بسم الله الرحمن الرحيم

HEART FAILURE

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HEART FAILURE

Clinical syndrome that can result from any structural or functional cardiac disorder that impairs the ability of the ventricle to fill with or eject blood

Causes of heart failure

- **Main causes**

Ischemic heart disease, Cardiomyopathy, Hypertension

- **Other causes**: Valvular heart disease, Congenital heart

disease, Alcohol and drugs, Hyperdynamic circulation (anaemia, thyrotoxicosis, haemochromatosis, Paget's disease), Right heart failure (RV infarct, pulmonary hypertension, pulmonary embolism, cor pulmonale (COPD)), Arrhythmia and Pericardial disease.

Mechanisms leading to heart failure

- Impaired cardiac contractility as in myocardial infarction and cardiomyopathy
- Ventricular outflow obstruction (pressure overload) as in hypertension and aortic stenosis
- Impaired ventricular fillings as in mitral stenosis and constrictive pericarditis
- Volume overload as in mitral regurgitation

PRECIPITATING FACTORS

- **Infections**
- **Arrhythmias**
- Physical, Dietary, Fluid, Environmental, and Emotional Excesses.
- Myocardial infarction
- Pulmonary embolism
- Anemia
- Thyrotoxicosis and pregnancy
- Aggravation of hypertension
- Rheumatic, Viral, and Other Forms of Myocarditis
- Infective endocarditis

Adaptive mechanisms

The heart depends on a number of adaptive mechanisms for maintenance of its pumping function:

1- The Frank Starling mechanism (cardiac dilatation)

2- Myocardial hypertrophy

3- Increased release of catecholamines, activation of renin-angiotensin-aldosteron system and other Neurohumoral adjustments

N.B.: These effects are compensatory at first, then they are overwhelmed and become pathophysiological

PATHOPHYSIOLOGICAL CHANGES

- Ventricular dilatation
- Myocyte hypertrophy
- Increased collagen synthesis
- Altered myosin gene expression
- Altered sarcoplasmic Ca2+-ATPase density
- Increased ANP secretion
- Salt and water retention
- Sympathetic stimulation
- Peripheral vasoconstriction

Neurohormonal changes

N/H changes	Favorable effect	Unfavor. effect
↑ **Sympathetic activity**	↑ HR ,↑ contractility, vasoconst. → ↑ V return, ↑ filling	Arteriolar constriction → After load →↑ workload →↑ O_2 consumption
↑ **Renin-Angiotensin – Aldosterone**	Salt & water retention→↑ VR	Vasoconstriction → ↑ after load
↑ **Vasopressin**	Same effect	Same effect
↑ **interleukins &TNFα**	May have roles in myocyte hypertrophy	Apoptosis
↑**Endothelin**	Vasoconstriction→↑ VR	↑ After load

Cardiac remodeling:

1. Hypertrophy & Dilatation

⇩

•↑ E.D.V

⇩ C.O.P

2. ↑Sympathetic activity:

- ↑ H.R.
- V.C

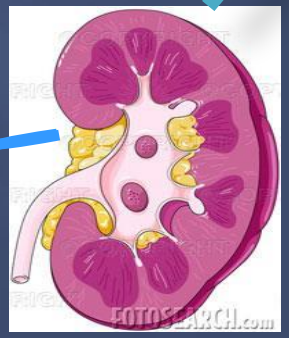
↑Pre-load.

↑After-load

Angiotensine

Aldosterone

Na^+ & water retention ↑



CLINICAL SYNDROMES OF HEART FAILURE

- Left ventricular systolic dysfunction (LVSD) is commonly caused by ischaemic heart disease but can also occur with valvular heart disease and hypertension.
- Right ventricular systolic dysfunction (RVSD) may be secondary to chronic LVSD but can occur with primary and secondary pulmonary hypertension, right ventricular infarction.
- Diastolic heart failure is a syndrome consisting of symptoms and signs of heart failure with preserved left ventricular ejection fraction above 45–50% and abnormal left ventricular relaxation assessed by echocardiography. Diastolic heart failure is more common in elderly hypertensive patients but may occur with primary cardiomyopathies (hypertrophic, restrictive, infiltrative).

SYMPTOMS & SIGNS OF HEART FAILURE

- **Left heart failure**

Symptoms are predominantly fatigue, exertional dyspnoea, orthopnoea and PND

*Physical sig*ns: Cardiomegaly, gallop, functional mitral regurgitation and crackles at the lung bases.

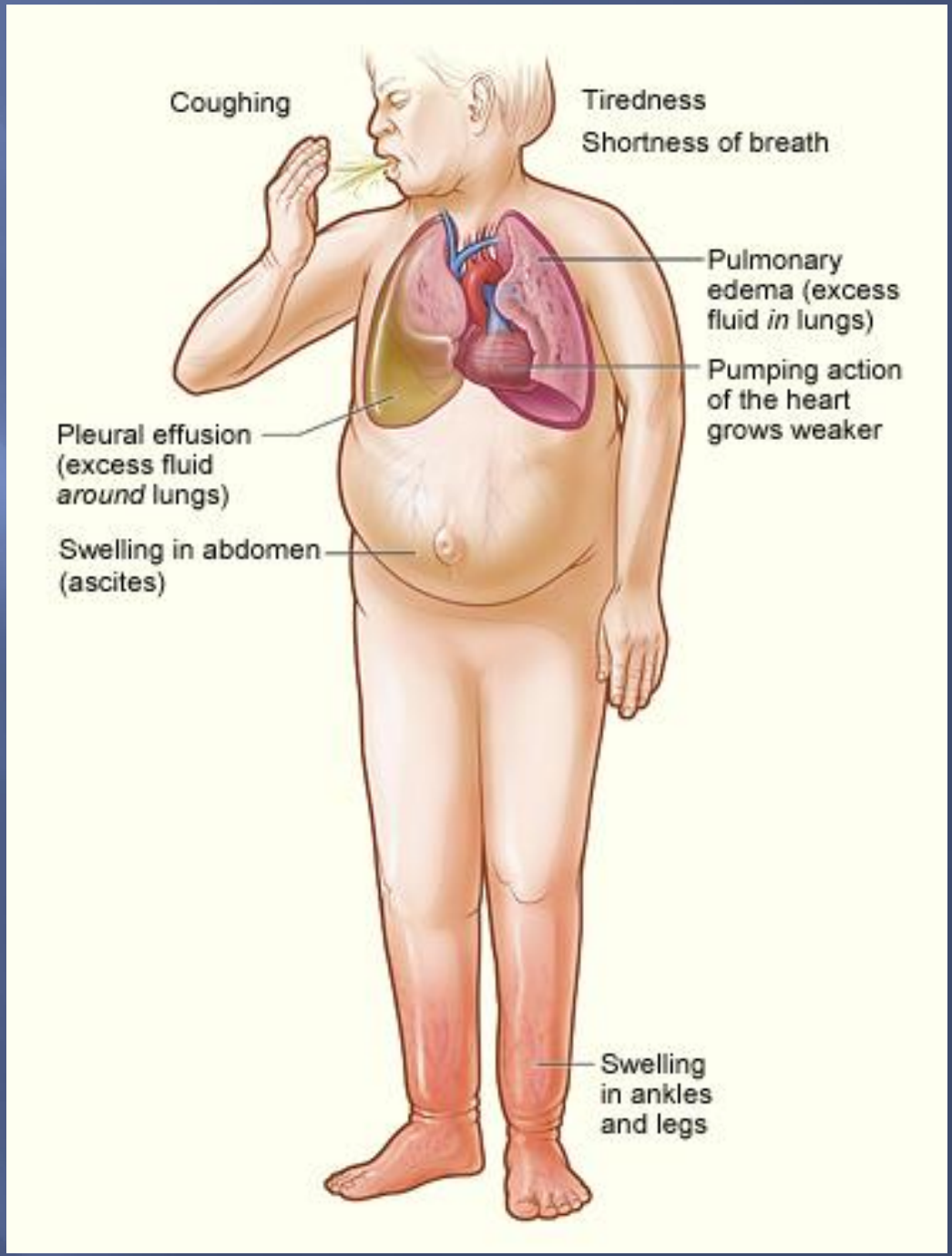
Right heart failure

Symptoms (fatigue, breathlessness, anorexia and nausea) relate to distension and fluid accumulation in areas drained by the systemic veins.

Physical signs are usually more prominent than the symptoms, with:

- jugular venous distension (± *v* waves of tricuspid regurgitation)
- tender smooth hepatic enlargement
- dependent pitting oedema
- development of free abdominal fluid (ascites)
- pleural transudates (commonly right-sided).
- Dilatation of the right ventricle produces cardiomegaly and may give rise to functional tricuspid regurgitation. Tachycardia and a right ventricular third heart sound are usual.

Major symptoms & signs of heart failure



New York Heart Association (NYHA) Classification of heart failure

- Class I No limitation. Normal physical exercise does not cause fatigue, dyspnoea or palpitations
- Class II Mild limitation. Comfortable at rest but normal physical activity produces fatigue, dyspnoea or palpitations
- Class III Marked limitation. Comfortable at rest but less gentle physical activity produces marked symptoms of heart failure
- Class IV Symptoms of heart failure occur at rest and are exacerbated by any physical activity

Framingham Criteria for Diagnosis of Congestive Heart Failure

- ***MAJOR CRITERIA*** PND, Neck vein distention, Rales, Cardiomegaly, Acute pulmonary edema, S_3 gallop, Increased venous pressure (>16 cmH_2O) and Positive hepatojugular reflux.
- ***MINOR CRITERIA*** Extremity edema, Night cough, Dyspnea on exertion, Hepatomegaly, Pleural effusion, Vital capacity reduced by one-third from normal and Tachycardia (≥120 bpm). Weight loss ≥4.5 kg over 5 days' treatment.
- To establish a clinical diagnosis of congestive heart failure by these criteria, at least one major and two minor criteria are required.

Diagnostic Investigations

- **Blood tests** - CBC, liver biochemistry, urea and electrolytes, cardiac enzymes , BNP or N-terminal portion of proBNF (NPproBNP), thyroid function.
- **Chest X-ray** - cardiac size and evidence of pulmonary congestion
- **Electrocardiogram** - evidence of ischaemia, hypertension or arrhythmia.
- **Echocardiography**. To establish the presence of systolic and/or diastolic impairment of the left or right ventricle. They may also reveal the aetiology

BRAIN NATRIURETIC PEPTIDE (BNP)

- Pre pro-BNP is formed in the ventricles and, with myocyte stretch, is broken down to N-terminal-pro-BNP (NT-pro-BNP) and BNP.
- These hormones are highly accurate for identifying or excluding HF with high sensitivity and specificity.
- BNP is particularly valuable in differentiating cardiac from pulmonary causes of dyspnea.
- The availability of a bedside assay makes BNP useful in evaluating patients in the Emergency Department.

- *Stress echocardiography. Exercise or pharmacological stress echocardiography has no radiation hazard and is a reliable technique for detecting ischaemia*
- *Nuclear cardiology. Radionuclide angiography (RNA) provides accurate measurements of left, and to a lesser extent, right ventricular ejection fractions, cardiac volumes and regional wall motion.*
- *Cardiac MRI.*
- *Positron emission tomography (PET). When other tests (e.g. stress echocardiography) cannot provide satisfactory results, PET scanning can be used, especially to identify potentially viable muscle that is hibernating.*
- *Cardiac catheterization*
- *Cardiac biopsy for infiltrative disease, e.g. amyloid.*

Treatment of Heart Failure

The treatment of HF may be divided into five components:

(1) general measures.
(2) Correction of the underlying cause.
(3) Removal of the precipitating cause.
(4) Prevention of deterioration of cardiac function.
(5) Control of the congestive HF state.

Treatment of Heart Failure

- **General measures:**

  Rest, salt restriction, stop smoking

- **Removal of the cause:**

  This deserves top priority in all cases and includes surgical measures correcting valvular lesions or congenital malformations and medical measures for treating hypertension or infective endocarditis when Present

- **Removal of precipitating causes of heart failure**

Treatment of heart failure

⇩C.O.P

1. Hypertrophy & Dilatation

⇩

•↑ E.D.V

Positive Inotropics

2. ↑Sympathetic activity:

- □ H.R.
- V.C

↑ Pre-load.

↑ After-load

vasodilators

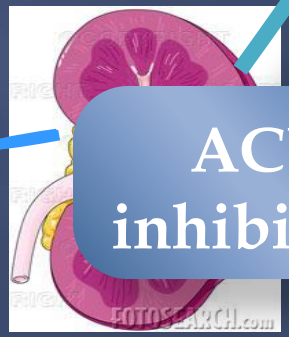
Angiotensine

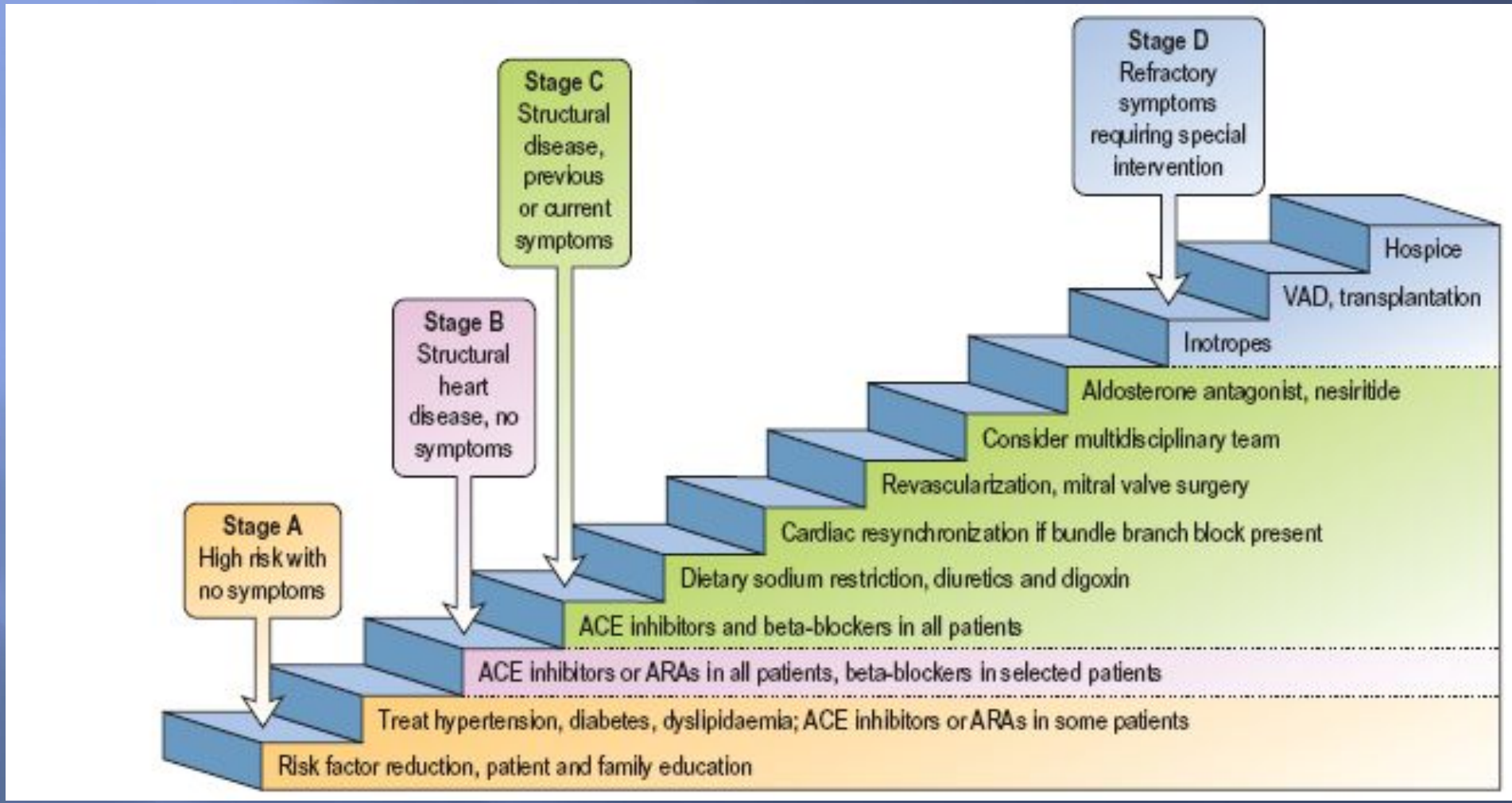
Diuretics

ACE inhibitors

Na⁺ & water retention

Aldosterone




Stage A
High risk with no symptoms
Stage B
Structural heart disease, no symptoms
Stage C
Structural disease, previous or current symptoms
Stage D
Refractory symptoms requiring special intervention
Risk factor reduction, patient and family education
Treat hypertension, diabetes, dyslipidaemia; ACE inhibitors or ARAs in some patients
ACE inhibitors or ARAs in all patients, beta-blockers in selected patients
ACE inhibitors and beta-blockers in all patients
Dietary sodium restriction, diuretics and digoxin
Cardiac resynchronization if bundle branch block present
Revascularization, mitral valve surgery
Consider multidisciplinary team
Aldosterone antagonist, nesiritide
Inotropes
VAD, transplantation
Hospice

Diuretics

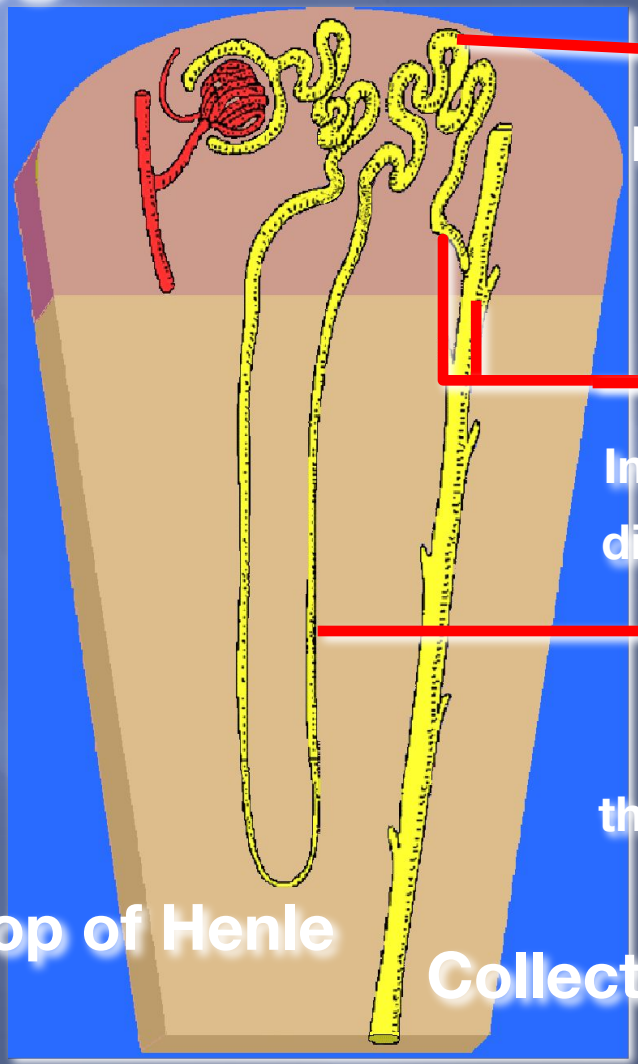
- Essential to control symptoms secondary to fluid retention
- Prevent progression from HT to HF
- Spironolactone improves survival
- New research in progress

Diuretics & ACEI reduces the number of sacks on the wagon

Diuretics

Cortex

Medulla



Loop of Henle

Collecting tubule

Thiazides

Inhibit active exchange of Cl-Na in the cortical diluting segment of the ascending loop of Henle

K-sparing

Inhibit reabsorption of Na in the distal convoluted and collecting tubule

Loop diuretics

Inhibit exchange of Cl-Na-K in the thick segment of the ascending loop of Henle

Pharmacological Treatment

- ***Diuretics***

(loop diuretics, thiazide diuretics and potassium sparing diuretics)

These act by promoting the renal excretion of salt and water by blocking tubular reabsorption of sodium and chloride. The resulting loss of fluid reduces ventricular filling pressures (preload), produces consistent haemodynamic and symptomatic benefits and rapidly improves dyspnoea and peripheral oedema.

Diuretics (continue)

Loop diuretics: such as furosemide and bumetanide

- Have a rapid onset of action (i.v. - 5 min; oral - 1-2 h) and generally short-lived (4-6 h) diuresis as the concentrating power of the kidney is reduced.
- These agents also produce potassium loss and promote hyperuricaemia, and renal function should be monitored.

Diuretics (continue)

Thiazide diuretics:

- Thiazides are less effective in patients with reduced glomerular filtration rates.
- Thiazide diuretics in combination with loop diuretics have a synergistic action and greater diuretic effect. Associated metabolic abnormalities are more likely and close supervision is needed.

Diuretics (continue)

Potassium-sparing diuretics:

- *Spironolactone* is a specific competitive antagonist to aldosterone, producing a weak diuresis but with a potassium-sparing action.
- The Randomized Aldactone Evaluation Study (RALES) showed a 30% reduction in all-cause mortality when spironolactone (up to 25 mg) was added to conventional treatment in patients with moderate to severe heart failure.
- Risk factors for developing hyperkalaemia include spironolactone dose > 50 mg/day, high-dose angiotensin-converting enzyme inhibitor (ACEI) and renal impairment.

Vasodilator therapy

Angiotensin-converting enzyme inhibitors ACEI:

- ACEI lower systemic vascular resistance and venous pressure, and reduce levels of circulating catecholamines, thus improving myocardial performance.
- These drugs should be carefully introduced in patients with heart failure because of the risk of first-dose hypotension.
- ACEI are contraindicated in patients with bilateral renal artery stenosis.
- Between 10% and 15% of patients develop a cough, owing to the inhibition of bradykinin metabolism.

Vasodilator therapy

Angiotensin receptor antagonists:

Angiotensin II receptor antagonists (ARA) (e.g. losartan, ibersartan, candesartan and valsartan) have similar haemodynamic effects to ACEI, but as they do not affect bradykinin metabolism, they do not produce a cough.

Arteriolar vasodilators:

- Drugs such as α-adrenergic blockers (e.g. prazosin) and direct smooth-muscle relaxants (e.g. hydralazine) are potent arteriolar vasodilators but are not very effective in heart failure.
- Calcium-channel blockers also reduce afterload, but first-generation calcium antagonists (diltiazem, nifedipine) may have a detrimental effect on left ventricular function in patients with heart failure.

Venodilators:

- Short- and long-acting nitrates act by reducing preload and lowering venous pressure, with resulting reduction in pulmonary and dependent oedema but tolerance occurs.
- Only combination therapy of nitrate with hydralazine has been shown to improve mortality and exercise performance, and may be useful when ACEI are contraindicated.

β-Adrenoceptor blocking agents

- There is considerable evidence to support the use of beta-blockers in patients with chronic stable heart failure. The current guidelines recommend that beta-blockers licensed for use (bisoprolol and carvedilol) in heart failure should be initiated in patients with confirmed heart failure due to left ventricular systolic dysfunction after diuretics and ACE inhibitor therapy, regardless of whether or not symptoms persist.
- Initial doses should be low, e.g. carvedilol 3.125 mg twice daily and should be titrated slowly.
- Nebivolol is used in the treatment of stable mild–moderate heart failure in patients over 70 years old.

ß-Blockers

Limit the donkey's speed, thus saving energy

Inotropic Agents

- Intravenous inotropes are frequently used to support myocardial function in patients with acute left ventricular failure and following cardiac surgery. Epinephrine (adrenaline), dobutamine, dopexamine and dopamine are intravenous adrenergic agonists.

Cardiac Glycosides

Cardiac glycosides in clinical use are:

- Digoxin,
- Digitoxin
- Ouabain.

Digitalis Compounds

Like the carrot placed in front of the donkey

Cardiac Glycosides

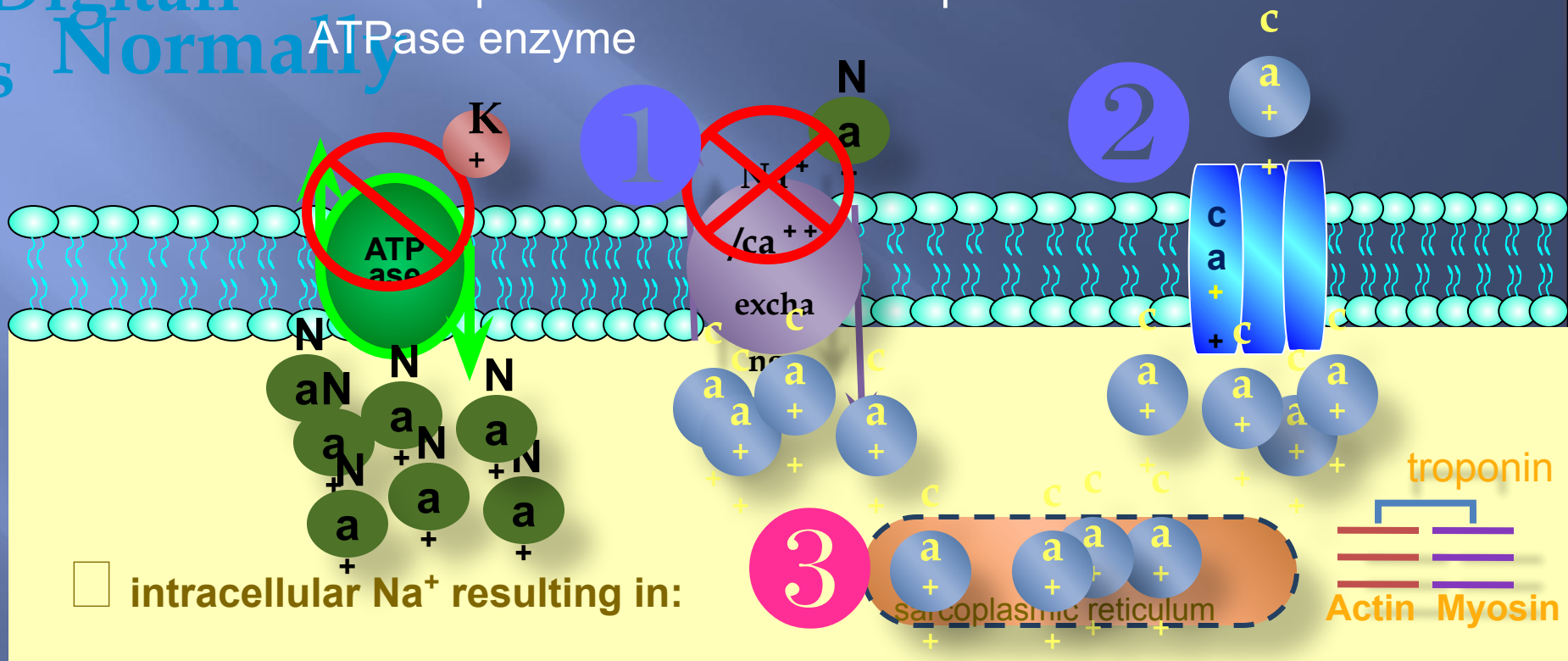
- Digitalis glycosides have been used for many years in patients with heart failure and atrial fibrillation.
- Digoxin acts as a positive inotrope by competitive inhibition of Na+/K+-ATPase, producing high levels of intracellular sodium. This is then exchanged for extracellular calcium. High levels of intracellular calcium result in enhanced actin-myosin interaction and increased contractility. Digoxin also improves baroreceptor responsiveness, and reduces sympathetic activity and circulating renin.

Digitali
s

Digitali
s Normally

Mechanism of the +ve inotropic action:

In therapeutic dose leads to partial inhibition of Na^+/K^+ ATPase enzyme

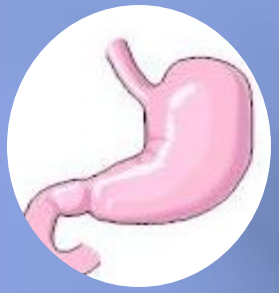


intracellular Na^+ resulting in:

Force Of Contractility

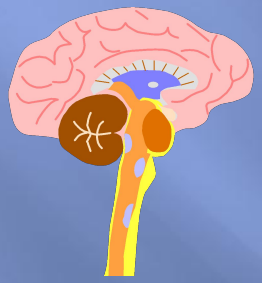
Digitalis - *Adverse (toxic) effects:*

They have narrow safety margin



GIT:

Anorexia, nausea, vomiting **(early toxicity)** abdominal discomfort or pain and diarrhea.



Neurological effects:

Headache, malaise, fatigue, drowsiness, confusion, delirium, hallucination or rarely convulsions.

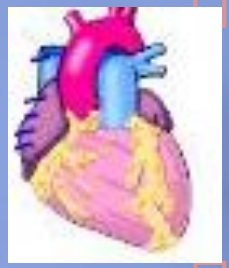
Vision:

Blurred vision, chromatopsia mostly for yellow and green vision, transient amblyopia or diplopia.

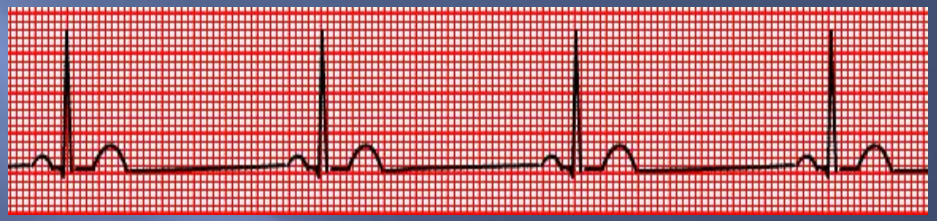
Digitalis - *Adverse (toxic) effects:*

C.V.S.:

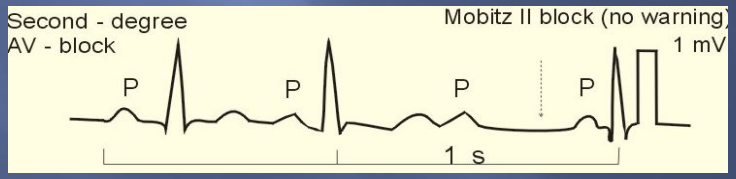
Any type of arrhythmia may be produced including:



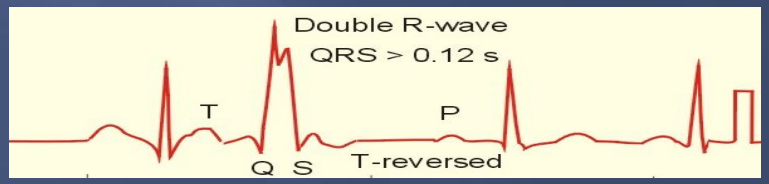
- Bradycardia,



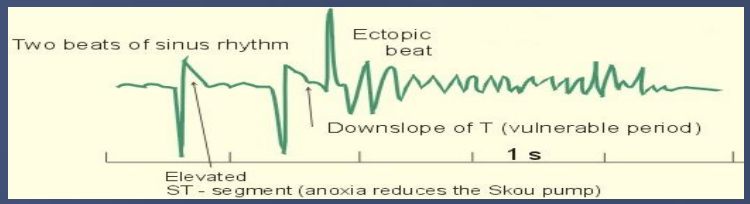
- Heart block,



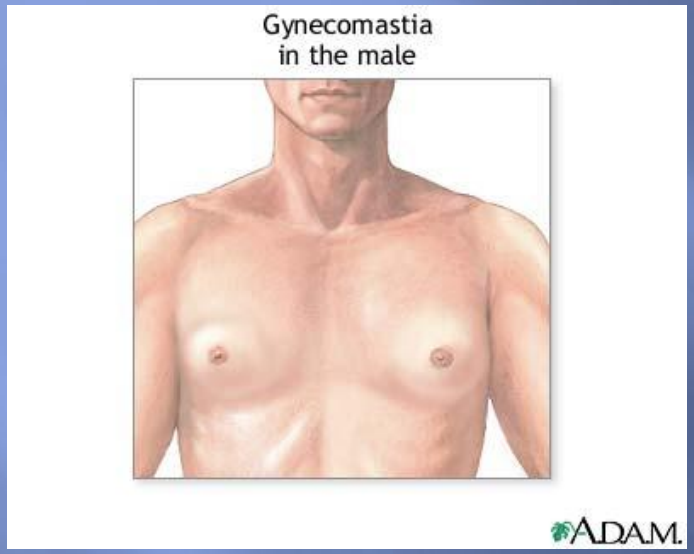
- Ventricular extrasystole



- Ventricular fibrillation.



Digitalis - *Adverse (toxic) effects:*



Gynecomastia:

May be due to steroidal structure.

Treatment Of Digitalis Toxicitiy:

1) Stop the responsible drug.

2) KCl syrup or slow release or I.V. with ECG monitoring if plasma potassium is low or normal.

3) If due to calcium injection give disodium edetate I.V. which is chelating agent for calcium.

4) Cholestyramine binds to digitalis in gut, thus inhibit absorption and decreases the toxicity especially of digitoxin.

5) In acute toxicity give specific digitalis antibodies (Fab fragment).

Treatment Of Digitalis Toxicitiy:

6) Treatment of associated arrhythmia:

- Partial heart block is treated by atropine.
- Ventricular arrhythmia without A-V block is treated by lidocaine I.V. or beta blockers.
- Ventricular arrhythmia with A-V block & atrial arrhythmia is treated by Diphenylhydantoin (phenytoin)

OTHER MEDICATIONS

- **Anticoagulants:**

  Oral anticoagulants are recommended in patients with atrial fibrillation and in sinus rhythm with a history of thromboembolism, endocardial thrombus or LV aneurysm.

- **Antiarrhythmic agents:**

  Precipitating factors should be treated, in particular electrolyte disturbance.

  Atrial fibrillation is common in heart failure and leads to a deterioration in symptoms.

  Restoration of sinus rhythm, either by electrical cardioversion or drugs, is desirable but less successful in the presence of structural heart disease and decompensated heart failure.

  Rate control with digoxin is often preferred.

- The administration of synthetic BNP (Nesritide) produces beneficial haemodynamic effects in acute heart failure (reducing pulmonary capillary wedge pressure and peripheral vascular resistance) but has been associated with an increased risk of death.
- Neutral endopeptidase (NEP) is a metallo-endopeptidase involved in the degradation of a variety of vasoactive peptides (including ANP, BNP, CNP and bradykinin). In animal studies NEP inhibitors can produce diuresis and natriuresis.
- Acute intravenous administration of endothelin antagonists improves haemodynamic abnormalities in patients with congestive cardiac failure, and oral endothelin antagonists are being developed.

NON-PHARMACOLOGICAL TREATMENT OF HEART FAILURE

- Revascularization
- Biventricular pacemaker or implantable cardioverter–defibrillator
- Cardiac transplantation

Cardiac Resynchronization Therapy

Increase the donkey's (heart) efficiency

Acute Heart FAILURE

- Acute heart failure (AHF) occurs with the rapid onset of symptoms and signs of heart failure secondary to abnormal cardiac function, causing elevated cardiac filling pressures.
- This causes severe dyspnoea and fluid accumulates in the interstition and alveolar spaces of the lung (pulmonary oedema).

Clinical Syndromes of Heart Failure

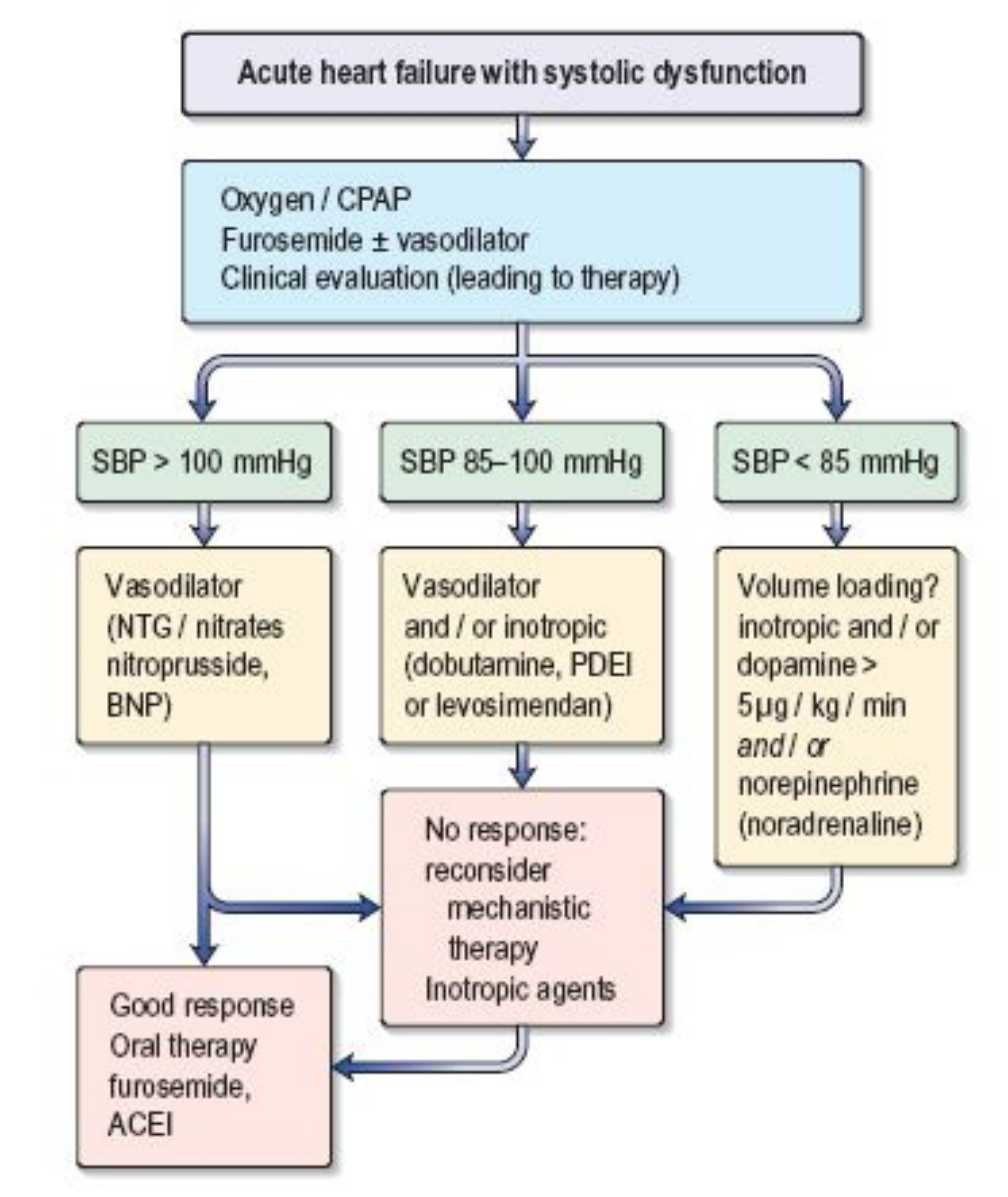
Type	Clinical features
Acute decompensated heart failure	Mild features of heart failure, e.g. dyspnoea
Hypertensive AHF	High blood pressure, preserved left ventricular function, pulmonary oedema on CXR
Acute pulmonary oedema	Tachypnoea, orthopnoea, pulmonary crackles, oxygen saturation <90% on air, pulmonary oedema on CXR
Cardiogenic shock	Systolic blood pressure <90 mmHg, mean arterial pressure drop >30 mmHg, urine output <0.5 mL/kg/hour, heart rate >60 b.p.m.
High output heart failure	Warm peripheries, pulmonary congestion, blood pressure may be low, e.g. septic shock
Right heart failure	Low cardiac output, elevated jugular venous pressure, hepatomegaly, hypotension

Diagnosis

Initial investigations performed in the emergency room should include:

- a 12-lead ECG for acute coronary syndromes, left ventricular hypertrophy, atrial fibrillation
- a chest X-ray (cardiomegaly, pulmonary oedema, pleural effusion, non-cardiac disease)
- blood investigations (serum creatinine and electrolytes, full blood count, blood glucose, cardiac enzymes and troponin, CRP and D-dimer)
- plasma BNP or NTproBNP (BNP > 100 pg/ml or NTproBNP > 300 pg/ml) indicates heart failure
- transthoracic echocardiography should be performed without delay to confirm the diagnosis of heart failure and possibly identify the cause.

Management of Acute Heart Failure



Pulmonary Oedema

PULMONARY OEDEMA

This is a very frightening, life threatening emergency characterized by

Extreme breathlessness.

Clinical features

- Patients with alveolar oedema are acutely breathless, wheezing, anxious and perspiring profusely.
- Cough productive of frothy, blood-tinged (pink) sputum, which can be copious with tachypnea and peripheral circulatory shutdown.
- Tachycardia, a raised venous pressure and a gallop rhythm.
- Crackles and wheezes are heard throughout the chest.
- The arterial *P*o2 falls and initially the *P*aco2 also falls, owing to overbreathing. Later, the *P*aco2 increases because of impaired gas exchange.
- The chest X-ray shows diffuse haziness, owing to alveolar fluid, and the Kerley B lines of interstitial oedema.

Pulmonary Oedema

TREATMENT

- The patient must be placed in a sitting position.
High-concentration oxygen is given. In severe cases, ventilation is necessary.

- *Intravenous diuretic treatment* with furosemide or bumetanide is given. These diuretics induce an acute venodilatory response with a reduction in preload that helps to relieve pulmonary congestion in addition to the more delayed diuretic response.

- *Morphine* (10-20 mg i.v.) together with an antiemetic such as metoclopramide (10 mg i.v.) is given.

- *Venous vasodilators*, such as glyceryl trinitrate, may produce prompt relief by reducing the preload.

- *Aminophylline* (250-500mg or 5mg/kg IV) is infused over 10 minutes

Cardiogenic Shock

- SHOCK is a severe failure of tissue perfusion, characterized by hypotension, a low cardiac output and signs of poor tissue perfusion such as oliguria, cold extremities and poor cerebral function. Cardiogenic shock is commonly due to myocardial infarction, acute massive pulmonary embolus, pericardial tamponade & sudden-onset valvular regurgitation.

TREEATMENT: Patients require intensive care

- General measures such as complete rest, continuous 60% oxygen administration and pain and anxiety relief are essential
- The infusion of fluid is necessary if the pulmonary capillary wedge pressure is below 18 mmHg.
- Short-acting venous dilators such as glyceryl trinitrate or sodium nitroprusside should be administered intravenously if the wedge pressure is 25 mmHg or more.
- Cardiac inotropes to increase aortic diastolic pressure.
- Emergency revascularization of occluded arteries


THANK YOU