

University of Thi-Qar
College of Nursing

Congestive Heart Failure



Prepared By:

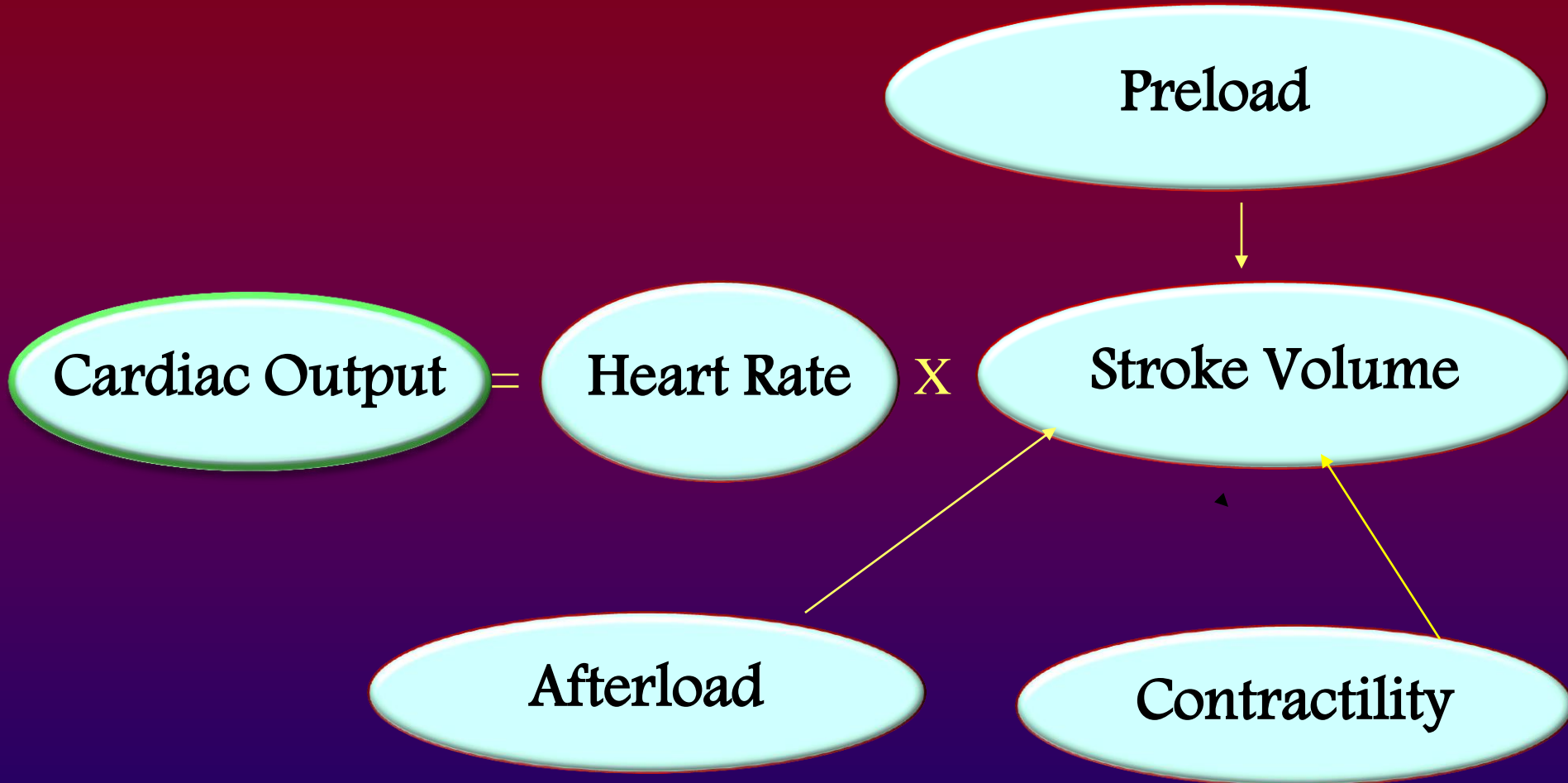
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Congestive Heart Failure

Definition

- ❑ Impaired cardiac pumping such that heart is unable to pump adequate amount of blood to meet metabolic needs
- ❑ Not a disease but a “**syndrome**”
- ❑ Associated with long-standing HTN and CAD

Factors Affecting Cardiac Output



Factors Affecting Cardiac Output

- Heart Rate

- In general, the higher the heart rate, the lower the cardiac

- E.g. $HR \times SV = CO$

- » $60/\text{min} \times 80 \text{ ml} = 4800 \text{ ml/min}$ (4.8 L/min)

- » $70/\text{min} \times 80 \text{ ml} = 5600 \text{ ml/min}$ (5.6 L/min)

- But only up to a point. With excessively high heart rates, diastolic filling time begins to fall, thus causing stroke volume and thus CO to fall

Heart Rate	Stroke Volume	Cardiac Output
60/min	80 ml	4.8 L/min
80/min	80/ml	6.4 L/min
100/min	80/ml	8.0 L/min
130/min	50/ml	6.5 L/min
150/min	40/ml	6.0 L/min

Factors Affecting Cardiac Output

- Preload
 - The volume of blood/amount of fiber stretch in the ventricles at the end of diastole (i.e., before the next contraction)

Factors Affecting Cardiac Output

- **Preload increases with:**
 - Fluid volume increases
 - Vasoconstriction (“squeezes” blood from vascular system into heart)
- **Preload decreases with**
 - Fluid volume losses
 - Vasodilation (able to “hold” more blood, therefore less returning to heart)

Factors Affecting Cardiac Output

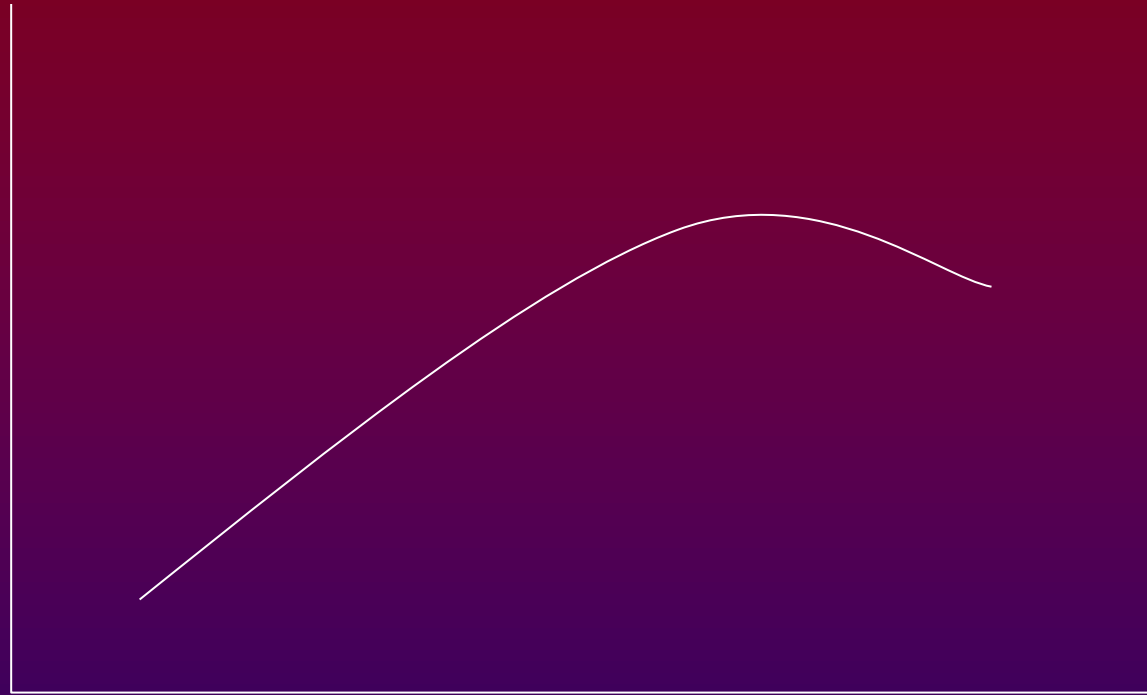
- **Starling's Law**

- Describes the relationship between preload and cardiac output

- The greater the heart muscle fibers are stretched (b/c of increases in volume), the greater their subsequent force of contraction – but only up to a point. Beyond that point, fibers get over-stretched and the force of contraction is reduced

- **Excessive preload = excessive stretch → reduced contraction → reduced SV/CO**

Cardiac
Output



End Diastolic Volume
(preload)

Factors Affecting Cardiac Output

- Afterload

- The resistance against which the ventricle must pump. Excessive afterload = difficult to pump blood
→ reduced CO/SV

- Afterload increased with:

- Hypertension
 - Vasoconstriction

- Afterload decreased with:

- Vasodilatation

Factors Affecting Cardiac Output

- **Contractility**
 - Ability of the heart muscle to contract; relates to the strength of contraction.

Factors Affecting Cardiac Output

- **Contractility decreased with:**
 - infarcted tissue – no contractile strength
 - ischemic tissue – reduced contractile strength.
 - Electrolyte/acid–base imbalance
 - Negative inotropes (medications that decrease contractility, such as beta blockers).
- **Contractility increased with:**
 - Sympathetic stimulation (effects of epinephrine)
 - Positive inotropes (medications that increase contractility, such as digoxin, sympathomimetics)

Pathophysiology of CHF

- Pump fails → decreased stroke volume /CO.
- Compensatory mechanisms kick in to increase CO
 - SNS stimulation → release of epinephrine/nor-epinephrine
 - Increase HR
 - Increase contractility
 - Peripheral vasoconstriction (increases afterload)
 - Myocardial hypertrophy: walls of heart thicken to provide more muscle mass → stronger contractions

Pathophysiology of CHF

- Hormonal response: ↓'d renal perfusion interpreted by juxtaglomerular apparatus as hypovolemia. Thus:
 - Kidneys release renin, which stimulates conversion of angiotensin I → angiotensin II, which causes:
 - Aldosterone release → Na retention and water retention (via ADH secretion)
 - Peripheral vasoconstriction

Pathophysiology of CHF

- Compensatory mechanisms may restore CO to near-normal.
- But, if excessive the compensatory mechanisms can worsen heart failure because . . .

Pathophysiology of CHF

- Vasoconstriction: ↑'s the resistance against which heart has to pump (i.e., ↑'s afterload), and may therefore ↓ CO
- Na and water retention: ↑'s fluid volume, which ↑'s preload. If too much "stretch" (d/t too much fluid) → ↓ strength of contraction and ↓'s CO
- Excessive tachycardia → ↓'d diastolic filling time → ↓'d ventricular filling → ↓'d SV and CO

Risk Factors

CAD

Age

HTN

Obesity

Cigarette smoking

Diabetes mellitus

High cholesterol

African descent

Congestive Heart Failure

Etiology

- May be caused by any interference with normal mechanisms regulating cardiac output (CO)
- Common causes
 - HTN
 - Myocardial infarction
 - Dysrhythmias
 - Valvular disorders

Congestive Heart Failure

Types of Congestive Heart Failure

- **Left-sided failure**

- Most common form

- Blood backs up through the left atrium into the pulmonary

- Pulmonary congestion and edema**

- Eventually leads to biventricular failure

Congestive Heart Failure

Types of Congestive Heart Failure

- **Left-sided failure**
 - Most common cause:
 - HTN
 - Cardiomyopathy
 - Valvular disorders
 - CAD (myocardial infarction)

Congestive Heart Failure

Types of Congestive Heart Failure

- **Right-sided failure**

- Results from diseased right ventricle
- Blood backs up into right atrium and venous circulation
- Causes

- LVF

- Cor pulmonale

- RV infarction

Congestive Heart Failure

Types of Congestive Heart Failure

- **Right-sided failure**
 - Venous congestion
 - Peripheral edema
 - Hepatomegaly
 - Splenomegaly
 - Jugular venous distension

Congestive Heart Failure

Types of Congestive Heart Failure

- **Right-sided failure**

- Primary cause is left-sided failure
- Cor pulmonale

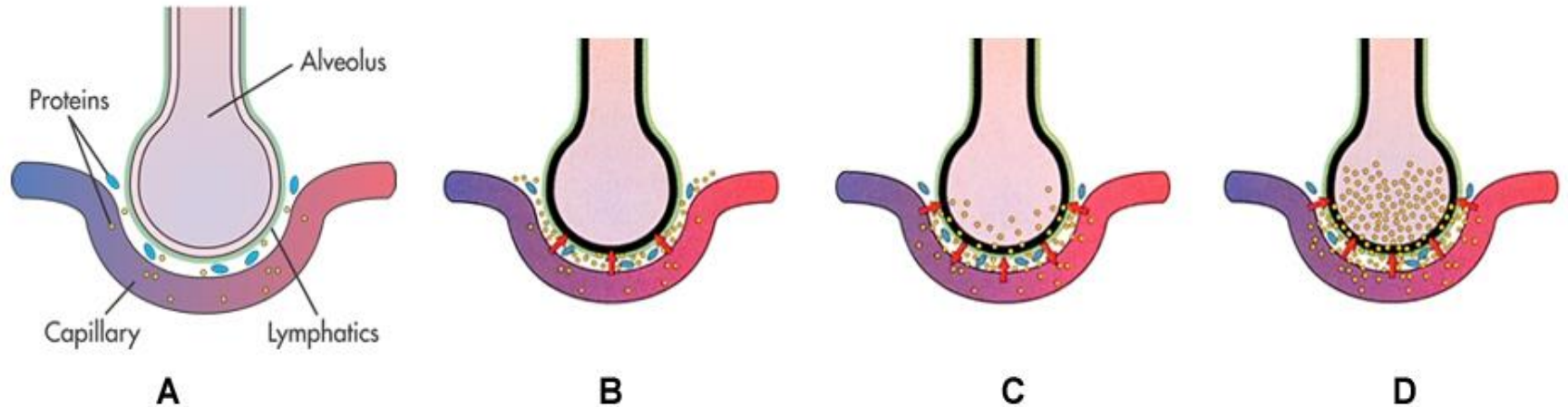
- RV dilation and hypertrophy caused by pulmonary pathology**

Acute Congestive Heart Failure

Clinical Manifestations

- Pulmonary edema (what will you hear?)
 - Agitation
 - Pale or cyanotic
 - Cold, clammy skin
 - Severe dyspnea
 - Tachypnea
 - Pink, frothy sputum

Pulmonary Edema



Redrawn from Urden LD, Stacy KM, Lough ME: *Thelan's critical care nursing: diagnosis and management*, ed 4, St Louis, 2002, Mosby.

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Fig. 34-2

Chronic Congestive Heart Failure

Clinical Manifestations

Fatigue

Dyspnea

▪ Paroxysmal nocturnal dyspnea (PND)

Tachycardia

Edema – (lung, liver, abdomen, legs)

Nocturia

Chronic Congestive Heart Failure

Clinical Manifestations

☐ Behavioral changes

- Restlessness, confusion, ↓ attention span

☐ Chest pain (d/t ↓ CO and ↑ myocardial work)

☐ Weight changes (r/t fluid retention)

☐ Skin changes

- Dusky appearance

Congestive Heart Failure

Classification

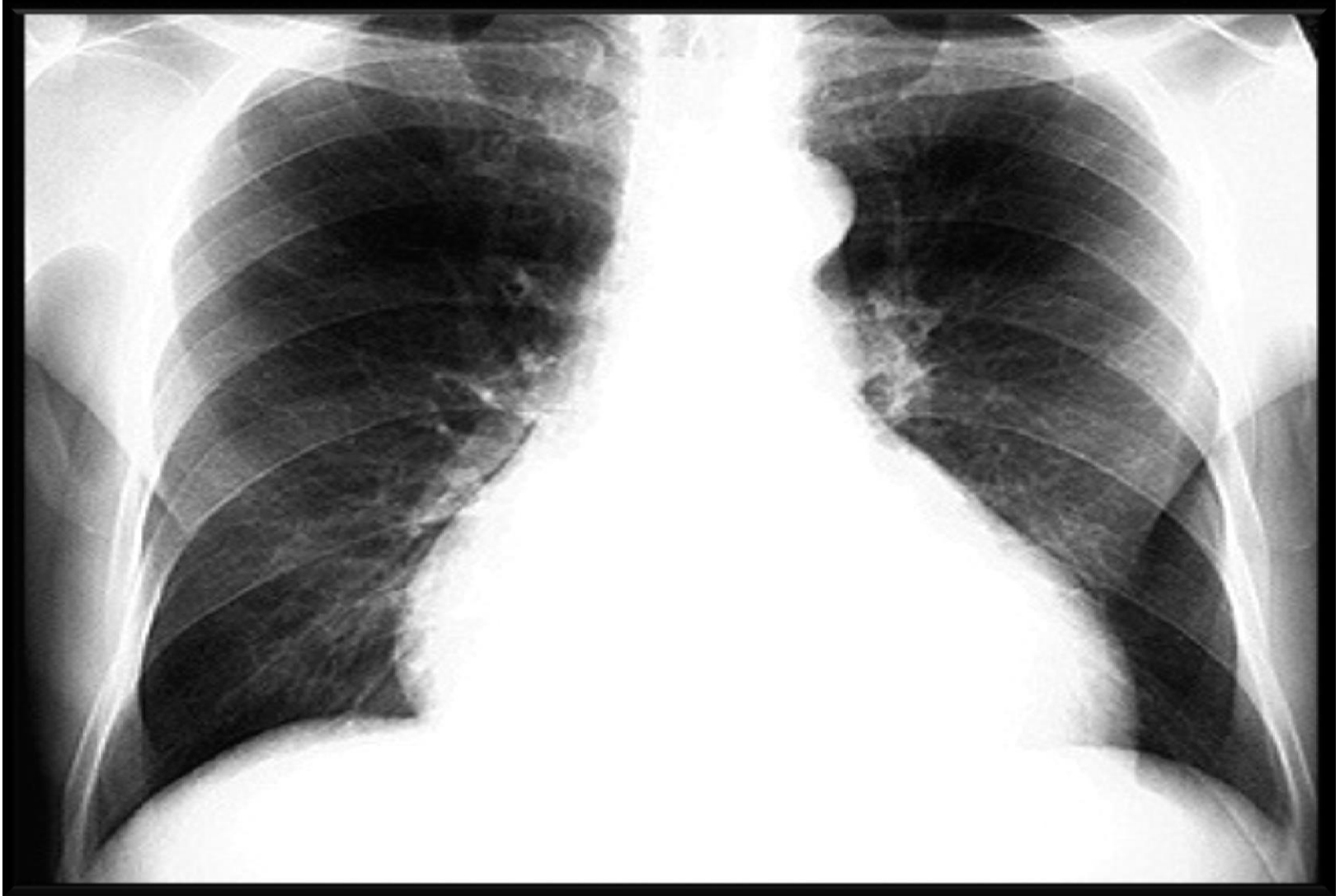
- **Based on the person's tolerance to physical activity**
 - ❑ **Class 1: No limitation of physical activity**
 - ❑ **Class 2: Slight limitation**
 - ❑ **Class 3: Marked limitation**
 - ❑ **Class 4: Inability to carry on any physical activity without discomfort**

Congestive Heart Failure

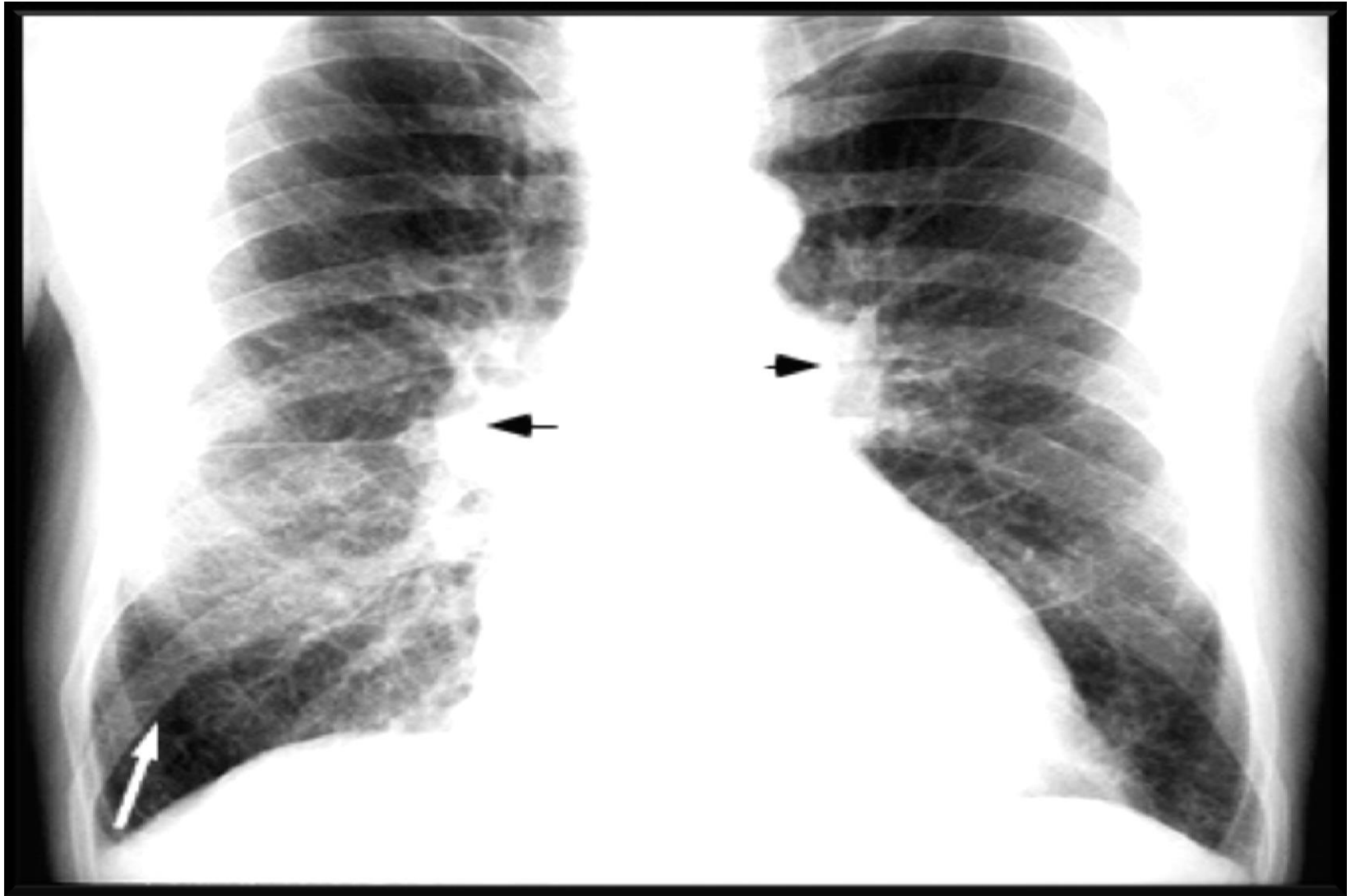
Diagnostic Studies

- **Primary goal is to determine underlying cause**
 - Physical exam
 - Chest x-ray
 - ECG
 - Hemodynamic assessment

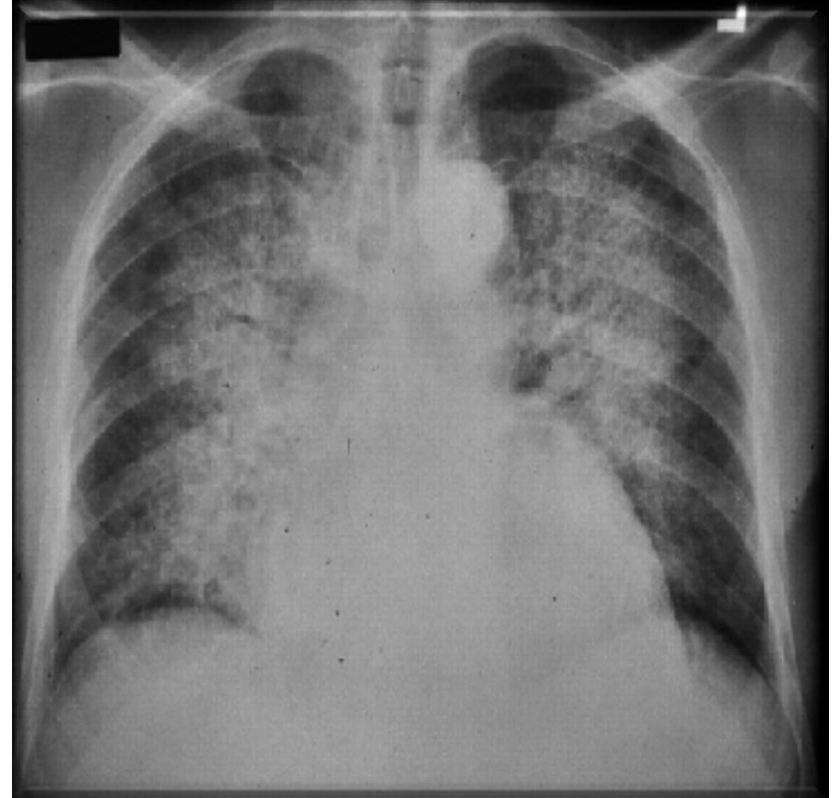
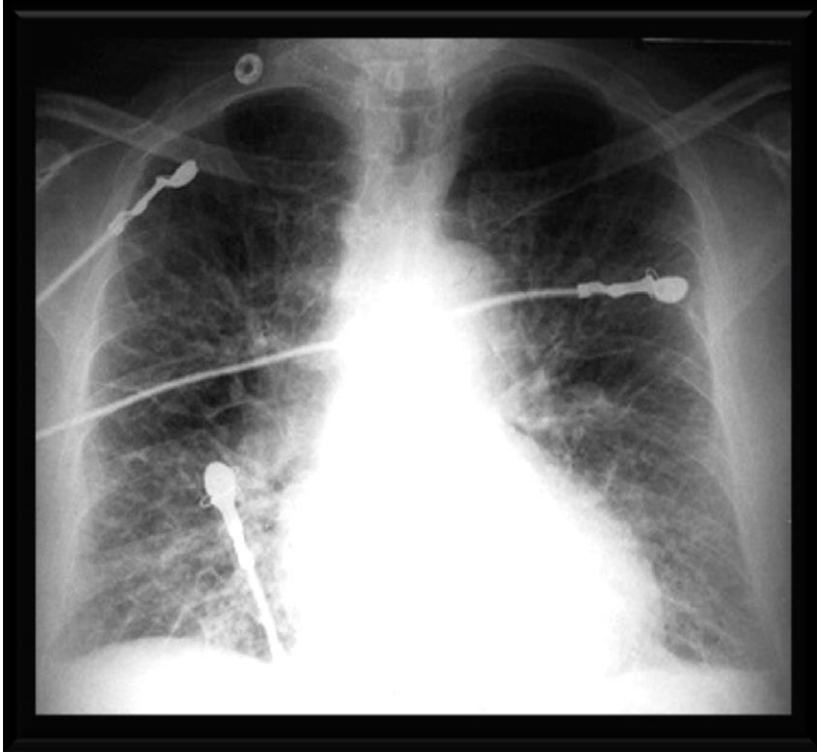
Cardiomegaly



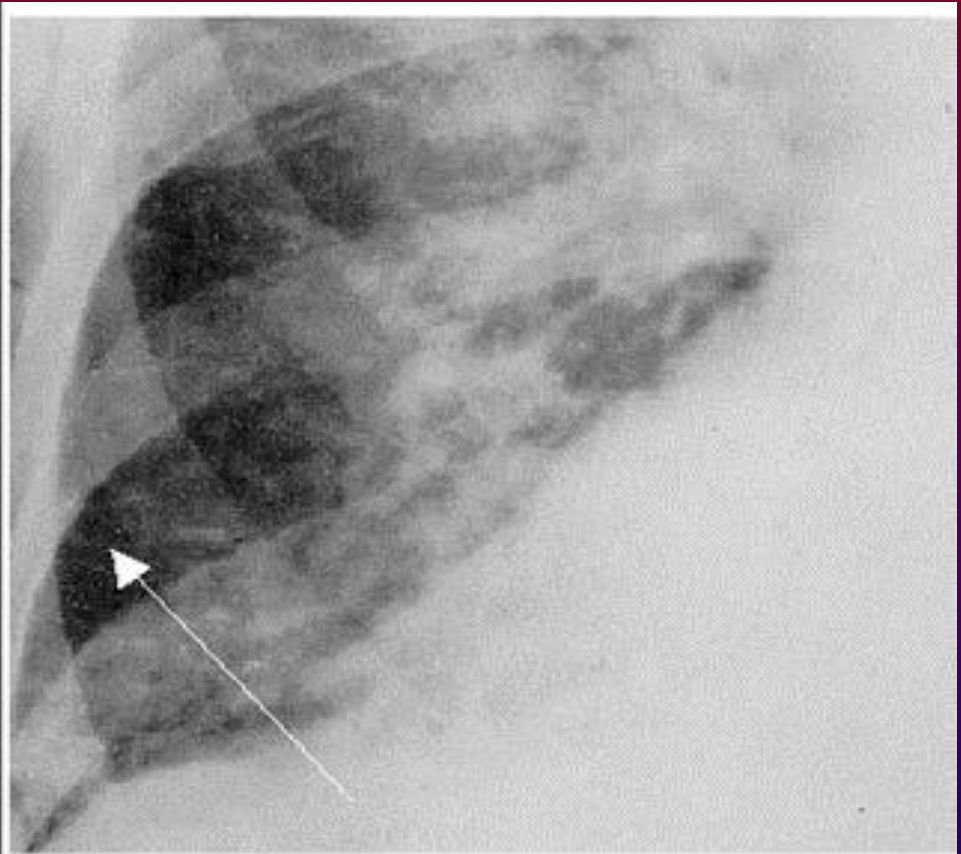
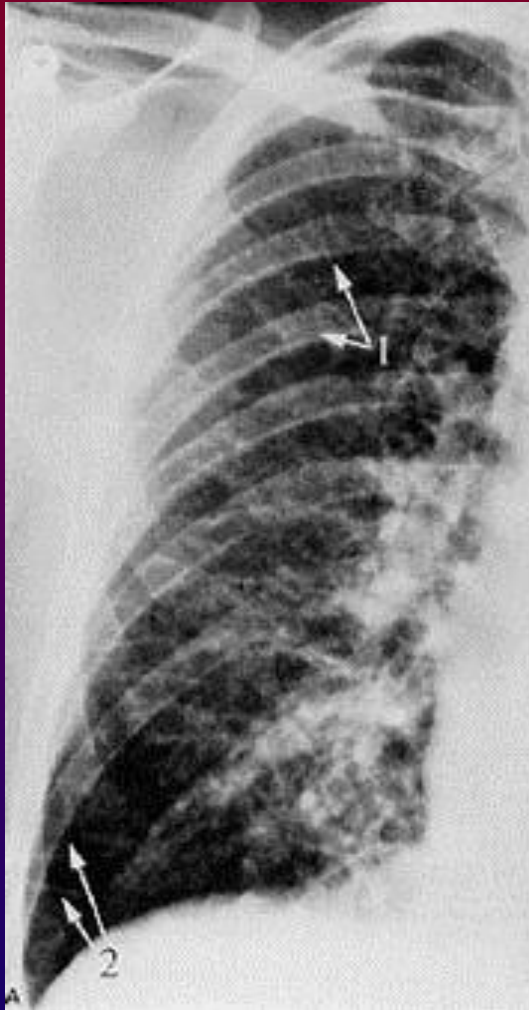
Pulmonary vessel congestion



Pulmonary Edema due to Heart Failure



Kerley B lines



Congestive Heart Failure

Diagnostic Studies

- **Primary goal is to determine underlying cause**
 - Echocardiogram (Uses ultrasound to visualize myocardial structures and movement, calculate EF)
 - Cardiac catheterization

Acute Congestive Heart Failure

Nursing and Collaborative Management

- Primary goal is to improve LV function by:
 - Decreasing intravascular volume
 - Decreasing venous return
 - Decreasing afterload
 - Improving gas exchange and oxygenation
 - Improving cardiac function
 - Reducing anxiety

Acute Congestive Heart Failure

Nursing and Collaborative Management

- Decreasing intravascular volume
 - Improves LV function by reducing venous return
 - Loop diuretic: drug of choice
 - Reduces preload
 - High Fowler's position

Acute Congestive Heart Failure

Nursing and Collaborative Management

- Decreasing afterload
 - Drug therapy:
 - vasodilatation, ACE inhibitors
 - Decreases pulmonary congestion

Acute Congestive Heart Failure

Nursing and Collaborative Management

- Improving cardiac function
 - Positive inotropes
- Improving gas exchange and oxygenation
 - Administer oxygen, sometimes intubate and ventilate
- Reducing anxiety
 - Morphine

Chronic Congestive Heart Failure

Collaborative Care

- Treat underlying cause
- Maximize CO
- Alleviate symptoms

Chronic Congestive Heart Failure

Collaborative Care

- Oxygen treatment
- Rest
- Biventricular pacing
- Cardiac transplantation

Chronic Congestive Heart Failure

Drug Therapy

- ACE inhibitors
- Diuretics
- Inotropic drugs
- Vasodilators
- β -Adrenergic blockers

Chronic Congestive Heart Failure

Nutritional Therapy

- ❑ Fluid restrictions not commonly prescribed
- ❑ Sodium restriction
 - 2 g sodium diet
- ❑ Daily weights
 - Same time each day
 - Wearing same type of clothing

Chronic Congestive Heart Failure

Nursing Management

Nursing Assessment

- Past health history
- Medications
- Functional health problems
- Cold, diaphoretic skin

Chronic Congestive Heart Failure

Nursing Management

Nursing Assessment

- Tachypnea
- Tachycardia
- Crackles
- Abdominal distension
- Restlessness

Chronic Congestive Heart Failure

Nursing Management

Nursing Diagnoses

- Activity intolerance
- Excess fluid volume
- Disturbed sleep pattern
- Impaired gas exchange
- Anxiety

Chronic Congestive Heart Failure

Nursing Management

Planning

- **Overall goals:**

- ↓ Peripheral edema
- ↓ Shortness of breath
- ↑ Exercise tolerance
- Drug compliance
- No complications

Chronic Congestive Heart Failure

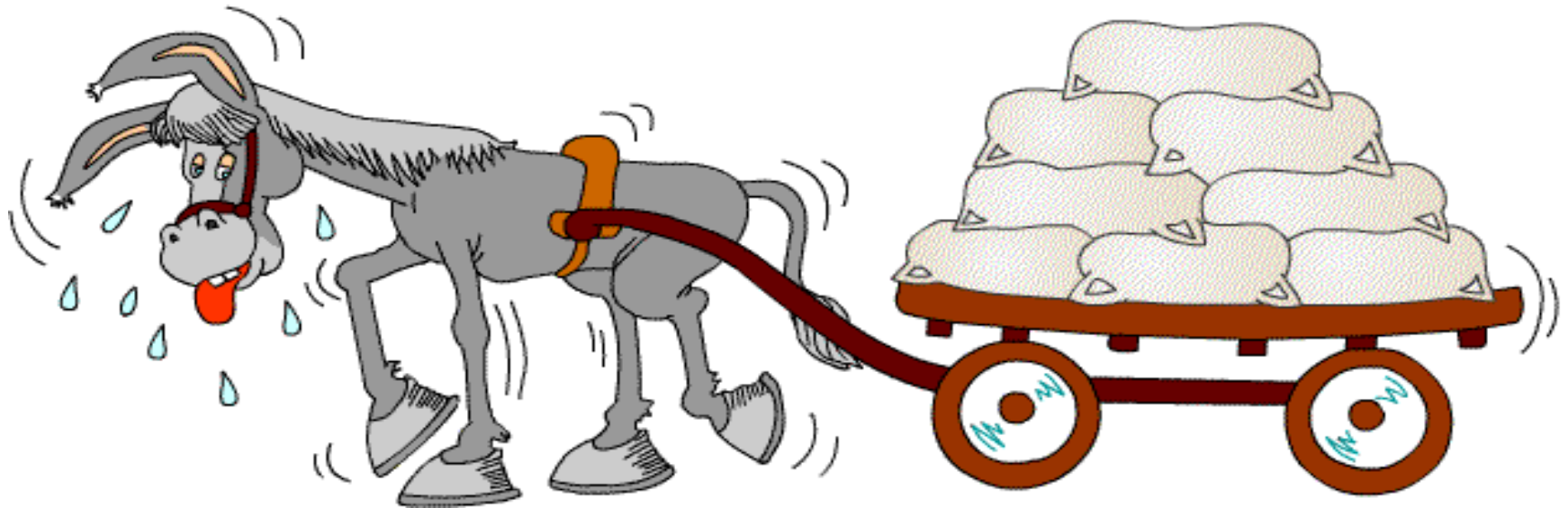
Nursing Management

Nursing Implementation

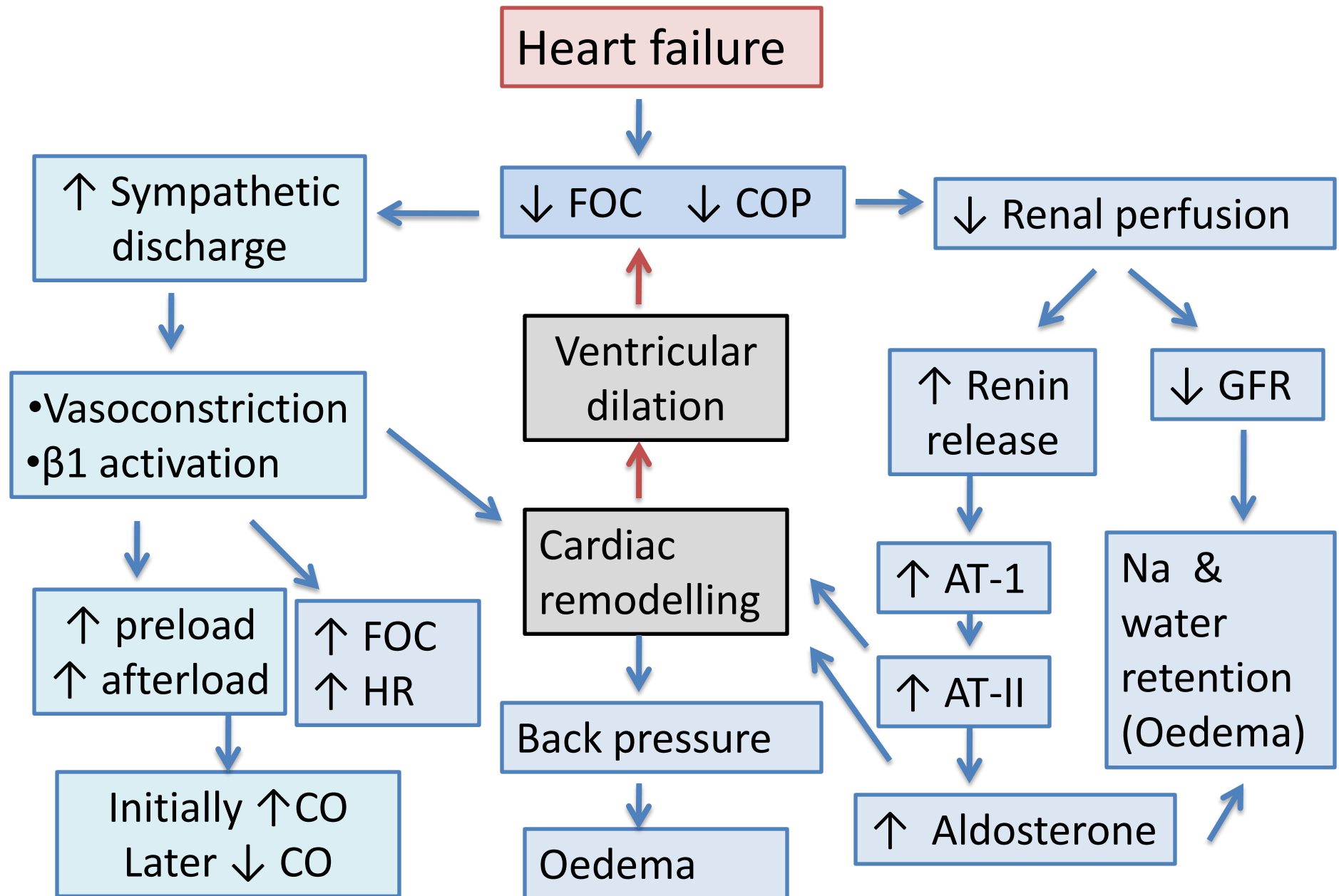
- **Acute intervention**
 - Establishment of quality of life goals
 - Symptom management
 - Conservation of physical/emotional energy
 - Support systems are essential

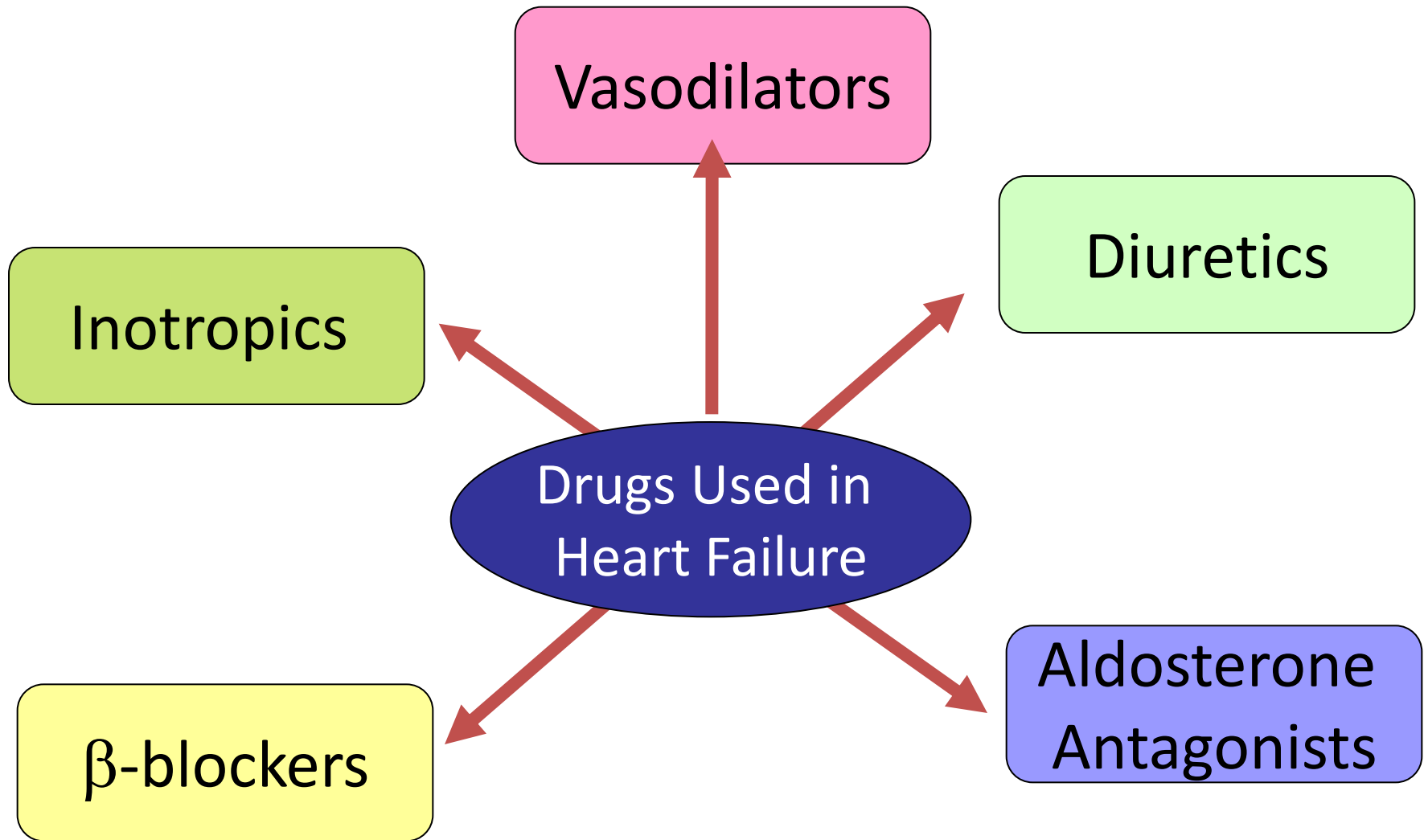
Pharmacological Management of Congestive Heart Failure

What is heart failure



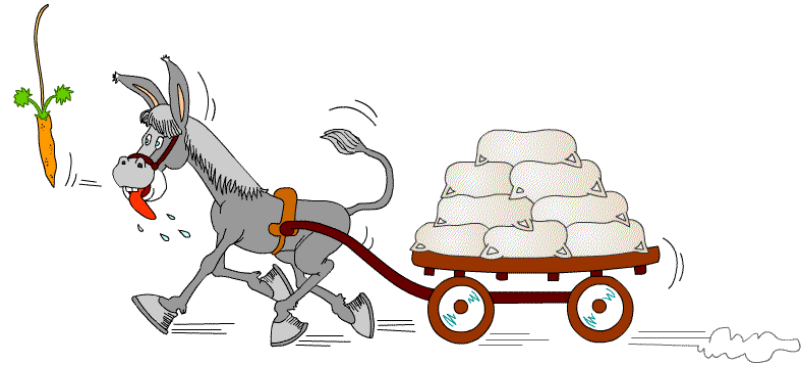
Compensatory responses during heart failure





Inotropic drugs

- **Cardiac glycosides:**
 - Digoxin, digitoxin
- **Sympathomimetic amines:**
 - Dopamine , dobutamine
- **Phosphodiesterase inhibitors:**
 - Amrinone , milrinone



Like the carrot placed in front of the donkey

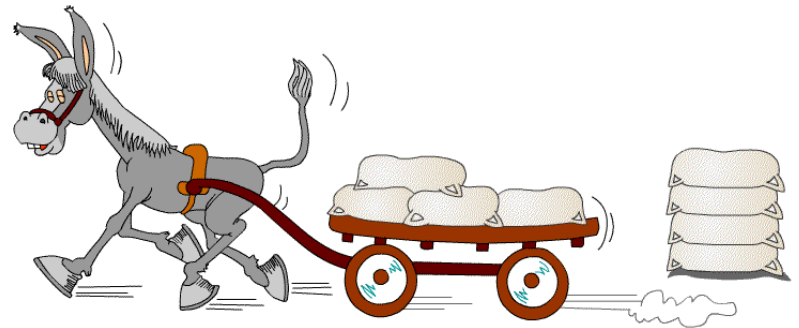
Vasodilators

- Arteriolar: hydralazine , minoxidil, nicorandil
- Venodilators: nitrates
- Arteriolar and venodilators: ACE inhibitors, angiotensin receptor blockers



Diuretics

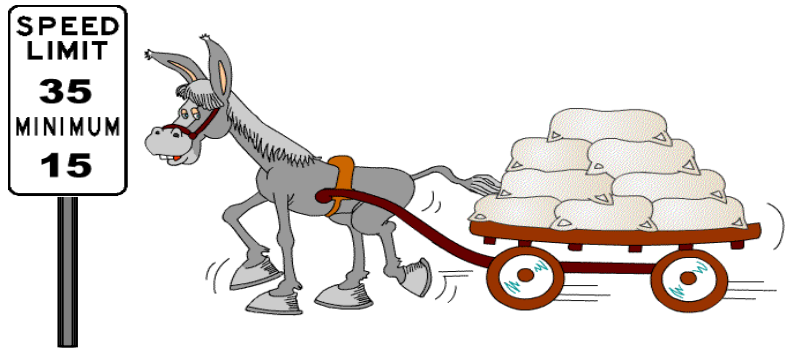
- Loop diuretics: furosemide, torsemide
- Thiazide diuretics: hydrochlorothiazide
- K⁺ Sparing diuretics:
 - Spironolactone (Also is aldosterone antagonist)
 - Amiloride



Reduce the number of
sacks on the wagon

Beta Blockers

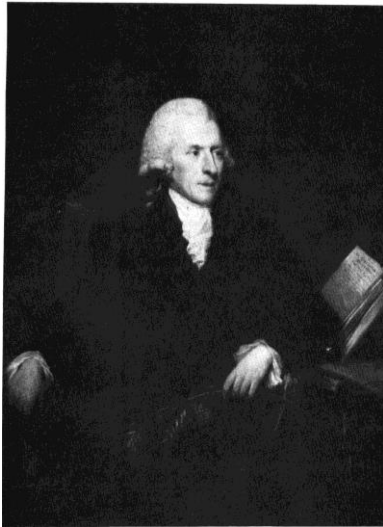
- Metoprolol, bisoprolol, carvedilol



Limit the donkey's speed,
thus saving energy

Inotropic Agents

- Cardiac glycosides: Digoxin

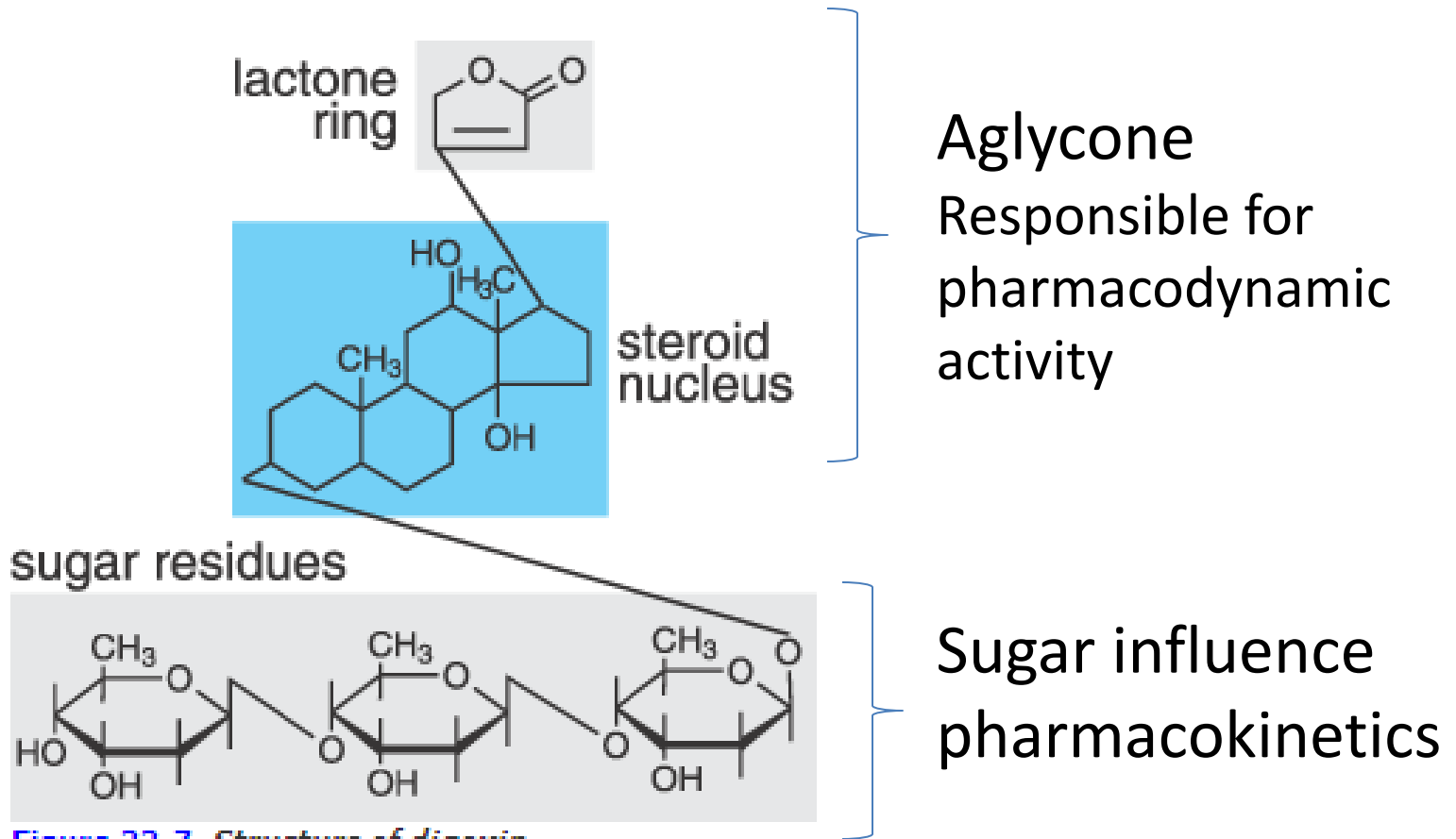


William Withering 1785



Foxglove plant

Chemistry of cardiac glycosides



Pharmacological actions

CARDIAC

- ↑force of contraction & Cardiac Output
- ↓ Heart rate
- ↓ Refractory period (RP) & ↑ Conduction velocity (CV) in atria/ventricles
- ↑ RP & ↓ CV in AV node
- Increased automaticity
- ECG: ↑PR interval , ↓ QT interval

EXTRA CARDIAC

- Kidney:
 - Due to improvement in circulation and renal perfusion
 - Retained salt and water is gradually excreted
- CNS:
 - Nausea, vomiting

Pharmacokinetic properties

Property	Digoxin	Digitoxin
Oral absorption	60 -80 %	90 -100 %
Plasma protein binding	25 %	95%
Onset of action	15 -30 min	½ to 1 hour
Duration of action	2-6 days	2-3 weeks
Plasma t ½	40 hrs	5-7 days
Route of elimination	Renal excretion	Hepatic metabolism
Time for digitalization	5-7 days	25-30 days
Daily maintenance dose	0.125 – 0.5 mg	0.05 -0.2 mg
Administration	Oral / IV	Oral

Cardiac Glycosides (Digitalis)

- Two glycosides:

Used

– Short acting **Digoxin** ($t_{1/2}$: **1.5** days)

– Long acting **Digitoxin** ($t_{1/2}$: **5** days)

Severely
limited Use

Uses of digoxin

- Congestive heart failure
- Cardiac arrhythmias
 - Atrial fibrillation
 - Atrial flutter
 - Paroxysmal supraventricular tachycardia

Adverse effects of digoxin

Extra-Cardiac

- GIT: Nausea & vomiting (first to appear)
- CNS: Vomiting
Restlessness,
Disorientation, Visual disturbance
- Endocrine:
Gynaecomastia

Cardiac

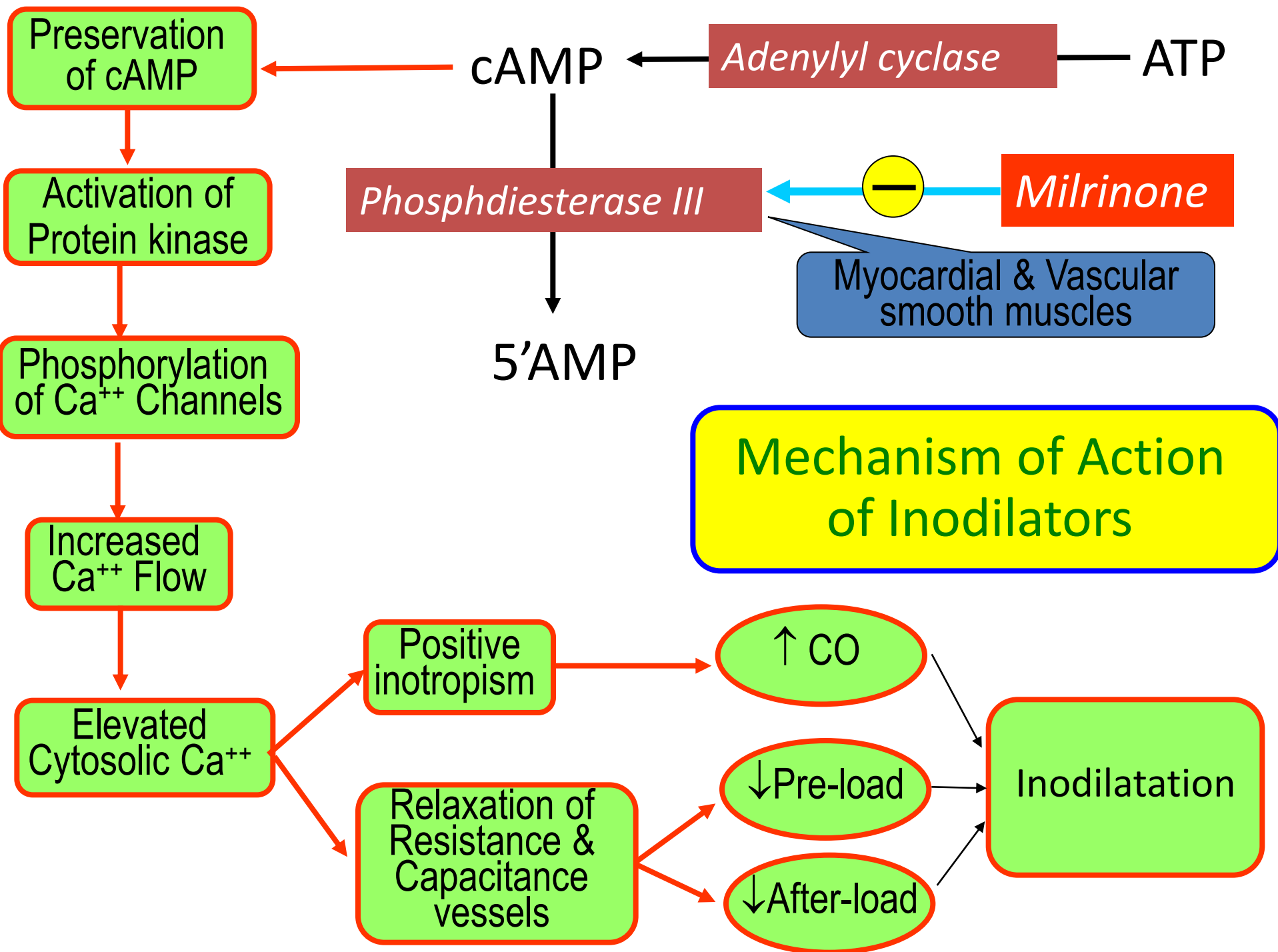
- Bradycardia (first cardiac toxic sign)
- Pulsus bigemini
- Atrial extra-systole → flutter → fibrillation
- Ventricular extra-systole → tachycardia → fibrillation
- Partial heart block → complete block

Treatment of toxicity

- Stop digitalis
- Oral or parenteral potassium supplements
- For ventricular arrhythmias:
 - Lidocaine IV drug of choice
- For supraventricular arrhythmia:
 - Propranolol may be given IV or orally
- For AV block and bradycardia
 - Atropine 0.6 -1.2 mg IM
- Digoxin antibody

Phosphodiesterase inhibitors in heart failure

- Amrinone & milrinone are selective phosphodiesterase III inhibitors
- ↑ cAMP levels
- The PDE III isoenzyme is specific for intracellular degradation of cAMP in heart, blood vessels and bronchial smooth muscles.
- Inodilators
- IV administration for short term treatment of severe heart failure
- Milrinone is more potent than amrinone and does not produce thrombocytopenia



Other inotropic drugs

- Dopamine
- Dobutamine

Role of diuretics in heart failure

- Almost all symptomatic Patients treated with a diuretic
- High ceiling diuretics (loop diuretics) preferred
 - Low dose therapy for maintenance
- They increase salt and water excretion & reduce blood volume
 - Reduce preload & venous pressure
 - Improve cardiac performance & relieve edema

ACE Inhibitors in heart failure

- Angiotensin converting enzyme inhibitors
 - Captopril, enalapril, ramipril, lisinopril
- Act by
 - Reduction of after load
 - Reduction of preload
 - Reversing the compensatory changes
- ACE inhibitors are the most preferred drugs for treatment of Congestive cardiac failure

Angiotensinogen



Renin

Angiotensin I

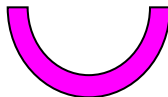
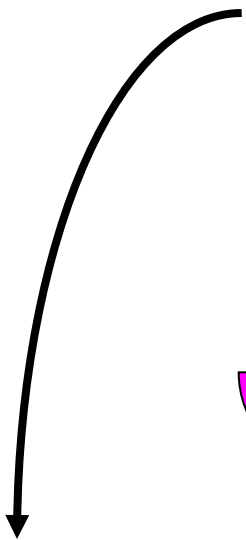


Angiotensin Converting Enzyme (ACE)

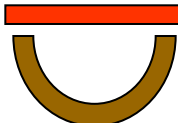
Synthesis Blocker

ACE inhibitor

Angiotensin II



AT2



AT1

Angiotensin Receptor Blocker

Receptor Blocker

Angiotensin III

Angiotensin receptor blockers in heart failure

- Losartan , candesartan, valsartan, telmisartan
- Block AT_1 receptor on the heart, peripheral vasculature and kidney
- As effective as ACE inhibitors
- Used mainly in patients who cannot tolerate ACE inhibitors because of cough, angioedema, neutropenia

Approach to the Patient with Heart Failure

Assessment of LV function (echocardiogram)

EF < 40%

Assessment of volume status

Signs and symptoms of fluid retention

Diuretic
(titrate to euvolemic state)

No signs and symptoms of fluid retention

ACE Inhibitor

β -blocker

Digoxin

Drugs used in heart failure

Chronic heart failure

- Diuretics
- Aldosterone receptor antagonist
- ACE inhibitors
- Angiotensin receptor blockers
- Cardiac glycosides
- Vasodilators

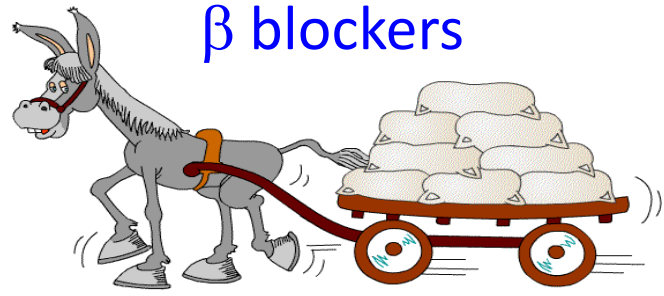
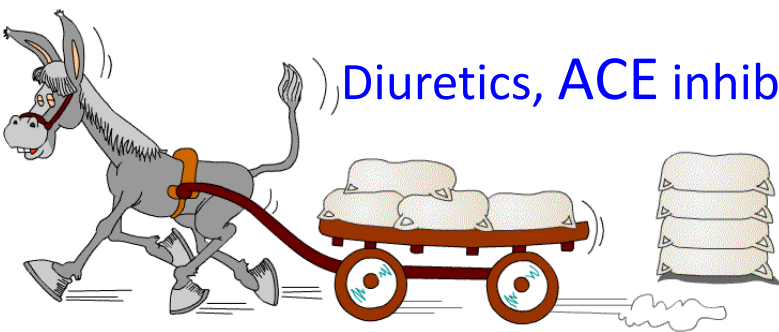
Acute heart failure

- Diuretics
- Vasodilators
- Dopamine, dobutamine
- Amrinone

Summary

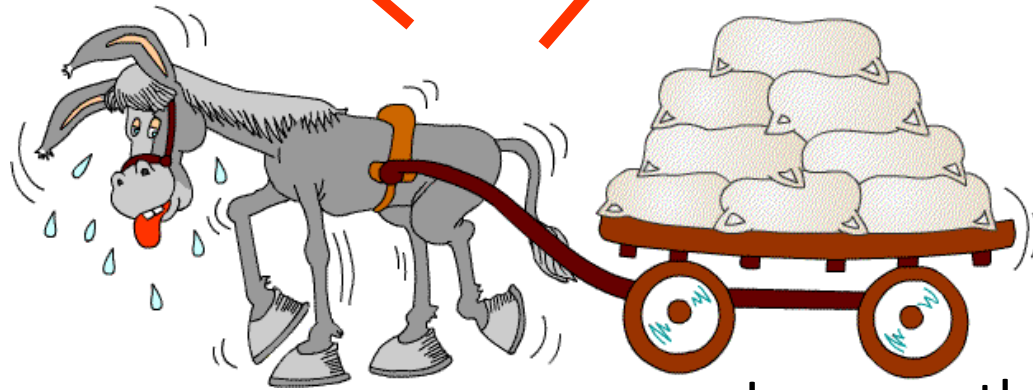
Diuretics, ACE inhibitors

β blockers



Reduce the number of sacks on the wagon

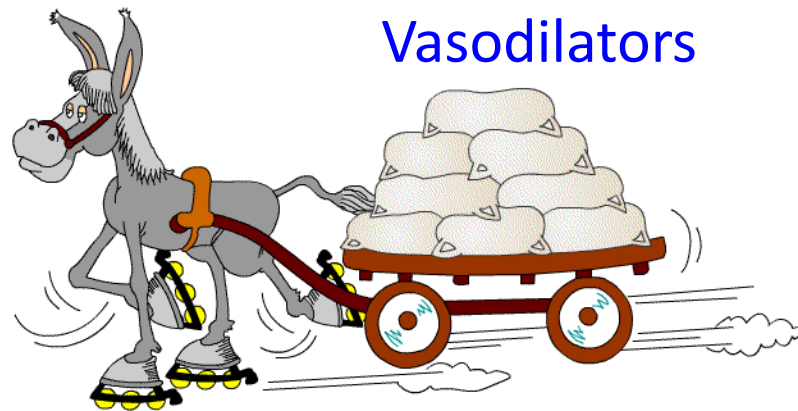
Limit the speed, thus saving energy



Increase the efficiency

Vasodilators

Like the carrot placed in front



Inotropes