Heart failure

Done by:
Tamador A. Zetoun
Heart Failure …

- Means failure of heart to pump enough blood to satisfy the need of the body.

- Due to an impaired ability of the heart to adequately to fill or eject blood.
Heart failure (HF) means decreased ability of the heart to perform its proper pumping action (due to decreased force of contraction of the ventricles).

HF may be left-sided HF or right sided HF or both (congestive HF).
# HEART FAILURE

<table>
<thead>
<tr>
<th>Left-sided heart failure</th>
<th>Right-sided heart failure</th>
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<tbody>
<tr>
<td><strong>Causes:</strong></td>
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<tr>
<td>Systemic hypertension (chronic or untreated).</td>
<td>Pulmonary hypertension</td>
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<td>Coronary heart disease → myocardial infarction in the LV.</td>
<td>Mitral stenosis pulmonary hypertension.</td>
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<tr>
<td>Aortic stenosis or incompetence (valvular disease).</td>
<td>Left sided heart failure</td>
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</tbody>
</table>
Diastole (filling):
- Normal: The ventricles fill normally with blood.
- Systolic Dysfunction: The enlarged ventricles fill with blood.
- Diastolic Dysfunction: The stiff ventricles fill with less blood than normal.

Systole (pumping):
- Normal: The ventricles pump out about 60% of the blood.
- Systolic Dysfunction: The ventricles pump out less than 40 to 50% of the blood.
- Diastolic Dysfunction: The ventricles pump out about 60% of the blood, but the amount may be lower than normal.
Pathophysiology of Heart Failure

- Increased blood pressure and cardiac work
- Increased afterload
- Vasoconstriction
- Increased intravascular volume
- Sodium and water retention

Heart failure:
- Reduced cardiac output

Myocyte loss

Neurohumoral activation:
- Sympathetic nervous system
- Renin–angiotensin system
- Vasopressin system
- Endothelin system
Pathophysiology of Heart Failure

Molecular & Cellular changes

Secondary myocardial effects
- LV remodelling
- Contractility
- Hypertrophy
- Fibrosis

Neurohormones
- ↑SNS activity
- ↑RAS
- ↑Endothelin
- ↑ANP/BNP
- ↑Cytokines

Endothelium
- Vasoconstriction
- Structural change
- Cytokines
HEART FAILURE

COMPENSATED ACUTE HF
1-Baroreceptor
2-Chemoreceptors
3-CNS Ischemic response
4-Activate of sympathetic activity
5-Increase circulatory filling pressure
6-Increase venous return
HEART FAILURE

CHRONIC STAGE OF FAILURE

1- Renal function, renin angiotensin
2- Increase aldosterone
3- Increase sympathetic stimulation
4- Fluid retention
5- Role of atrial Natriuretic peptide
Decompensate HF

- The heart become severely damage even with all compensatory mechanism of the body.
• Decompressed Heart Failure

  a. If severely damaged, no amount of compensation can make the heart pump a normal output

Fig. 22.2
TYPES OF HEART FAILURE

1-Left, right and congestive HF
2-Acute and chronic
3-Systolic and diastolic HF
4-High cardiac output HF
Clinical assessment

1-LVF: Dyspnea, orthopnoea, PND, inspiratory crepitation and pulmonary edema. Poor tolerance, cold extremities, low BP, oliguria and uraemia.

2-RVF: Increase jugular venous pressure, peripheral edema, hepatomegaly, splenomegaly, ascites, and pleural effusion.

3- Congestive HF: 1+2
COMPLICATIONS

1- Renal failure
2- High or low K, by the effect of treatment (drugs)
3- Hyponatremia
4- Impair liver function
5- DVT and pulmonary embolism
6- Arrhythmias
SHOCK

Shock is a clinical syndrome characterized by inadequate tissue perfusion due to decreased cardiac output and decreased ABP (hypotension). (Cold skin, Hypotension and confusion or coma).

It is generally classified into 4 types:
1. Hypovolemic shock
2. Low-resistance shock
3. Cardiogenic shock
4. Obstructive shock
HYPOVOLAEMIC SHOCK
- It occurs as a result of excessive loss of blood or plasma, e.g.
- Haemorrhagic shock
  - Traumatic shock
  - Surgical shock
  - Burn shock

CARDIOGENIC SHOCK
- It occurs as a result of decreased pumping action of the left ventricle
  e.g. due to:
  - Myocardial infarction.
  - Severe ventricular tachycardia
- It is also called cold shock.
**OBSTRUCTIVE SHOCK**

This occurs as a result of obstruction of blood flow in the lungs or Heart e.g. due to a large pneumotorax. Cardiac tamponade or massive embolism pulmonary embolism.

**LOW-RESISTANCE SHOCK**

- It occurs as a result of massive vasodilatation → ↑ circulatory capacity

   and → dec venous return

   → ↓ COP → ↓ ABP e.g. neurogenic shock, anaphylactic shock (histamine shock)
SEPTIC SHOCK

1- High temp
2- Normal BP
3- Normal Co
4- Warm skin
DEGREES OF SHOCK

1- Nonprogressive or compensated shock
2- Progressive or noncompensated shock
3- Irreversible shock
The following effects are produced by haemorrhage:

- Hypotension: because the loss of blood → ↓ blood volume
  → ↓ C.O.P. → ↓ A.B.P.
- Rapid and weak pulse: and in severe haemorrhage, the pulse is hardly felt.
- ↑ Respiration in rate and depth.
- Pale and cold skin: the skin is pale due constriction of skin capillaries and it is cold due to constriction of skin arterioles → ↓ blood volume passing through the skin.
- ↓ Urine Formation: due to ↓ renal blood flow and ↑ secretion of antidiuretic hormone.
- Fainting (=loss of consciousness) and death may occur in severe haemorrhage due to brain ischaemia.
EFFECTS OF HAEMORRHAGE

Hypotension (↓ ABP) → inadequate perfusion
Cerebral hypoxia (ischaemia) → depression of brain (cortex and centers) → coma.

BODY REACTION TO HAEMORRHAGE

Compensatory reactions in acute haemorrhage

Immediate compensatory reactions
Immediate reactions aim at rapid elevation of the arterial B.P.

Reactions that correct the hypovolaemia: (a) 1. fluid shift from the tissue spaces to the Capillary (b) Mobilization of the labile tissue protein into bloodstream (c) Splenic contraction (which adds the stored blood in the spleen to the circulating blood).
**Summary**

**BODY REACTION TO HAEMORRHAGE**

**Delayed Compensatory Reactions**
- Secretion of ADH & aldosterone → retention of water → ↑ plasma volume.
- Mobilization of labile and reserve proteins from the tissues to the → ↑ plasma proteins.

**Immediate Compensatory Reactions**
- ↑ heart rate → ↑ COP
- Vasoconstriction of arterioles (↑PR)
- Venoconstriction → ↑ VR
- Contraction of spleen.
- ↑ secretion of adrenaline & Noradrenaline
↑ formation of erythropoietin → ○ Activation of renin-angiotensin sys
↑ production of RBCs (ADH).
Capillary fluid shift
↓ urine formation.

↑ secretion of vaso-pressin

These reactions restore blood pressure and blood volume in mild or moderate haemorrhage.
TREATMENT OF SHOCK