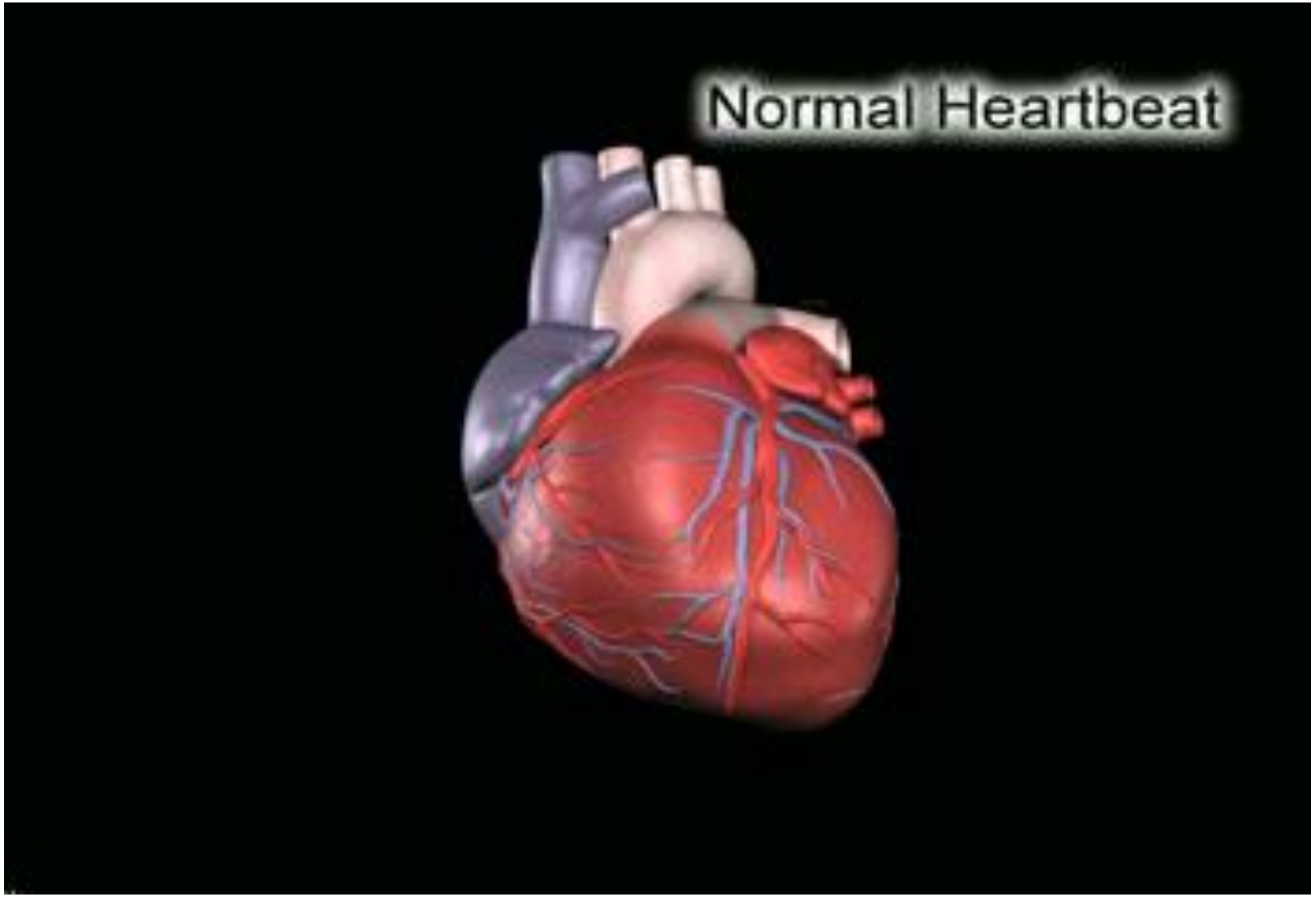
Heart Failure

- **Heart (or cardiac) failure** is the state in which the heart is unable to pump blood at a rate commensurate with the requirements of the tissues or can do so only from high pressures

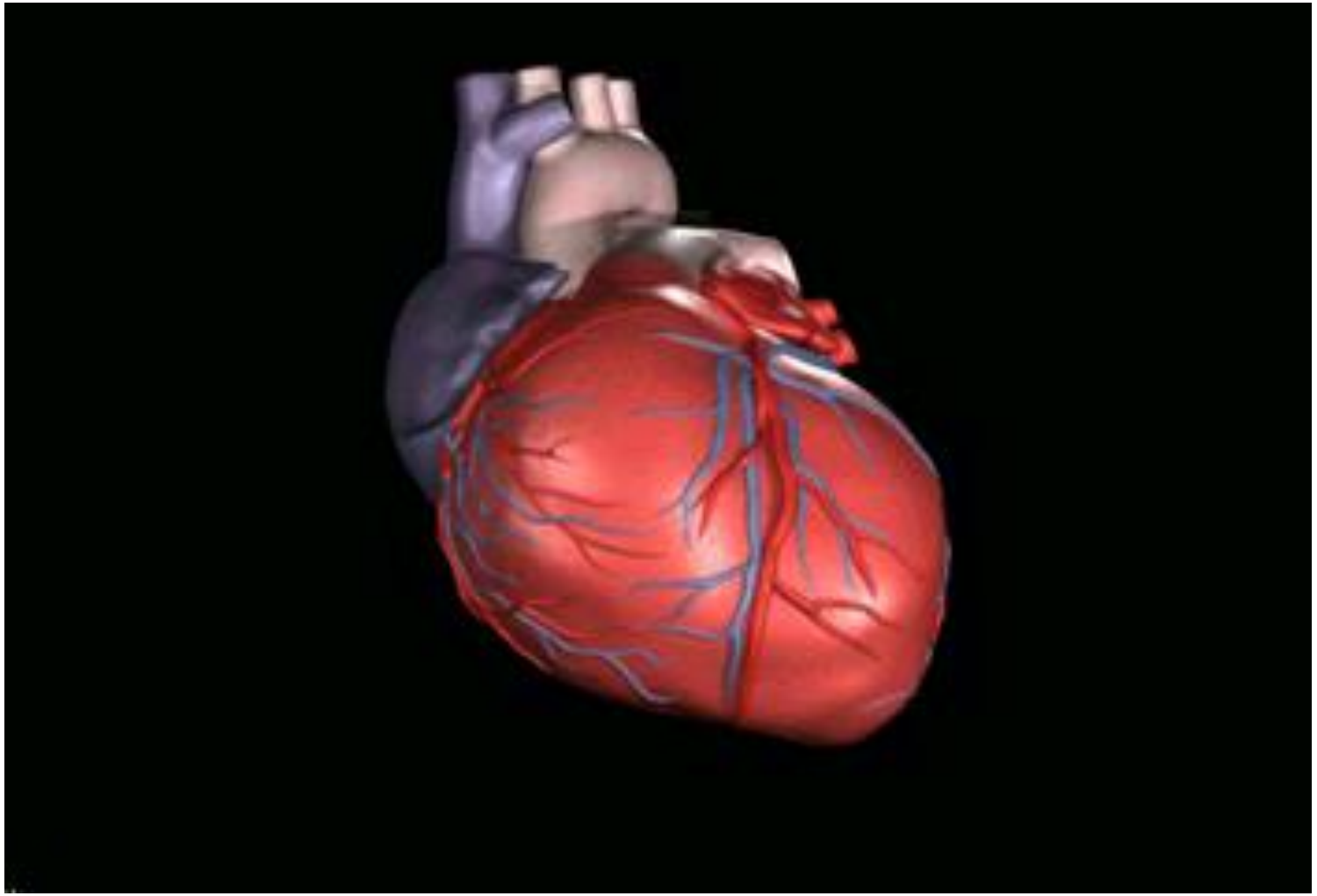
- **Heart failure is a clinical syndrome usually due to ventricular dysfunction,** resulting in acute or chronic symptoms of cardiac pump failure.
- **The most common causes of heart failure are** coronary heart disease, hypertension, alcohol abuse, and idiopathic dilated cardiomyopathy ,pulmonary diseases
- **Other causes are** valvular and pericardial disease; or non-cardiac diseases causing high-output cardiac failure, such as anaemia, thyrotoxicosis, septicaemia, Paget's disease of bone, and arteriovenous fistulae.

- **The male to female ratio** is about 2:1.
- **The median age of presentation is 76 years.**
- **The prevalence of heart failure is increasing** because of the improved treatment of coronary heart disease (e.g. thrombolysis resulting in more people surviving a myocardial infarct but left with residual left ventricular dysfunction), and the ageing population



A normal heart pumps blood in a smooth and synchronized way.

Heart Failure Heart



A heart failure heart has a reduced ability to pump blood.

Types of Heart Failure

- Systolic (or squeezing) heart failure
  - Decreased pumping function of the heart, which results in fluid back up in the lungs and heart failure

- Diastolic (or relaxation) heart failure
  - Involves a thickened and stiff heart muscle
  - As a result, the heart does not fill with blood properly
  - This results in fluid backup in the lungs and heart failure

Left-sided heart failure. There is a reduction in the left ventricular output and/or an increase in the left atrial or pulmonary venous pressure. An acute increase in left atrial pressure may cause pulmonary congestion or pulmonary oedema; a more gradual increase in left atrial pressure, as occurs with mitral stenosis, may lead to reflex pulmonary vasoconstriction, which protects the patient from pulmonary oedema at the cost of increasing pulmonary hypertension.

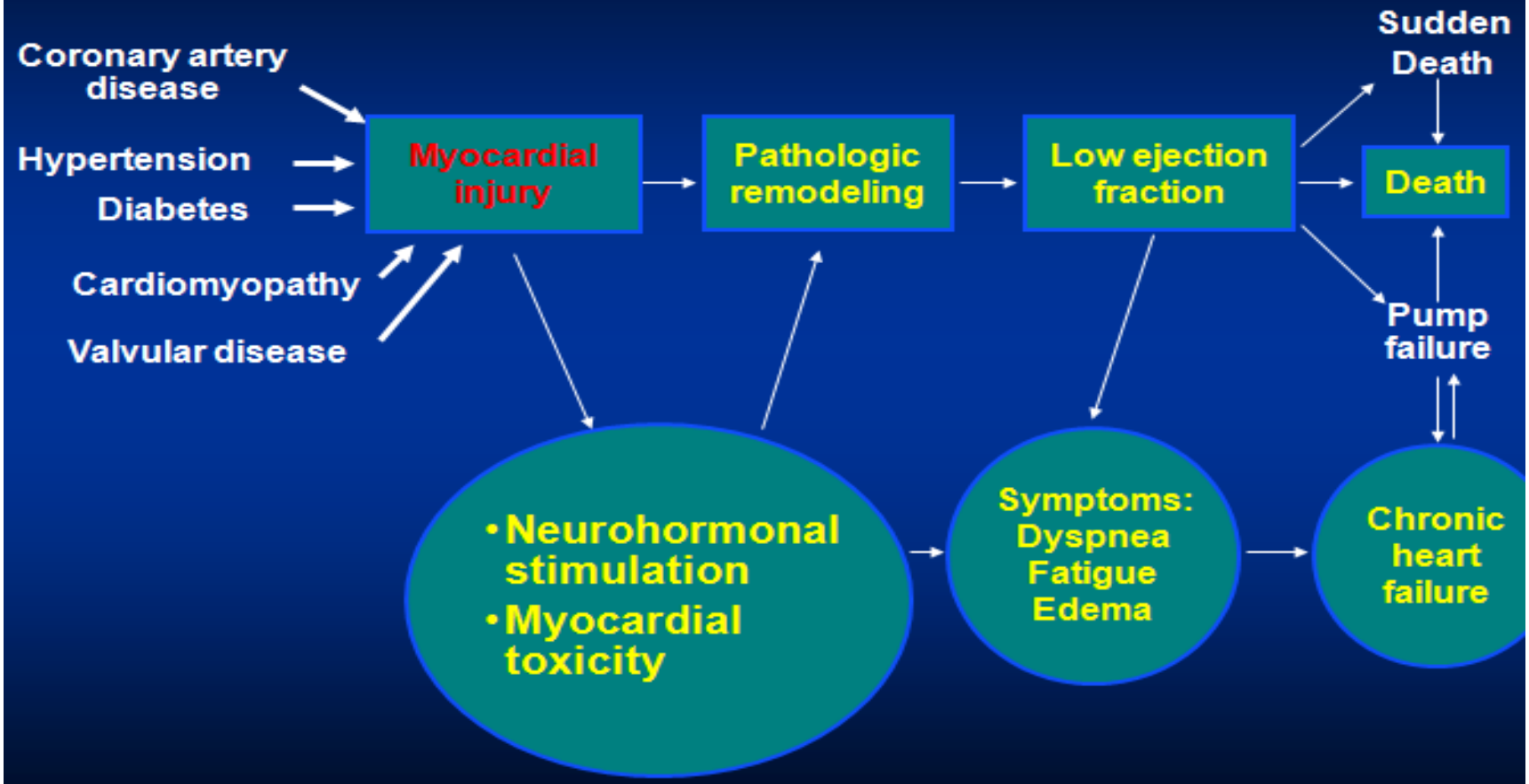
Right-sided heart failure. There is a reduction in right ventricular output for any given right atrial pressure. Causes of isolated right heart failure include chronic lung disease (cor pulmonale), multiple pulmonary emboli and pulmonary valvular stenosis.

Biventricular heart failure. Failure of the left and right heart may develop because the disease process (e.g. dilated cardiomyopathy or ischaemic heart disease) affects both ventricles, or because disease of the left heart leads to chronic elevation of the left atrial pressure, pulmonary hypertension and right heart failure

Acute Heart Failure

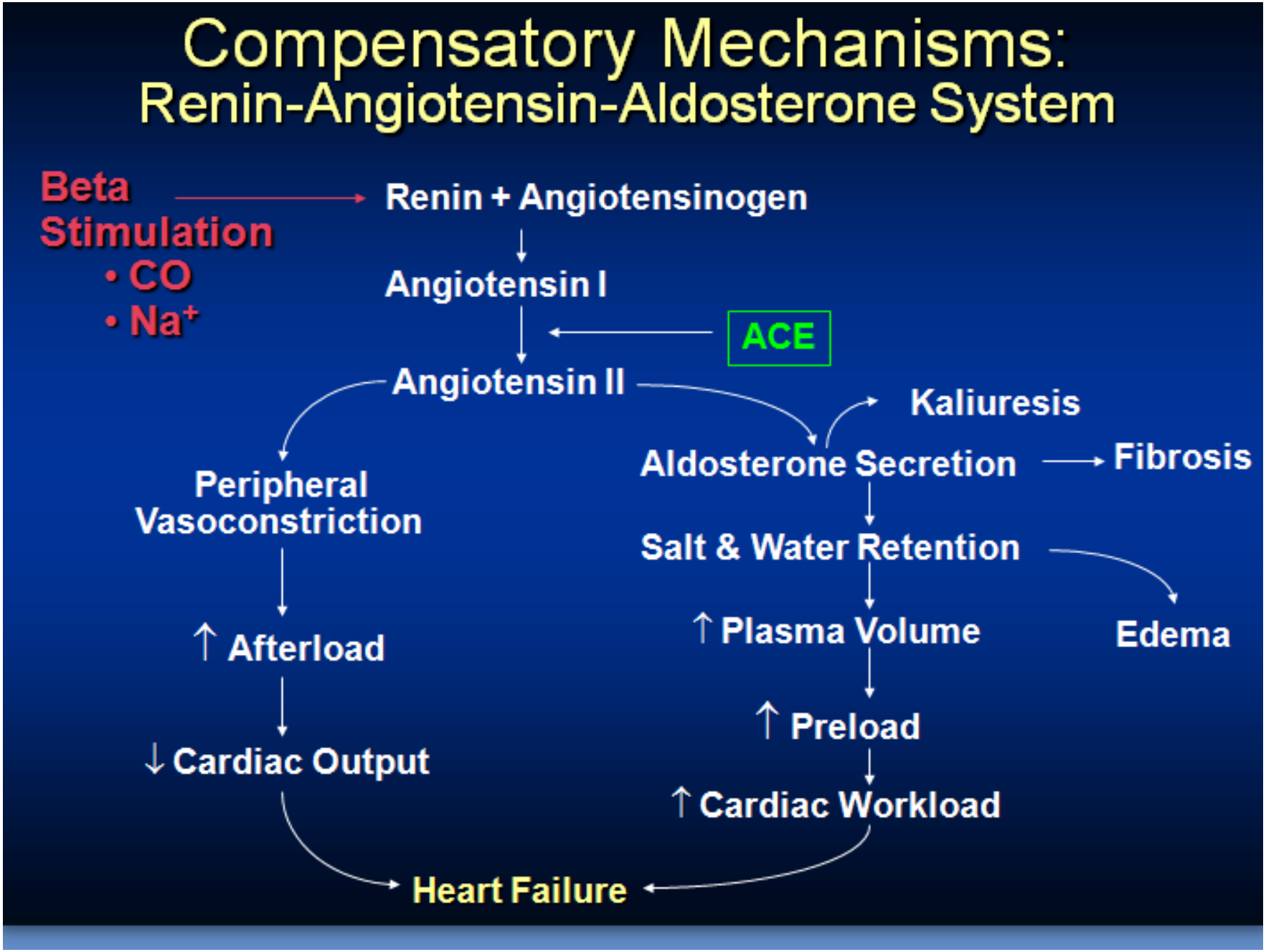
- Often precipitated by a myocardial infarction.
- **Signs include:**
  - Severe breathlessness
  - Frothy pink sputum
  - Cold clammy skin
  - Tachycardia
  - Low blood pressure
  - Lung crepitations
  - Raised jugular venous pressure
  - Third heart sound
  - Confusion

Pathologic Progression of CV Disease



Adapted from Cohn JN. *N Engl J Med.* 1996;335:490–498.

Compensatory Mechanisms:
Renin-Angiotensin-Aldosterone System



Chronic Heart Failure

- **The most specific signs are:**
- Laterally displaced apex beat
- Elevated jugular venous pressure
- Third heart sound
- **Less specific signs include:**
- Tachycardia
- Lung crepitations
- Hepatic engorgement (tender hepatomegaly)
- Peripheral oedema

FACTORS THAT MAY PRECIPITATE OR AGGRAVATE HEART FAILURE IN PATIENTS WITH PRE-EXISTING HEART DISEASE

Myocardial ischaemia or infarction

Intercurrent illness, e.g. infection

Arrhythmia, e.g. atrial fibrillation

Inappropriate reduction of therapy

Administration of a drug with negative inotropic properties (e.g. β-blocker) or fluid-retaining properties (e.g. non-steroidal anti-inflammatory drugs, corticosteroids)

Pulmonary embolism

Conditions associated with increased metabolic demand, e.g. pregnancy, thyrotoxicosis, anaemia

Intravenous fluid overload, e.g. post-operative i.v. infusion

DIFFERENTIAL DIAGNOSIS OF PERIPHERAL OEDEMA

Cardiac failure: right or combined left and right heart failure, pericardial constriction, cardiomyopathy

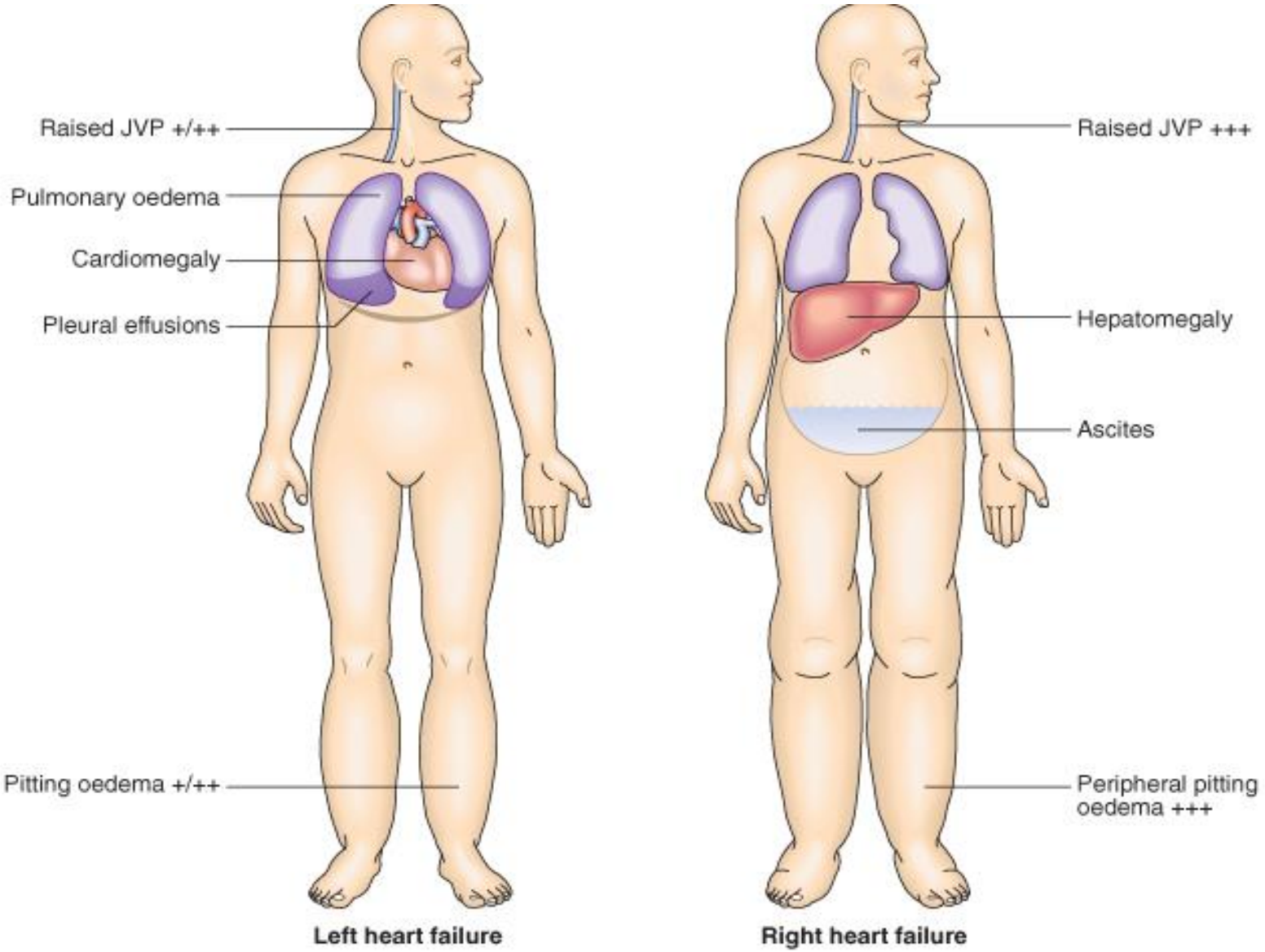
Chronic venous insufficiency: varicose veins

Hypoalbuminaemia: nephrotic syndrome, liver disease, protein-losing enteropathy; often widespread, can affect arms and face

Drugs
Sodium retention: fludrocortisone, non-steroidal anti-inflammatory agents
Increasing capillary permeability: nifedipine, amlodipine

Idiopathic: women > men

Chronic lymphatic obstruction


Raised JVP +/++
Pulmonary oedema
Cardiomegaly
Pleural effusions
Pitting oedema +/++
Left heart failure
Raised JVP +++
Hepatomegaly
Ascites
Peripheral pitting oedema +++
Right heart failure

COMPLICATIONS

: *Renal failure* is caused by poor renal perfusion due to a low cardiac output and may be exacerbated by diuretic therapy, ACE inhibitors and angiotensin receptor blockers.

Hypokalaemia may be the result of treatment with potassium-losing diuretics or hyperaldosteronism caused by activation of the renin-angiotensin system and impaired aldosterone metabolism due to hepatic congestion. Most of the body's potassium is intracellular and there may be substantial depletion of potassium stores, even when the plasma potassium concentration is in the normal range.

Hyperkalaemia may be due to the effects of drug treatment, particularly the combination of angiotensin-converting enzyme (ACE) inhibitors and spironolactone (which both promote potassium retention), and renal dysfunction.

Hyponatraemia is a feature of severe heart failure and may be caused by diuretic therapy, inappropriate water retention due to high ADH secretion, or failure of the cell membrane ion pump. It is a poor prognostic sign,

Impaired liver function is caused by hepatic venous congestion and poor arterial perfusion, which frequently cause mild jaundice and abnormal liver function tests; reduced synthesis of clotting factors may make anticoagulant control difficult.

Thromboembolism. Deep vein thrombosis and pulmonary embolism may occur due to the effects of a low cardiac output and enforced immobility, whereas systemic emboli may be related to arrhythmias, atrial flutter or fibrillation, or intracardiac thrombus complicating conditions such as mitral stenosis or LV aneurysm.

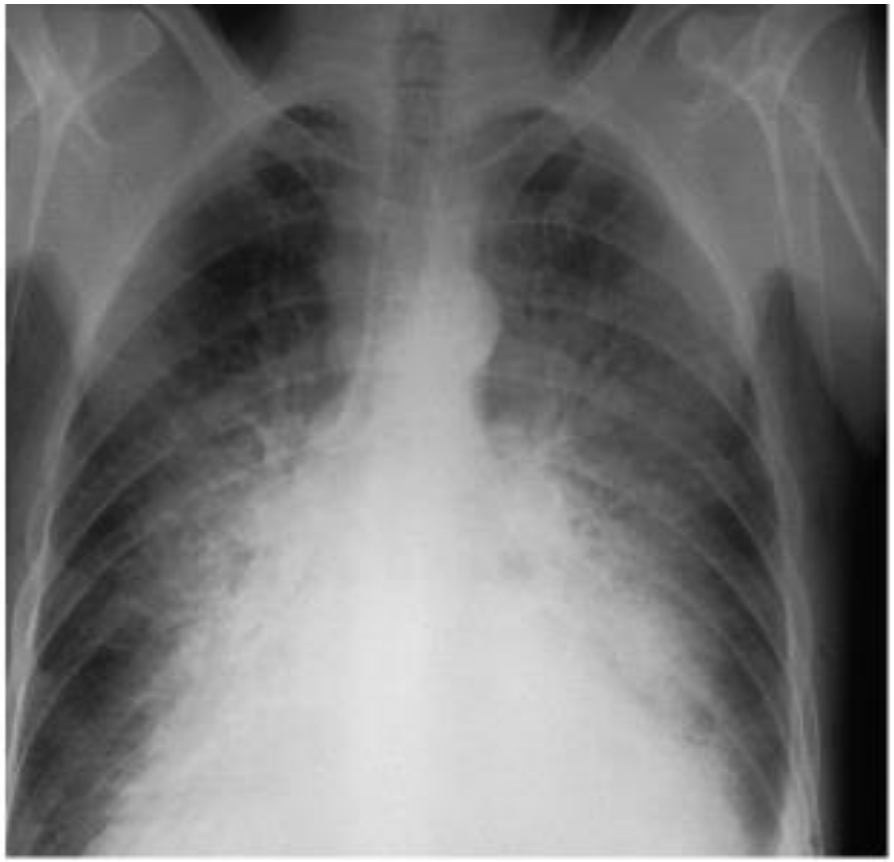
Atrial and ventricular arrhythmias are very common and may be related to electrolyte changes (e.g. hypokalaemia, hypomagnesaemia), the underlying structural heart disease, and the pro-arrhythmic effects of increased circulating catecholamines and some drugs (e.g. digoxin). Sudden death occurs in up to 50% of patients with heart failure and is often due to a ventricular arrhythmia. Frequent ventricular ectopic beats and runs of non-sustained ventricular tachycardia are common findings in patients with heart failure and are associated with an adverse prognosis.

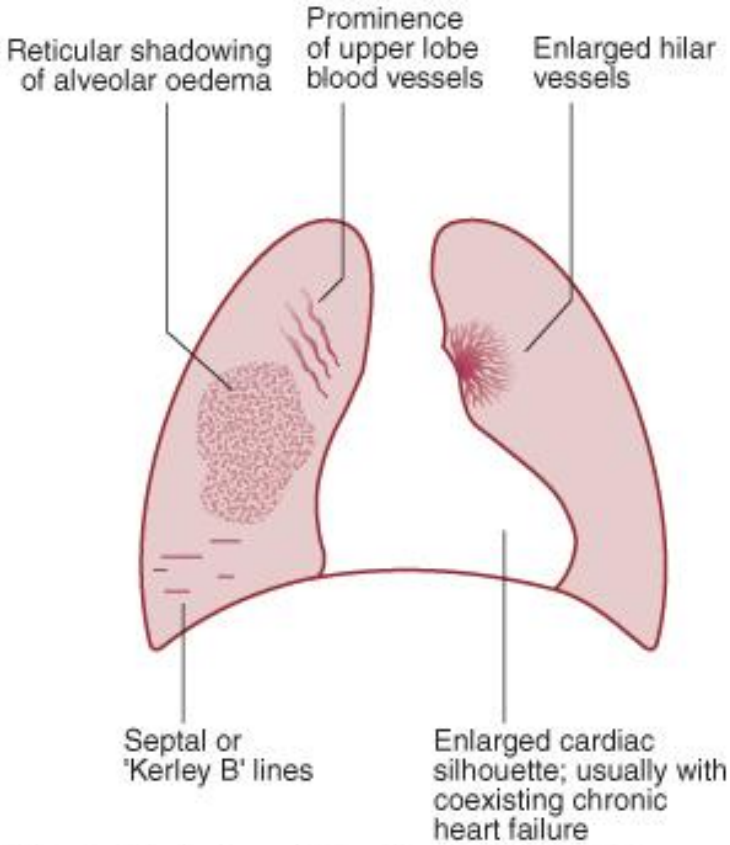
Investigations

- **Electrocardiogram (ECG)** may show acute ischaemia, arrhythmias, left ventricular hypertrophy, left bundle branch block, or prior MI.
- **Heart failure is unlikely if the ECG is normal,** and the diagnosis should be reconsidered in this situation.
- **Chest X-ray (CXR)**
  - pulmonary vascular congestion (upper lobe diversion),
  - pulmonary oedema
  - effusions
  - cardiomegaly

Chronic Heart Failure

- **B-type natriuretic peptide (BNP) and its N-terminal fragment (NTproBNP)**
- New diagnostic test
- A raised concentration of either has been shown to have a sensitivity of greater than 90% and a specificity of 80-90% for the diagnosis of heart failure, [de Lemos et al, 2003].
- Heart failure is unlikely if the level of BNP or NTproBNP is normal, especially if the ECG is also normal, and the diagnosis should be reconsidered in this situation.




Reticular shadowing of alveolar oedema
Prominence of upper lobe blood vessels
Enlarged hilar vessels
Septal or 'Kerley B' lines
Enlarged cardiac silhouette; usually with coexisting chronic heart failure

Differential Diagnosis

- **Other causes of shortness of breath on exertion** - e.g. pulmonary disease, obesity, unfitness, volume overload from renal failure or nephrotic syndrome, angina, anxiety.
- **Other causes of peripheral oedema** - e.g. dependent oedema, nephrotic syndrome.
- **Non-cardiac diseases causing high-output cardiac failure** - e.g. anaemia, thyrotoxicosis, septicaemia, Paget's disease of bone, arteriovenous fistulae

Classification

- **The New York Heart Association (NYHA) has classified chronic heart failure** according to the following functional criteria:
  - **Grade I** - no limitation of physical activity
  - **Grade II** - slight limitation of physical activity; comfortable at rest, but ordinary physical activity results in fatigue, palpitation, or dyspnoea.
  - **Grade III** - marked limitation of physical activity; comfortable at rest, but less than ordinary activity causes fatigue, palpitation, or dyspnoea.
  - **Grade IV** - unable to carry out any physical activity without discomfort; symptoms of cardiac insufficiency at rest; if any physical activity is undertaken.
- [European Heart Journal, 1997; NZMJ, 1997; NHS CRD, 1998; DH, 2000]

Management of acute pulmonary oedema
This needs urgent treatment:

Sit the patient up in order to reduce pulmonary congestion.

Give oxygen (high flow, high concentration).

Non-invasive positive pressure ventilation (continuous positive airways pressure, CPAP, of 5-10 mmHg) by a tight-fitting face mask results in a more rapid improvement in the patient's clinical state.

Administer nitrates (e.g. i.v. glyceryl trinitrate 10-200 µg/min or buccal glyceryl trinitrate 2-5 mg) titrated upwards every 10 minutes, until clinical improvement occurs or systolic blood pressure falls to < 110 mmHg.

Administer a loop diuretic such as furosemide 50-100 mg i.v.

Management

- **1-Manage other risk factors**
- **Manage coexisting coronary heart disease**
- **Avoid aggravating factors**
  - Non-steroidal anti-inflammatory drugs
  - Short-acting calcium-channel blockers
  - Advise low salt diet
  - Advise a moderate alcohol intake
  - Limiting fluid intake may be appropriate in advanced heart failure, but care is needed to avoid dehydration.
- **Vaccinate people against influenza annually and pneumococcus as a one-off,** as they are at increased risk of infective complications.
- **Consider cardiac rehabilitation, palliative care, and long-term social support** if appropriate.

Medication

- **2-Drug treatments should be initiated in the following order:**
- ACE inhibitor - with diuretic if needed - for NYHA Grades I-IV.
- Angiotensin-II receptor antagonist - if intolerant of ACE inhibitor.
- Beta-blocker - for NYHA Grades I-IV.
- Spironolactone - for NYHA Grades III-IV.
- Digoxin - for NYHA Grades II-IV
- TREATMENT of complication .

ACE inhibitors

- **Angiotensin-converting enzyme inhibitors (ACE inhibitors) relieve symptoms and improve prognosis** and should be considered in all people with heart failure [Eccles et al, 1998; SIGN, 1999; DH, 2000]. Twenty-six people need to be treated for 3 years to prevent one death [SIGN, 1999].
- **ACE inhibitors are cost-effective** [Andersson and Swedberg, 1998; Eccles et al, 1998]. In a health authority of 250,000 people, around 40 deaths and 300 hospital admissions could be prevented each year using ACE inhibitors [Bandolier, 1997].
- **All ACE inhibitors are effective in treating heart failure,** although most evidence is from clinical trials of enalapril [Medicines Resource, 1996; Eccles et al, 1998].
- **Treatment with an ACE inhibitor alone** can be considered in people with NYHA grades I-II who do not have symptoms or signs of fluid overload. Diuretics should be added if fluid overload is present [Eccles et al, 1998].
- **Cough** is common in heart failure but is also caused by an ACE inhibitor in a small percentage of people. Cough is not a reason to stop an ACE inhibitor unless it is troublesome [SIGN, 1999; DTB, 2000].

Diuretics

- **Diuretics give rapid symptom relief** and should be started early in symptomatic people with signs of fluid overload. Their long-term effects on mortality rates and other endpoints when given alone are not known (excluding spironolactone). An ACE inhibitor should always be added to diuretic therapy, unless contraindicated, as this improves prognosis.
- **Loop diuretics are usually preferred to thiazide diuretics.** Thiazides may be as effective as loop diuretics in treating oedema in people with mild failure who have preserved renal function.
- **The combination of a thiazide with a loop diuretic** gives a synergistic effect and may be useful in people with severe, persistent symptoms. Close monitoring of electrolytes is required and such treatment should usually be specialist initiated.
- [MeReC, 1990; DTB, 1994; European Heart Journal, 1997; NZMJ, 1997; Andersson and Swedberg, 1998; Eccles et al, 1998; Heart Failure Society of America, 1999; SIGN, 1999; DH, 2000; DTB, 2000; Krum, 2001; Remme et al, 2001]

B-Blockers

- **Beta-blockers are recommended for all people with heart failure (NYHA grades I-IV) whose failure is stable,** on standard treatment, unless there is a contraindication.
- **Beta-blockers in combination with other treatments,** such as ACE inhibitors, diuretics and digoxin, improve survival by more than 30% compared to standard treatment alone in people with stable heart failure.
- **Bisoprolol, carvedilol, and modified-release metoprolol have been shown to be beneficial.** Bisoprolol and carvedilol are the only beta-blockers that are licensed for the treatment of heart failure.

Spironolactone

- **Spironolactone,** should be considered for people with moderate to severe heart failure (NYHA grades III-IV) who are already on an ACE inhibitor and a loop diuretic [SIGN, 1999; DH, 2000; Samuel, 2003].
- **The Randomised Aldactone Evaluation Study (RALES)** compared treatment with low-dose spironolactone (25 mg daily) added to standard care with other diuretics, ACE inhibitors and digoxin against standard care alone, in people with moderate to severe heart failure (NYHA III-IV) [Pitt et al, 1999]. Mortality was reduced by 30%, the risk of hospitalization for worsening heart failure was reduced by 35%, and there was a significant improvement in symptoms. Over 2 years, one death was avoided for every 9 people treated with spironolactone in addition to standard therapy.
- **Careful monitoring for hyperkalaemia and hypovolaemia is required.** [Heart Failure Society of America, 1999; Krum, 2001; Remme et al, 2001]

Digoxin

- **Digoxin,** given in combination with a diuretic and an ACE inhibitor to people with heart failure (NYHA grades II-IV) in normal sinus rhythm, has been found to reduce hospitalization and clinical deterioration, but not mortality [Hood et al, 2002].
- **Consider digoxin** if the person continues to be symptomatic despite adequate doses of diuretic and ACE inhibitor [DH, 2000].
- **Give digoxin to all people with heart failure and atrial fibrillation** who need control of the ventricular rate.
- [Heart Failure Society of America, 1999]

Angiotensin II antagonists

- **Candesartan, losartan, and valsartan are recommended in PRODIGY for people intolerant of an ACE inhibitor** (especially when that intolerance is due to ACE inhibitor-induced cough). Initial trial data appear comparable with ACE inhibitors
- Candesartan is now licensed for heart failure and impaired left ventricular dysfunction. Valsartan is now licensed for heart failure in post myocardial infarction patients. Losartan is not currently licensed for the treatment of heart failure.