ PND: Due to pulmonary congestion.

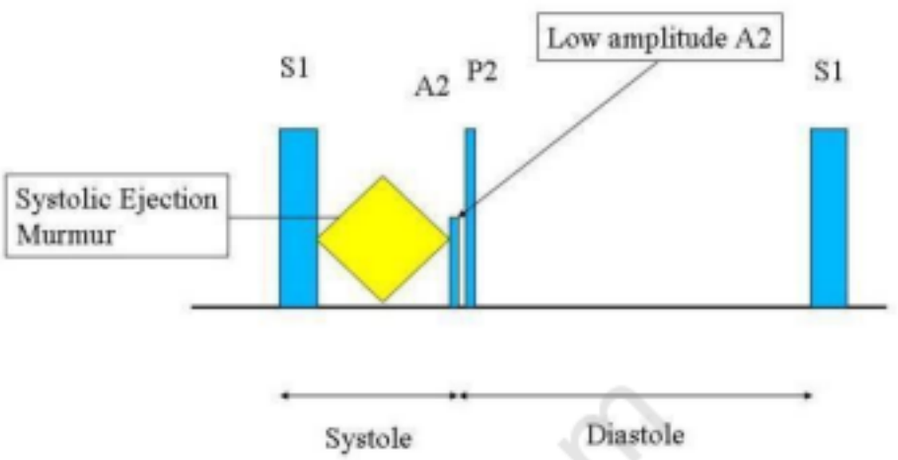
Signs:

1. Low volume pulse and low BP.
2. Apical impulse:
   - Forceful and well sustained (heaving in nature).
     Mechanism:
     Forceful due to increased LV force of contraction and well sustained because of increased LV systolic ejection time.
   - May be shifted down and out.

Examination of aortic area

1. A soft, muffled A2 is present.
   Mechanism:
   Due to atherosclerosis of aortic valve cusps, their mobility is impaired. So, they can't close properly; resulting in a soft/ absent A2.
2. Murmur in AS:
   - A mid-systolic ejection murmur.
   - Loud in intensity (harsh blowing murmur).
   - It usually propagates towards carotid.
   - Ejection click (Opening snap of AS):
     The ejection click usually precedes the mid-systolic murmur and occurs due to ***sudden vibration produced during opening of thickened aortic valve***. Therefore, it is nothing but the opening snap of AS.

Heart Sounds (Aortic Stenosis)



Investigation

1. CBC, TC, DC, ESR/ CRP.
2. Urea- creatinine Na+ K+.
3. ECG: May show LVH.
4. CXR: May show cardiomegaly.
5. Echo:
   a. Confirmation of diagnosis.
   b. Identify the type of damage to the aortic valve.
   c. Assessment of cardiac function.
   d. Measures the severity of AS (Severe AS is defined as a transvalvular pressure gradient> 60 mm Hg).
6. Often these patients will need further investigations to evaluate the cause of angina pain (coronary angiogram).

Treatment

The treatment of AS may be divided into 2 groups:

a. Medical management (treatment of complications) and,
b. Interventional treatment.

Medical management

LHF

Acute stage

M. MORPHINE.

N: NITRATE.

O. OXYGEN.

D. DIURETIC.

Chronic/ stable stage

A. ACE INHIBITORS/ ARB.
B. BETA BLOCKERS.
C. CCB.
D. DIURETICS.

Interventional treatment

- The procedure of choice is aortic valve replacement.
- If there is significant coronary artery disease, then coronary revascularization is preferred. It is done using either of the following options when indicated, along with/ before valve replacement:
  a. Percutaneous intervention with stenting.
  b. Coronary artery bypass graft (CABG).

Complications of AS

1. LHF: May be acute/ chronic/ acute on chronic.
2. Syncope.
3. Sudden death:
   Mechanism:
   a. Stenosed aortic valve refuses to open at all, leading to sudden irreversible fall of CO.
   b. Sudden fatal ventricular arrhythmia arising from stretched ventricular myocardium.

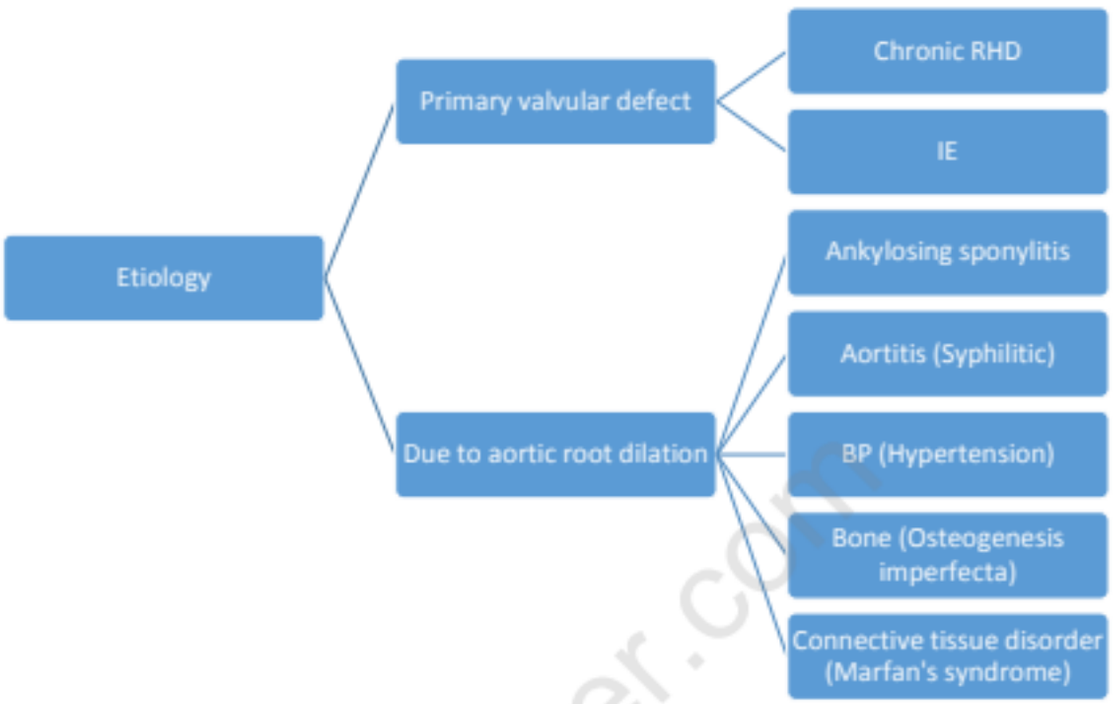
Causes of mid-systolic murmur over aortic area

1. Valvular AS (Carotid propagation + Ejection click).
2. Functional AS (Carotid propagation – Ejection click):
   It occurs when abnormal amount of blood flows through a normal aortic valve orifice.
   Example:
   a. AR.
   b. Any hyperkinetic circulatory state:
      ✓ Fever,

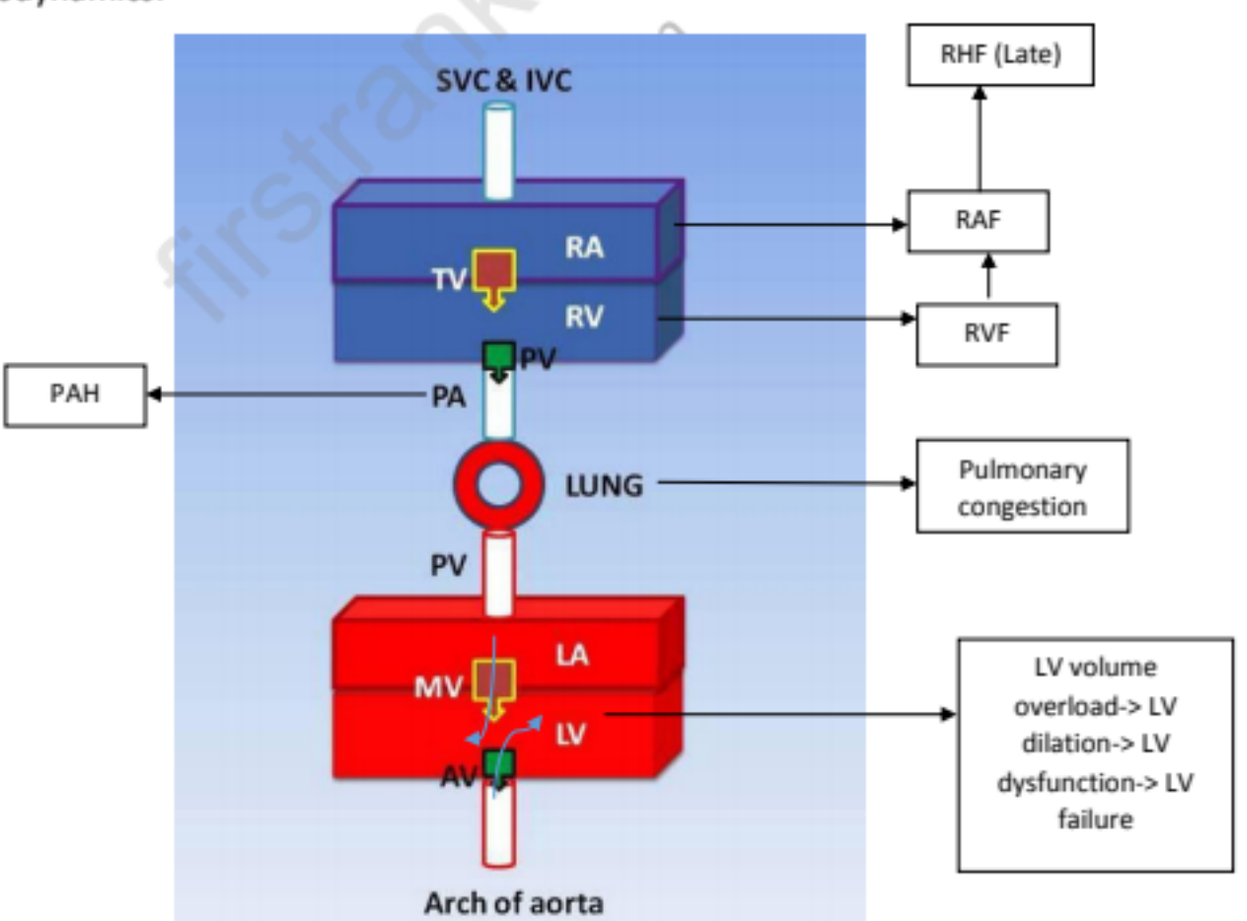
- ✓ Anemia,
- ✓ Thyrotoxicosis,
- ✓ Pregnancy etc.
  - These are often called "*flow murmur/ innocent murmur*" as they don't arise from a valvular pathology.

Aortic regurgitation (AR)

It is defined as incomplete closure of aortic valve.



Hemodynamics:



Hemodynamics of aorta and other arteries:

1. *Abrupt systolic distension due to increased stroke volume.*
2. *Rapid diastolic collapse due to diastolic backflow of blood towards the LV.*
   - This hemodynamic effect explains the peripheral signs of AR.

Symptoms:

1. Due to pulmonary congestion:
   a. *Shortness of breath,*
   b. *Orthopnoea,*
   c. *PND.*
2. *Palpitation in left lateral position*: When the patient feels the forceful LV contraction.
3. *Uneasy throbbing sensation at different places of body*: Due to abrupt systolic distension of the arteries.

Signs:

JVP↑ (in late stage).

Examination of aortic area

- Soft A2 (due to incomplete closure of aortic valve).
- Murmur:
  a. Actual murmur of AR is best heard at neo-aortic area (Left 3rd intercostal space). It is an *early-diastolic murmur*, soft blowing in quality; the sound may be accentuated by asking the patient to sit in a *leaning forward position holding the breath, after a full deep expiration*. It propagates along the left sternal border towards mitral area.
  b. A *mid-systolic murmur* may be heard due to functional AS (during ventricular systole, the LV contracts forcefully to expulse the abnormal amount of blood in LV to circulation).

Peripheral signs

1. Episodic head nodding with each systole (De Musset sign).
2. Prominent carotid pulsation (Dancing carotid/ Corrigan's sign).
3. High volume collapsing pulse (Water hammer pulse/ Corrigan's pulse).

4. BP:
   - SBP ↑/Normal.
   - DBP ↓.

   As the severity of AR progresses, SBP may be normal/ low normal and DBP doesn't fall to that extent it was falling in the initial stage.

   Hill's sign:

   Normally femoral arterial SBP is higher than brachial arterial SBP. The difference is usually <20 mm Hg. But in AR, this difference becomes >20 mm Hg.

5. *Prominent capillary pulsation (Quinche's sign)*: On exerting pressure over the tip of the nail, there is alternate flushing (due to systole) and blanching (due to backflow of blood) of nail bed.
6. Auscultation of femoral artery:
   a. *Pistol shot sound*: A booming sound with each systole with abrupt systolic distension of femoral artery.
   b. Durozey murmur: On distal compression over femoral artery, a diastolic murmur can be heard with the diaphragm of the stethoscope.
   c. Durozey sign:
      Diastolic murmur heard on distal compression of femoral artery + systolic murmur heard on proximal compression of femoral artery.

Investigation

1. CBC.
2. Urea-creatinine Na+ K+.
3. CXR: May show cardiomegaly.
4. ECG.
5. Echocardiogram:
   a. Confirms the diagnosis.
   b. Can tell the underlying etiology.
   c. Visualizes cardiac chambers.
   d. Assess cardiac function.

Treatment

It comprises of 2 parts:

a. Medical management (Treatment of complications).
b. Interventional treatment.

Medical management

Treatment of LHF

In acute stage

M: Morphine.

N: Nitrate.

O: Oxygen.

D: Diuretics.

In chronic stage

A: ACE inhibitors/ ARB.

B: Beta blocker.

C: CCB.

D: Diuretic.

Interventional treatment

Interventional treatment of choice is aortic valve replacement.

Causes of a mid-systolic murmur (MSM)

There are usually 2 causes:

a. Valvular AS.
b. Functional AS (Ex.: AR and several hyperkinetic circulatory conditions).

How to differentiate between the 2 etiologies?

MSM of valvular AS	MSM of functional AS (as in AR)
Ejection click is also heard with the murmur.	Ejection click is not heard with the murmur.
As the turbulence of blood flow is high, so carotid propagation of the murmur is present.	Carotid propagation does not occur.

Congenital heart diseases

In this section, we will discuss about the following congenital heart diseases:

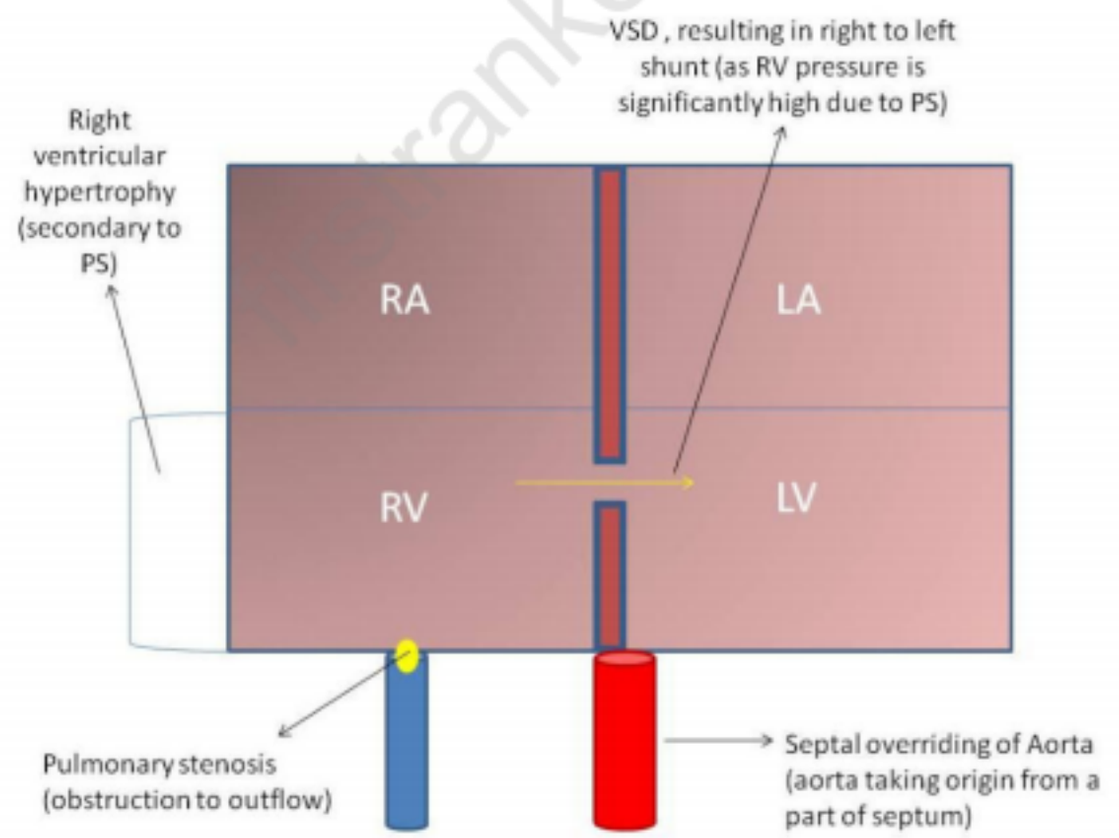
1. Tetralogy of Fallot.
2. Ventricular septal defect (VSD).
3. Atrial septal defect (ASD).
4. Patent ductus arteriosus (PDA).
5. Coarctation of aorta.

Tetralogy of Fallot

4 components of the "tetralogy":

1. Pulmonary stenosis (PS).
2. Right ventricular hypertrophy (RVH).
3. VSD.
4. Septal overriding of aorta (in some patients, there may be right ventricular origin of aorta).

Hemodynamics:

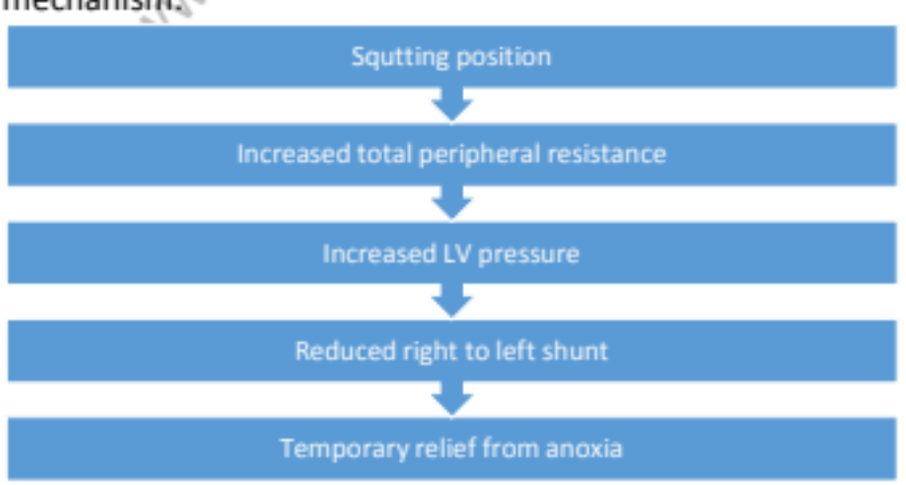


Hemodynamics at different levels: (See picture above)

Level	Hemodynamics
Pulmonary valve	Turbulent blood flow.
Right ventricle	1. RVH (secondary to PS). 2. Due to PS, RV pressure remains significantly high since the time of birth and it becomes> LV pressure soon after birth/ during early stages of life; which is responsible for *right to left shunt* through the ventricular septal defect. 3. During systole, RV gets decompressed faster as there are 2 outlets through which blood can flow out of RV (to pulmonary artery and LV). So, *RV dysfunction/ failure is relatively uncommon in TOF.* 4. The severity of PS is the indicator of the severity of the disease.
Left heart	It is largely unaffected as there is neither volume nor pressure overload.

Symptoms:

1. Anoxic spells:
    - Duration, frequency and severity of anoxic spells depend on the amount of right to left shunt.
    - The child is usually restless, irritable, drowsy and cyanotic.
    - Exercise often precipitate severe anoxic spells (as venous return is increased in exercise and that results in increased right to left shunt).
    - Squatting may temporarily relief the baby by the following mechanism:



Signs:

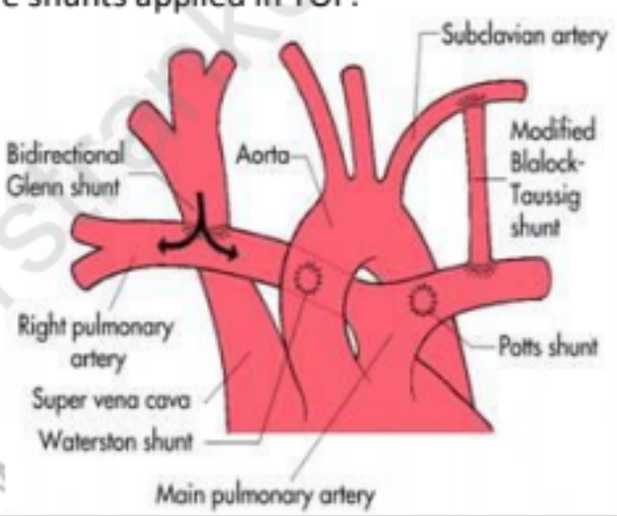
1. Cyanosis,
2. *Mid-systolic murmur* over pulmonary area due to PS.
3. *Parasternal hip* due to RVH.

Investigation

Echocardiogram: Confirms the diagnosis.

Treatment

1. Symptomatic relief during anoxic spells:
   a. Put the baby on a squatting posture.
   b. Give the baby high flow oxygen.
2. Surgical correction of the underlying defect.
3. Palliative surgery:
   Anastomosis is created between a systemic artery and one of the branches of pulmonary artery so that deoxygenated blood can be redirected to lungs for oxygenation.
   Various palliative shunts applied in TOF:



Name of the shunt	Applied in between
Potts shunt	Descending aorta connected to left pulmonary artery.
Waterson shunt	Ascending aorta connected to main/ right pulmonary artery.
Modified BT shunt* (procedure of choice)	A tube graft placed between subclavian artery and one of the pulmonary arteries.
Glenn shunt	Bidirectional connection between aorta and right pulmonary artery.

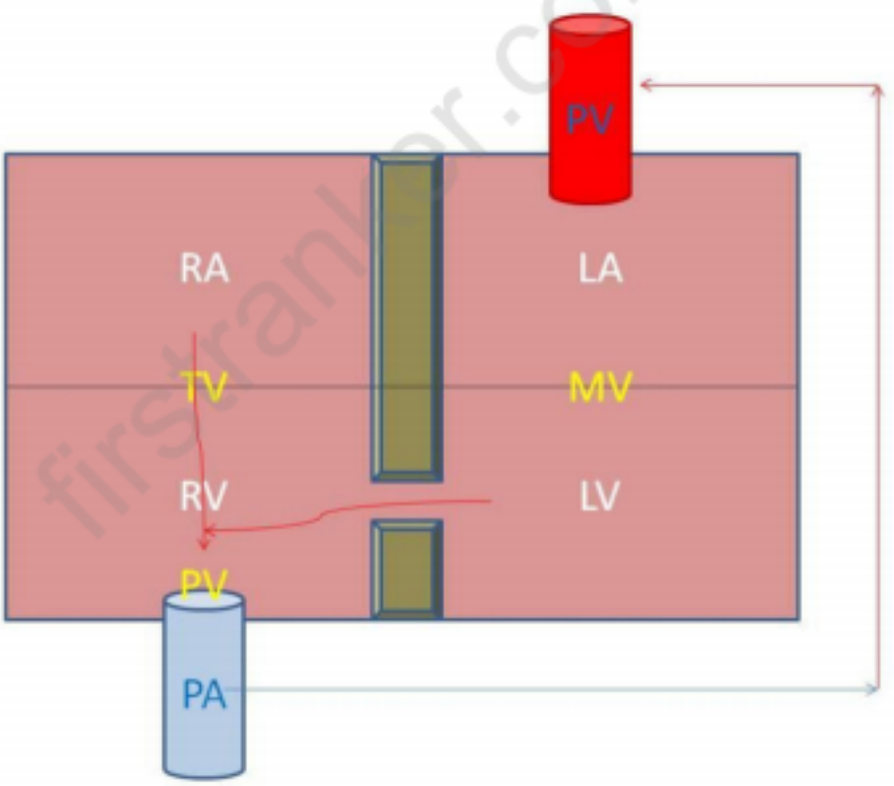
Complications

1. Severe anoxic spells.
2. Infective endocarditis.
3. Recurrent broncho-pulmonary infection.

VSD (Ventricular septal defect)

Basic pathology:

Left to right shunt which usually continues throughout the systole along the pressure gradient.



Hemodynamics at different levels:

Level	Hemodynamics
Pulmonary valve	Functional PS (abnormal blood flow through a normal valve).
Pulmonary artery	PAH.
RV	• Volume overload---→ RV dysfunction/ failure.

	• Eventually a time might come when RV pressure will be greater than LV pressure; causing reversal of shunt.
LA	Volume overload---→ LA failure.
MV	Functional MS (abnormal blood flow through a normal valve).
LV	Volume overload---→LV failure.

Symptoms:

1. Symptoms due to LHF and pulmonary congestion:
   - Shortness of breath,
   - Orthopnoea,
   - PND.
2. Symptoms due to RHF (at very late stage):
   Swelling.

Signs:

1. Murmur:
   a. Due to VSD:
      A loud *pan-systolic murmur* best heard over left 4th intercostal space.
      It's so loud that it is often audible at other cardiac areas of heart.
   b. Associated murmurs:
      - MSM at pulmonary area (due to functional PS).
      - MDM at mitral area (due to functional MS).
2. Sign of RHF (JVP↑)/ LHF (signs of pulmonary congestion).

Investigation

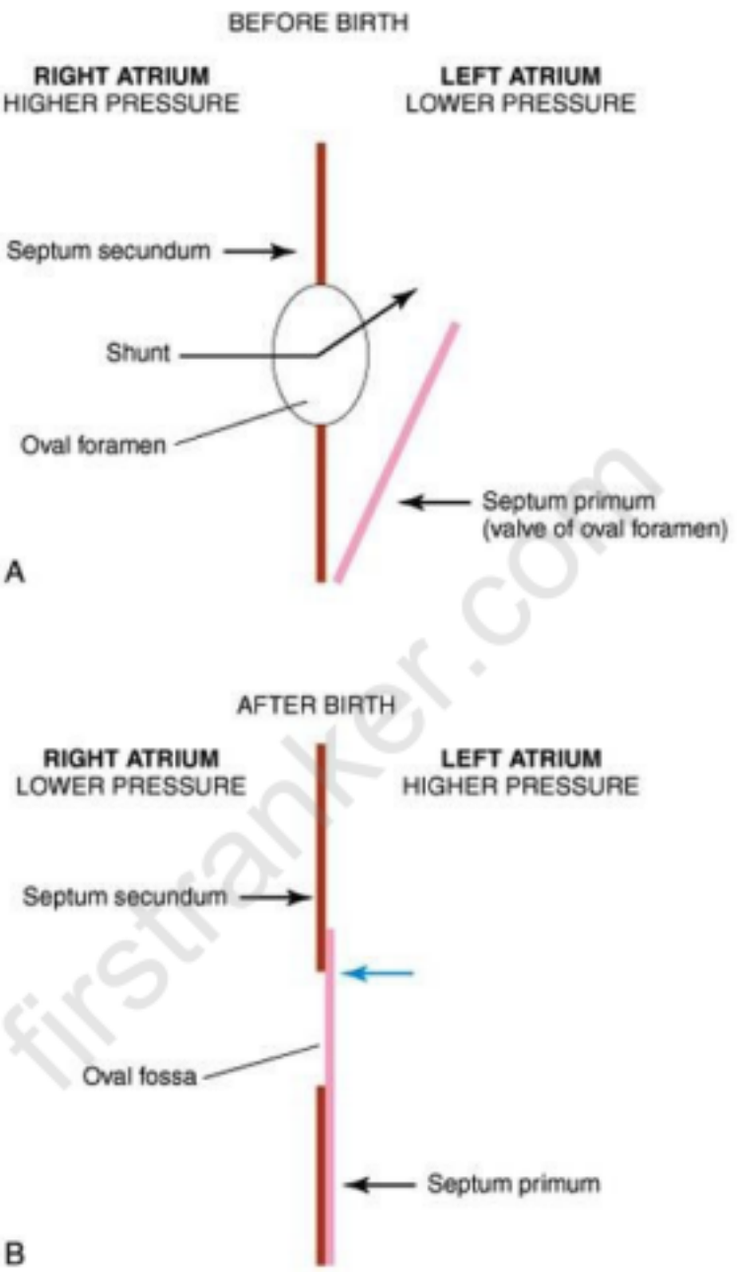
Echocardiogram: Confirms the diagnosis.

Treatment

1. Medical management of underlying heart.
2. Surgical correction of VSD.

ASD (Atrial septal defect)

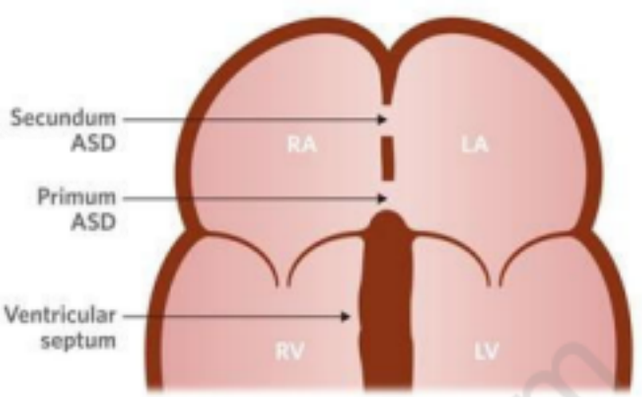
Development of atrial septum:



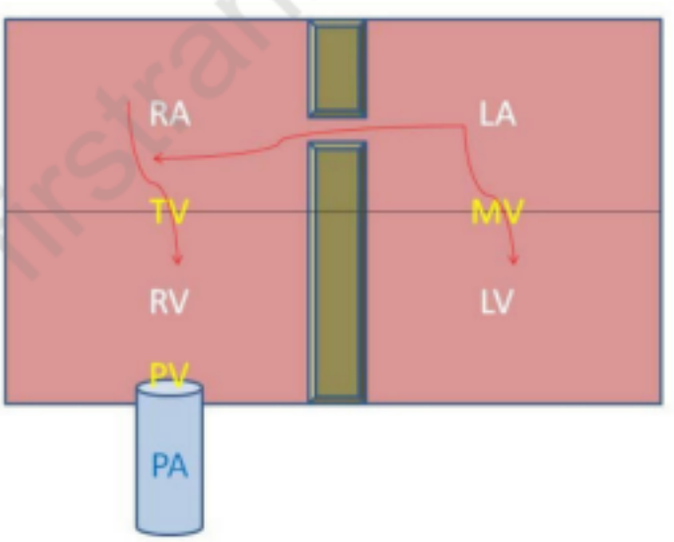
Types of ASD:

1. *Ostium secondum type*: Common variety; defect lies at the level of fossa ovalis.
2. *Ostium primum type*: Defect lies inferior to fossa ovalis. Sometimes associated with other congenital cardiac structural abnormality (Ex.: congenital MR).

Atrial septal defect (ASD)



Hemodynamics:



Basic hemodynamics:

There is *left to right shunt* along the pressure gradient.

As the pressure gradient is not significant, it usually does not produce a murmur. The hemodynamic abnormality takes a long time before it becomes symptomatic.

Hemodynamics at different levels:

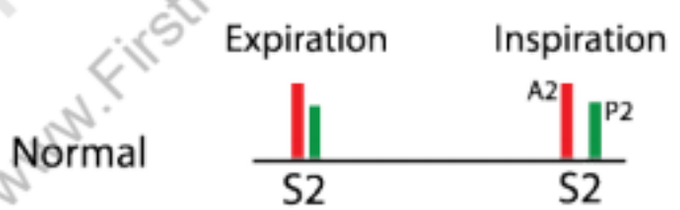
Level	Hemodynamics
RA	Volume overload------→ RA dilation-----→ Dysfunction and failure (if occur, after a long time).
RV	Volume overload------→ RV dilation-----→ Dysfunction and failure (if occur, after a long time).
Pulmonary valve	• Functional PS (due to abnormal blood flow through a normal valve). • Delayed closure of pulmonary valve due to prolonged systolic ejection time may lead to a delayed P2.
Pulmonary artery	PAH.
LA	LA volume overload and dilation may occur after a long time.

Symptoms:

- Often asymptomatic, diagnosed incidentally.
- Swelling may occur in late stages of ASD as a result of RHF.

Signs:

- A normal S1.
- S2: Usually normal in intensity, P2 may be loud if PAH develops.
- Split:
  Mechanism of physiological splitting in second heart sound (during inspiration):



  - During inspiration, a negative intrathoracic pressure is created to inhale air into the lungs.
  - This creates a vacuum effect which results in increased venous return to the right side of the heart.
  - Hence the RV takes a longer time to eject the blood into the pulmonary system.
  - Also since pulmonary vasculature expands in capacity, lower amount of blood flows into the LA.

- Hence the LV takes a shorter time to eject blood into the aorta.
- Because of these 2 factors, the aortic valve closes before that of the pulmonary valve and we can appreciate the split during inspiration.

Mechanism of wide and fixed split in ASD:

- ***The split is wide because*** RA is dealing with extra amount of blood (in addition to the normal venous return, it receives blood from LA) and this extra blood is received by RV, RV systolic ejection time gets prolonged and consequently there is a delayed occurrence of P2.
- ***The split is fixed because:***
  - During inspiration, venous return increases through vena cava and so, RA receives more amount of blood. So, amount of L-R shunt decreases.
  - During expiration, capacity of pulmonary vasculature decreases and more amount blood flows into LA. So, amount of L-R shunt increases.
  - So, venous return and L-R shunt varies reciprocally during inspiration and expiration; resulting in a fixed split.

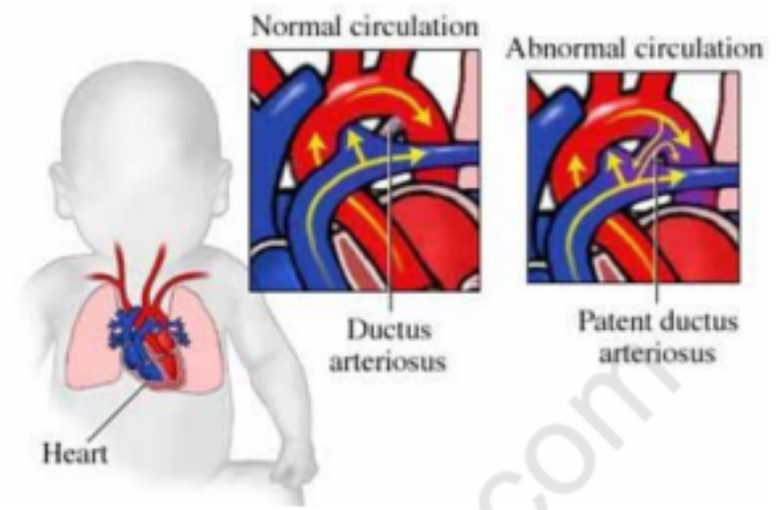
Investigation: Echocardiogram confirms the diagnosis.

Treatment:

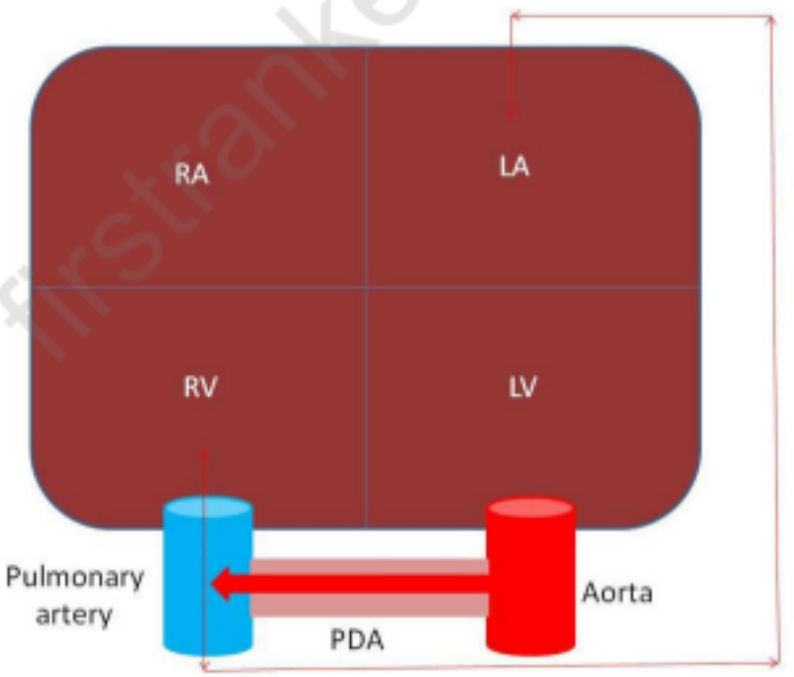
1. Management of heart failure.
2. Surgical correction of defect.

Patent ductus arteriosus (PDA)

PDA is defined as non obliteration of ductus arteriosus (which is a communicating blood vessel between aorta and pulmonary artery and usually obliterates by the time of birth).



Hemodynamics:



Hemodynamics at different levels:

- Pulmonary artery: PAH.
- RV/RA pressure overload may occur from PAH at late stage.
- LA/LV volume overload may occur at late stage.
  - Both of which may result in RHF/LHF at a late stage.

Symptoms:

- Often asymptomatic.
- May develop symptoms of RHF/LHF at a late stage.

Signs:

- A ***continuous machinery murmur continuing throughout the systole and diastole*** is usually present over the ***pulmonary area***.
- As PA pressure rises and shunt eventually rises (which may occur after a long time), both the components of the murmur (particularly the diastolic component) becomes less intense.

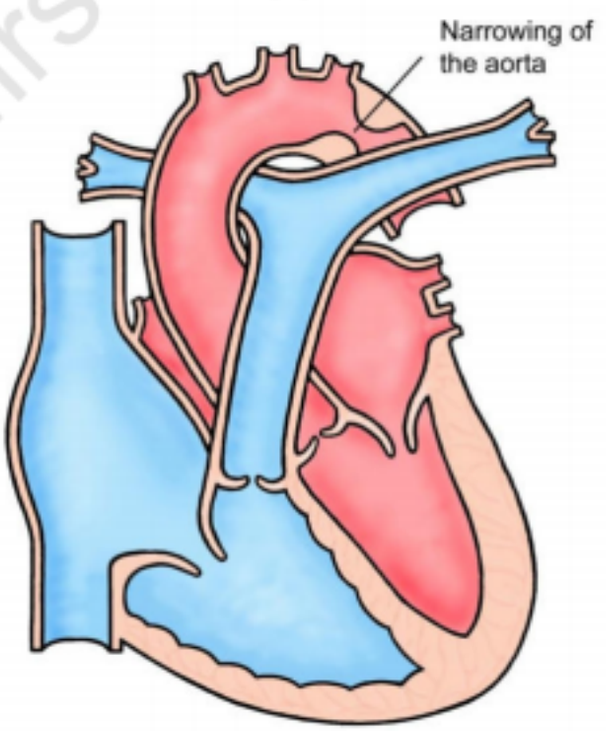
Investigation: Echocardiogram confirms the diagnosis.

Treatment:

1. Surgical correction of defect.
2. Obliteration of ductus arteriosus can be induced by ***Indomethacin***.
3. Symptomatic management of HF, if present.

Coarctation/ stenosis of aorta

This condition is defined as narrowing of aorta usually between arch of aorta and descending thoracic aorta.

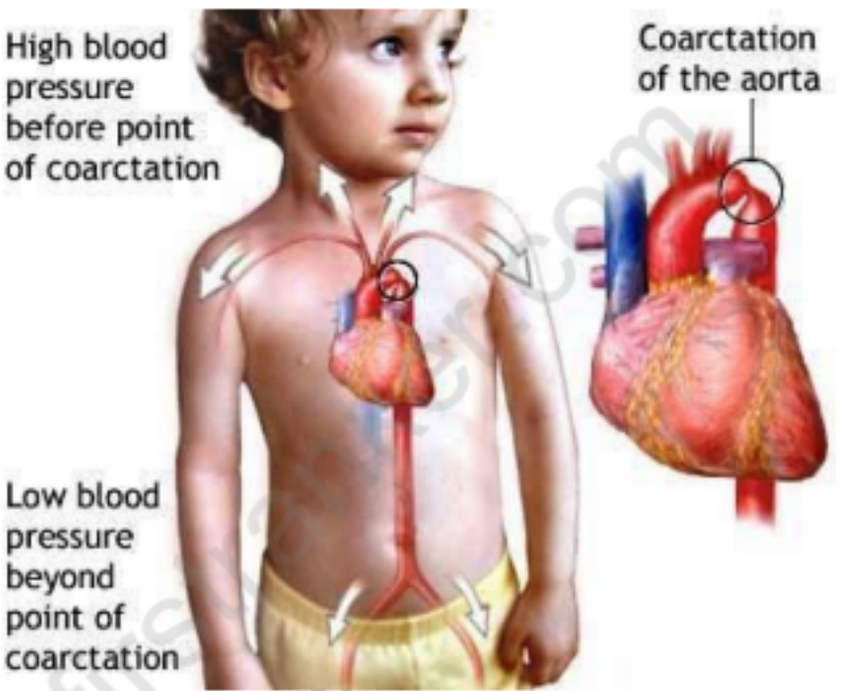


This condition is sometimes associated with:

a. Aneurysm of circle of Willis.
b. Mitral valve prolapse.

Hemodynamics:

- At the pre-stenotic part (upper part of the body), there is high BP.
- At the post-stenotic part (lower part of the body), there is low BP.
- The pulse volume may be slightly weaker at the lower extremity while compared to upper extremity.

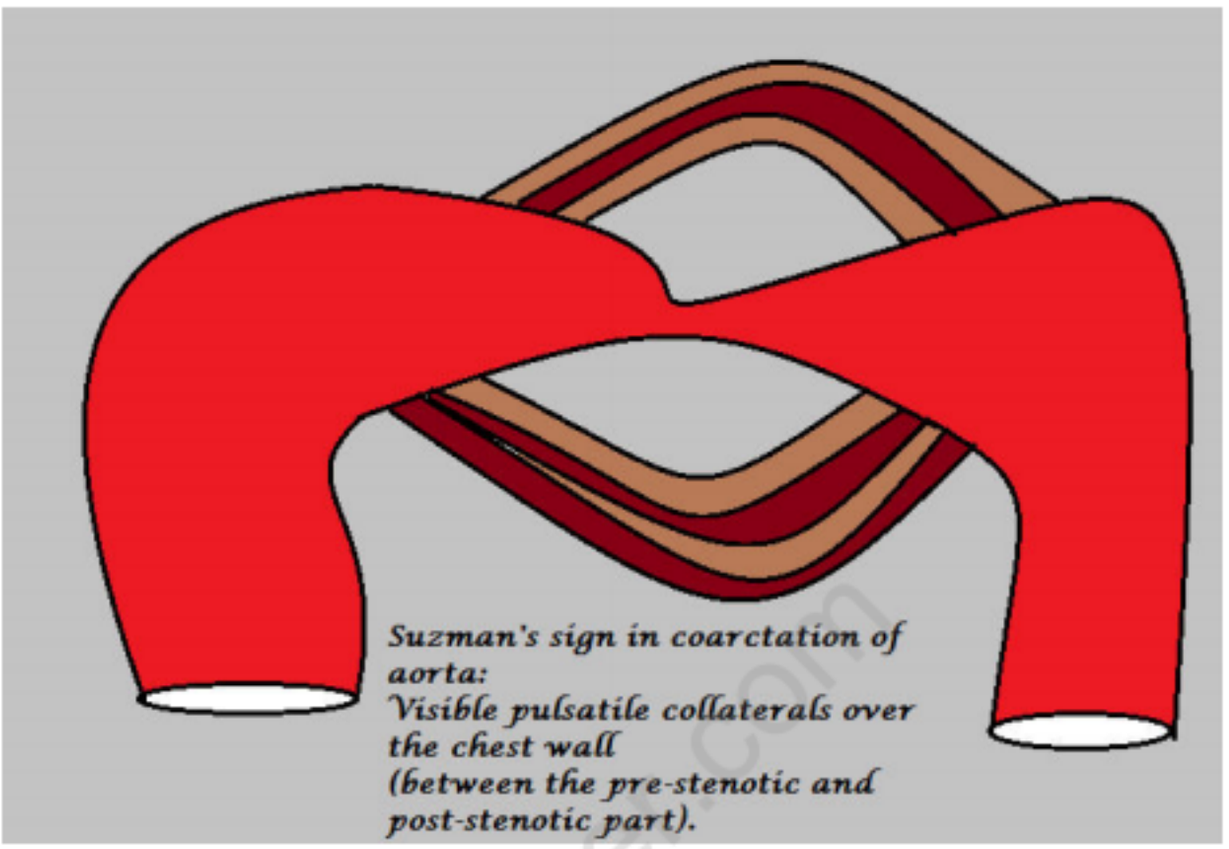


Symptoms:

- May be asymptomatic.
- Symptoms due to hypertension/ its complications.

Signs:

1. Asymmetry of radial and femoral pulse volume with radio-femoral delay.
2. Brachial artery BP disproportionately higher than femoral artery BP.
3. Visible, pulsatile collaterals may be present over the chest wall (Suzman's sign).
4. A prominent suprasternal pulsation may appear.
5. Rarely, a systolic bruit can be heard over the interscapular area.



Investigation:

1. Echocardiogram confirms the diagnosis.
2. Cardiac MRI scan for better visualization.

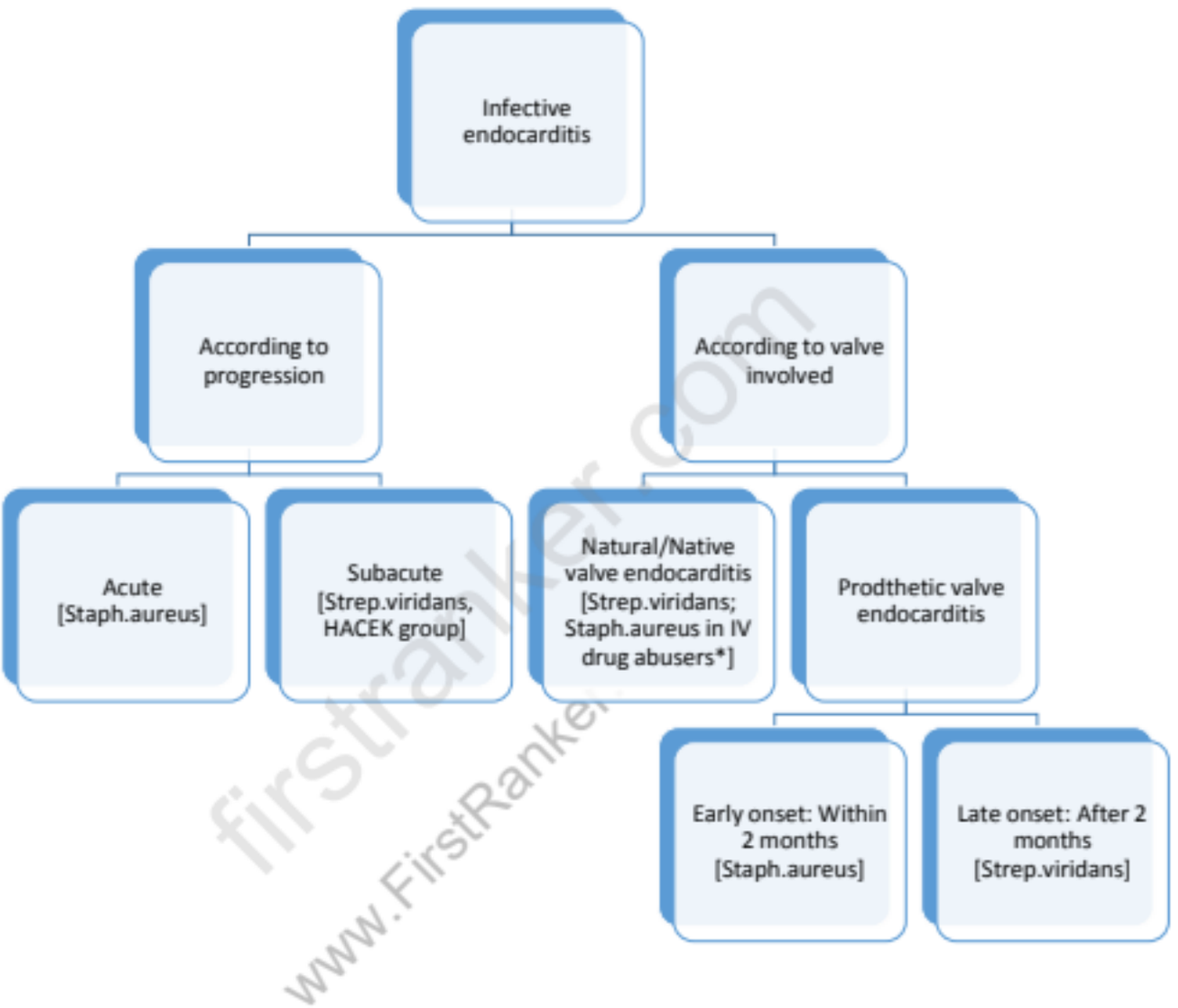
Treatment:

1. Surgical correction of coarctation.
2. Symptomatic treatment for hypertensive complications.

INFECTIVE ENDOCARDITIS

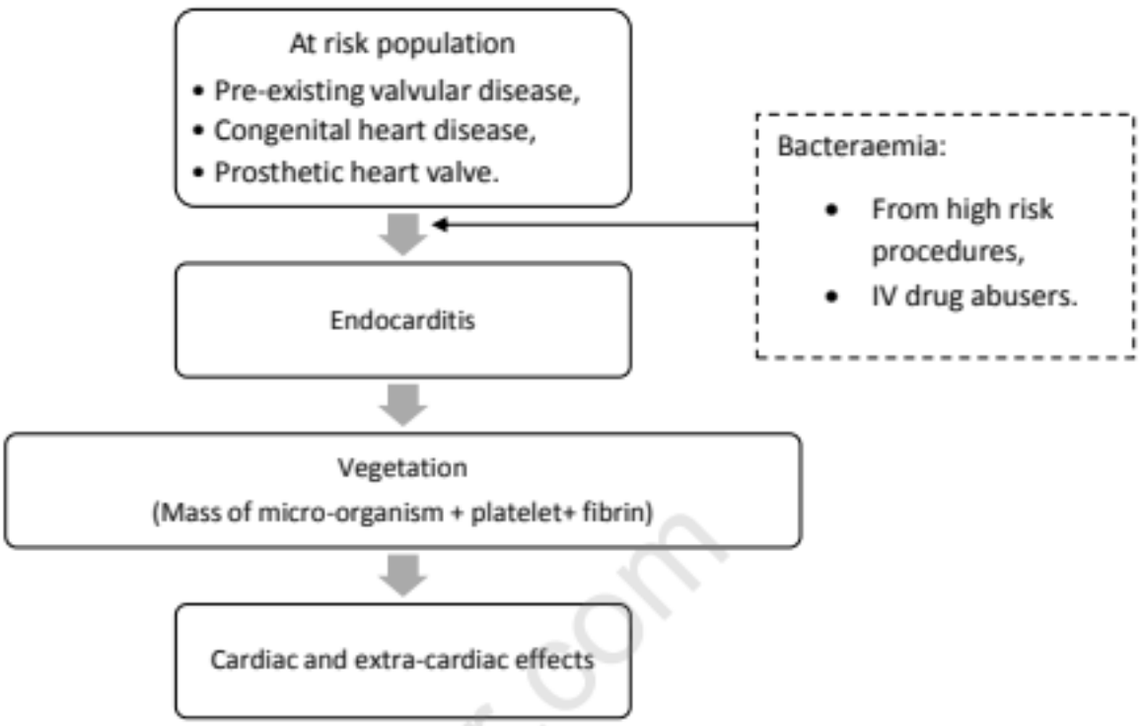
It is defined as the infection of cardiac valves and mural endocardium.

Etiology:



[* In IV drug abusers, predominantly tricuspid valve is affected.]

Pathophysiology with clinical manifestations:



Cardiac effects:

- Worsening of pre-existing symptoms/ appearance of new symptoms.
- Change in the quality/ intensity of a pre-existing murmur/ appearance of a new murmur [usually a regurgitant murmur].
- Formation of myocardial abscess, resulting in conduction abnormality.

Embolic manifestations:

- ***Involvement of CNS:***
  - Transient ischemic attacks (TIA),
  - Brain abscess.
- ***Involvement of kidney:***
  - Acute loin pain.
- ***Involvement of spleen:***
  - Acute left upper quadrant pain due to splenic infarction.
  - Splenic rub is found if splenic capsule is involved.
- ***Involvement of digital arteries:***
  - Sub-ungual/ splinter hemorrhage (linear streaks of hemorrhage best seen in nail bed).
  - Janeway lesion (small hemorrhagic spots seen on palm and sole).

Effects due to immune complex deposition:

- In kidney: Acute glomerulonephritis.
- In joints: Arthritis.
- Retina: Roth spots (Retinal hemorrhages typically observed by an ophthalmoscope).

Note: In general, patients usually have background valvular heart disease/ congenital heart disease/ prosthetic heart valve and features of toxaemia (fever, malaise, appetite loss etc.).

Investigation

1. Complete blood count: Hb, TC, DC, ESR/CRP.
2. ECG: To rule out any conduction abnormality.
3. CXR: May show cardiomegaly due to pre-existing valvular disease.
4. Echo-cardiogram:
   a. Conventional/ Trans thoracic Echo: May/ may not show vegetations as they are usually deep-seated.
   b. ***Trans-oesophageal Echo:*** It is the best modality and the gold standard of diagnosis of a case of IE.
5. Blood culture:
   The blood culture samples should be taken before the first dose of antibiotic. At least 3 sets of sample should taken from 3 different sites. The interval between taking 2 samples should be at least 1 hour.

Treatment

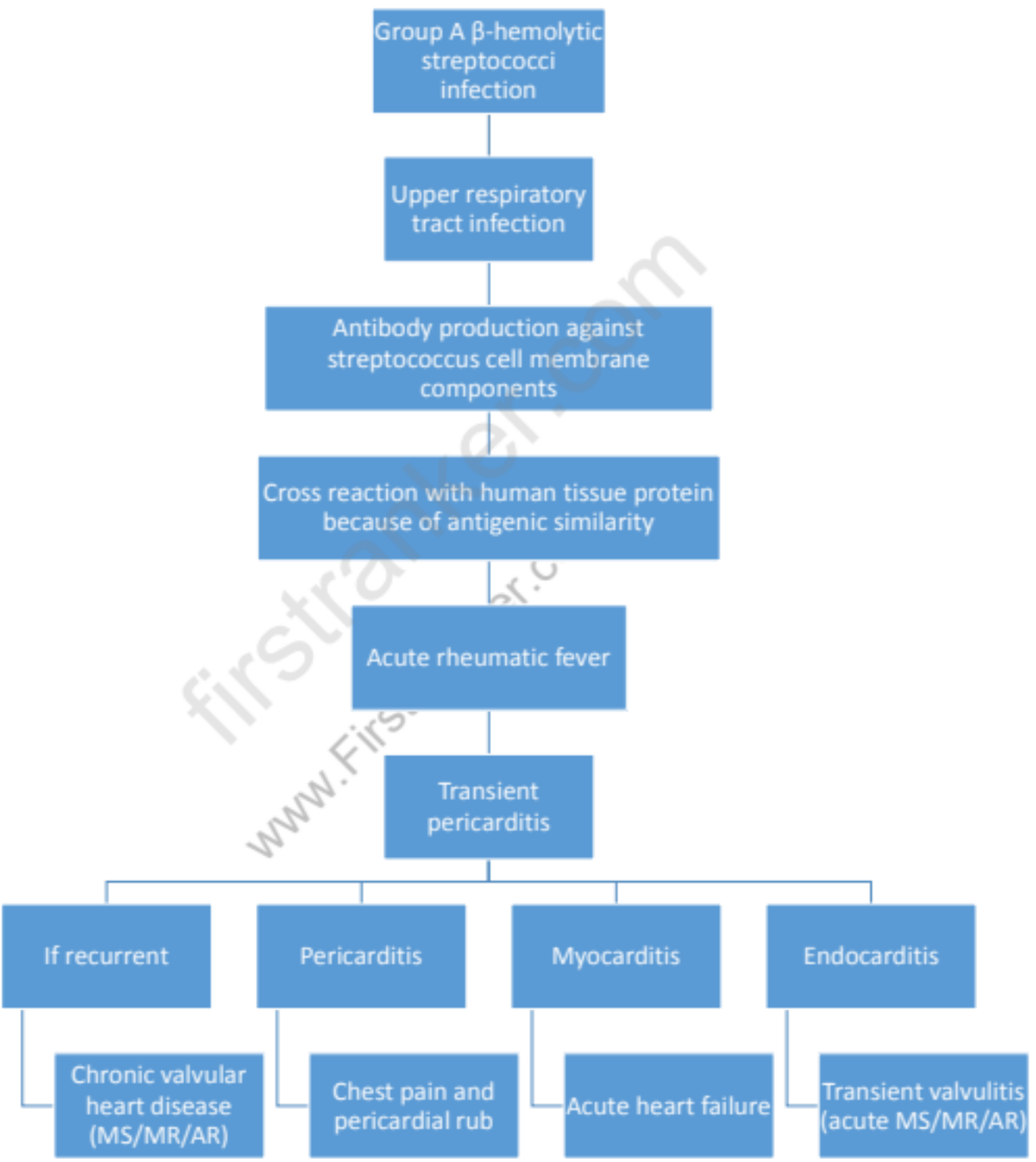
- An empirical regimen (IV Ceftriaxone + IV Vancomycin) is usually administered before the result of blood culture comes. This regimen may further be modified according to the culture-sensitivity result. Therapy should be continued for at least ***4-6 weeks duration***.
- Symptomatic treatment of underlying valvular heart disease/ complication should be given concurrently.
- Prophylactic antibiotic should be given to all patients who are at high risk of developing endocarditis (valvular heart disease/ congenital heart disease/ prosthetic heart valve).

- Before undertaking any types of invasive procedures, which can potentially cause Staph./Strep. bacteraemia in the patient (like orodental/ respiratory tract/ skin and soft tissue procedures); a single prophylactic dose of ***ampicillin/ amoxicillin/ clindamycin*** should be administered just before the procedure.

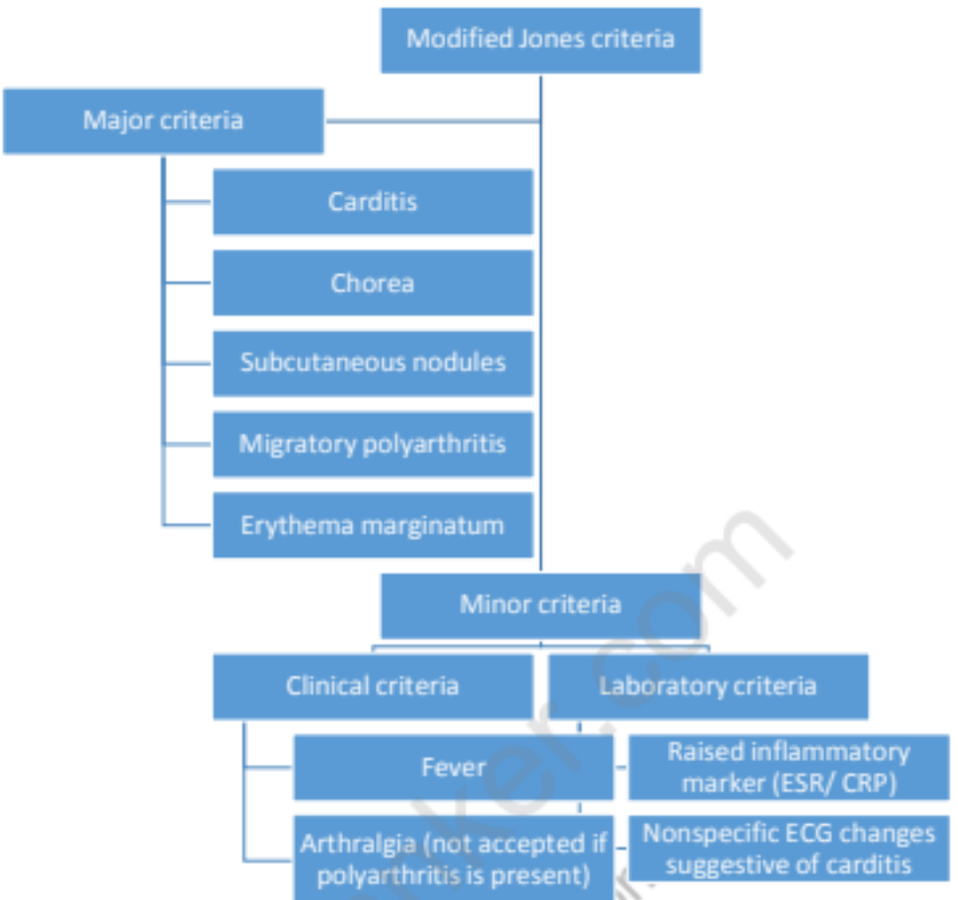
Acute rheumatic fever (ARF)

It is an immunologically mediated multisystem disease triggered by group A beta haemolytic streptococci.

Pathogenesis:



Diagnosis:



At least 2 major/ (1 major+ 2 minor) criteria should be present in a patient having ***unequivocal evidence of a recent streptococcal infection (includes a recent history of sore throat along with positive streptococcal serology [Ex.: ASO titre/ Anti DNAase/ Anti-hyaluronidase])***.

Investigation

1. Complete blood count, CRP/ ESR.
2. ECG.
3. Echocardiogram.

Treatment

Acute rheumatic fever

The treatment consists of 3 "A"gents:

a. Aspirin: High dose aspirin is the drug of choice for rheumatic polyarthritis which dramatically response to aspirin.

b. Anti-inflammatory agents (like corticosteroids): It is reserved for certain cases of polyarthritis and may be used to treat rheumatic carditis.
c. Antibiotics:
    - Benzyl penicillin IM 1.2 million units to get rid of any residual streptococcal infection.
    - Long term prophylactic antibiotic should always be considered to prevent recurrent streptococcal infection. Usually Benzyl penicillin IM 1.2 million units every 4 weeks is given.
    - Duration of prophylactic therapy:
      Duration of the prophylaxis depends on initial presentation of the patient:
        - Carditis in any form: Lifelong prophylaxis.
        - No carditis: For the next 5 years/ Till the patient reaches 25 years of age (whichever comes later).

Ischemic Heart Disease (IHD)

The spectrum of IHD:

It includes the following groups:

1. Asymptomatic patients of IHD.
2. Stable angina.
3. Acute coronary syndrome (ACS):
   a. Simple ACS,
   b. ST segment elevated myocardial infarction (STEMI),
   c. Non ST segment elevated myocardial infarction (NSTEMI).

Etiology of IHD:

The main ***underlying cause*** behind IHD is coronary artery atherosclerotic disease (CAAD).

There are certain ***risk factors*** of CAAD. They are:

A: ABDOMINAL OBESITY,

B: BLOOD LIPID ABNORMALITY,

C: CIGARETTE,

D: DIABETES,

E: EXCESS ALCOHOL,

F: FAMILY HISTORY,

G: GENDER (MALE> FEMALE),

H: HYPERTENSION/ HOMOCYSTEINEMIA/ HYPOOESTROGENEMIA,

I: INFLAMMATORY MARKERS (CRP↑).

There are also some ***precipitating factors*** which usually come into play only in the presence of CAAD. They may precipitate an ischemic event either acutely or chronically by increasing the myocardial oxygen demand. Ex.:

- Activity:
  - ✓ Physical,
  - ✓ Emotional.
- Anemia.
- Aortic stenosis (or any other cause of significant LVH).

- Pregnancy.
- Thyrotoxicosis.

There are also some rare causes of IHD, where an unusual picture of IHD in the absence of significant CAAD is seen. Ex.:

- Coronary vasculitis.
- Coronary vasospasm (Prinzmetal angina).
- Syndrome X.

STABLE ANGINA/ ANGINA PECTORIS

Stable angina is defined as a relative coronary insufficiency particularly when myocardial oxygen demand increases.

Symptoms:

Chest pain:

- Site: Precordial/ retrosternal.
- Nature: It is characterized by more a discomfort rather than a pain.
- Common expression used by the patients:
  - Tightness/ heaviness of chest.
  - Sense of indigestion etc.
- Duration: Usually doesn't last more than 15-20 minutes and resolves within 3-4 minutes.
- Aggravating factor: Physical/ emotional activity (but any factor that increases myocardial oxygen demand can precipitate angina in a patient with CAAD).
- Relieving factor: Avoidance/ cessation of the aggravating factor(s); usually a dramatic relief is observed when short acting nitrates are administered.
- Radiation: Neck/ jaw/ left shoulder/ inner aspect of left upper limb, occasionally pain may even start at these areas.
- Localization: **Anginal pain is NEVER sharply localized.**

Signs:

Clinical examination may be absolutely normal but thorough assessment should be done for any evidence of risk factors of atherosclerosis.

Investigations:

The investigations of IHD may be divided into 2 categories for better understanding and control of etiological agent(s):

a. To establish the diagnosis.
b. To assess the cardiovascular risk.

To establish the diagnosis of IHD:

1. ***Resting ECG***: May be normal/ may show ischemic changes.
2. ***Exercise ECG*** (also called exercise tolerance test/ Tread mill test): Ischemia is provoked by exercising the patient and ECG changes are monitored.
3. ***Myocardial stress scan:***
   Ischemia is provoked and myocardial perfusion defect is imaged using 2 modalities:
   a. Myocardial perfusion scan (Thallium scan),
   b. Stress echocardiogram (with Dobutamine).
4. ***Visualization of coronary artery:***
   This is also done using 2 modalities:
   a. CT coronary angio.
   b. Coronary angiogram.

Treatment:

This again can be divided into 2 categories:

1. Risk factor modification,
2. Symptomatic treatment.

Risk factor modification

It includes:

1. Lifestyle modification and,
2. Pharmacotherapy.

A: ABDOMINAL OBESITY: Regular exercise and reduction of abdominal fat.

B: BLOOD LIPID ABNORMALITY: Regular monitoring and control of LDL levels.

C: CIGARETTE: Reduction/ cessation of cigarette smoking.

D: DIABETES: Strict monitoring and control of blood sugar.

E: EXCESS ALCOHOL: Avoidance/ complete cessation of alcohol ingestion.

F: FAMILY HISTORY: Non-modifiable risk factor.

G: GENDER (MALE> FEMALE): Non-modifiable risk factor.

H: HYPERTENSION: Treat hypertension adequately and monitor BP on a regular basis.

Symptomatic treatment

- ***Antiplatelet drug***: Aspirin.
- ***β-blocker***: Metoprolol/ Carvedilol/ Bisoprolol.
- ***CCB***: Not commonly used, if they have to be used, then either Amlodipine or Diltiazem has to be used.
- Nitrate/ Nitroglycerine.
- Ranolazine (Alteration of the transcellular late sodium current).
- Nicorandil (K+ channel opener).

Antianginal drugs can be used either singly/ in combination with other drugs. Usually a (β-blocker + Nitrate) is the initial treatment.

Interventional treatment

This is of 2 types:

1. PCI (Percutaneous coronary intervention) and,
2. CABG (Coronary artery bypass grafting).

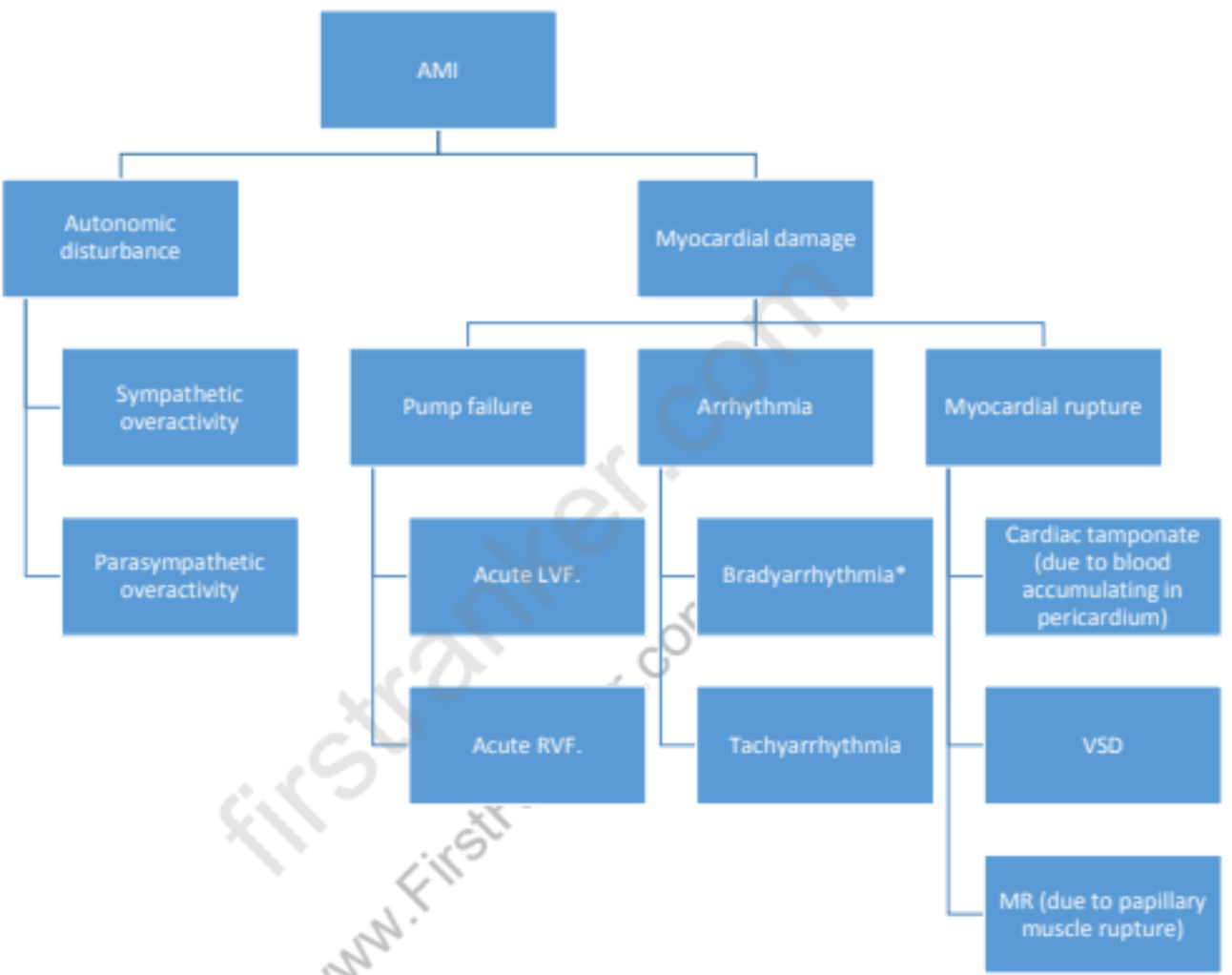
ACUTE CORONARY SYNDROME (ACS)

It is an acute, unstable, cardiac ischemic state with/ without myocardial damage due to acute thrombotic occlusion of coronary artery.

Types:

1. ACS with myocardial damage:
   - STEMI,
   - NSTEMI.
2. ACS without myocardial damage.

Pathophysiology:



[*: Bradyarrhythmia usually occurs in case of inferior wall AMI, in which the Right coronary artery (RCA) is affected. RCA supplies the ventricular septum; so involvement of RCA causes ischemic damage to septum (and also to the conduction pathway), resulting in conduction disturbance.]

Symptoms:

1. Chest pain:
    - Site: Retrosternal.
    - Character: A discomfort rather than a pain.
    - Expressions used by the patient:
        - Heaviness/ tightness of chest,
        - Sense of indigestion etc.
    - Duration: Usually last for more than 15-20 minutes.
    - Precipitating factor: May/ may not be present however physical/ emotional activity may precipitate an event of ACS.
    - Relieving factor: Pain usually DOES NOT subside with cessation of activity/ rapidly acting nitrate.
    - Radiation: Neck/ jaw/ left shoulder/ inner aspect of left upper limb.
    - Localization: Pain is usually not sharply localized.
2. Associated symptoms:
    a. Severe sweating (due to sympathetic overactivity).
    b. Few episodes of involuntary defecation/ vomiting (usually a feature of inferior wall MI, due to parasympathetic overactivity as vagus nerve is situated near the inferior wall of heart, so features of vagal stimulation are usually seen).
    c. Acute shortness of breath (due to acute LVF).
    d. Palpitation/ syncope (due to arrhythmia).
    e. Sudden collapse/ sudden death (due to fatal arrhythmia/ severe cardiogenic shock).

Signs:

The following signs may be present:

1. Signs of acute LVF: Gallop rhythm (S3/S4), Bibasal crepts.
2. Signs of acute RVF: Raised JVP, Hemodynamic instability.
3. Signs acute cardiogenic shock: Low pulse, low BP.
4. Signs of acute MR/ acute VSD: A pansystolic murmur.
5. Signs of tachy/ brady-arrhythmia.

Coexistent inflammatory conditions (like fever/ pericarditis etc.) may also be present.

Investigation

To establish AMI and its immediate complications, the following investigations are to be done:

1. ECG:
   A. STEMI:
      - ST elevation.
      - Abnormal Q wave.
      - T inversion (T↓).
      - Reciprocal ST depression.
   B. NSTEMI:
      - ST depression.
      - T inversion (T↓).

      In all patients with suspected MI, serial ECG every 15-30 minutes must be done, particularly ***during first 2 hours*** post admission – to look for any dynamic ECG changes suggestive of ongoing ischemic event.

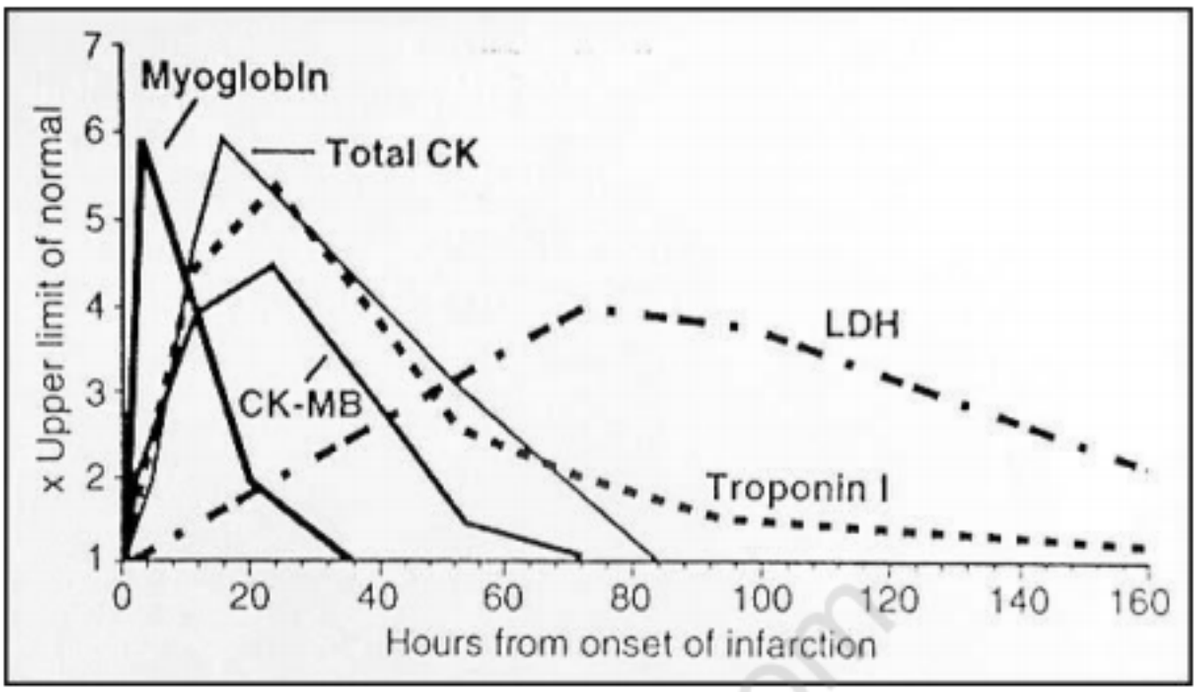
2. Cardiac biomarkers:
   A. Cardiac specific:
      - Trop-T/ Trop-I.
      - CK-MB.
   B. Non cardiac markers:
      - LDH,
      - Total CPK,
      - AST.

      Time of elevation and normalization of cardiac specific markers:

Markers	Time of elevation	Time of normalization
Trop-T/Trop-I	6 hours	7-10 days; upto 14 days
CK-MB	8 hours	72 hours

      Cardiac markers are often sent serially at an interval of ***8 hours*** during first 24-48 hours to see the trend.

      The most sensitive cardiac marker which is detected earliest in blood is myoglobin.

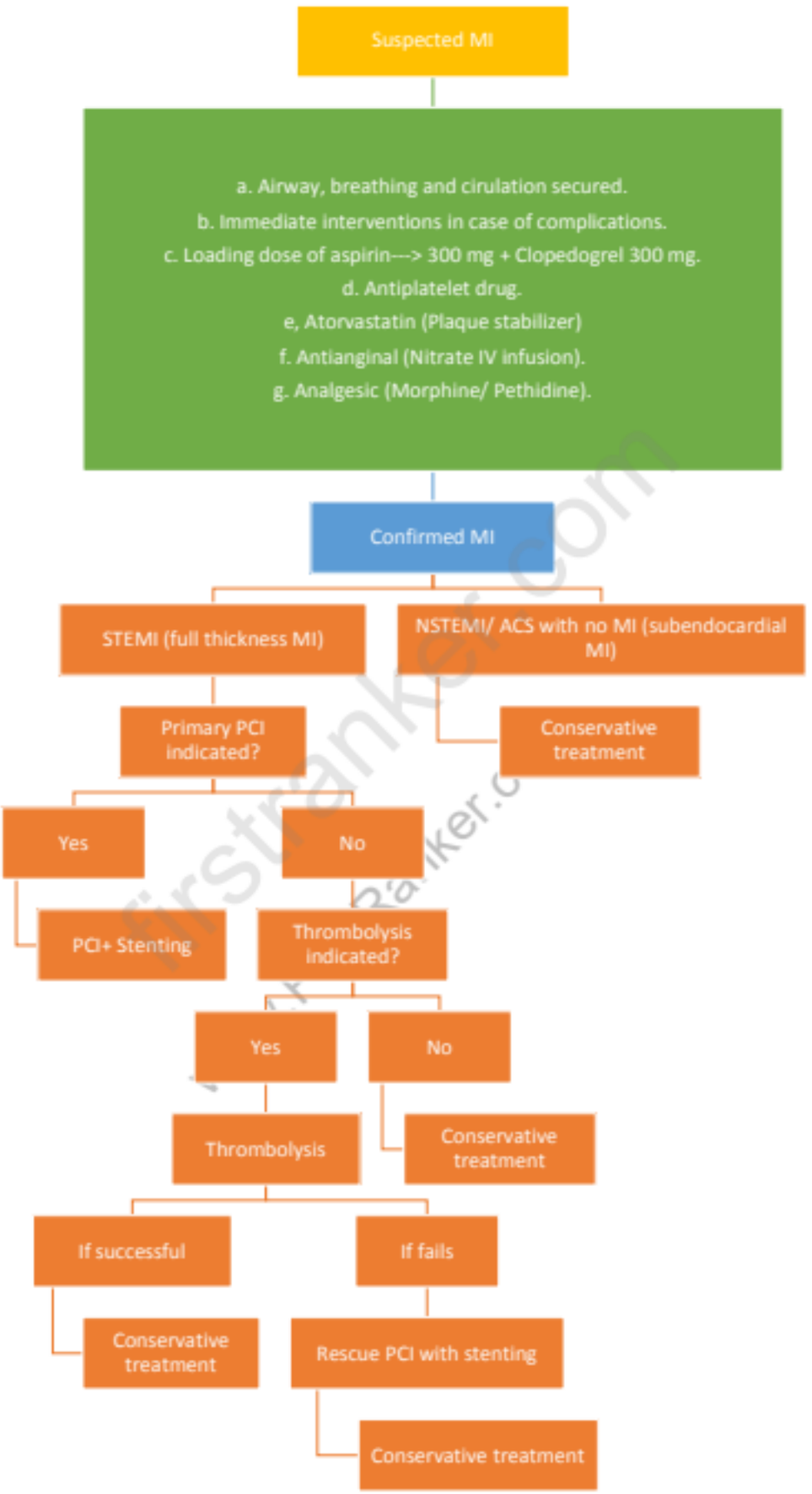


3. Echocardiogram:
   It's one of the most sensitive tests to diagnose AMI. It can potentially show:
   a. Regional wall motion abnormality/ akinetic wall.
   b. Degree of cardiac dysfunction.
   c. Acute MR/ VSD/ Tamponade.
4. Coronary angiogram.

Risk assessment

1. Hb/ TC/ DC.
2. Urea-creatinine Na+ K+.
3. Serum Mg++ and Ca++.
4. Lipid profile.
5. Fasting and postprandial blood sugar.

Treatment protocol of a suspected case of MI



What do you mean by conservative treatment of MI?

It is long term treatment, usually started from the very beginning.

It consists of 2 parts:

a. Risk factor modification.
b. Specific treatment.

Risk factor modification

It includes:

1. Lifestyle modification and,
2. Pharmacotherapy.

A: ABDOMINAL OBESITY: Regular exercise and reduction of abdominal fat.

B: BLOOD LIPID ABNORMALITY: Regular monitoring and control of LDL levels.

C: CIGARETTE: Reduction/ cessation of cigarette smoking.

D: DIABETES: Strict monitoring and control of blood sugar.

E: EXCESS ALCOHOL: Avoidance/ complete cessation of alcohol ingestion.

F: FAMILY HISTORY: Non-modifiable risk factor.

G: GENDER (MALE> FEMALE): Non-modifiable risk factor.

H: HYPERTENSION: Treat hypertension adequately and monitor BP on a regular basis.

Specific treatment

1. Antiplatelet:
    - Aspirin: Lifelong.
    - Clopidogrel/ Prasugrel/ Ticagrelor: Continued for 9-12 months after stenting (upto 24 months).
2. Anticoagulant:
    - Unfractionated heparin.
    - Low molecular weight heparin (LMW-Heparin).
    - Fondaparinux.
3. Atorvastatin/ any other statins.
4. ACE inhibitor/ ARB.
5. Anti-anginal agents: Nitrate/ Nicorandil/ Ranolazine.

6. Beta blocker: Metoprolol/ Bisoprolol/ Carvedilol.
7. CCB: Very limited role in MI. If at all to be used, then following are used:
   a. Amlodipine.
   b. Diltiazem.

Thrombolysis in MI

Indications:

1. Ongoing chest pain with >2 mm of ST segment elevation in 2 inferior leads/ at least 2 contagious precordial leads (Ex.: V1, V2; V5, V6 etc.).
2. Ongoing chest pain with new onset left bundle branch block (LBBB), as a STEMI may disguise in this condition.

- Thrombolysis is strongly indicated if the above ECG changes are present in a patient with ongoing chest pain if the patient presents *within 3 hours of onset of chest pain*. After this period, effectiveness of thrombolysis progressively declines; however the period can be stretched maximum upto 12 hours.

Drugs used:

1. Recombinant tissue plasminogen activator (Recombinant tPA).
   Ex.:
   Alteplase,
   Reteplase,
   Tenecteplase.
2. Streptokinase.

Contraindication:

1. H/O intracerebral hemorrhage.
2. H/O recent ischemic stroke.
3. Known case of intracerebral space occupying lesion (SOL).
4. Any active bleeding (excluding menstruation).

Complication:

Minor/ severe bleeding manifestations.

Criteria for successful thrombolysis:

1. Significant relief of chest pain.
2. 90 minutes post-thrombolysis ECG shows at least 50% resolution of ST elevation.

3. Appearance of re-perfusion arrhythmia.

Primary PCI in MI

Immediate revascularization of blocked coronary artery by putting a coronary artery stent in a patient of STEMI is called Primary PCI with stenting.

It is usually considered if the patient presents within 90 minutes (upto maximum 2 hours) of onset of chest pain.

Complications of MI

- Immediate/ early (within few days):
  a. Acute cardiogenic shock.
  b. Acute heart failure (RHF/ LHF).
  c. Arrhythmia.
  d. Acute MR/ Acute VSD.
  e. Acute cardiac tamponade.
  f. Acute pericarditis.
- Delayed (after few weeks):
  a. Dressler's syndrome#.
  b. Aneurysmal dilation of ventricular wall, which may lead to systemic embolism.

Dressler's syndrome/ Post MI syndrome is a secondary form of pericarditis that occurs typically 2-3 weeks after an episode of MI due to an autoimmune reaction against the leaked myocardial proteins; the classical presentation includes fever, pleuritic chest pain and pericardial effusion, usually treated with colchicine.

Heart failure

It is a condition where heart is unable to meet the metabolic need of the tissue (in spite of a normal venous return).

Congestive cardiac failure (CCF): Cardiac failure with congestion behind the chamber that has failed.

Ex.:

In case of LHF, there is pulmonary congestion (congestion behind the LA).

In case of RHF, there is systemic congestion (congestion behind the RA).

Types:

1. On the basis of onset:
   a. Acute HF.
   b. Chronic HF.
2. On the basis of chamber:
   a. LHF.
   b. RHF.
3. On the basis of mechanism of clinical manifestation:
   a. Forward failure (features of suboptimal CO, less prominent).
   b. Backward failure (congestive features, more prominent).
4. On the basis of cardiac output:
   a. Low output failure.
   b. High output failure (Ex.: anemia/ thyrotoxicosis/ pregnancy).

An important concept about HF:

By the time diastole ends, each ventricle has filled up with blood. This amount of blood is the end diastolic volume or EDV (~120 ml).

The amount of blood ejected during the systole is the stroke volume (~70 ml).

At the end of systole the volume of blood remaining in each ventricle is the end systolic volume or ESV (~50 ml). So,

SV (70 ml) = EDV (120 ml) - ESV (50 ml)

$$Ejection\ fraction = \frac{SV}{EDV} = \frac{70}{120} = 58\%\ (Range\ 55 - 70\%).$$

5. So, according to ejection fraction, HF is of 2 types:
   a. *Preserved ejection fraction*: Also referred to as diastolic heart failure. The heart muscle contracts normally but the ventricles do not relax as they should during ventricular filling (or when the ventricles relax).
   b. *Reduced ejection fraction:* Also referred to as systolic heart failure. The heart muscle does not contract effectively and less oxygen-rich blood is pumped out to the body.

Causes of heart failure:

- Causes of LHF:
  1. Systemic hypertension,
  2. Valvular heart disease,
  3. Cardiomyopathy,
  4. Ischemic heart disease,
  5. Myocarditis.
- Causes of RHF:
  1. Secondary to LHF.
  2. Secondary to chronic lung disease:
     a. Diseases of airway: COPD, Bronchiectasis.
     b. Diseases of parenchyma: Interstitial lung disease.
  3. Pulmonary vascular disease:
     a. Acute/ chronic pulmonary thromboembolism.
     b. Primary/ idiopathic PAH.
  4. Congenital heart disease.
  5. Valvular heart disease.
  6. Cardiomyopathy.
  7. IHD.
  8. Myocarditis.

Signs and symptoms of heart failure

LHF

Symptoms:

- Due to pulmonary congestion:
  1. Dyspnoea.
  2. Orthopnoea.

3. PND.

- Due to low cardiac output:
  1. Exertional fatigue.
  2. Muscle pain.
  3. Reduced endurance.
- Due to underlying etiology:
  Symptoms due to underlying etiology and any complication that may arise from it.

Signs:

- Gallop rhythm (S3/S4).
- Bibasal fine respiratory crepitation.
- Patient may be hemodynamically unstable in case of acute LVF (which may give rise to cardiogenic shock).

RHF

Symptoms:

- Swelling of the body usually starting in the lower limb.
- Symptoms of LHF.
- Symptoms of underlying chronic lung disease.
- Symptoms of any underlying etiology responsible for RHF.

Signs:

- Raised JVP.
- Bilateral pitting edema.
- Soft tender hepatomegaly.
- Signs due to LHF/ CLD/ any other etiology.

Investigations

1. Hb/ TC/ DC/ ESR or CRP.
   Note: As anemia is an aggravating factor of HF, Hb estimation is important.
2. Urea-creatinine Na+ K+: It should be monitored regularly.
3. LFT: Non-specifically deranged due to *congestive hepatitis.*
4. Arterial blood gas: It is used to assess the degree of hypoxia in acute HF.

5. BNP (Brain natriuretic peptide)/ N terminal pro BNP:
   Usually elevated if ventricular filling pressure rises, which almost invariably occurs in HF. However, it is also raised in presence of LV dilation/ stretching/ significant stress.
6. ECG: May show signs of underlying etiology/ complication(s)/ chamber enlargement.
7. CXR: Helpful in diagnosing:
   a. Acute pulmonary edema.
   b. Cardiomegaly.
8. Echocardiogram: It may show:
   a. Any systolic/ diastolic dysfunction.
   b. Degree of cardiac dysfunction.
   c. Any structural abnormality.
9. Other relevant investigations must be done to diagnose the etiology/ cardiovascular risk factors.

Treatment of HF

It may be divided into 3 groups:

1. Treatment of HF.
2. Treatment of etiology.
3. Treatment of any aggravating/ precipitating factor(s).

Treatment of acute LVF

A. Airway:
   If not patent, (suction +/ intubation) is indicated.
B. Breathing:
   - Moist oxygen inhalation.
   - Assisted ventilation: Non-invasive/ invasive.
   - Nebulised bronchodilator (to treat cardiac asthma/ bronchospasm).
C. Circulation:
   In case of cardiogenic shock, contractility enhancing drugs (inotropes) like IV Dopamine/ Dobutamine is indicated.
   Milrinone is a phosphodiesterase 3 (PDE-3) inhibitor that has both inotropic and vasodilator properties, so it is called an "*inodilator*" drug. It also may be used.

D. Drugs:
   1. Diuretics (Furosemide/ Torsemide/ Bumetanide).
   2. Dilators:
      There are 2 types of dilators used in treatment of acute LVF:
      a. Venodilators: Decreases preload.
         Ex.: IV Nitroglycerine infusion.
      b. Arteriolar dilator: Decreases afterload.
         Ex.: Nitroprusside, Nesiritide (It is a recombinant BNP stimulating cGMP, resulting in cardiac smooth muscle relaxation).

   Diet:
   Salt and fluid restriction.
   Daily monitoring of fluid intake and renal function (Urea-creatinine Na+ K+).
E. Etiological treatment.
F. IV Morphine:
   - Analgesia.
   - Reduction of anxiety.
   - Reduction of heart rate.
   - Peripheral venous pooling and reduction of preload.
   - Peripheral vasodilation and reduction of afterload.
   - Reduction in pulmonary congestion.
     - All of which results in a reduction in myocardial oxygen demand.

Treatment of chronic heart failure

1. ACE Inhibitors:
   - Captopril.
   - Ramipril.
2. ARB:
   - Losartan.
   - Telmesartan.
3. Aldosterone antagonist:
   - Spironolactone.
   - Eplerenone.
4. β-blocker:
   - Carvedilol (α+β1 blocker).
   - Bisoprolol.

They should be started cautiously at a low dose and then uptitrated gradually.

5. Contractility enhancing agent:
   - Digoxin.

   However, its role is very limited and it is indicated only in patients having severe systolic dysfunction with coexisting atrial fibrillation.

6. Calcium channel blocker:
   It is usually AVOIDED in patients with chronic HF because of its negative inotropic effect.
7. Diuretics:
   - Furosemide.
   - Torsemide.
   - Bumetanide.
8. Vasodilators:
   - Venodilator: Reduces preload: Oral nitroglycerine/ GTN.
   - Arteriolar dilator: Reduces afterload: Hydralazine.
9. Diet:
   Salt and fluid restriction.
10. Etiologic treatment.
11. Treatment of exacerbating factors.
12. External interventions:
    - Implantation of biventricular pacemaker (Resynchronization).
    - Implantation of implantable defibrillator.
    - Cardiac transplantation.

Treatment of RHF

1. Diuretics.
2. Dietary salt and fluid restriction.
3. Monitoring of volume status:
   - Regular body weight estimation.
   - Monitoring of fluid intake and output.
   - Regular monitoring of renal function (Urea-creatinine Na+ K+).
4. Treatment of LHF, if present.
5. Treatment of chronic lung disease, if present.
6. Treatment of underlying etiology.

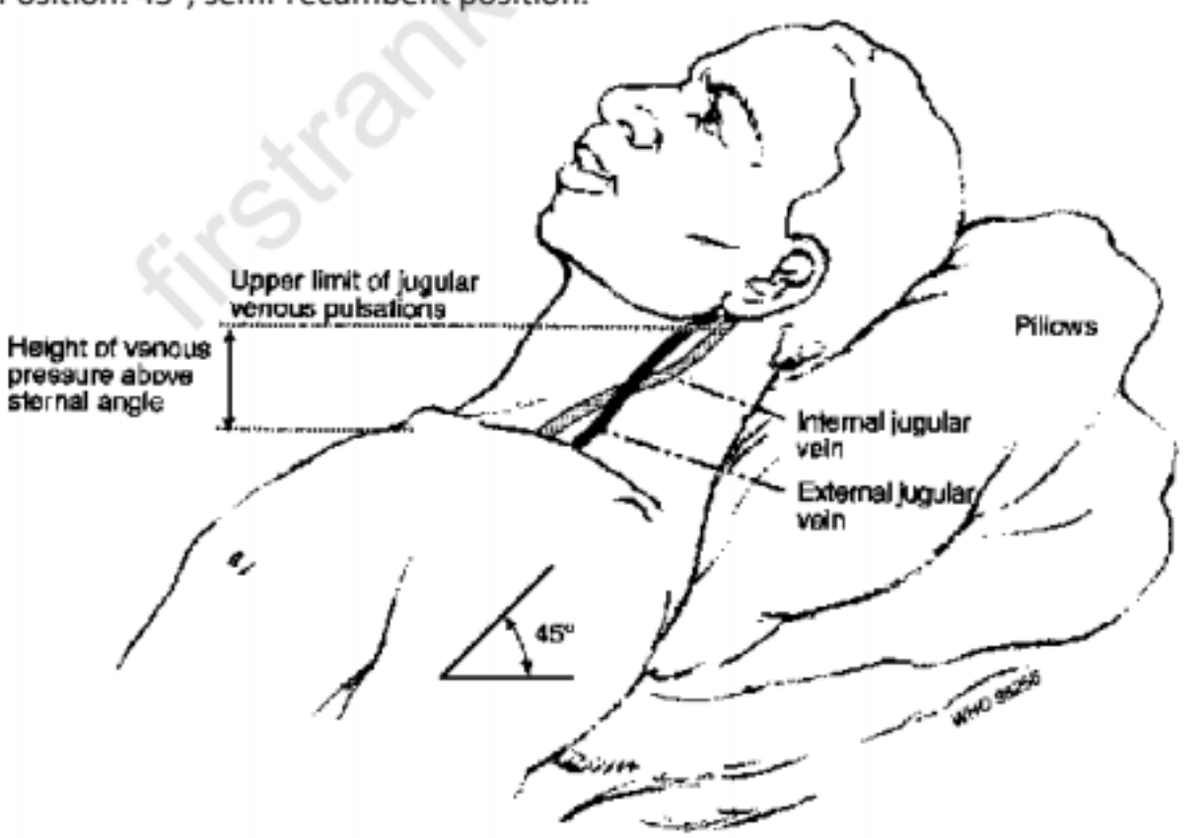
Important signs of CVS pathology

Neck vein

- Importance:
  It reflects any internal abnormality in right side of heart, which may be mechanical/ electrophysiologic/ hemodynamic.
- Which vein is examined?
  Internal jugular vein (IJV).
- Why IJV is preferred over EJV?

IJV	EJV
IJV is a direct branch of SVC.	EJV is a not a direct branch of SVC.
There are no valves in IJV.	There are 2 pairs of valves in EJV.
Venous wave form can propagate continuously.	Venous wave form can't propagate continuously.
	It lies in a more superficial course, so may engorge due to peripheral factors.

- Position: 45°, semi-recumbent position.



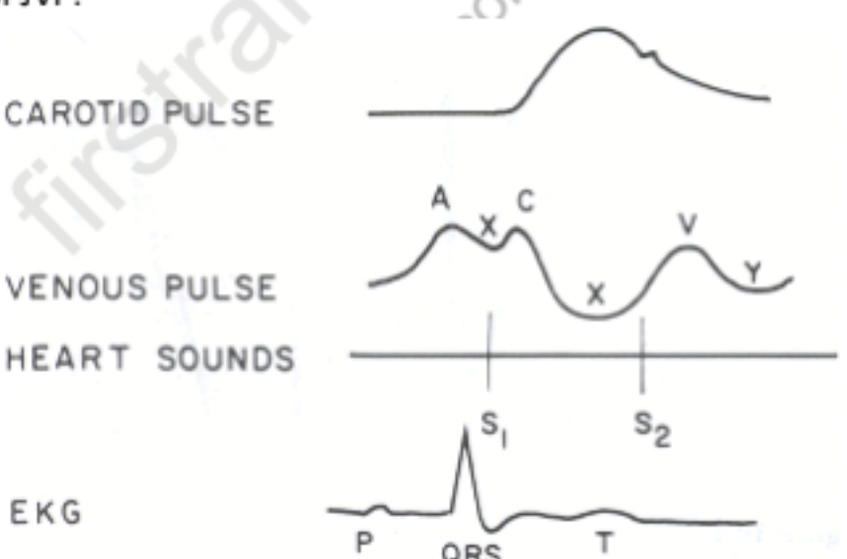
- How will you differentiate between jugular venous pulse and carotid pulsation?

Jugular venous pulse	Carotid pulse
Better seen than felt.	Definite feel of thrust.
Can be obliterated by pressing between two heads of sternocleidomastoid.	No change.
The upper border of waveform shifts with inspiration and expiration.	
Abdominojugular reflux positive.	Negative.
May/ may not coincide with arterial pulsation and S1.	Definitely coincide.

- Abnormalities of JVP:

Pathology	Conditions
Volume overload	Right heart failure.
	Chronic kidney disease.
Impaired venous return	Constrictive pericarditis.
	Pericardial effusion.
Raised JVP with a non-pulsatile wave (no wave is seen, only engorgement is seen)	SVC obstruction.

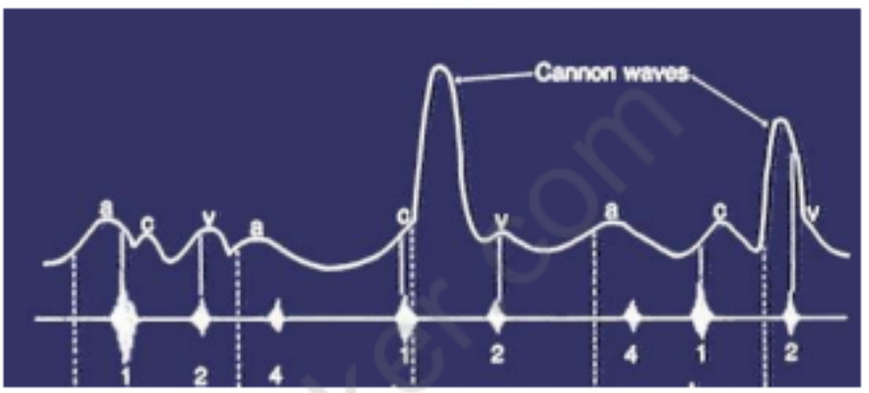
- Waves of JVP:

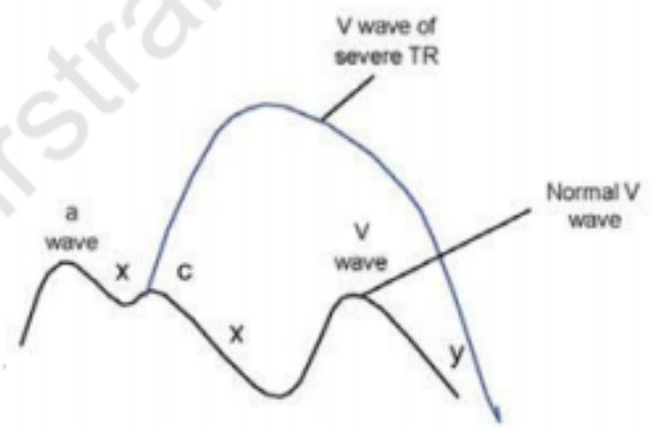


Name of wave	Event in the heart	Pathology
A wave	Right atrial systole.	Elevated in TS/PS/PAH. Absent in AF. Cannon A wave in AV dissociation.
C wave	Transient bulging of TV into RA	

	at the beginning of ventricular systole. (Usually not seen)	-
X descent	Right atrial relaxation.	Absent in TR.
V wave	Right atrial venous filling.	Elevated in TR.
Y descent	Rapid fall of RA pressure at the beginning of ventricular systole.	Rapid in TR.

AF





Cyanosis

- It is defined as bluish discoloration of skin and mucous membrane due to presence of abnormal quantity of deoxygenated Hb.
- Amount of deoxygenated Hb required to cyanosis become evident is 5gm/dl. However, in clinical life, 2.5 gm/dl is often enough to produce it.

- Causes of cyanosis:

A. *Impaired oxygen supply:*
   1. High altitude.
   2. Hypoxemic respiratory condition.
   3. Hyperventilation.
   4. Right to left shunt:
      - Intra-cardiac:
        - ✓ Tetralogy of Fallot.
        - ✓ VSD with reversal of shunt.
      - Great vessels:
        - ✓ Transposition of great vessels.
        - ✓ PDA with reversal of shunt.

B. *Circulatory failure:*
   Cardiogenic shock.

C. *Circulatory obstruction:*
   - Arterial thrombosis.
   - Venous thrombosis.

D. *Peripheral vasoconstriction:*
   1. Raynoud's phenomenon.
   2. Transient cyanosis.

- It is of 2 types:

A. *Central cyanosis:* Impaired oxygenation and circulatory failure will lead to cyanosis both in central and peripheral sites.
   Sites:
   1. Tongue.
   2. Buccal mucosa.
   3. Inner aspect of lips.

   Note:

   Central cyanosis may/ may not disappear on oxygenation depending on cause.

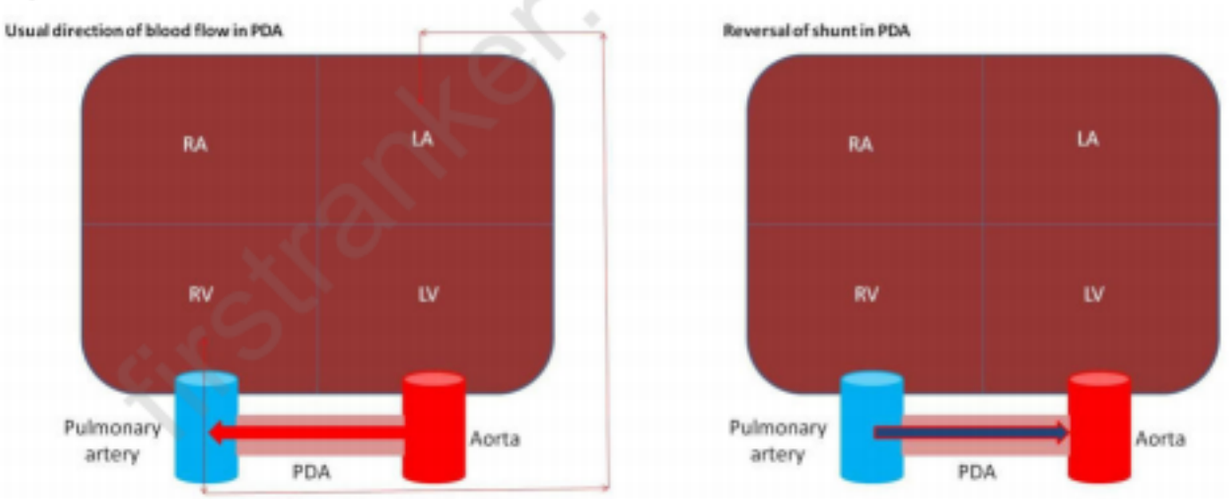
B. *Peripheral cyanosis:* Circulatory obstruction and local vasoconstriction will lead to cyanosis in peripheral sites.
   Sites:
   1. Extremities.

2. Finger tips.
3. Tip of nose.
4. Ear lobule.
5. Outer surface of lips.

Note:

Peripheral cyanosis usually disappears on oxygenation.

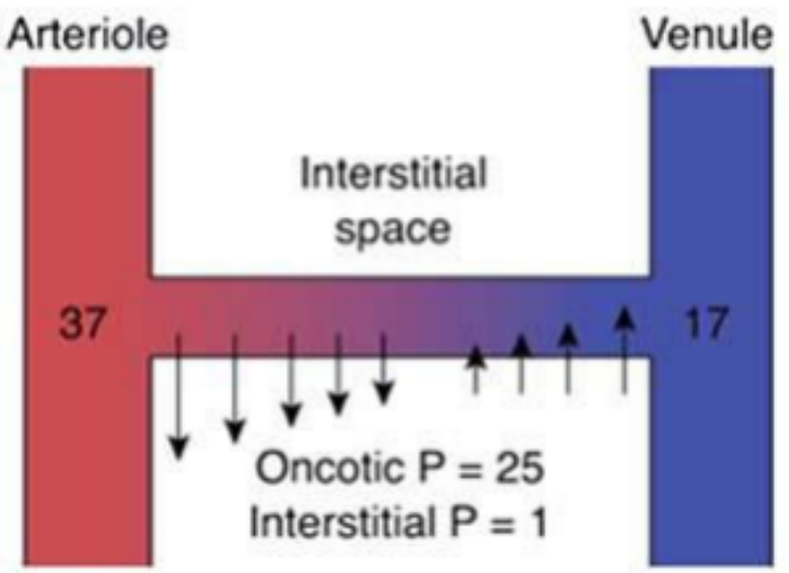
- Unilateral cyanosis can occur due to local causes:
  A. Circulatory obstruction.
  B. Local vasoconstriction.
- *Differential cyanosis:*
  It is characterized by cyanosis present in lower limb but not in upper limb.
  It is seen in reversal of shunt in case of PDA, as the deoxygenated blood from the pulmonary circulation will shunt through the PDA to the descending aorta causing the cyanosis to be seen in the lower limbs only.



- *Methemoglobinemia:*
  In this condition, patient looks bluish and therefore, looks cyanosed.

Oedema

- It is defined as collection of fluid in the subcutaneous tissue.
- Pathogenesis:
  - The diagram below shows the actual change in pressure gradients at the capillary level. The numbers represent the pressure in the vessels in mmHg.



- At the arteriolar end, the hydrostatic pressure (outward force) is 37 mmHg while the oncotic pressure and interstitial pressure (inward forces) are 25 and 1 mmHg respectively. Thus the net outward force is 37 – (25+1) = +11 mmHg. Since the net outward force is positive fluid moves from the capillary to the interstitial spaces.
- At the venular end, the hydrostatic pressure is lower and has a value of 17 mmHg. The oncotic and interstitial pressure on the other hand remain the same i.e. a total of 26 mmHg (25+1). Thus the net outward force is 17 – (25+1) = -9 mmHg. Since the outward force is negative, it means fluid is not getting out of the capillary but instead it is moving into it.
- Either increased hydrostatic pressure, diminished colloid osmotic pressure or inadequate lymphatic drainage can result in an abnormally increased interstitial fluid i.e. edema.

- Causes:
  A. Increased capillary hydrostatic pressure:
     1. RHF.
     2. Chronic kidney disease.
     3. Constrictive pericarditis.
     4. Pericardial effusion.
  B. Decreased colloidal oncotic pressure:
     1. Chronic liver disease.
     2. Nephrotic syndrome.
     3. Protein losing enteropathy.

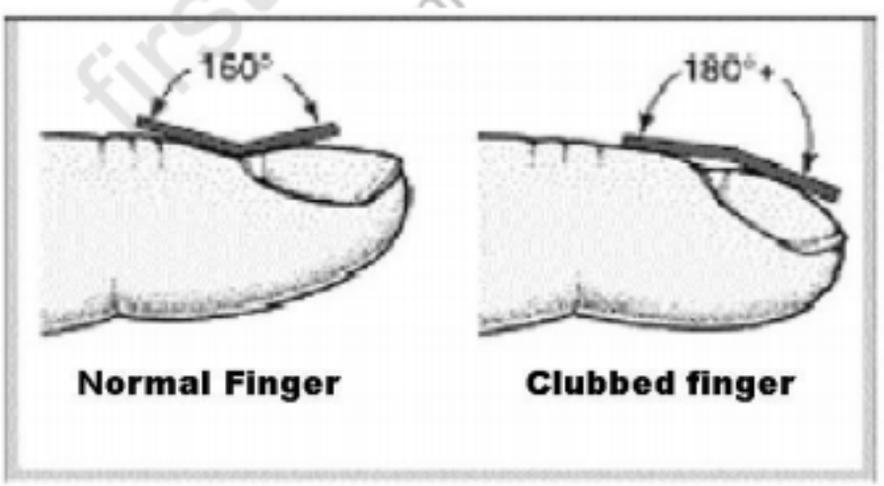
C. Unilateral/ localized edema:
   1. Deep vein thrombosis.
   2. Cellulitis.
   3. Thrombophlebitis.
   4. Lymphatic obstruction and myxoedema (non-pitting edema).

Clubbing

Degrees of clubbing:

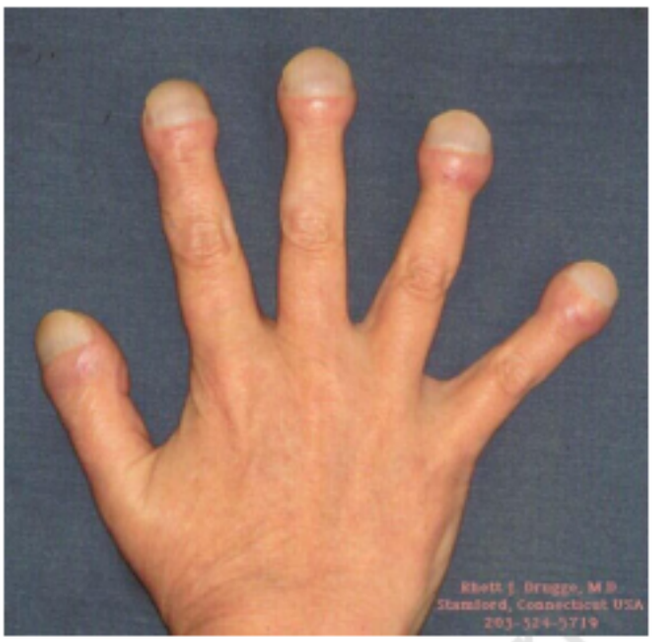
1°: Fluctuating nail bed (obliteration of onycho-dermal angle).

2°: Increased antero-posterior and transverse diameter of nail bed (obliteration of Lovibond angle: the angle between the base of the nail and the nail itself).

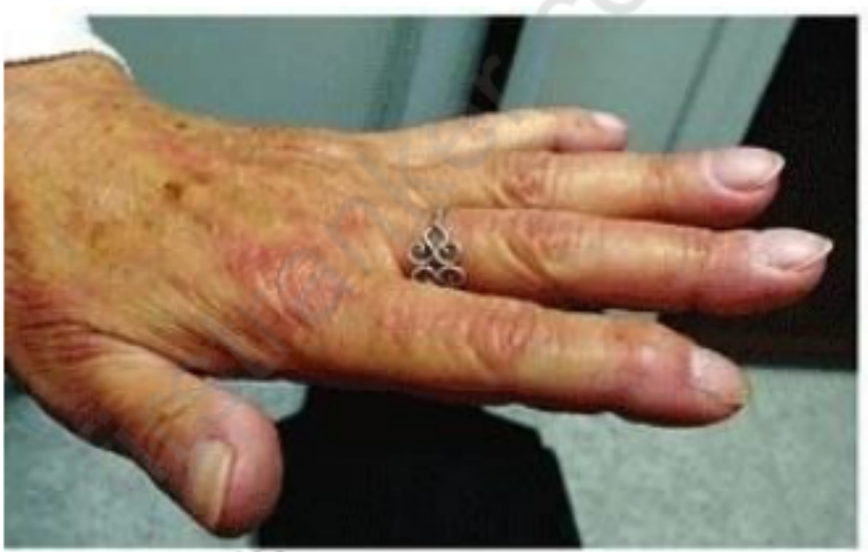


Lovibond's angle

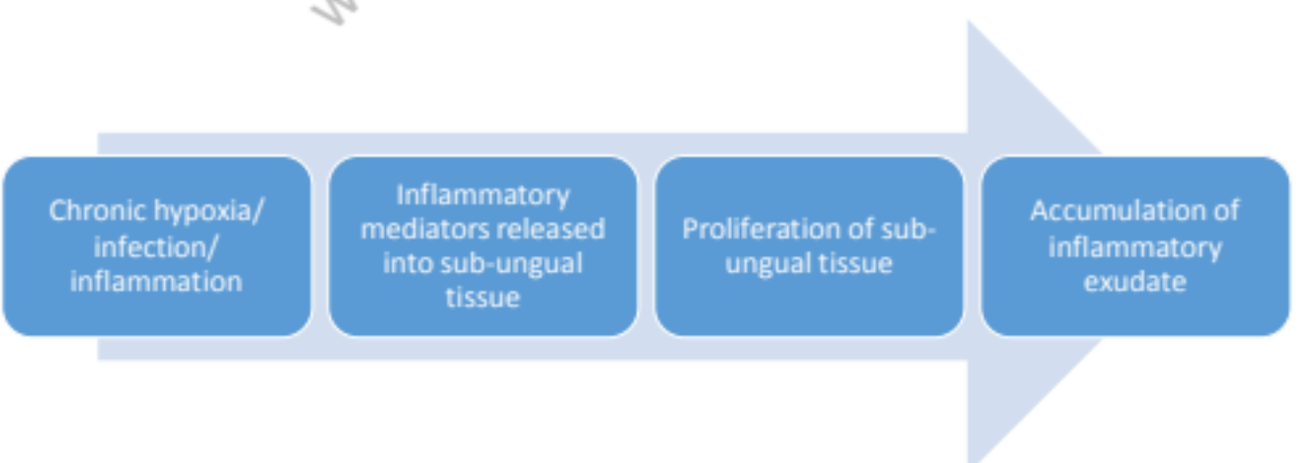
3°: Bulbous enlargement of pulp of the finger (Parrot beak/ drum stick appearance).



4°: Hypertrophic Osteo Arthropathy (HOA).



Pathophysiology of clubbing:



Causes:

1. Respiratory disease:
   a. Bronchiectasis,
   b. Lung abscess,
   c. Bronchogenic CA,
   d. Diffuse interstitial lung disease,
   e. TB.
2. Cardiovascular disease:
   a. Right to left shunt (Ex.: Tetralogy of Fallot),
   b. Subacute bacterial endocarditis.
3. GIT:
   a. Ulcerative colitis,
   b. Cirrhosis of liver,
   c. Primary biliary cirrhosis.
4. Idiopathic.

Note: Hypertrophic osteo-arthropathy (HOA):

(Clubbing + HOA representing 2 extreme points of a same spectrum).

Cause:

Bronchogenic CA.

Clinical feature:

In HOA, there is sub-periosteal new bone formation at the distal ends of long bones (Radius-ulna/ Tibia-fibula etc.), leading to bone pain.

Diagnosis: X-Ray.