

Cardiac Output (CO) & Cardiac reserve

Physiology department merit university

ILOS

- Describe cardiac output and state components controlling it.
- Define various related terms, including stroke volume, cardiac index, end diastolic and end systolic volumes.
- Explain the importance of cardiac reserve, mechanisms and limitations, and heart failure (pathophysiology, causes and consequences).

Cardiac output

- **Cardiac output (COP):** Is the volume of blood pumped by each ventricle per minute.

About 5L\min during rest.

$$\text{COP} = \text{SV} \times \text{HR} = 70 \times 70 = 4900 \text{ ml}\backslash\text{min}$$

- The CO depends on:
 - 1- stroke volume (SV)
 - 2- heart rate (HR)

definitions

- **Stroke volume:** is the volume of blood pumped by each ventricle per beat.

About 70-9- ml\beat during rest

- **Cardiac output (COP):** Is the volume of blood pumped by each ventricle per minute.

About 5L\min during rest.

definitions

- **Cardiac Index (CI):** Volume of blood pumped by each ventricle per minute **per** square meter body surface area.

Normally = 3.2 L/min/m²

- **End Diastolic Volume:** is the volume of blood at the ventricle at the end of the diastole.
About (110-130 ml).
- **End systolic ventricle:** is the volume of blood at the ventricle at the end of the systole.
About (40-60 ml).
- $SV = EDV - ESV = 130 - 60 = 70ML$

definitions

Ejection Fraction (EF):

Definition: EF is the fraction of the EDV that is ejected with each beat

Calculation:

$$EF = \frac{\text{Stroke volume}}{\text{End diastolic volume}} \times 100$$

Normally: Ejection fraction is 55-75%

Importance : Assessment of Contractility.

Variations of cardiac output (CO):

UNCHANGED

1- SLEEP

2- MODERATE CHANGES IN EXTERNAL TEMPRATURE.

Variations of cardiac output (CO):

INCREASED IN

- 1- EXIETMENT 50-100%
- 2- EXERCISE 300%
- 3- EXPOSURE TO HIGH TEMPRATURE
- 4- EATING
- 5- EPINEPHRINE
- 6- END OF PREGNANCY
- 7- INSPIRATION
- 9- SHIFTING FROM STANDING TO RECOMBINANT POSITION. (Gravity→ decreases VR)

Variations of cardiac output (CO):

DECREASED IN

- 1- SITTING OR STANDING FROM LYING POSITION (gravity)
- 2- ARRHYTHMIA
- 3- HEART FAILURE

Control of the cardiac output

1. HEART RATE REGULATION.
2. CHANGES IN PRELOAD.
3. EFFECTS OF ATERLOAD.
4. EFFECTS OF CONTRACTILITY.

NB: 2,3,4 = factor regulating Stroke volume(preload, afterload, contractility).

1-Heart Rate(HR)

- An increase of HR up to 200/min. (eg during exercise) leads to an increase of CO (?????) → during exercise both HR & SV(increased VR) increase → COP increase.
- Moderate changes in heart rate(50-100 beat\min) : no change in COP ?????? The increase or decrease in HR is corrected by SV, **EXAMPLE**:
When heart rate increased to 100 beat \min → shortening of the diastole → SV decreases to 50 ml so $cop = SV \times HR = 100 \times 50 = 5000 \text{ ml}\backslash\text{min}$

1-Heart Rate(HR)

- Pathological or excessive change in HR → decrease COP ???

In paroxysmal tachycardia → heart rate is increased but stroke volume is decreased → shortened diastole & decreased contractility (over use of energy).

2-Change of Preload

- Preload determines muscle length before start of contraction.
 - Ventricular preload is determined by EDV
 - EDV depends on venous return
 - According to Starling's Law:
 - The more the end diastolic volume the more is the force of contractions; within limits.
 - An increase of EDV (preload) leads to increase SV with consequent increase of CO

2-Change of Preload

- Preload is affected mainly by venous return
- **Factors that increase venous return:** Means increase filling of ventricles increased EDV → increased myocardial contractility → increased SV → increased cardiac output

2-Change of Preload:

- Factors that affect ventricular preload:

1. **Venous Return (VR):** Increased venous return will increase preload
2. **Ventricular compliance:** Decreased ventricular compliance will decrease preload eg myocardial infarction
3. **Heart rate:** Increased rate more than 200/min leads to a decrease of preload due to shortening of diastole decreased ventricular filling

3- Effect of afterload on SV

- After load is the load against which the ventricle contracts and ejects blood
- The main after load for left ventricle is the **aortic pressure**.
- An **increase** of after load leads to a **decrease** of SV → decrease CO.
- **Decreased** after load leads to **increase** of SV → increase CO.

4- Effect of contractility (inotropic state) on SV

- Positive inotropic → increase contractility of the ventricle → ejecting more blood → increase SV → increase CO.

Cardiac Reserve "CR"

- **Definition:** It is difference between cardiac output pumped by the heart under ordinary circumstances & its maximum capacity for pumping blood

Assessed by:

Low-dose dobutamine stress echocardiography

Mechanisms of Cardiac Reserve CR

1- +ve chronotropic effects

(Heart rate reserve)

During rest:

HR = 70

During exercise:

HR = 200 (so, COP can increase 3 time)

Limitation: Diastolic period is shortened

Mechanisms of Cardiac Reserve CR

2- factors that increase the preload

- During rest: = 135 ml (EDV reserve)
- During exercise: **Venous return increase** led to **increase** ventricular filling (increase in the initial length) → **increase** EDV → lead to **increase** force of contraction
- **Limitation:** over stretch as in heart failure → decrease contractility

3- factors that decrease the afterload

- Factors that decrease the peripheral resistance (will be discussed later)

Mechanisms of Cardiac Reserve CR

4- +ve inotropic conditions:

During rest the ESV is 50-60 ml.

During exercise sympathetic stimulation increase the force of contraction → the ESV decrease → increase of SV → increase in COP.

Limitation: too much decrease in ESV → injury to endocardium.

Mechanisms of Cardiac Reserve CR

5-Cardiac hypertrophy: physiological (athletes) or pathological (hypertension)
hypertrophy → increase contractility → increase SV → increase COP.

Limitation: relative ischemia as ventricular capillaries do not proliferate

FINALLY;

*If there is no matching between COP & body needs: so,
this Heart is Failed (HF)*

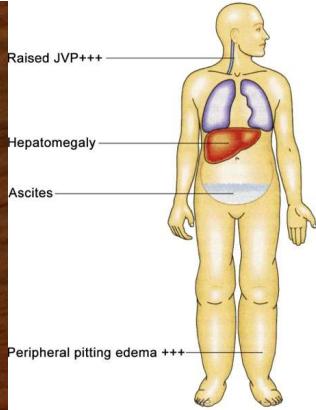
Heart Failure

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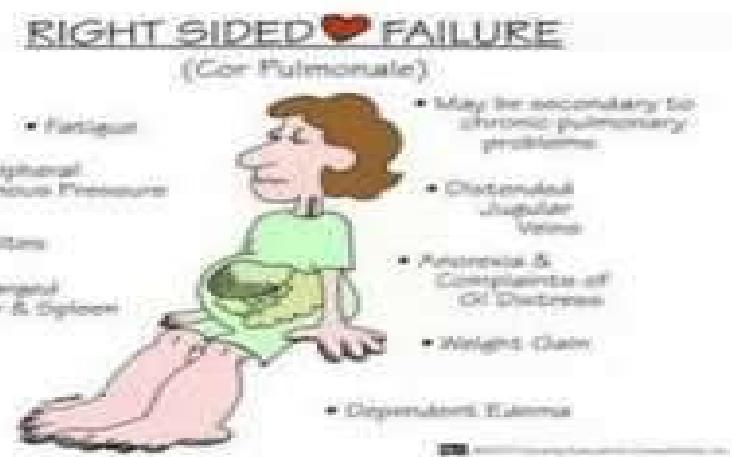
Heart Failure

- Definition: It is a clinical syndrome in which the heart is unable to pump an adequate amount of blood to satisfy metabolic requirements of the body
- Etiology:
 1. Ischemic heart diseases (most common)
 2. Systemic hypertension
 3. Valvular disease
 4. Myocardial disease as cardiomyopathy.



Clinical presentation of HF

Right-Sided Heart Failure		Left-Sided Heart Failure	
① Backward manifestations (Systemic congestion)	: eg: - Lower Limb edema - Ascites - Congested neck veins ($\uparrow\uparrow$ JVP) - Congested liver	① Backward manifestations (Pulmonary congestion)	: eg - Dyspnea - Cough - Hemoptysis - Recurrent chest infection
② Forward manifestations (Low COP manifestations)	: eg syncope, fatigue	② Forward manifestations (Low COP manifestations)	: eg syncope, fatigue
③ Cardiovascular manifestations	<u>S₃</u> & gallop	③ Cardiovascular manifestations	<u>S₃</u> & gallop



INVESTIGATIONS

1. Chest Xray → cardiac size
2. echocardiography: → asses ventricular function → ejection fraction (55-70% normal)

Treatment

① Medical:

① ↓ Preload:

① Rest: Semi-sitting or sitting

② Diet: Salt restriction (< 2 gm/day)

③ Diuretics

④ VenoDilators: eg Nitrates “Nitroglycerin”

② ↓ Afterload: Arteriodilators: eg Ca channel blocker (CCB) : Diltiazem

③ ↑ Contractility: Inotropic drugs: eg:

1. Digitalis

1. Dobutamine

Treatment

② Interventional:

① ↓ Preload:

Mechanical removal of fluids: By Dialysis

② ↓ Afterload:

Mechanical assistance of circulation: By
IABP “Intra-Aortic Balloon Pump”

③ ↑ Contractility: CRT (Cardiac Resynchronization Therapy)

③ Surgical:

Cardiac transplantation

Thank you

Dr\dina monir

**KEEP
WORKING
HARD.
LET THE
RESULTS
SPEAK FOR
THEMSELVES.**