

**IN THE NAME OF GOD**

# HEART FAILURE



# Congestive Heart Failure

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# DEFINITION:

- ⦿ The heart cannot deliver adequate cardiac output to meet the metabolic needs of the body.
- ⦿ when the demand for cardiac output exceeds the ability of the heart to respond.
- ⦿ Ineffective compensatory mechanisms result in clinical manifestations.

- The heart can be viewed as a pump with an output proportional to its filling volume and inversely proportional to the resistance against which it pumps.
- The primary determinants of stroke volume are:
  - Afterload* (pressure work)
  - Preload* (volume work)
  - Contractility* (intrinsic myocardial function).

- ◉ **Systemic oxygen transport :**

- ◉ cardiac output \* systemic oxygen content.

- ◉ **Cardiac output :**

- ◉ Heart rate \* stroke volume.

# Etiology of Heart Failure

## FETAL

- ١- Severe anemia
- ٢- Supraventricular tachycardia
- ٣- Ventricular tachycardia
- ٤- Complete heart block

# Etiology of Heart Failure

## FULL-TERM NEONATE:

- ١-Asphyxial cardiomyopathy
- ٢-AV malformation (vein of Galen, hepatic)
- ٣-Left-sided obstructive lesions (CO of aorta, HLHS)
- ٤-Complex defects (SV, truncus-arteriosus)
- ٥-Viral myocarditis



# Etiology of Heart Failure

## PREMATURE NEONATE

- ١ - Fluid overload
- ٢ - Patent ductus arteriosus
- ٣ - Ventricular septal defect
- ٤ - Cor pulmonale (bronchopulmonary dysplasia)

# Etiology of Heart Failure

## INFANT-TODDLER

- Left-to-right cardiac shunts (VSD, ASD, PDA..)
- Hemangioma (arteriovenous malformation)
- Anomalous left coronary artery
- Metabolic cardiomyopathy
- Acute hypertension (hemolytic-uremic syndrome)
- Supraventricular tachycardia
- Kawasaki disease (most common in developed countries)
- Viral myocarditis

# Etiology of Heart Failure

## CHILD-ADOLESCENT

- ⊙ Rheumatic fever (most common in developing countries)
- ⊙ Acute hypertension (glomerulonephritis)
- ⊙ Viral myocarditis
- ⊙ Thyrotoxicosis
- ⊙ Hemochromatosis-hemosiderosis
- ⊙ Cancer therapy (radiation, doxorubicin)
- ⊙ Sickle cell anemia
- ⊙ Endocarditis

# Types of Heart failure:

१- Low cardiac out put

○ Decreased cardiac function

२- Normal cardiac out put

○ Lt to Rt shunt

३- High cardiac out put

○ Anemia

○ Hyper metabolic state

○ Systemic arteriovenous fistula

High cardiac output heart failure => decreased systemic oxygen content (secondary to anemia) or increased oxygen demands (secondary to hyperventilation, hyperthyroidism, or hypermetabolism).

It is also seen with large systemic arteriovenous fistulas. These conditions reduce peripheral vascular resistance and cardiac afterload and increase myocardial contractility.

Severe high cardiac output failure may lead to myocardial dysfunction .

# COMPENSATORY MECHANISMS:

## ↑ - Increased sympathetic tone

Secretion of Epinephrine , Norepinephrine.

### Side effects:

hypermetabolism increased  $O_2$  consumption

myocardial toxicity

arrhythmogenesis

peripheral vasoconstriction

- ② - Activation of Renin-Angiotensin-Aldosterone
- ③ - Ventricular dilatation
- ④ - Ventricular hypertrophy
- ⑤ - Atrial natriuretic peptide
- ⑥ - Excitation contraction

# ⦿ Pathophysiology:

- ⦿ ١ - Cardiac dysfunction
- ⦿ ٢ - Systemic venous congestion
- ⦿ ٣ - Pulmonic venous congestion



# Clinical manifestation

Depend on the degree of cardiac reserve under various conditions.

A thorough history is extremely important .

In infants focus on feeding .

In older child ask about the activity.

# Clinical manifestation in INFANTS

- ⊙ Tachypnea, feeding difficulties, poor weight gain, excessive perspiration, irritability, weak cry, and noisy, labored respirations.
- ⊙ wheezing
- ⊙ Pneumonitis with or without atelectasis
- ⊙ Hepatomegaly and Cardiomegaly
- ⊙ Edema : the eyelids as well as the sacrum and less often the legs and feet.

## Clinical manifestation in CHILDREN

- Similar to adults, fatigue, effort intolerance, anorexia, abdominal pain, dyspnea, and cough.
- Older children and adolescents may have primarily abdominal symptoms and a surprising lack of respiratory complaints.
- Prominent jugular venous pressure and liver enlargement.

## Clinical manifestation in CHILDREN cont.

- ⦿ Orthopnea and basilar rales
- ⦿ Edema (at dependent portions or anasarca)
- ⦿ Cardiomegaly is invariably noted.
- ⦿ A gallop rhythm; when LVE is advanced.
- ⦿ The holosystolic murmur (MR-TR)

# Ross Classification for HF in Children:

- ◉ **Class I => Asymptomatic**

- ◉ **Class II**

Mild tachypnea or diaphoresis with feeding in infants, dyspnea on exertion in older children

- ◉ **Class III**

Marked tachypnea or diaphoresis with feeding in infants; prolonged feeding times with growth failure resulting from HF; marked dyspnea on exertion in older children

- ◉ **Class IV**

Symptoms such as tachypnea, retractions, grunting, or diaphoresis at rest

# NYHA Functional Classification for HF :

- **Class I**

Symptoms of HF are only present at exertion that would limit normal individuals

- **Class II**

Symptoms of HF are present at ordinary levels of exertion; comfortable at rest

- **Class III**

Symptoms of HF are present at less-than-ordinary levels of exertion; comfortable at rest

- **Class IV**

Symptoms of HF are present at rest

# AHA Stages of Heart Failure

## ◉ Stage A

At high risk for HF but without structural heart disease or symptoms of HF

## ◉ Stage B

Structural heart disease but without signs or symptoms of HF

## ◉ Stage C

Structural heart disease with prior or current symptoms of HF

## ◉ Stage D

Refractory HF requiring specialized interventions

# Diagnosis

## 1-CXR:

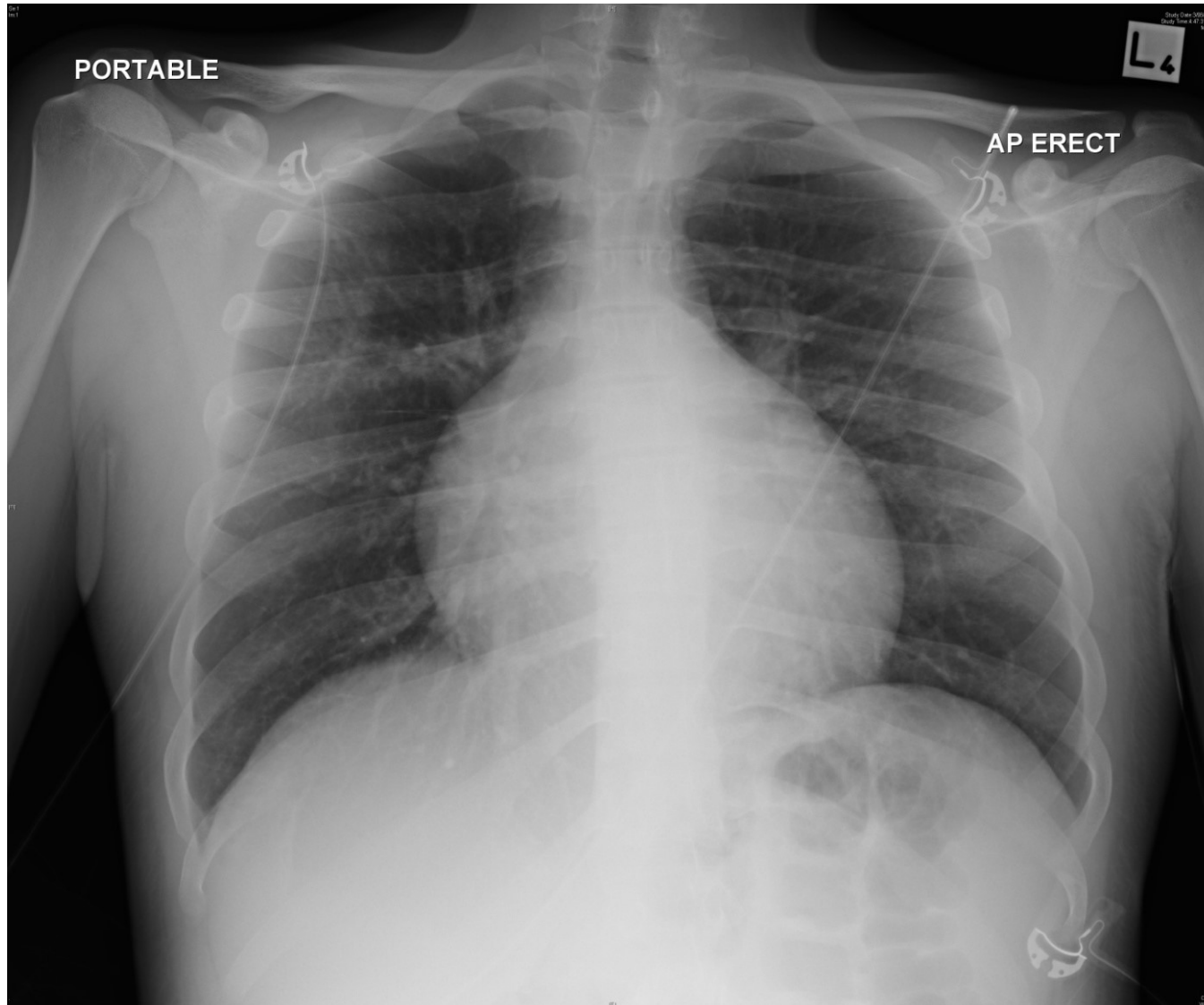
- 1: Pulmonary vascularity : venous congestion
- 2: CT Ratio : Increased

## 2-ECG:

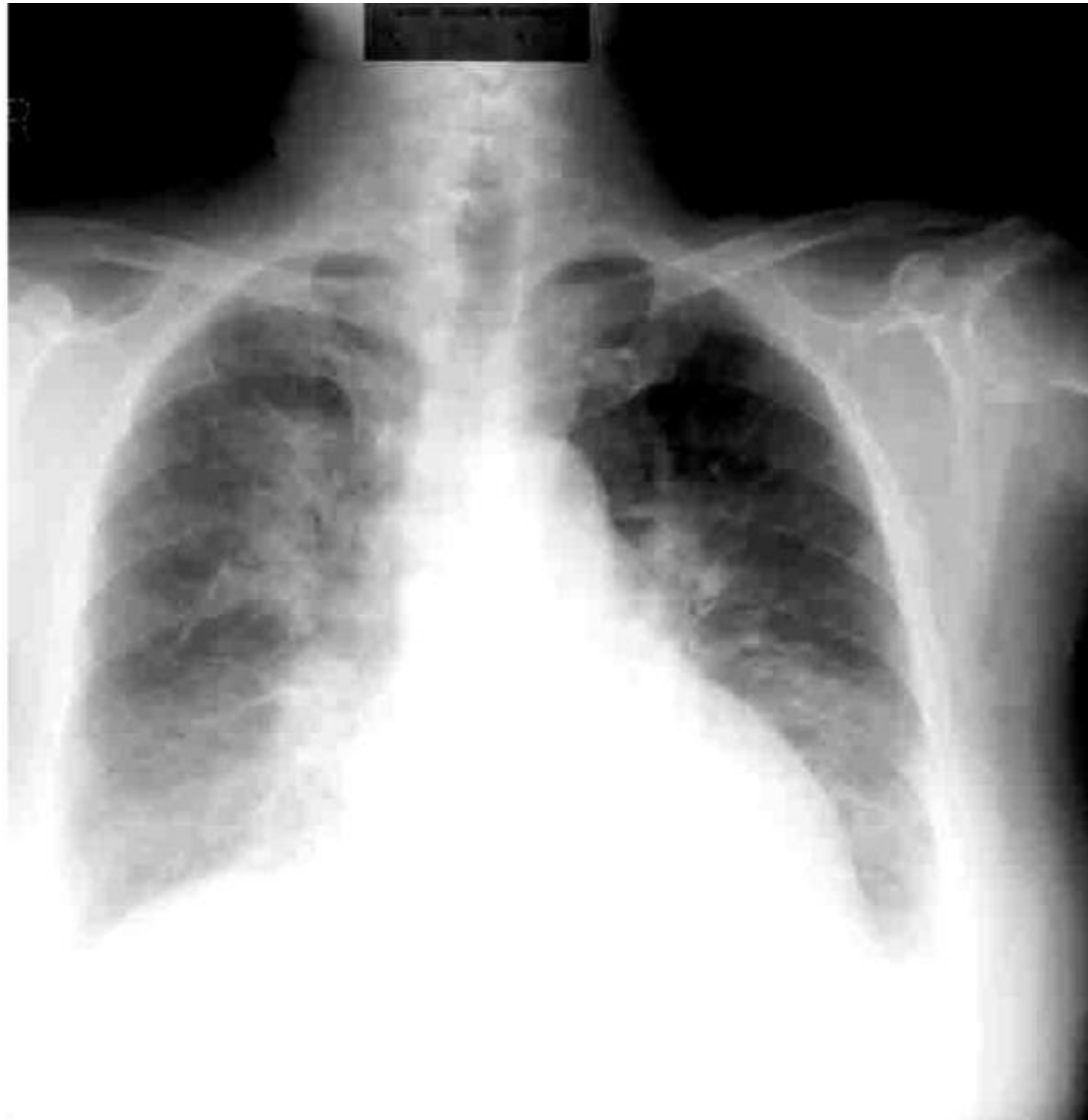
- 1: To evaluate rhythm disorders
- 2: Ischemic changes (CMP)
- 3: ST-T changes (myocarditis, pericarditis )
- 4: Hypertrophic changes



# CARDIOMEGALY



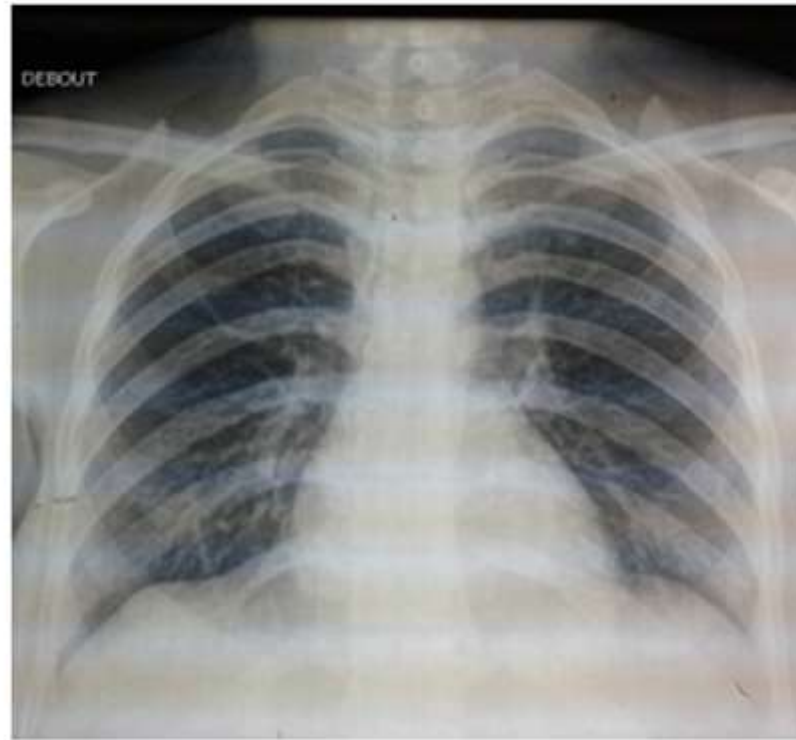
# PULMONARY CONGESTION



# BEFORE AND AFTER TREATMENT



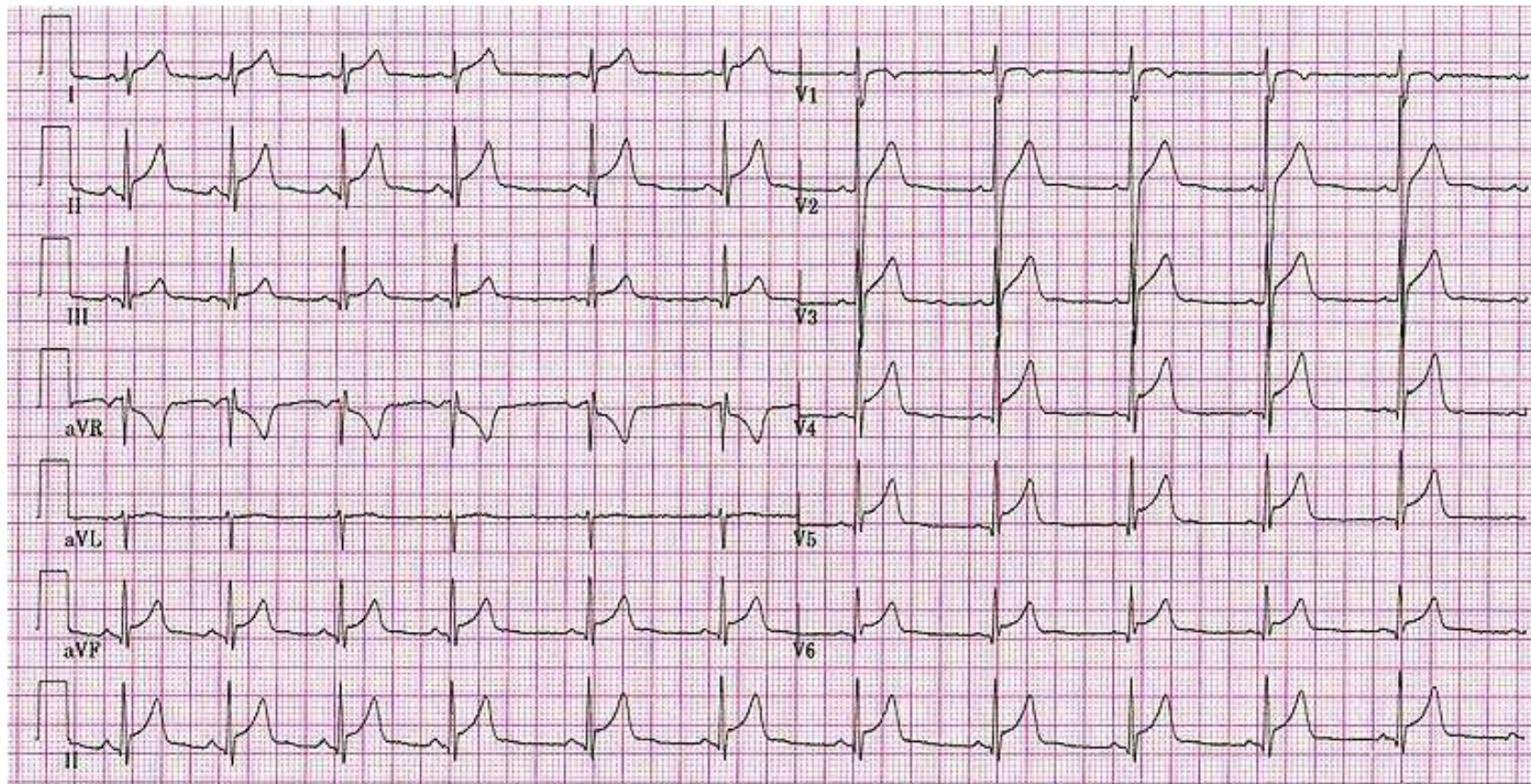
**a**



**b**

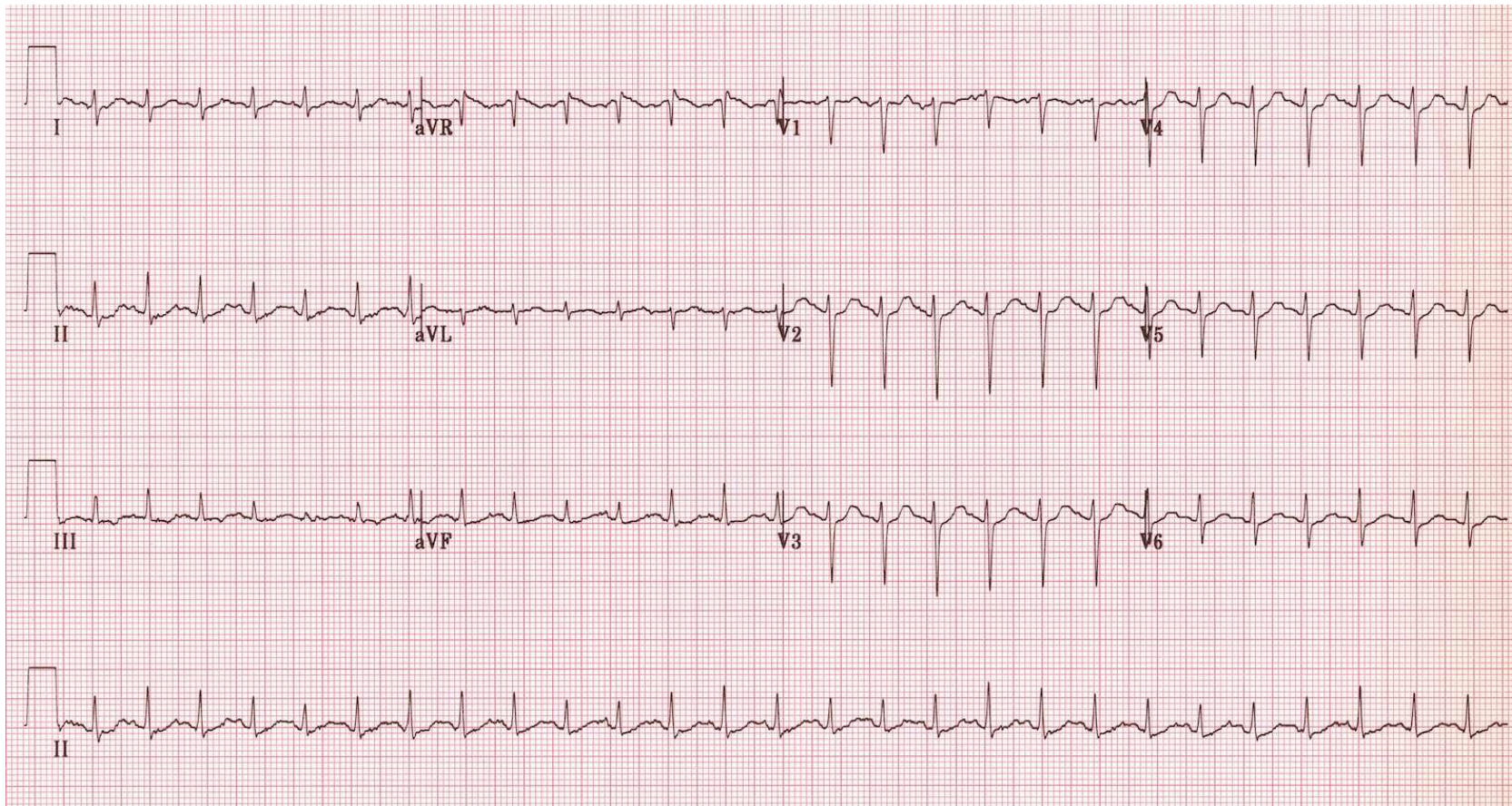


# ST-T CHANGES





# LOW VOLTAGE-SINUS TACHYCARDIA



# Diagnosis

## ३- Echocardiography:

- १- Systolic dysfunction
- २- Diastolic dysfunction

## ४- LAB data :

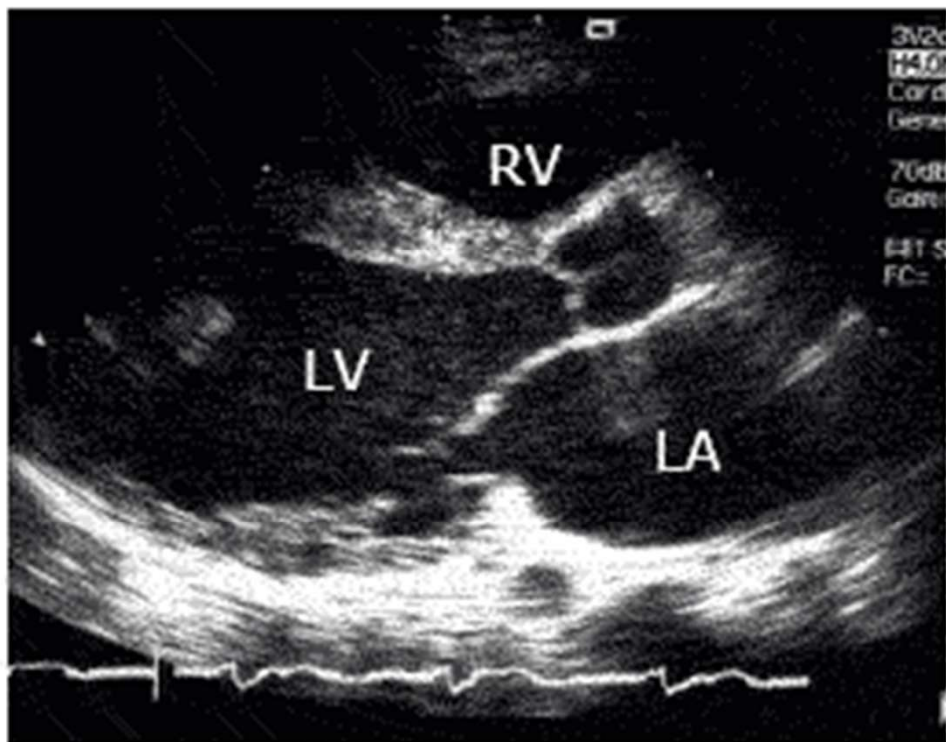
- १- Arterial O<sub>2</sub> level (Decreased due to pulm. edema)
- २- Acidosis
- ३- Hyponatremia (dilutional)
- ४- Biomarkers :BNP,NT-proBNP

- Biomarkers have assumed a prominent role in the diagnosis and management of chronic heart failure.
- NT-proBNP levels are generally higher than BNP levels.
- NT-proBNP are affected more by renal dysfunction than are BNP levels
- Normal values for BNP and NT-proBNP vary significantly with age, and gender, with women having higher levels than men.

- ⦿ Natriuretic peptides are elevated in recipients of anthracycline chemotherapy, with or without ventricular dysfunction.
- ⦿ Other commonly encountered conditions in which BNP and/or NT-proBNP levels may be elevated include end-stage renal disease, sepsis and brain death



# DILATED LA/LV



# PERICARDIAL EFFUSION



# Treatment:

- The underlying cause of cardiac failure must be removed or alleviated if possible.
- Strict bed rest is rarely necessary except in extreme cases, but it is important that the child rest often and sleep adequately.
- Competitive and strenuous sports activities are usually contraindicated

# Treatment

- ⊙ Positive pressure ventilation : In severe pulmonary edema.
- ⊙ Calorie intake:
  - ⊙ Increasing the number of calories per ounce.
  - ⊙ May not tolerate an increase beyond ۲۴ calories/oz due to diarrhea or large solute load for compromised kidneys

# Treatment

- ◉ In severely ill infants : NG feeding
- ◉ In patients with GE Reflux :  
Continuous drip nasogastric feedings at night,  
administered by pump.
- ◉ Occasionally medical or surgical intervention (Nissen fundoplication).

# Treatment

- Low sodium formulas is not recommended routinely as these are often poorly tolerated and may exacerbate diuretic-induced hyponatremia .
- Human breast milk is the ideal low sodium nutritional source.

# Drugs:

↳ **Digoxin:** Digital glycoside (PO-IV)

⊙ Half life : 36 hour, may exceed to 6 days in aneuric patient

⊙ Initial effect: PO : 30 min

IV : 15-30 min

Peak effect: PO : 2-6 hour

IV : 1-4 hour

Crosses the placenta

Adjustment in renal failure

## Consider :

- ⊙ 1-ECG monitoring: Prolonged PR interval  
ST-T change  
Q wave
- ⊙ 2- Serum electrolyte level : Ca, K
- ⊙ Be aware of Hypokalemia , hypomagnesemia and hypercalcemia
- ⊙ Max dose should not exceed 0.2-0.5 mg/day
- ⊙ Increase the IV dosage about 20-25% while orally used



- ◉ *In case of suspected Dig toxicity:*
- ◉ Check Dig level 4 Hrs after the last dose
- ◉ Appropriate level : infants : 2-4 ng/ml  
children: 1-2 ng/ml

## 2-Diuretic :

2-1:**Furosemide** : 1-4 mg/kg/day

- Most commonly used
- Inhibits the reabsorption of sodium and chloride in distal tubule and the loop of Henle
- Careful monitoring of electrolytes
- May cause metabolic alkalosis

2-2:**Spirinolactone** : 2-3 mg/kg/day

- K sparing
- Aldosterone inhibitor
- Improves survival in adult

## ٢-٣: Chlorothiazide : ١-٢ mg/kg/day

- Hydrochlorothiazide, metolazone
- Less potent
- Potassium supplement is required

## ٢-٤: Afterload reducing agent and ACE I :

- Decreased the SVR
- May decrease the systemic venous tone which decreases the preload
- Beneficial in CMP, severe AR, MR and Lt to Rt shunt.
- Not used in obstructive lesions.
- Decreases cardiac remodeling

**Captopril : 0.3-6 mg/kg/day**

- ⊙ Arterial and veno dilatation
- ⊙ Side effects: hypotension, maculopapular pruritic rash , renal toxicity , chronic cough , neutropenia

**Enalapril :**

- ⊙ long acting ACE I

**2-5-Angiotensin receptor blocker : Losartan**

**3-Nesiritide (recombinant BNP) :**

- ⊙ Arterial, veno and coronary dilatation

## ☿-Nitroprusside:

- ⊙ Arterial and venous dilation
- ⊙ Contraindicated in pre existing hypotension
- ⊙ Risk of thiocyanate toxicity (fatigue, nausea, disorientation, acidosis, and muscular spasm).
- ⊙ Toxic thiocyanate level :  $> 10 \text{ g/dl}$

## ♁-Hydralazine :

- ⊙ Arteriolar vasodilation

## **ϕ-Angiotensin Receptor-Neprilysin Inhibitors :**

- ⊙ sacubitril-valsartan

## **∇- Beta blocker :**

- ⊙ Metoprolol, carvedilol, bisoprolol
- ⊙ Improve exercise tolerance, decrease hospitalizations, and reduce overall mortality
- ⊙ In chronic heart failure not the acute phase

## **α-Alpha and Beta adrenergic agents :**

### **Dopamine :**

- ⊙ Beta adrenergic with Alpha adrenergic effect in higher doses ( $1.5$  micro/kg/min), dopaminergic

### **Dobutamine : $2-20$ micro/kg/min**

- ⊙ Inotrope , decreased SVR, adjunct to dopamine

## **Isoproterenol :**

- ⦿ Pure Beta adrenergic, chronotrope , increase HR
- ⦿ May cause arrhythmia

## **Epinephrine :**

- ⦿ Mixed Alpha and Beta adrenergic
- ⦿ Increases SVR
- ⦿ Used in cardiogenic shock , hypotension



## 9- Phosphodiesterase inhibitors :

- **Milrinone** : 0.25-1 micro/kg/min
- Inotrope, adjunct to dopamine and dobutamine
- Hypotension
- **Amrinone** :
- Thrombocytopenia

THANK YOU

