Physiology Team 431

Team leaders

- **Mohammed Asiri**
- **Nour Al-Khawaja**

- **Yafa Al-shamlan**
- **Sara Al-anazy**
- **Lama Mokhlis**
- **Tmader alaofi**
- **Hayfa alabdulkareem**
- **Dalal fatani**
- **Jomanah alshammari**
- **Shehanah alomair**

- **Abdullah Al-Towim**
- **Khalid Al Mohaimedi**
- **AbdulRahman Al-Bakr**
- **Fahad Al-showishi**
- **Saad Al-Mdemig**
- **Ahmad Al-Zuhair**
- **Mohammed Al Numeir**
- **Majid Al-Oriny**
- **Abdullrahman Alshahrani**
- **Tariq Al-Otaibi**
- **Abdulmalik Almufarrih**
- **Nasser Al-moosa**
- **Abdulaziz Al-hamad**
- **Ahmed Almarzuqi**
- **Nasser Al-moosa**

* Cardiac contractility and stroke volume

Systolic function of the heart is controlled by:

1. Contractile state of the myocardium.
2. Preload of the ventricle.
3. Afterload applied to the ventricle.
4. Heart Rate.

* How to measure the cardiac output (CO) ?

- **Echocardiographic** techniques:
  will give the Ejection fraction= SV/EDV X 100
- **Radionuclide imaging** techniques can be used to estimate real-time changes in ventricular dimensions, thus computing stroke volume, which when multiplied by heart rate, gives cardiac output.
- **Fick's Principle :** An old technique can be used to compute cardiac output (CO) indirectly from whole body oxygen consumption (VO2) and the mixed venous (O2ven) and arterial oxygen contents (O2art); however, this technique is rarely used.
  - So, **CO = VO2/(O2art – O2ven)**
  - The blood contents of oxygen VO2= 250 ml O2/min
  - O2art contents=0.2 ml/ml blood
  - O2ven contents=0.15 ml O2/ml blood
  - CO = 5000 ml/min, or 5 L/min.
    *Ventricular stroke volume would simply be the cardiac output divided by the heart rate.

* Heart Failure :

→What is heart failure ?

It is a pathological process in which systolic and /or diastolic function of the heart is impaired as a result, CO is low and unable to meet the metabolic demands of the body.

→Pathophysiology of heart failure :

Heart failure can be caused by factors originating from within the heart (i.e., intrinsic disease or pathology) or from external factors that place excessive demands upon the heart.

- **Intrinsic factors:**
  dilated cardiomyopathy and hypertrophic cardiomyopathy, myocardial infarction..
- **External factors**
  That make the heart does extra work either by: pressure load or volume load
  - long-term, uncontrolled hypertension "pressure load"
    - High blood pressure (heart contract against high pressure →Fatigue →hypertrophy→need more blood supply →heart failure.)
  - Increased **stroke volume** "volume load" maybe caused by:
    - **arterial-venous shunts**, hormonal disorders such as hyperthyroidism, and pregnancy.

→Causes :

- **Myocardial infarction**
- **Coronary artery disease**
- **Valve disease**
- **Idiopathic cardiomyopathy**
- **Viral or bacterial cardiomyopathy**
- **Myocarditis**
- **Pericarditis**
- **Arrhythmias**
- **Chronic hypertension**
- **Thyroid disease**
- **Septic shock**
- **Aneamia**
- **Arterio-venous shunt.**

- Intrinsic factors
- Extrinsic factors

- **Acute Heart Failure** :

Acute heart failure develops rapidly and can be immediately life threatening because the heart does not have time to undergo compensatory adaptations.

Duration: hours/ days

Causes: cardiopulmonary by-pass surgery, acute infection (sepsis), acute myocardial infarction, severe arrhythmias

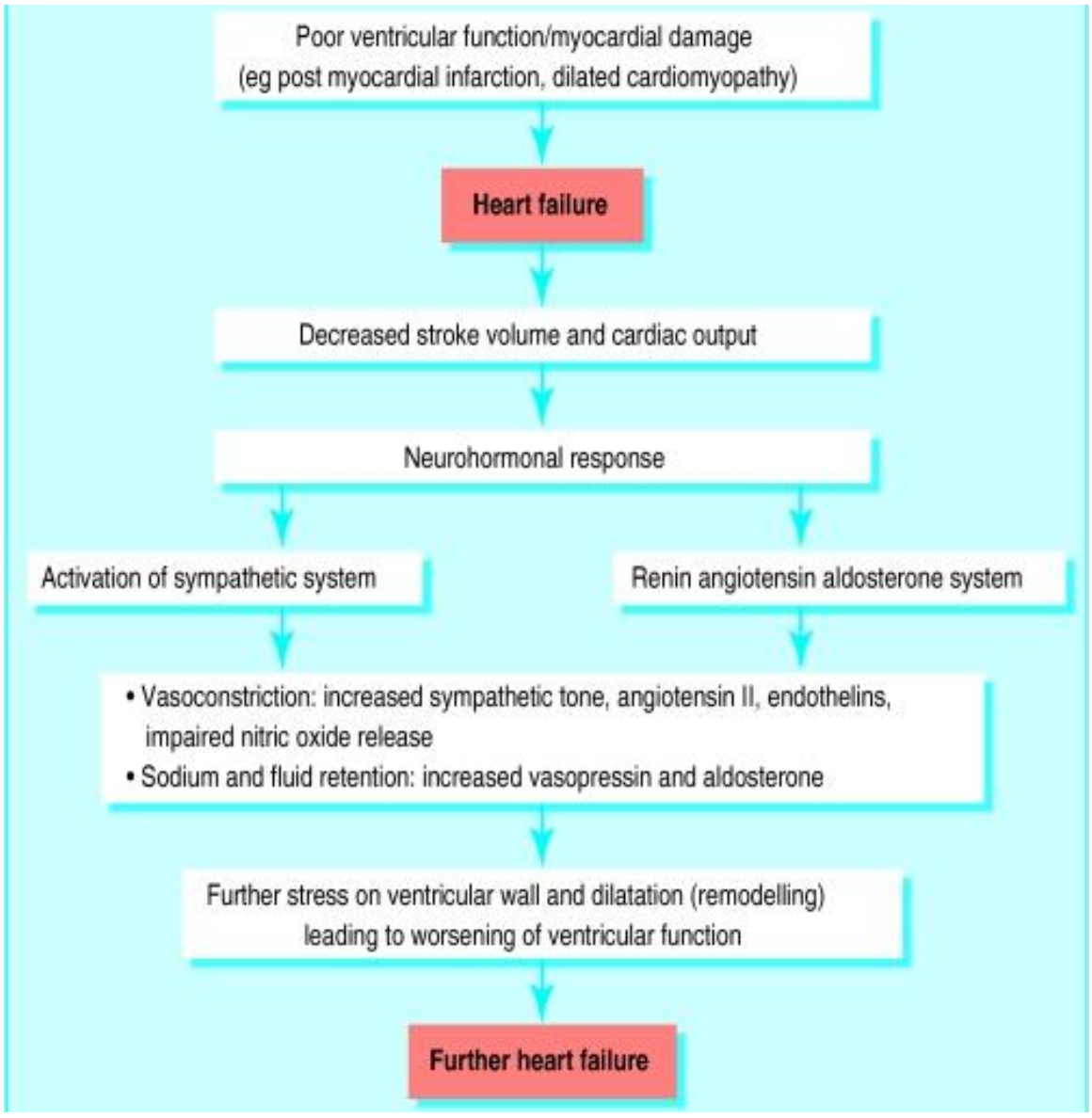
Management: pharmacological or surgical interventions.

- **Chronic Heart Failure** :

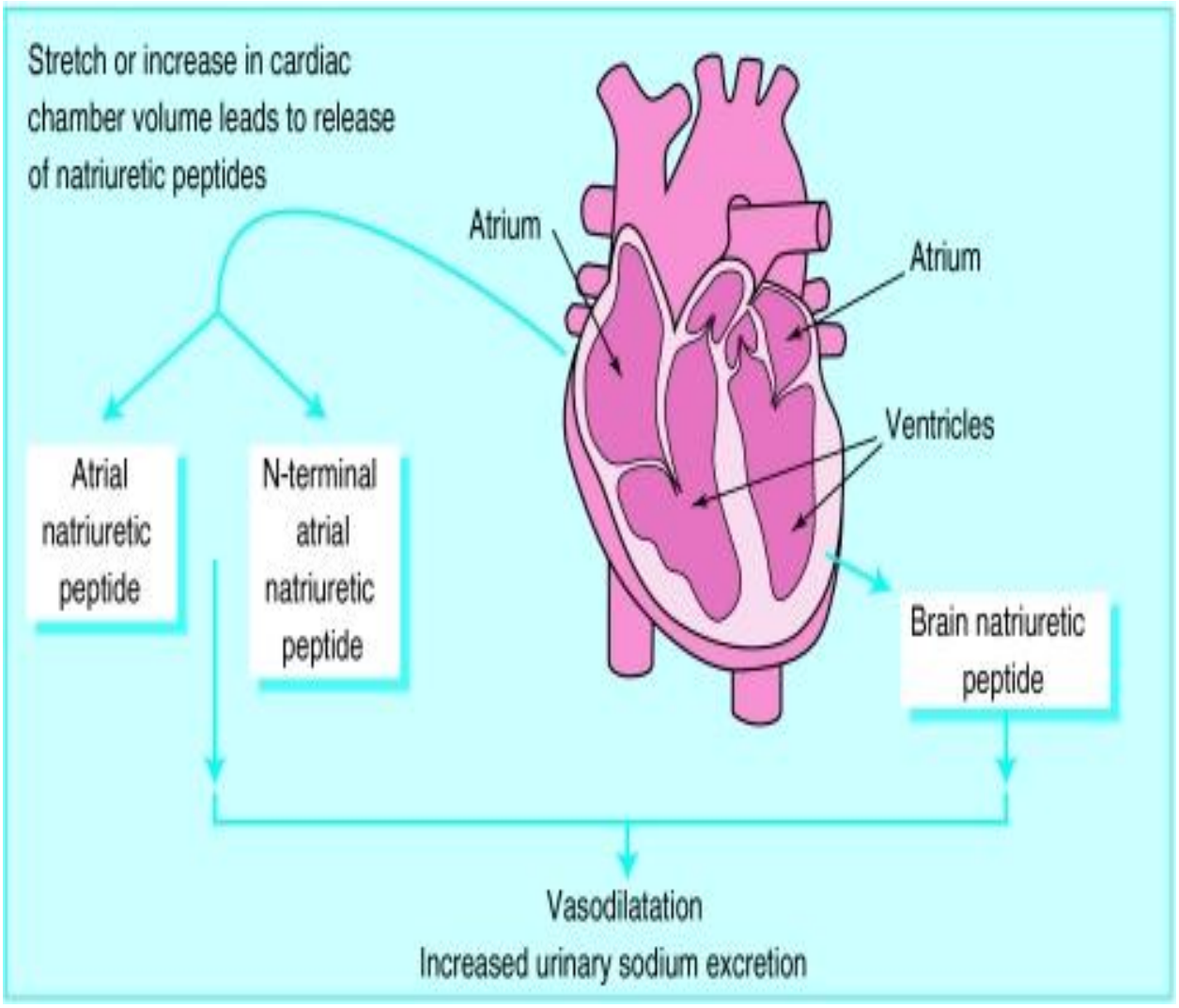
is a long-term condition that is associated with the heart undergoing adaptive responses (such as dilation and hypertrophy) to a precipitating cause. These adaptive responses, however can have a harmful effect (deleterious) .

Duration : months / years

➢ **Neurohormonal mechanisms and compensatory mechanisms in heart failure:**



➢ Effect of natriuretic peptide:



➢ Summary of the consequences to the neurohormonal responses to impaired cardiac performance

Responses	Short-term effects	Long-term effects
Salt & water retention	Increase preload	Pulmonary congestion Systemic congestion
Vasoconstriction	Maintain BP for perfusion of vital organs	-Exacerbate pump dysfunction by increasing afterload -Increase cardiac energy expenditure
Sympathetic stimulation	Increase heart rate and ejection	Increase energy expenditure, Risk of dysrrhythmia, Sudden death

❖ Types of heart failure :

➢ Left-sided heart failure :

- Oxygenated blood from the lungs is pumped by the left side of the heart to the rest of the body. Failure of the left side of the heart results in insufficient delivery of oxygenated blood to the body.

→**Signs and symptoms :**

(are due to pulmonary congestion and low CO)

- **Tachypnea** (increased *rate* of breathing) and increased *work* of breathing.
- **pulmonary edema can develop** (fluid in the alveoli).
- **Cyanosis:** which suggests severe hypoxemia, is a late sign of extremely severe pulmonary edema.
- Additional signs indicating **left ventricular failure** include:
  - a laterally displaced apex beat (which occurs if the heart is enlarged)
  - gallop rhythm (additional heart sounds) may be heard as a marker of increased blood flow, or increased intra-cardiac pressure.

➢ **Right-sided heart failure :**

→**Signs and symptoms :**

- **Pitting peripheral edema,**
- **Ascites** (an abnormal accumulation of fluid in the abdomen)
- **Hepatomegaly** (enlargement of the liver)
- **Elevated Jugular venous pressure** .JVP is frequently assessed as a marker of fluid status, which can be marked by the hepatojugular reflux.

Signs/Symptoms	Left-Sided Heart Failure	Right-Sided Heart Failure
Pitting Edema(Legs,Hands)	Mild to moderate.	Moderate to severe
Fluid Retention	Pulmonary edema (fluid in lungs) and pleural effusion (fluid around lungs).	Abdomen (ascites).
Organ Enlargement	Heart.	Liver. Mild jaundice may be present
Neck Veins	Mild to moderate raised jugular venous pressure (JVP).	Severe jugular venous pressure (JVP). Neck veins visibly distended.
Shortness of Breath	Prominent dyspnea. Paroxysmal nocturnal dyspnea (PND).	Dyspnea present but not as prominent.
Gastrointestinal	Present but not as prominent.	Loss of appetite. Bloating. Constipation. Symptoms are significantly more prominent than LVF

Male notes

➢ Cardiac output

Stroke volume : amount of blood pomp out of each ventricle per beat.

(Norma=70ml/beat)

Co= HR*SV normal 5L/min

When we are doing exercise CO became 25-30 L/min . why?

Because circulating became faster. Blood volume is same

Cardiac index :-how much blood is going per square meter of body surface area.

Normal 3.2L/M

Cardiac index has no great meaning

Ejection fraction:- equal to stroke volume but more accurate and measured by echocardiography

Ejection fraction:- (SV÷ EDV)*100 = normal 45-60%

SV= EDV-ESV

ESV depend on contraction on ventricle and pressure of aorta and contractility. EDV: depend on venous return

If ejection fraction is low →low cardiac output e.g. .heart failure

What are factors affecting cardiac output ?

1. Preload.
2. contractility.
3. after load.

If increase blood pressure what happen to cardiac output ?

Decrease cardiac output due to increase after load

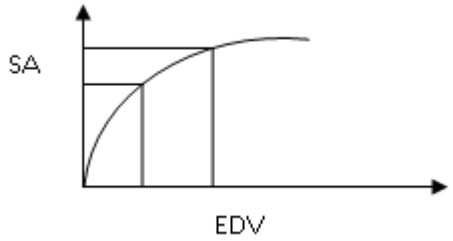
- **Contractility tow type**

1. Isotonic . contraction of heart. E.g. maximum ejection phase
2. Isovolumetric .closed chamber ,ventricle start contraction

Frank-Starling mechanism:

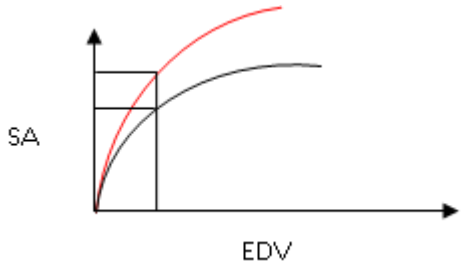
if there is increase in initial length of cardiac muscle fiber (due to increase venous return) there will be increased strength of contraction.

If EDV high→ length of fiber more→ more contraction→increase SA



It is also known as positive isotropic affect

If we stimulate sympathetic system increase inotropic affect. In these case EDV do not change



Red curve in case of sympathetic

Result

- Stimulation of sympathetic →increase SV.
- Stimulation of sympathetic + increase EDV = maximum SV.
- Increase EDV → increase SA.

➢ Contractility

If we stimulate sympathetic nerve what happen to contractility ?

Increase contractility . also, speed of contraction faster ,rate of pacemaker potential will be increase and conduction velocity increase.

Why during exercise do not follow staling law ?

Because sympathetic nerve stimulation and length of cardiac muscle fiber is same.

Starling law without nerve supply . if we cut nerve of heart ,it will completely work. E.g. heart transplant . these called intrinsic control.

There are two result of contraction

1- Positive ionotropic effect.
   a. Sympathetic stimulation
   b. Adrenaline & Noradrenaline
   c. Calcium ion
   d. Caffeine
   e. Drugs eg. Digitalis (Digoxin)

2- Negative ionotropic effect
   a. Parasympathetic stimulation. SA node and NA node supply by right and left vagus nerve. negative ionotropic mostly limited on right atrium but if we stimulate Parasympathetic forcefully, ventricle will be affected (because there is more Ach release which came from atrium to ventricle and affect myocardium)
   b. Acetyl choline
   c. Potassium ion; has got membrane stabilization function. Hyperkalemia more than Hypokalemia. (both can effect)

d. Hypoxia (Decrease oxygen)
e. Acidosis
f. Bacterial toxin
g. Drugs eg. Calcium channel blockers, β- Blockers

➢ Heart failure

Heart failure is when heart is unable to maintain sufficient output to meet the body's normal metabolic needs.

❖ if starling law go down that mean heart failure.

HF can be due to reduction of cardiac output. e.g. damage on myocardium ,infarction
HF can occur due to increase demand of oxygen in periphery. (SA is normal) e.g. thyrotoxicosis (mean metabolic is high → need more oxygen . arteriovenous fistula.

▸ Acute heart failure

Sudden peripheral vasodilatation or sudden opening peripheral vessel. Venus return is less

▸ Right heart failure

Ventricle do not push blood . superior vena cava (SVC)and inferior vena cave (IVC) full of blood.
Patient has edema in the legs and hepatomegaly .

▸ Left heart failure

Blood accumulate in ventricle →pulmonary congestion →lung full of fluids→dyspnea and orthopnea .

▸ Systolic failure

myocardium damage

▸ Diastolic failure

Because of compliance.

Causes of chronic heart failure

- More Venues return.
- Contraction not power full.
- Heart attack . Patient lose 2/3 of myocardium.
- After load high. e.g. hypertension →hypertrophy of left ventricle→ failure.
- Arrhythmia