PERICARDIAL DISEASES

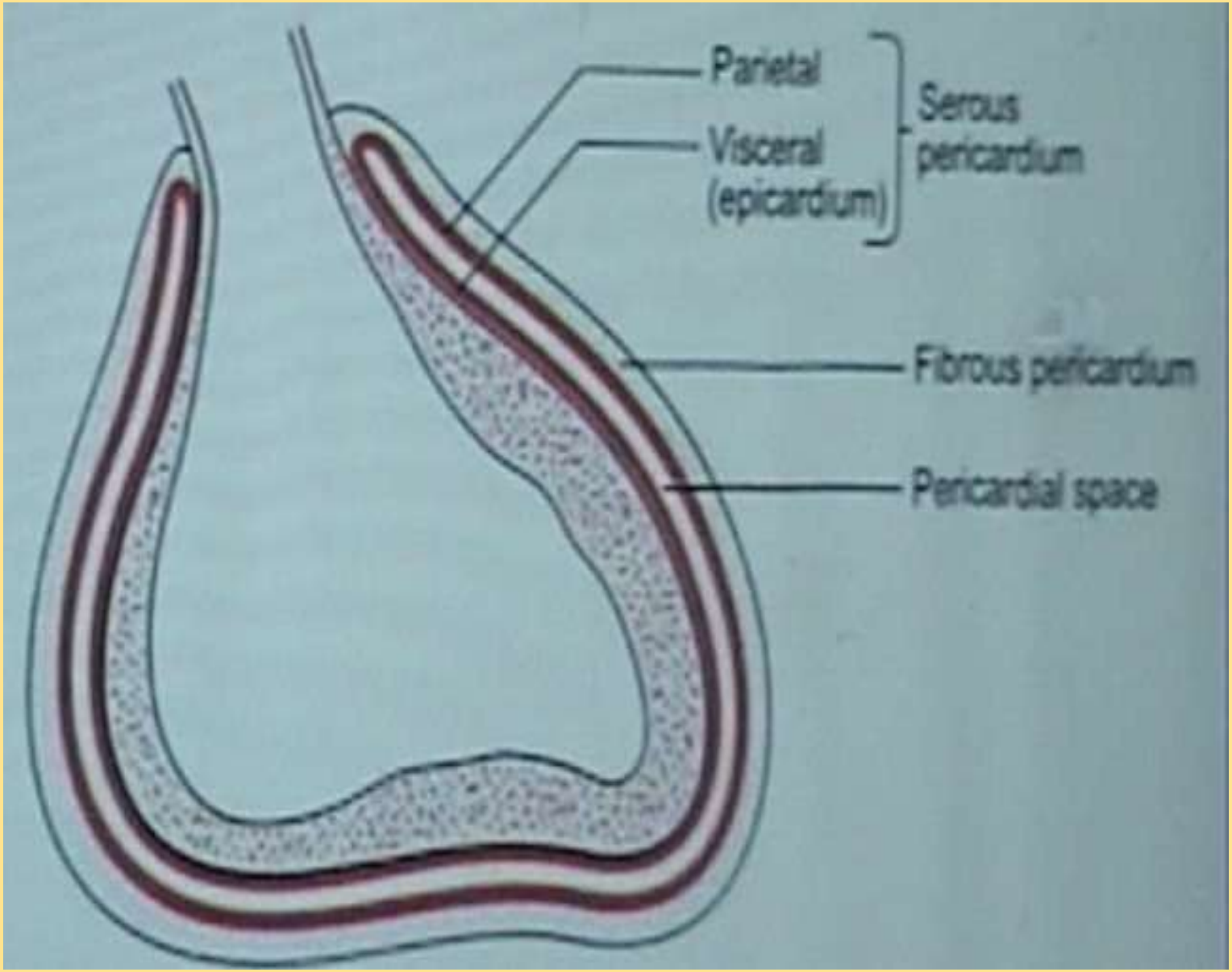
OVERVIEW

- Introduction
- Acute pericarditis
- Constrictive pericarditis
- Pericardial effusion
- Cardiac tamponade

PERICARDIUM IS THE SAC THAT SURROUNDS THE HEART MADE UP OF

- OUTER FIBROUS PERICARDIUM
- inner serous pericardium (parietal & visceral)

- **Pericardial fluid :**
  - up to 50 ml of clear plasma ultrafiltrate between the two layers of the serous pericardium


Parietal
Visceral
(epicardium)
Serous
pericardium
Fibrous pericardium
Pericardial space

FUNCTIONS

1.Stabilization of the heart within the thoracic cavity by virtue of its ligamentous attachments -- limiting the heart's motion.

2.Protection of the heart from mechanical trauma and infection from adjoining structures.

3.The pericardial fluid functions as a **lubricant** and decreases friction of cardiac surface during systole and diastole.

4.Prevention of excessive dilation of heart especially during sudden rise in intra-cardiac volume (e.g. acute aortic or mitral regurgitation).

ASSESSMENT

1. WHAT IS PERICARDIUM ?

2. MENTION THE FUNCTIONS OF PERICARDIUM

ACUTE PERICARDITIS

PERICARDITIS –DEFINITION

- Inflammation Of Pericardium Due To External Causes With No Defect In Primary Pericardium.
- Inflammation of the pericardium , the membranous sac enveloping the heart.

ETIOLOGY

COMMON CAUSES	LESS COMMON CAUSES
Tuberculosis	Rheumatic fever
Viral – Mumps, Varicella, Rubella ,	Uremia
Post myocardial infarction syndrome	Malignant Disease
Connective tissue –Diseases -SLE,RA	Hypothyroidism
Acute myocardial infarctions	Trauma
	Radiation Therapy

ACUTE PERICARDITIS

- Most common pathologic process involving the pericardium.

- Classification Of Pericarditis :
  - Clinical
  - Etiological

CLASSIFICATION

Acute Pericarditis : -< 6 Weeks

Sub Acute – 6 Weeks : – 6 months

Chronic pericarditis : > 6 months

CLASSIFICATION – CONT…

BASED ON UNDERLYING CAUSE :

- Acute Fibrinous Pericarditis
- Acute serous pericarditis
- Acute caseous pericarditis
- Acute Purulent pericarditis

CLASSIFICATION CONT

- ACUTE FIBRINOUS PERICARDITIS
- CAUSES: Post MI, Uremia, Chest Radiation, Rheumatic fever, SLE
- Thick , creamy fluid in pericardial sac
- Pericardium with fine Granular Roughning

CLASSIFICATION CONT

- Acute caseous pericarditis
- Cause : Tb
- Grey, white cheese like material accumulated in pericardial sac

- ACUTE SEROUS PERICARDIUM
- CAUSES: Non infectious inflammation ,tumours, uremia
- 50-200 ML serous fluid accumulates in pericardial space,
- Microscopically inflammatory infiltrate comprising neutrophils,, lymphocytes ad macrophages seen.

- ACUTE PURULENT PPERICARTIS :
- CAUSS: Bacterial infections
- 400-500 ml pus in pericardial sac,
- Red Granular surface pericardium

CLASSIFICATION

- ACUTE HEMORRHAGIC PERICARDITIS
- CAUSES: Tumours, TB , Bleeding
- Blood mixed serous ,or fibrinous fluid accumulated in pericardial sac .

CHRONIC PERICARDITIS

- CHRONIC ADHESIVE PERICARDITIS
- CAUSES: Post TB , Post cardiac interventions, Post radiation, Post Bacterial infections
- Dense adhesion between the layers of pericardium

CHRONIC PERICARDITIS

- CHRONIC CONSTRICTIVE PERICARDITIS
- Resembles RCM
- No Specific Cause
- Limited diastolic expansion

ETIOLOGICAL CLASSIFICATION

T = Trauma, Tumour
U = Uremia
M = Myocardial infarction (acute, post)
Medications (hydralazine)
O = Other infections (viral,bacterial, fungal, TB)
R = Rheumatoid, autoimmune disorder Radiation

CLINICAL FEATURES

Preceded by fever, malaise and myalgia

Common characteristics of pain

retrosternal or precordial with radiation to the trapezius ridge, neck, back, left shoulder or arm

Special characteristics of pericardial pain

more likely to be sharp

↑ with coughing, inspiration, swallowing

worse by lying supine, relieved by sitting and leaning forward

PERICARDITIS VS MI

Characteristic/Parameter	Pericarditis	Myocardial infarction
Pain description	Sharp, pleuritic, retro-sternal (under the sternum) or left precordial (left chest) pain	Crushing, pressure-like, heavy pain. Described as "elephant on the chest."
Radiation	Pain radiates to the trapezius ridge (to the lowest portion of the scapula on the back) or no radiation.	Pain radiates to the jaw, or the left or arm, or does not radiate.
Exertion	Does not change the pain	Can increase the pain
Position	Pain is worse in the supine position or upon inspiration (breathing in)	Not positional
Onset/duration	Sudden pain, that lasts for hours or sometimes days before a patient comes to the ER	Sudden or chronically worsening pain that can come and go in paroxysms or it can last for hours before the patient decides to come to the ER

CLINICAL FEATURES

- Triphasic friction rub is pathognomonic; scratching or grating sound;
- Best heard in the lower LSB with the patient sitting and leaning forward

Pericardial rub	Pleural rub
Can be heard even after cessation of breathing	Can be heard only during inspiration and expiration
Heard mostly over the sternum or sternal borders	Heard mostly over the lateral parts of the chest
Intensity doesn't increase with increased pressure of the steth	Intensity of rub increases with increased pressure of the steth over the chest wall

ELECTROCARDIOGAPHICDIFFERENTIATION OF PERICARDITIS

Acute pericarditis	Acute myocardial infarction	Early repolarization
ST-segment elevation in many leads, with no ST-segment depression	ST-segment elevation in anatomically contiguous leads, with possible reciprocal ST-segment depression	ST-segment elevation in middle and left precordial leads, but may be widespread
Upward concave ST-segment elevation	Upward convex ST-segment elevation	Upward convex ST-segment elevation
No T-wave inversion in leads with ST-segment elevation	T-wave inversion in leads with ST-segment elevation as myocardial infarction evolves	May have T-wave inversion in leads with ST-segment elevation
PR-segment depression		No PR-segment depression

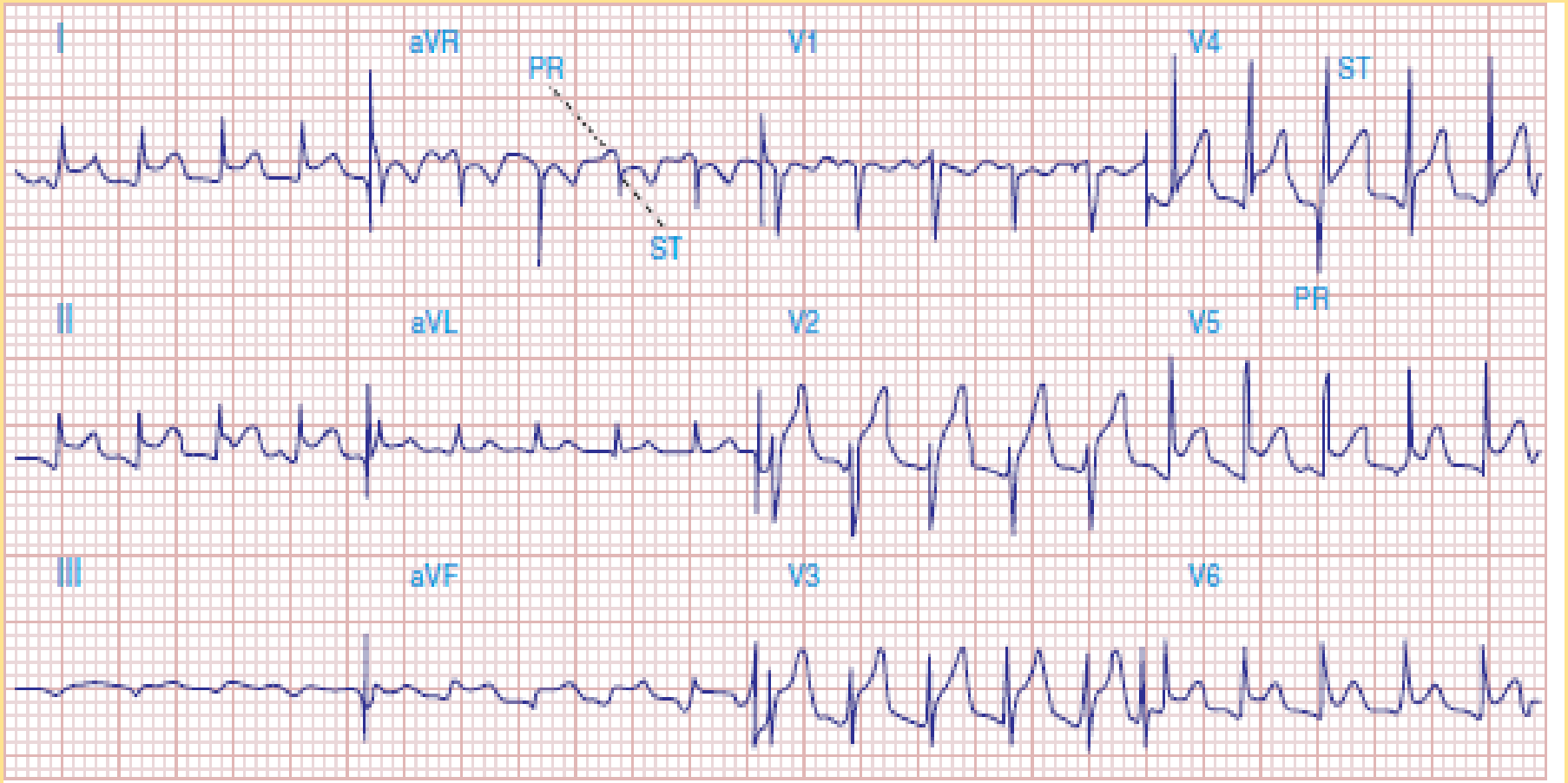
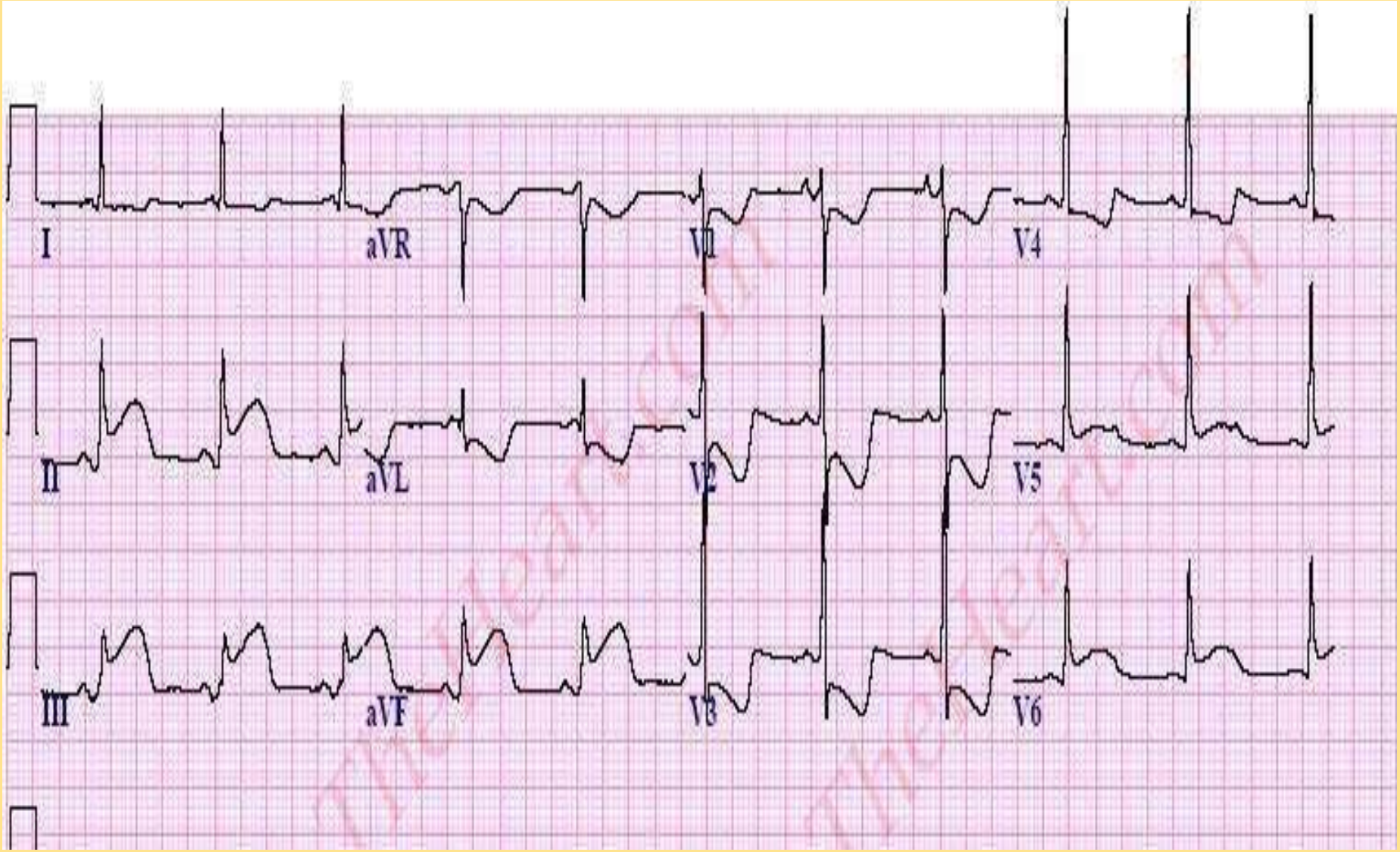


FIGURE 232-1 Acute pericarditis often produces diffuse ST-segment elevations (in this case in leads I, II, aVF, and V_2 to V_6) due to a ventricular current of injury. Note also the characteristic PR-segment deviation (opposite in polarity to the ST segment) due to a concomitant atrial injury current.


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6

ACUTE PERICARDITIS MANAGEMENT

- Treat underlying cause
- Analgesic agents
  - codeine 15-30 mg q 4-6 hrs
- Anti-inflammatory agents
  - Aspirin
  - NSAID (indomethacin 25-50 mg qid)
  - Corticosteroids are symptomatically effective , but preferably avoided

PERICARDITIS AFTER AMI

Early

- Occurs - 1 to 3 days (no more than a week
- due to transmural necrosis with pericardial inflammation
- 40% of patients with large, Q-wave MIs have pericarditis
- Benign
- aspirin doses (650 mg orally three or four times per day for 2 to 5 days) or acetaminophen is usually effective

Late (Dresslers Syndrome)

- Occurs - 1 week to a few months after AMI .
- autoimmune etiology
- 3% to 4%.
- Polyserositis with pericardial or pleural effusions

- Aspirin , Colchicine .
- Prednisone, 40 to 60 mg /d with a 7- to 10-day taper(If not responding to treatment or for recurrent symptoms)

CONSTRICTIVE PERICARDITIS

ETIOLOGY

- Idiopathic or viral — 42 to 49 %
- Post cardiac surgery — 11 to 37 %
- Post radiation therapy — 9 to 31 %
- Connective tissue disorder — 3 to 7 %
- Postinfectious (tuberculous or purulent pericarditis) — 3 to 6 %
- Miscellaneous causes (malignancy, trauma, drug-induced, asbestosis, sarcoidosis, uremic pericarditis) — 1 to 10 %

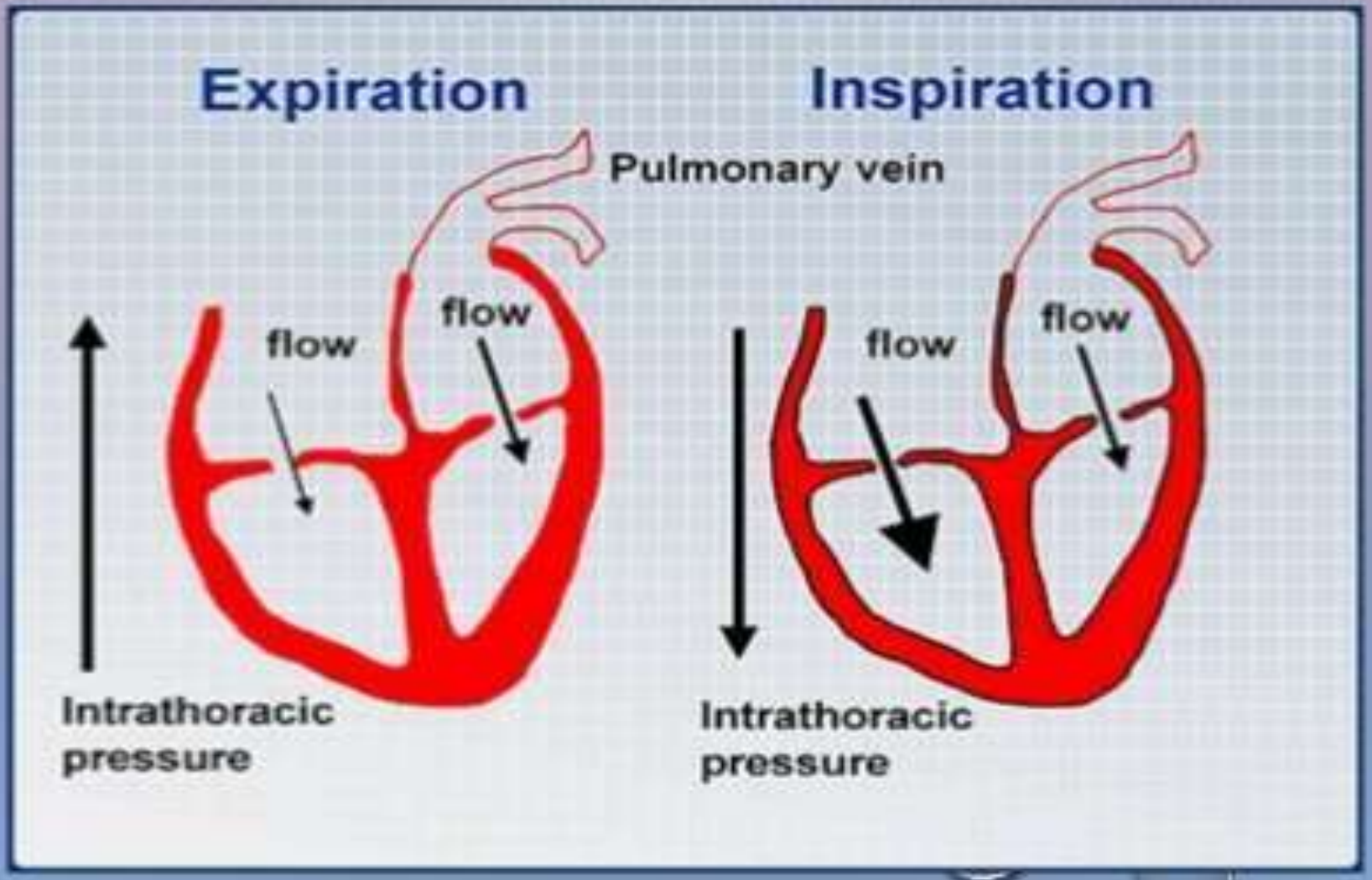
PATHOPHYSIOLOGY

- A thickened, fibrotic pericardium forms a non-compliant shell around the heart ("constricts it").
- Ventricles are like a fixed cavity; encased by the thick pericardium
- This shell prevents the heart from expanding when blood enters it.
- So it interferes with ventricular filling

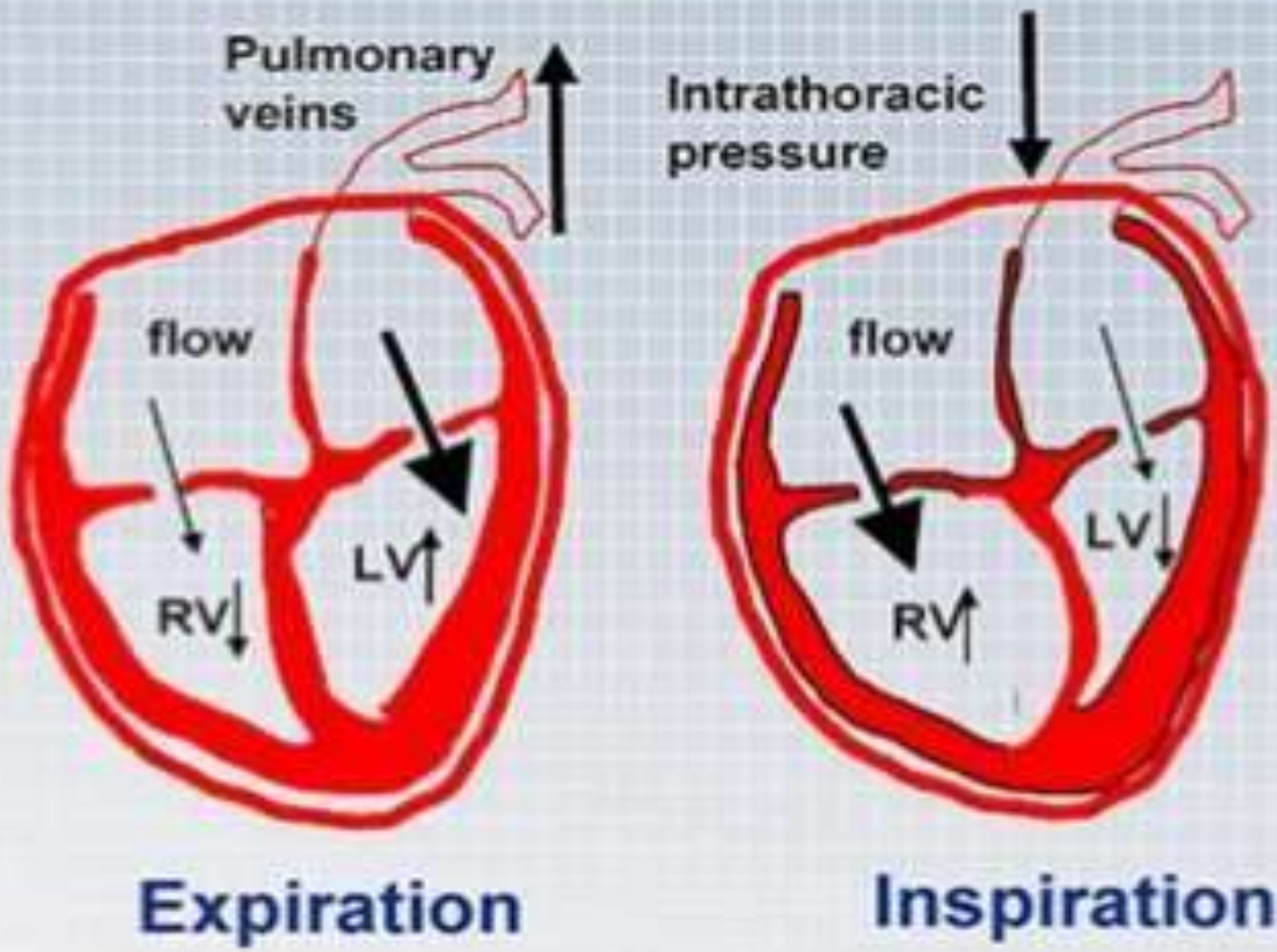
PATHOPHYSIOLOGY

- In early diastole the ventricle relaxes (expands) to a certain extent and stops abruptly because it cannot expand any more
- **Almost all of the ventricular filling occurs in early diastole; very little filling in late diastole because the ventricle cannot expand any more**
- CP → restriction of filling in late diastole
- **Filling of one ventricle occurs at the expense of the other ventricle**

NORMAL- RESPIRATORY VARIATION



CP- RESPIRATORY VARIATIONS



Pericardium – Rigid and Scarred

↓

SYSTEMIC > PULMONARY VENOUS CONGESTION

↓

Hepatic congestion, peripheral edema, ascites , anasarca, and cardiac cirrhosis.

↓

Reduced cardiac output :

↓

Consequence of impaired ventricular filling
causes fatigue, muscle wasting, and weight loss

CLINICAL FINDINGS

Rigid, scarred pericardium encircles heart:

Systolic contraction normal

Inhibits diastolic filling of **both** ventricles

↓ SV

↓

↓CO

↓

Hypotension/shock

Reflex tachycardia

↑venous pressures

systemic

↓

JVP

hepatomegaly

ascites

peripheral edema

pulmonary

↓

lung signs

PHYSICAL EXAMINATION

↓BP, ↑HR ↑ JVP

ascites, edema, hepatomegaly

early diastolic "knock"

after S2

sudden cessation of ventricular diastolic filling imposed by rigid pericardial sac

Kussmaul's sign

inspiratory increase in JVP

KUSSMAUL'S SIGN

- In Inspiration :
  - Normal :
    - RV Volume increases without increase in RA pressure.
  - In Constrictive pericarditis :
    - RV volume increases , as the RV cannot expand due to thickened pericardium ,this results in increase in RA pressure which causes Elevated JVP in inspiration.

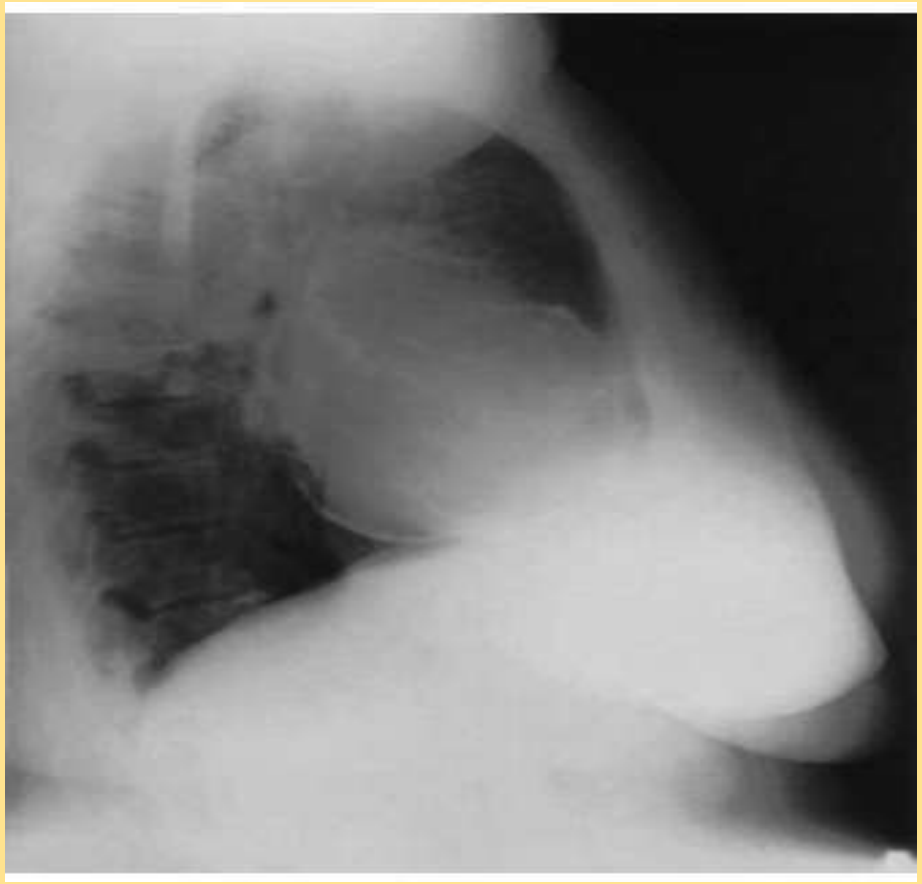
DIAGNOSIS

- Clinical suspicion followed by confirmation with certain diagnostic tests

  (many patients are initially seen for abdominal symptoms)
- ECG : AF in 1/3rd of patients

  flattened or inverted T waves

CHEST X RAY



Pericardial calcification

- **Echocardiogram**

  - pericardial thickening

- septal bounce : abrupt displacement of IVS during early diastole

  - restrictive filling pattern

- >25% increase in mitral E velocity during expiration compared with inspiration

CP VS RCM (ECHO)

Constriction

2 D

- Pericardial thickening
- Normal chamber wall thickness
- Septal bounce

Doppler

- > 25% ↓ in mitral E velocity with inspiration
- ↑ tricuspid flow with insp
- ↑HV diastolic flow reversal with inspiration
- Tissue doppler E' : > 8 cm /sec

Restriction

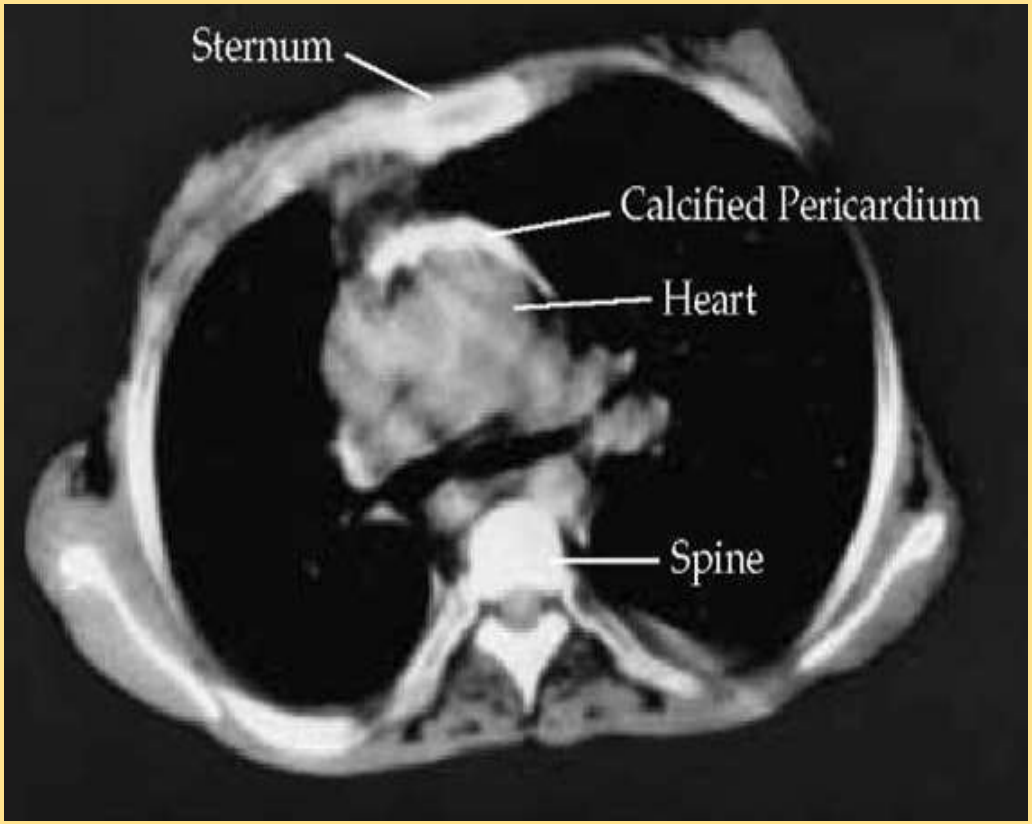
2D

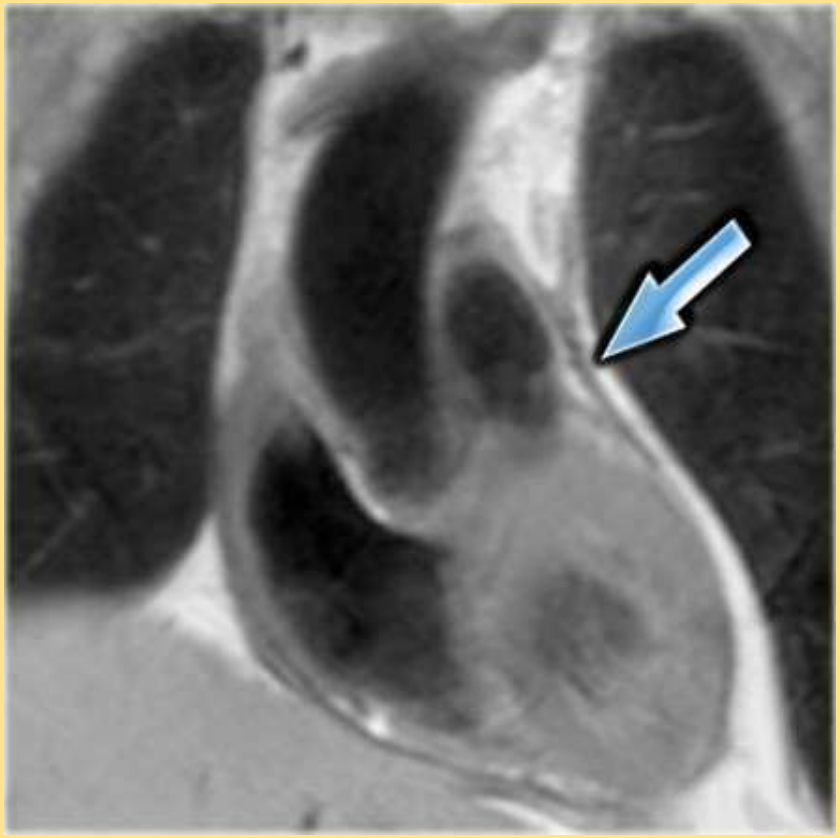
- Increased wall thickness
- Thickened valves
- Atrial enlargement
- Speckling

Doppler

- MR, TR
- ↑ HV diastolic diastolic flow reversal with expiration
- Tissue doppler E' : < 8 cm/sec

CONFIRMATION IS USUALLY THROUGH CT / MRI





MANAGEMENT

- Cautious diuretics and salt restriction
- In patients with AF digoxin is recommended as initial treatment to slow the ventricular rate before resorting to beta blockers or calcium antagonists. In general, the rate should not be allowed to drop 80 -90 / min

Definitive treatment : surgical pericardiectomy

PERICARDIAL EFFUSION

- Accumulation of fluid between the visceral and parietal layers of serous pericardium

- Serous
  - Transudative – CHF , Renal failure
- Suppurative
  - Pyogenic infection
- Hemorrhagic
  - occurs with any type of pericarditis
  - especially with infections and malignancies

ETIOLOGY

1. Inflammation from infection, immunologic process.
2. Trauma causing bleeding in pericardial space.
3. Noninfectious conditions such as:
    a.increase in hydrostatic pressure e.g. congestive heart failure.
    b. increase in capillary permeability e.g. hypothyroidism
    c. decrease in plasma oncotic pressure e.g. cirrhosis.
4.Decreased drainage of pericardial fluid due to obstruction of thoracic duct as a result of malignancy or damage during surgery.

QUANTIFICATION

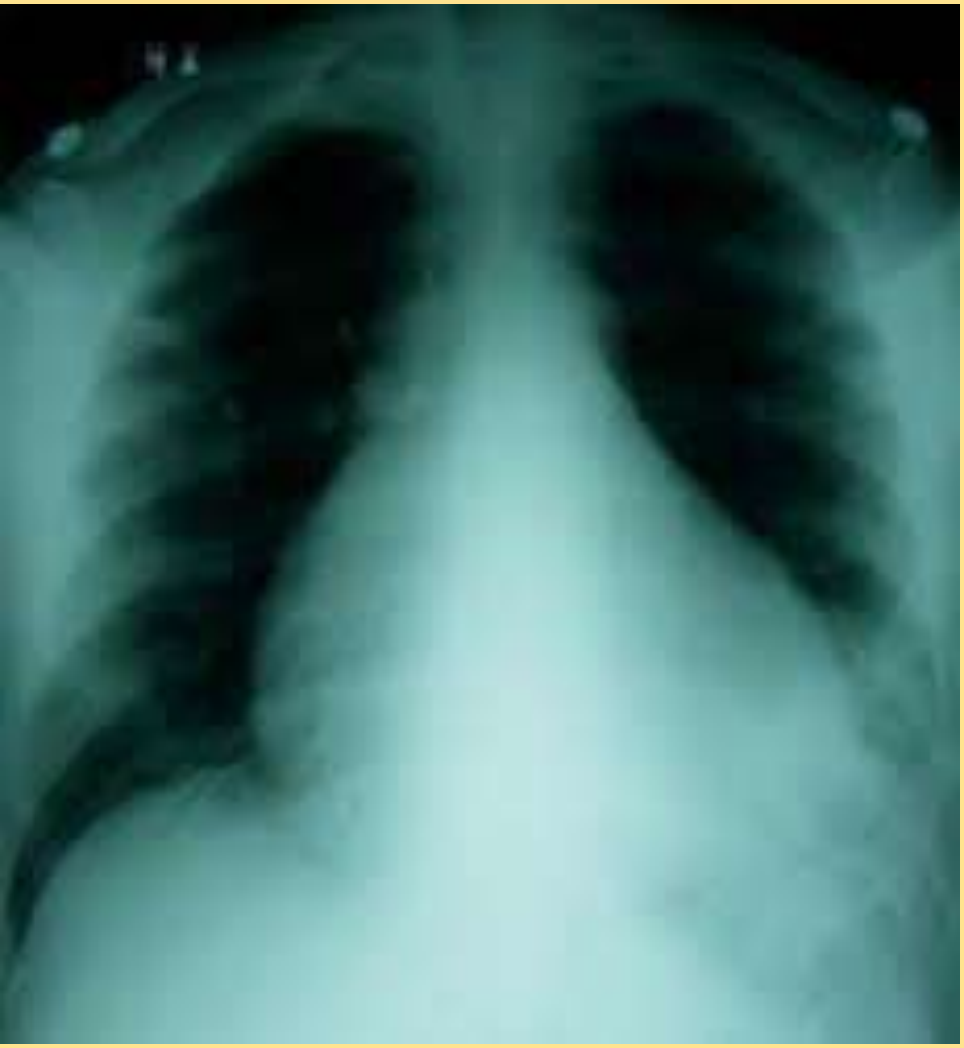
- Trivial : 50 – 100 cc
- Small : 100 cc
- Moderate : 500 cc
- Large : 1000 cc

CLINICAL FEATURES

- Usually asymptomatic
- Can have signs of compression
  - dyspnoea, dysphagia, hoarseness of voice, hiccoughs, nausea

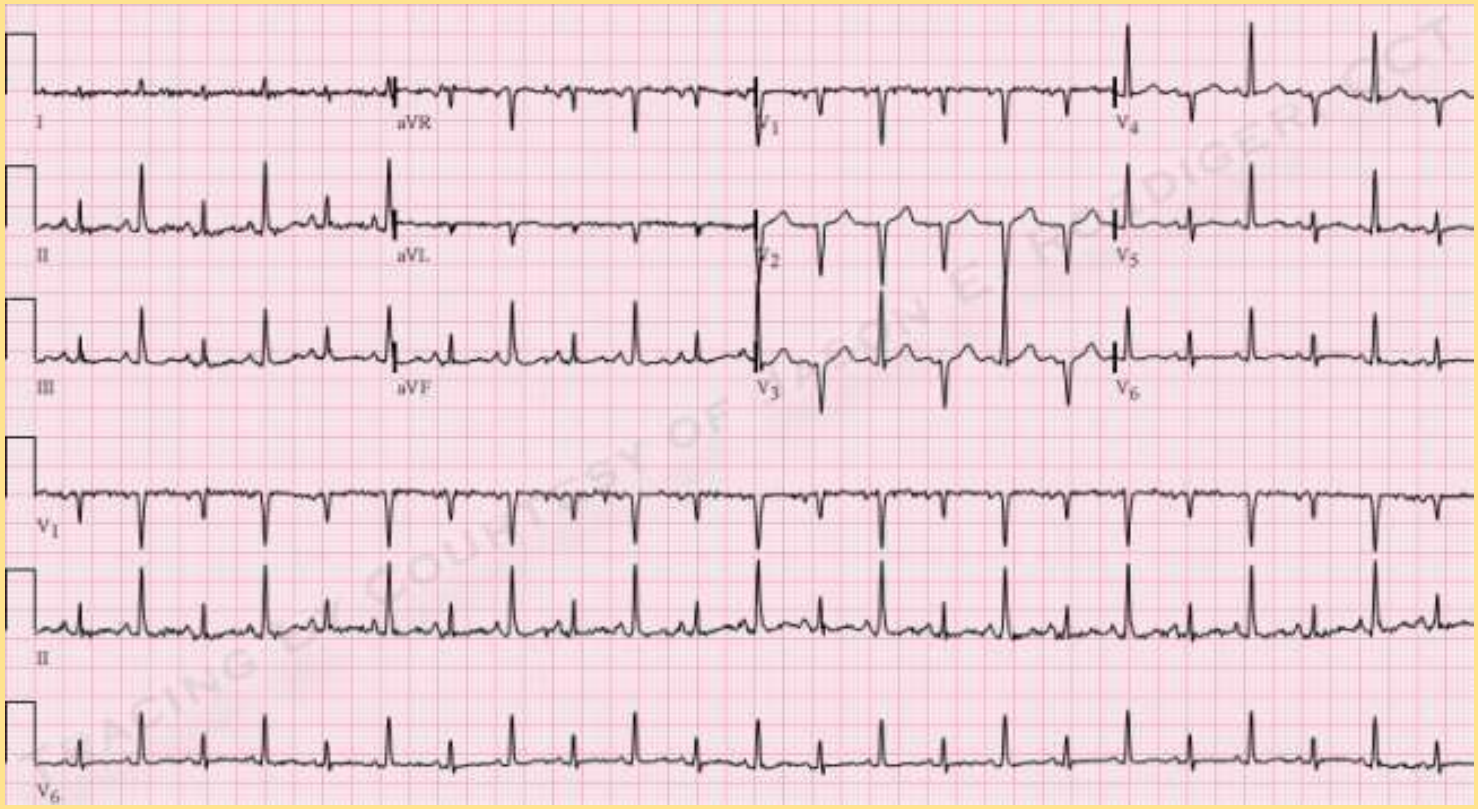
- Signs : muffled heart sounds
  paradoxically reduced intensity of rub

CHEST X RAY



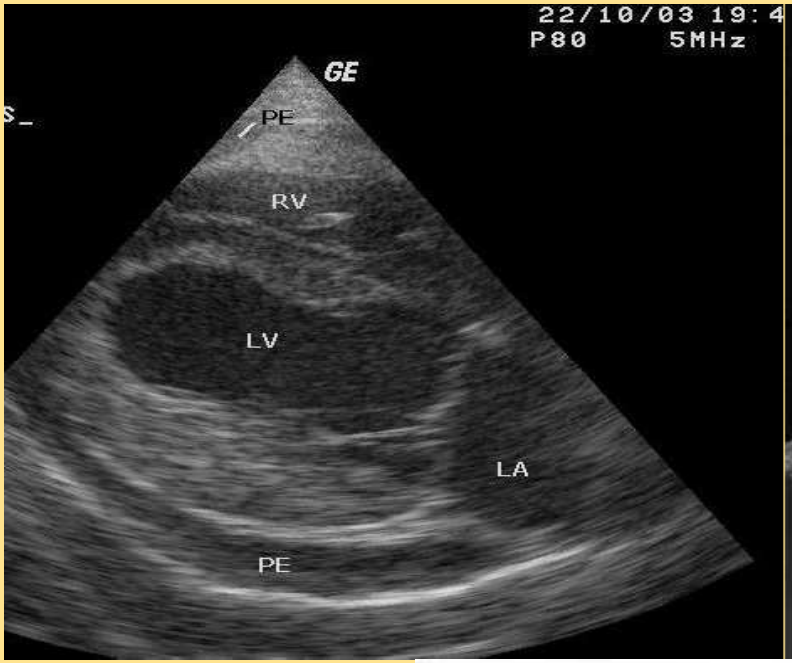
- usually requires > 200 ml of fluid
- cannot distinguish between pericardial effusion and cardiomegaly

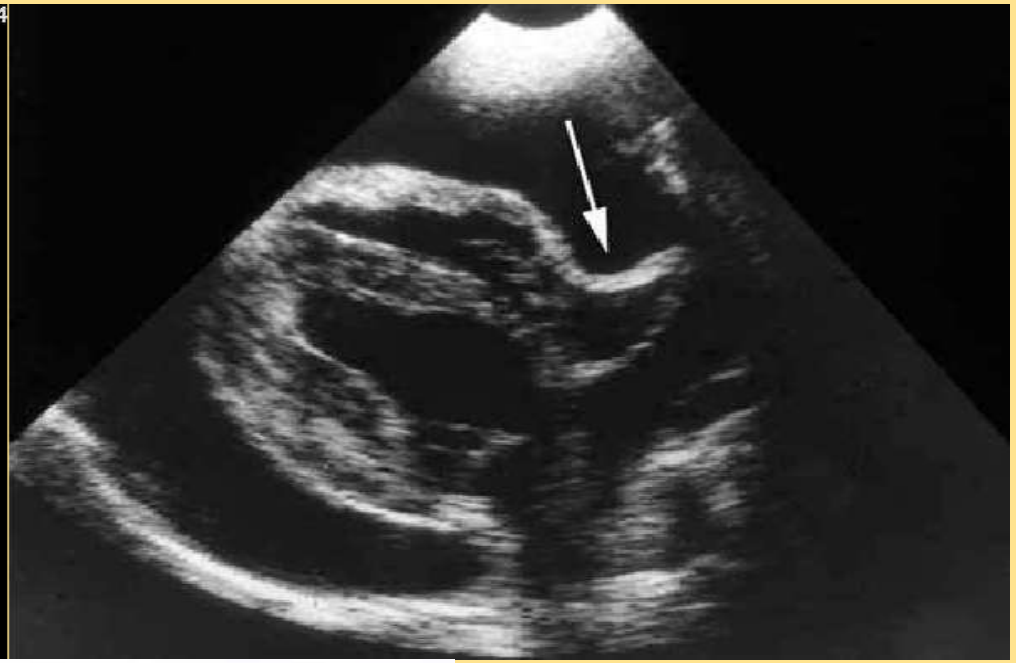
ELECTROCARDIOGRAM

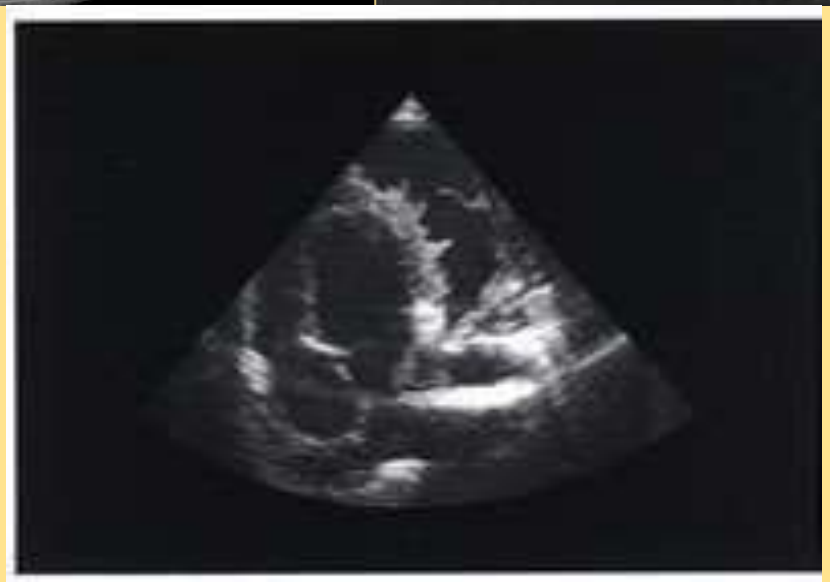


Low voltage complexes

ECHOCARDIOGRAM







MANAGEMENT

- Depends on the etiology , presence of hemodynamic compromise and the volume of fluid.
- No role for diuretics
- Pericardiocentesis is not always necessary.
- Pericardiocentesis if
  - Malignancy or Purulent pericarditis is suspected
  - Hemodynamic compromise present

CARDIAC TAMPONADE

WHAT IS TAMPONADE ?

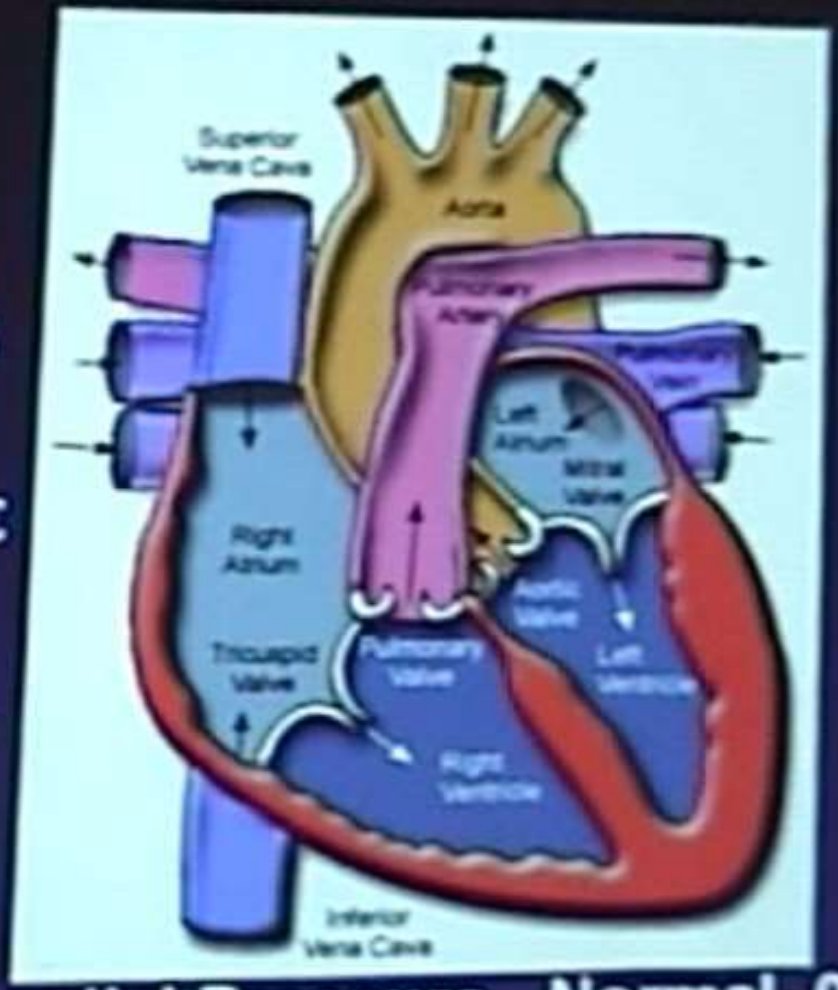
- Accumulation of fluid in the pericardial space causing increase in pressure with subsequent cardiac compression.
- Pericardial pressures > intracardiac pressures
- Most common causes :
  - Malignancy
  - Idiopathic pericarditis
  - Renal failure.
  - Bleeding following cardiac Sx and trauma , TB & Hemopericardium

Normal Cardiac Pressures

RIGHT ATRIUM
a 7 v 5 m 5

RIGHT VENTRICLE
S 25 ED 7

PULMONARY ARTERY
S 25 D 15 M 18



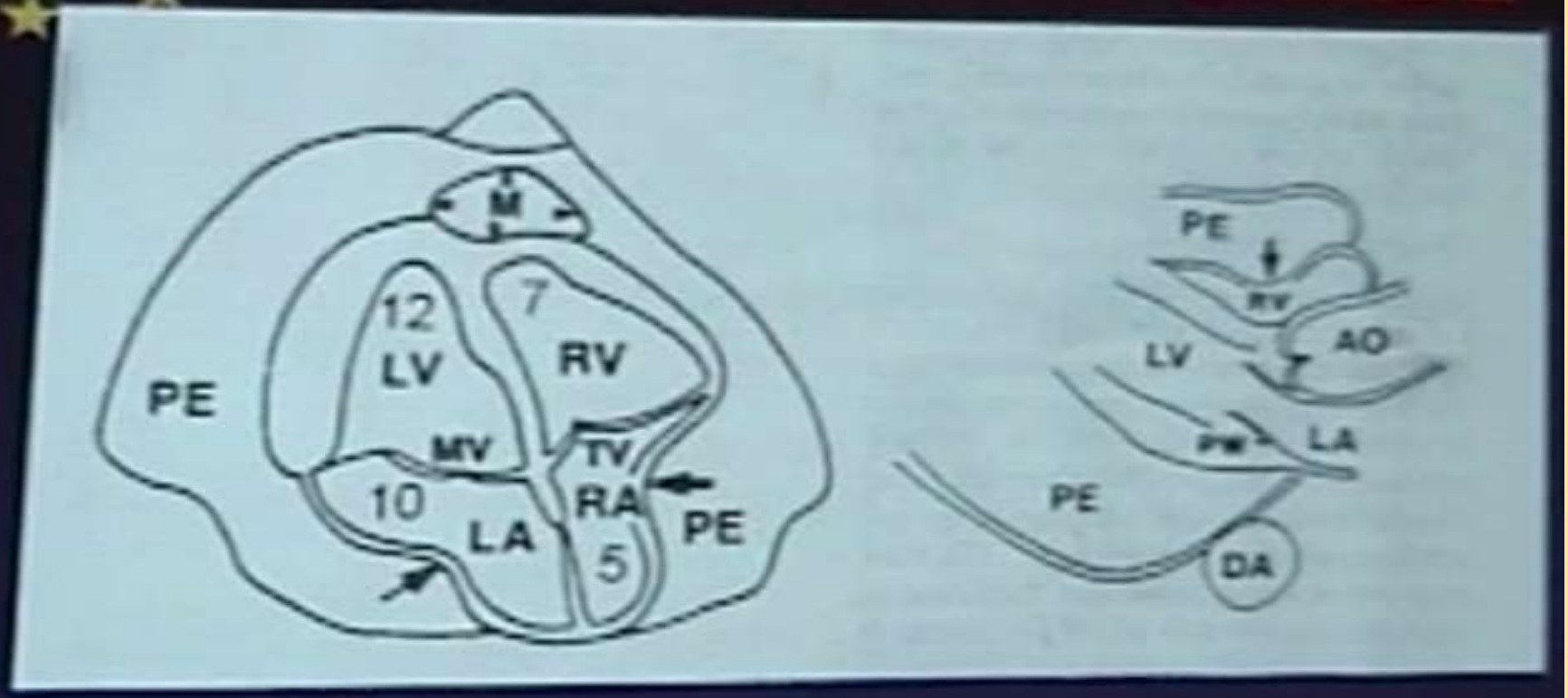
LEFT ATRIUM
a 12 v 10 m10

LEFT VENTRICLE
S 120 ED 12

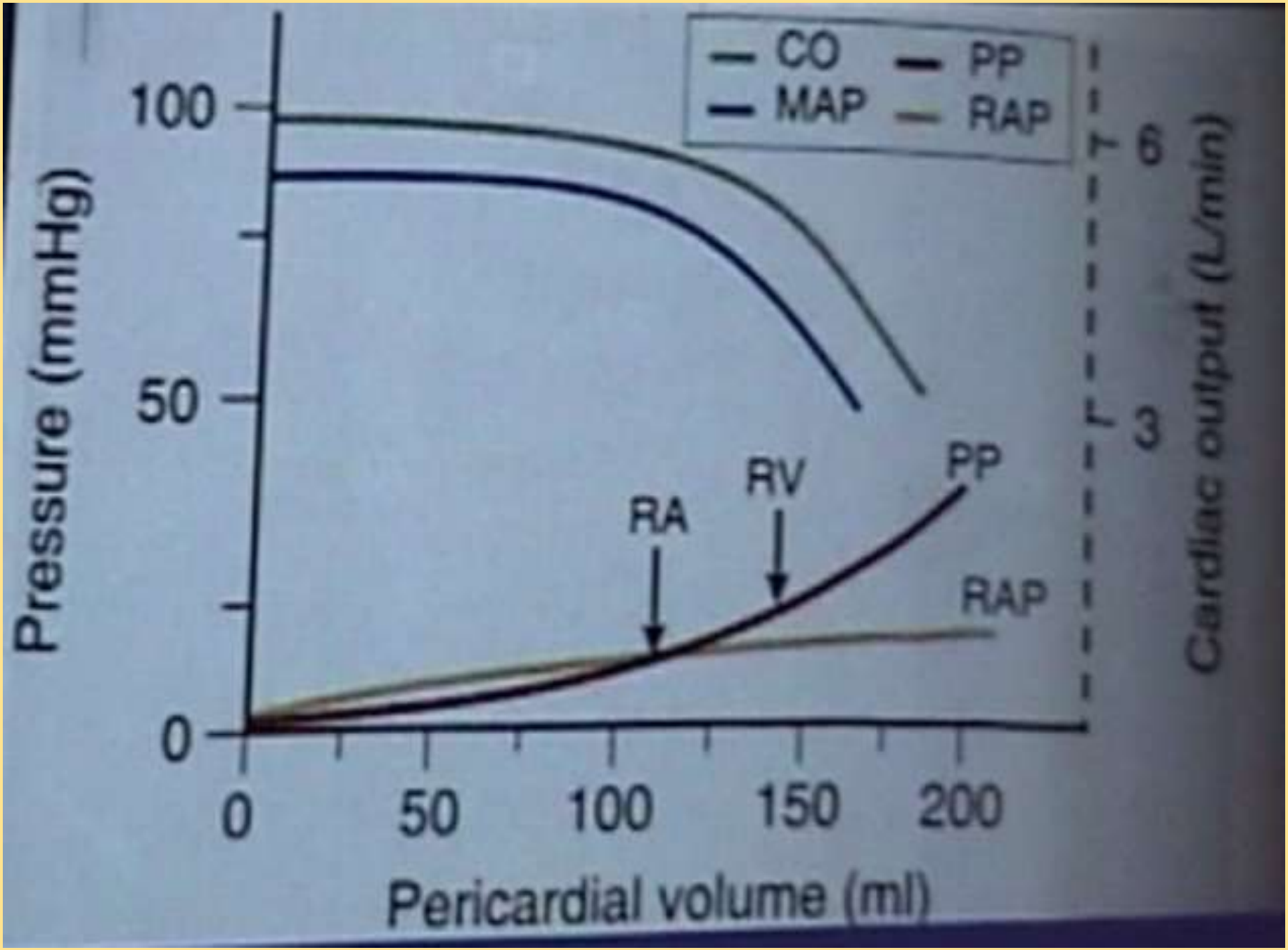
AORTA
S 120 D 80

Intra Pericardial Pressure - Normal- 0 mm
Inspiration –2 to-3 mm
Expiration Upto +5mm

PERICARDIAL EFFUSION
with CARDIAC TAMPONADE



Mild to Moderate Tamponade-IPP = 5-10 mm of Hg
Moder to Severe Tamponade-IPP=10-15 mm of Hg
Severe Cardiac Tamponade –IPP=15-20 mm of Hg

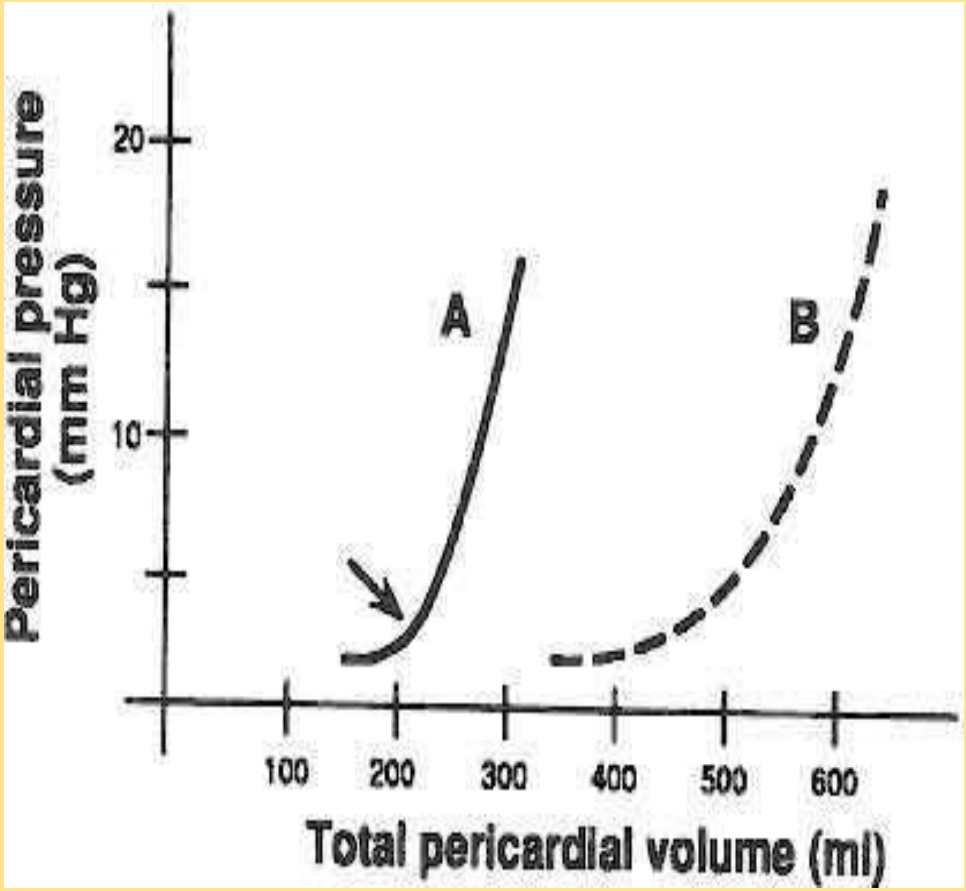

CO
PP
MAP
RAP
100
50
0
Pressure (mmHg)
RA
RV
PP
RAP
0
50
100
150
200
Pericardial volume (ml)
6
3
Cardiac output (L/min)

PATHOPHYSIOLOGY

- Most critical point occurs when an effusion reduces the volume of the cardiac chambers such that cardiac output begins to decline
- Mainly by impeding right-sided heart filling, with much of the effect on the left side of the heart due to secondary under filling.

PATHOPHYSIOLOGY

- A) Modest amounts of rapidly accumulating fluid can have major effects on cardiac function.
- B) Large, slowly accumulating effusions are often well tolerated, presumably because of chronic changes in the pericardial pressure-volume relation described earlier.



CARDIAC TAMPONADE -- PATHOPHYSIOLOGY

- **ACCUMULATION OF FLUID UNDER HIGH PRESSURE: COMPRESSES CARDIAC CHAMBERS & IMPAIRS DIASTOLIC FILLING OF BOTH VENTRICLES**

- **↓SV**
- **↓CO**

Hypotension/shock
Reflex tachycardia

↑VENOUS PRESSURES

SYSTEMIC

JVP
hepatomegaly
ascites
peripheral edema

CLINICAL FEATURES

- Symptoms
  acute : confusion / agitation

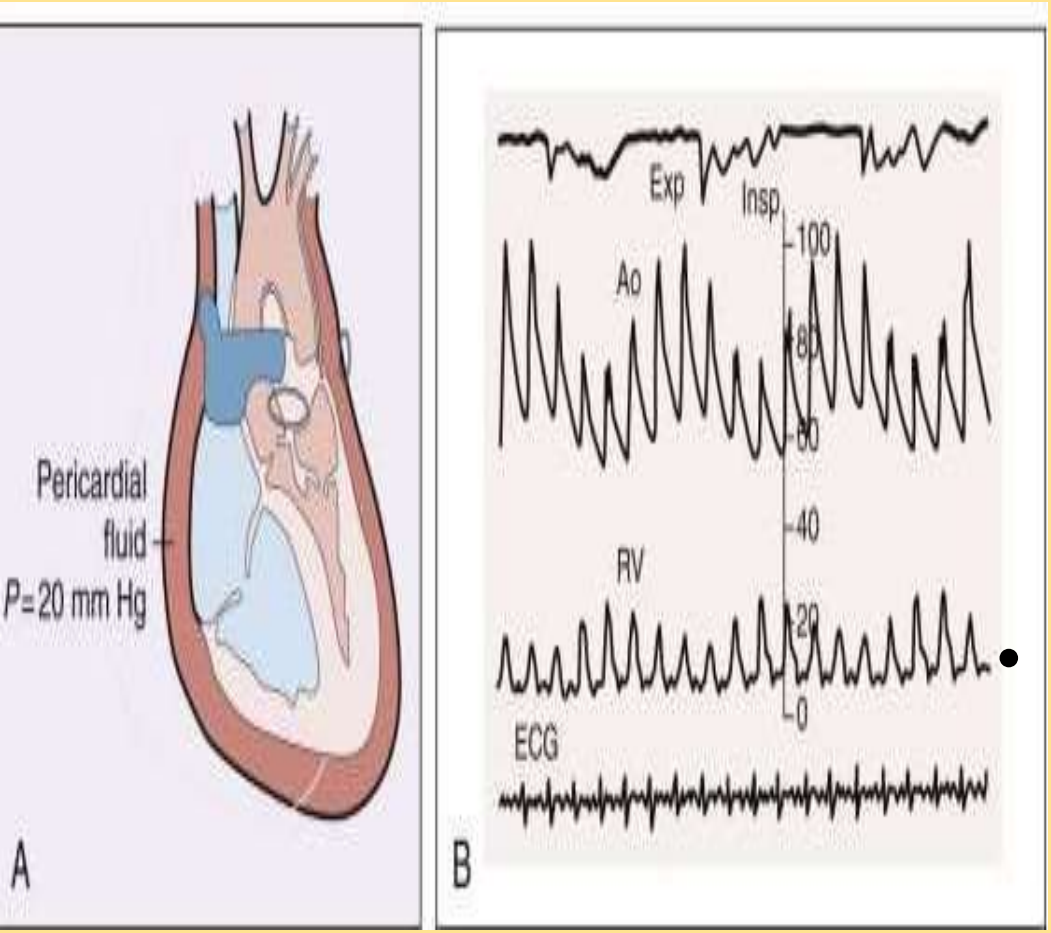
- Signs (Becks triad)
  - hypotension
  - elevated JVP
  - muffled heart sounds

Pulsus paradoxus : insp drop in SBP > 10 mmhg

Pulsus paradoxus also seen in CP, COPD, asthma

PULSUS PARADOXUS

- When severe, it may be detected by palpating weakness or disappearance of the arterial pulse during inspiration.
- Measured by noting the difference between the systolic pressure at which the Korotkoff sounds are first heard (during expiration) and the systolic pressure at which the Korotkoff sounds are heard with each beat, independent of respiratory phase
- Between these two pressures, the sounds are heard only intermittently (during expiration).

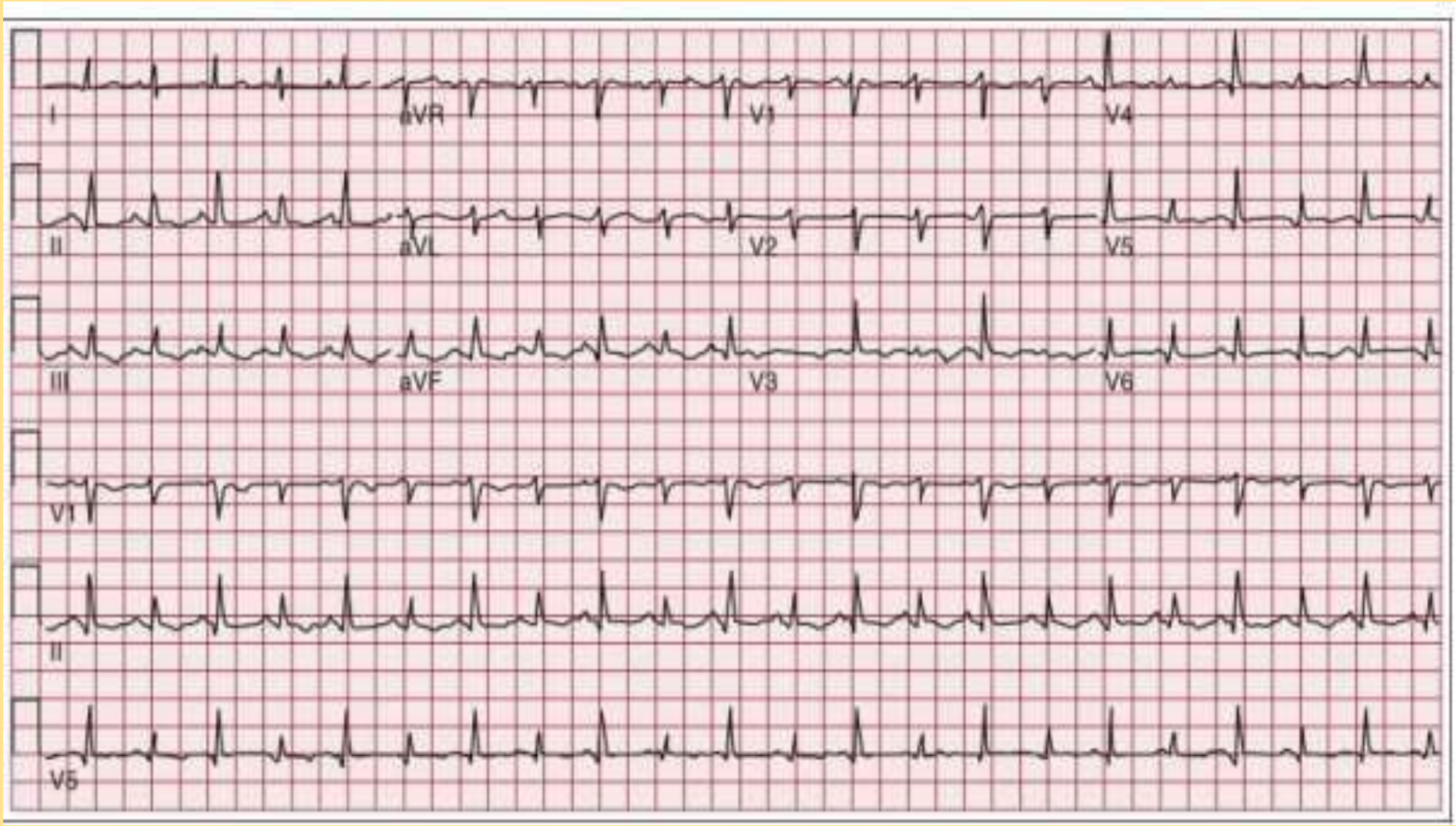


- Since both ventricles share a tight incompressible covering, the inspiratory enlargement of the RV compresses and reduces LV volume; leftward bulging of the IVS further reduces the LV cavity as the RV enlarges during inspiration
- The normal inspiratory augmentation of RV volume causes an exaggerated reciprocal reduction in LV volume.

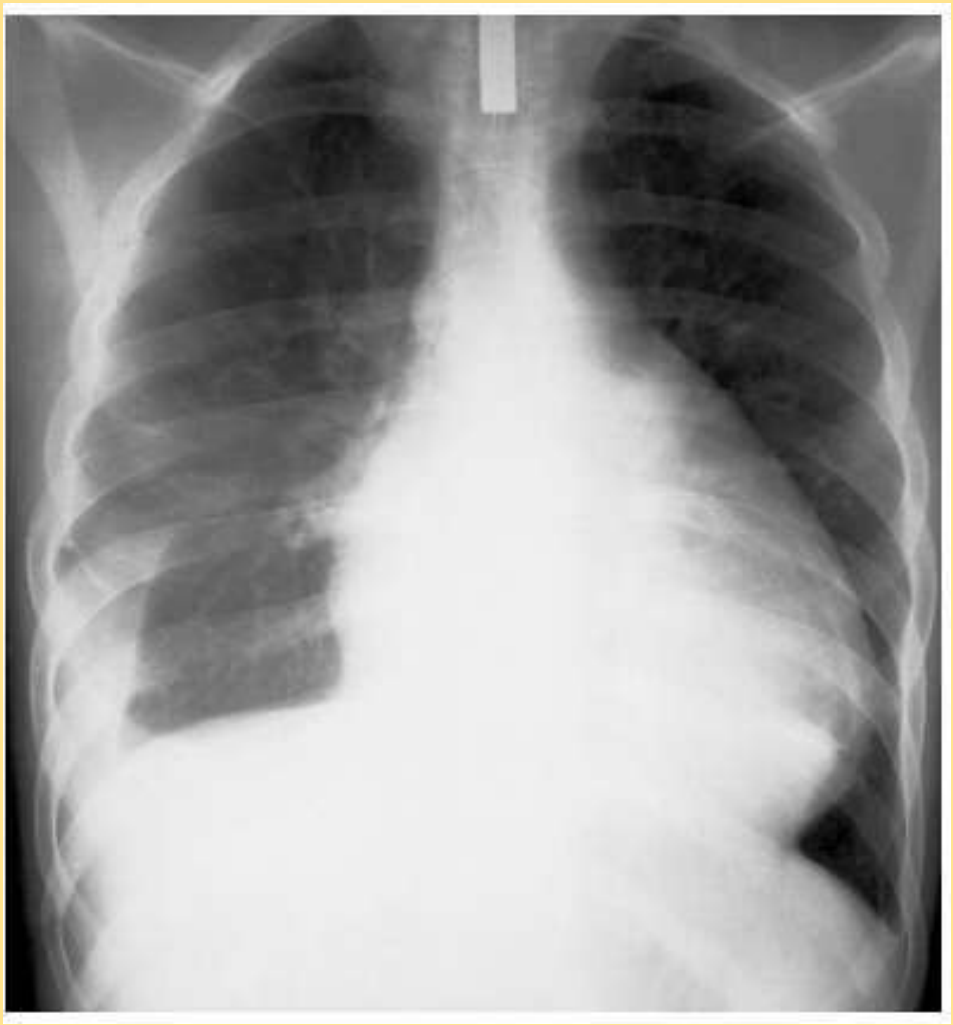
JVP

- Elevated JVP with prominent positive ways : heart failure
- Elevated JVP with prominent negative ways : pericardial disease
- Elevated JVP with absent pulsations : SVC obstruction

ECG



CHEST X RAY

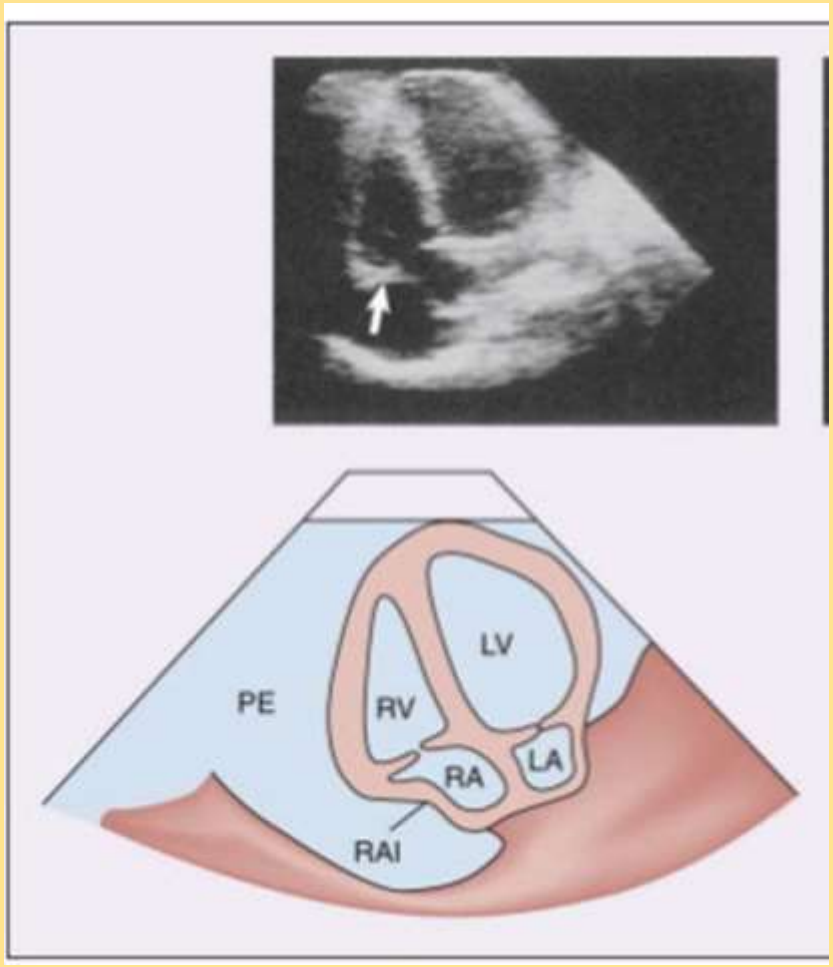


- Cardiac shadow rounded ; Flask like appearance
- Lungs appear oligemic

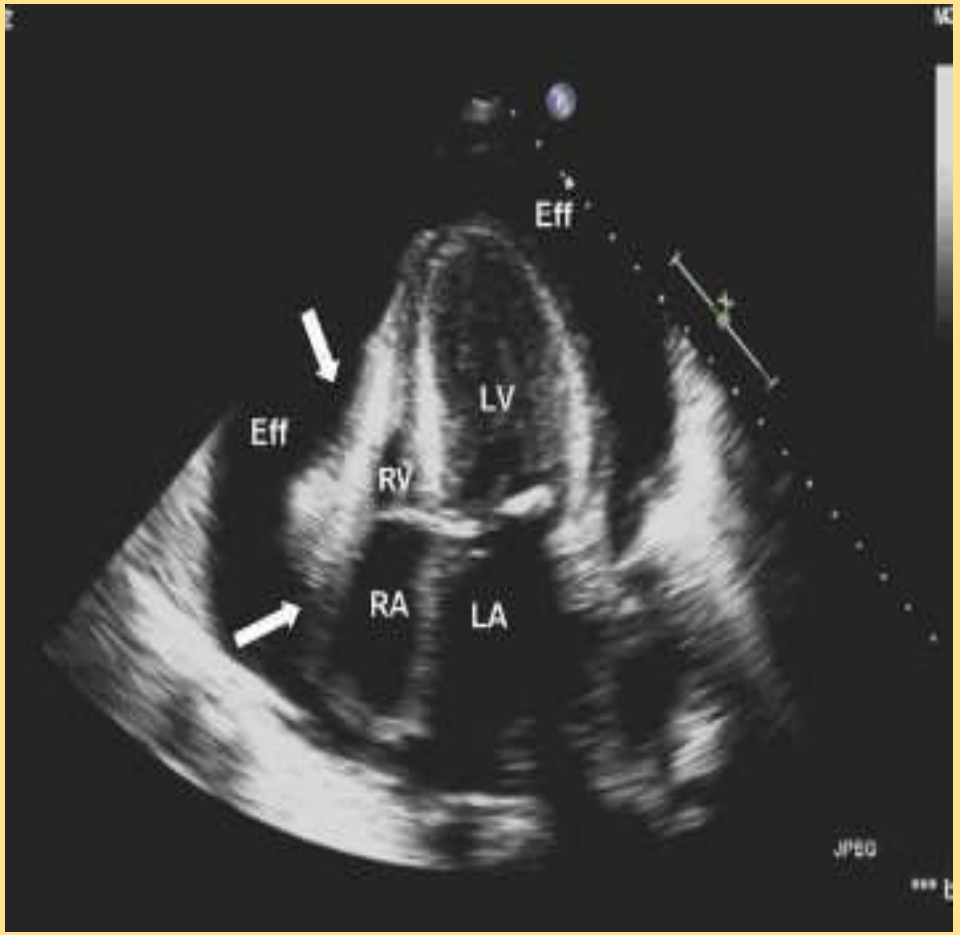
ECHOCARDIOG

RAM

RA collapse

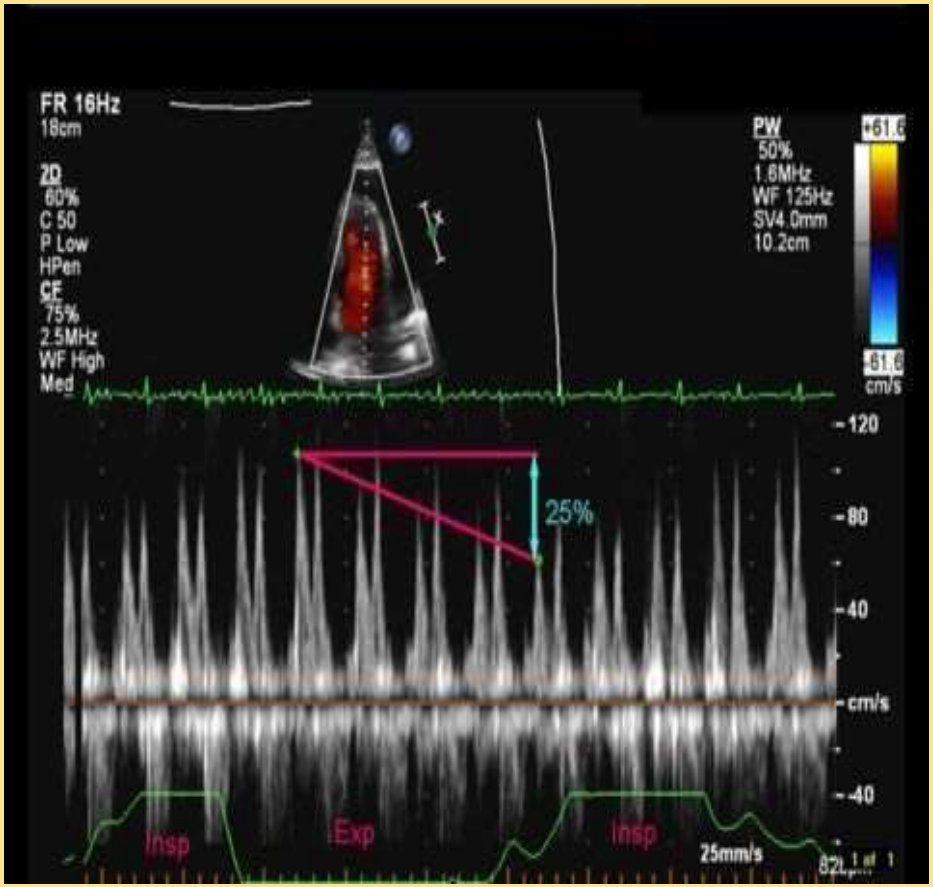


RV collapse

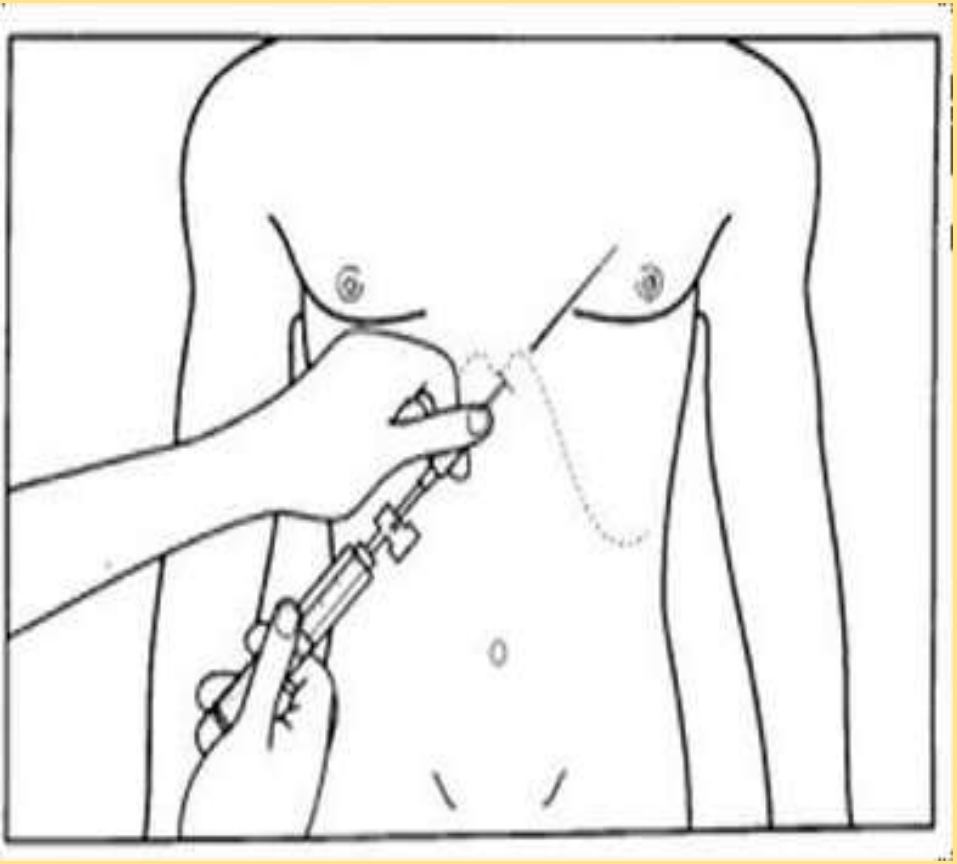


ECHOCARDIOGRAM

- Dilated IVC with minimal or no collapse
- Restrictive filling pattern
- Significant respiratory variation in filling pattern
- Tamponade : equal and continuous restriction throughout diastole
- CP : Restriction to filling in late diastole



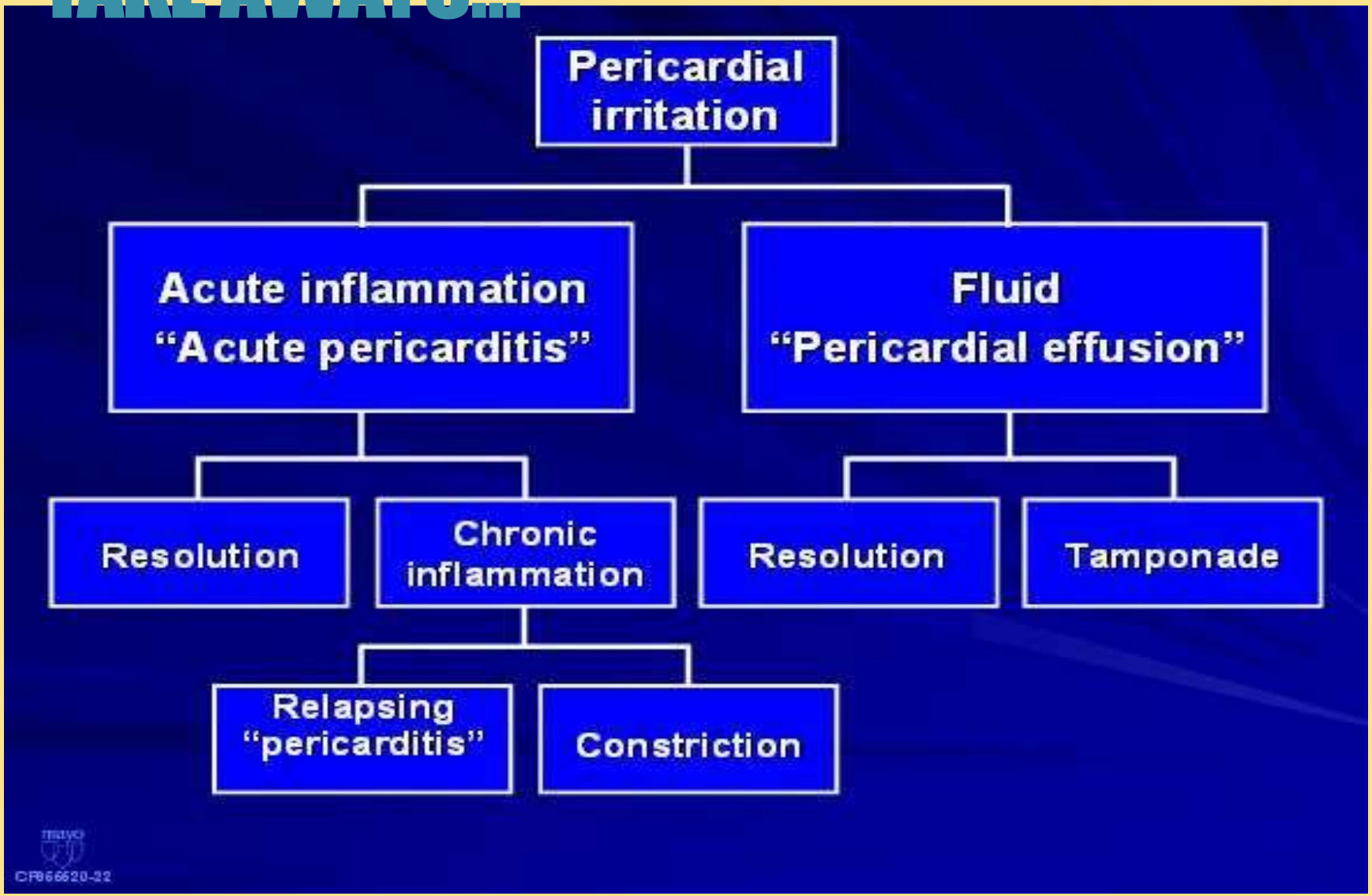
MANAGEMENT



- Avoid diuretics
- Adequate preload

- Pericardiocentesis
- Pericardial window
- Pericardiectomy in select cases

TAKE AWAYS...


Pericardial irritation
Acute inflammation "Acute pericarditis"
Fluid "Pericardial effusion"
Resolution
Chronic inflammation
Resolution
Tamponade
Relapsing "pericarditis"
Constriction
mayo
CR066620-22

TAKE AWAYS…

- Symptoms may be non cardiac
- CP and PE will mimic right heart failure
- In any RHF symptoms rule out pericardial disease
- Because treatment is completely different (diuretics,digoxin)

- Clinical suspicion is essential for diagnosis

- Correct diagnosis is imperative

- Potential for permanent cure

ECG

ECG IN MI

- ST ELEVATIONS ARE CONVEX,
- RESTRICTED TO ARTERIAL TERRITORY
- RECIPROCAL DEPRESSION - MORE PROMINENT
- QRS CHANGES OCCUR(Q WAVES, AS WELL AS NOTCHING AND LOSS OF R-WAVE AMPLITUDE)
- T-WAVE INVERSIONS ARE USUALLY SEEN WITHIN HOURS *BEFORE THE ST SEGMENTS HAVE* BECOME ISOELECTRIC.

ECG IN PERICARDITIS

- ST ELEVATIONS ARE CONCAVE.
- NOT RESTRICTED TO ARTERIAL TERRITORY.
- RECIPROCAL ST DEPRESSION IN AVR /V1
- T WAVE INVERSIONS AFTER ST
- SEGMENT BECOMES ISOELECTRIC.
- ELEVATED ST SEGMENTS RETURN TO NORMAL WITHIN HOURS

REFERENCE

MEDICINE, PREP MANUAL FOR UNDERGRADUATES

BY K. GEORGE MATHEW & PRAVEEN AGGARWAL

REVIEW IN PATHOLOGY WITH COLOUR PLATES

BY NITIN CHAWLA, SANDIP KUDESIA

TEXT BOOK OF MEDICAL-SURGICAL NURSING

BY SUZANNE C. SMELTZER, BRENDA G .BARE