Cardiovascular

(CVS)

By

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Cardiac Output (CO)

- **Definition:** It is the volume of blood pumped by each ventricle per minute
- About 5 L/min in normal resting young adult male

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Cardiac output

= SV x HR

= 70 ml x 72 bpm

= 5 L/min

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CO under various conditions:

Conditions increase ↑ CO

1. Anxiety and excitement (50 – 100%)
2. Food intake (30%)
3. Physical activity (up to 700%)
4. Increased environmental temperature
5. Pregnancy

Conditions decrease ↓ CO

1. Sitting or standing from lying position (30%)
2. Heart diseases.

❑ Determinants of the CO:

Cardiac output = SV x HR = 5 L/min

Stroke volume: volume of the blood pumped by each ventricle per beat (70 ml)

SV = EDV – ESV = 130 – 60 = 70 ml

Heart rate: number of heart contractions per minutes (72 bpm)

Factors controlling cardiac output:

(SV x HR)

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I. Stroke volume:

- Is affected by:

1. Preload
2. Afterload
3. Inotropic state of the heart
4. Size of cardiac muscle

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1. Preload

- Preload = EDV
- According to starling law, the more the preload is, the more the force of contraction will be within limits
- The right atrial pressure is the central venous pressure (CVP)

2. Afterload:

- Afterload = aortic pressure
- The **more** the **afterload** (aortic pressure) is, the **less** the **force of contraction** of the heart will be, so the **less** the **SV** will be and the **more** will be the **ESV** provided that the EDV (preload) is constant.

3. Inotropic state (contractility) of the heart:

- At constant preload and afterload, the force of contraction can be increased and consequently increase the SV and CO.
- Because contractility of the heart is determined by the amount of Ca++ accumulation inside the cardiac myocyte during activity and normally it is not the maximum load (the more the Ca++ IC ⟶ the more the force of contraction).
- Positive inotropes: as sympathetic stimulation
- Negative inotropes: as parasympathetic stimulation

4. Size of the cardiac muscle

- In case of eccentric cardiac hypertrophy as in athletes ⟶ the SV will be increased. EDV also will be increased which may lead to 700% increase in CO as in case of marathon (long distance) runners.

II. Heart rate (HR):

- The increase in heart rate will increase CO within limits (N.B. very rapid HR above 180 bpm will affect the diastolic filling time leading to decreased CO)
- Sympathetic stimulation (Catecholamines) increases HR (+ve chronotropic) and hence CO
- Parasympathetic stimulation decreases HR (-ve chronotropic) and hence CO

Venous return (VR)

It is the volume of the blood returning to the heart per min.

Under steady conditions, the VR = CO

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The regulation of Venous Return (VR) will consequently affect end diastolic volume (Preload) which regulates the SV (EDV – ESV)

- **According to Ohm's law of flow:**
  - **Flow (F) = Pressure gradient (ΔP) / resistance (R)**
    - **So, VR = ΔP (MSFP – RAP) / RVR**
      - **MSEF = mean systemic filling pressure**
      - **RAP = right atrial pressure**
      - **RVR= resistance to venous return**
- **Therefore, factors regulating venous return are MSFP, RAP and RVR, that will be discussed using venous return curves.**

Factors affecting VR

According to Ohm's law

Flow = ΔP / R

1. Mean systemic filling pressure (MSFP)
2. Right atrial pressure
3. Resistance to VR

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1. Mean systemic filling pressure (MSFP):

- It is the equilibrium pressure throughout the whole systemic circulation if the heart stops pumping.

❖ *It is measured using experimental animal in which the heart is removed and replaced by pump oxygenator perfusing the peripheral circulation. If the pump stops (i.e. cardiac output reaches zero), arterial pressure drops and venous pressure rises until reaching the equilibrium pressure which represents the venous pressure because the capacitance of the veins is higher than that of the arteries (about 20 times).*

- Therefore, equilibrium pressure represents the distending pressure of all blood vessels at certain blood volume which is the MSFP.

1. Mean systemic filling pressure (MSFP):

- It ranges normally between 6 – 8 mmHg and it is the pressure driving VR to the heart. So, the more MSFP is , the more will be the VR and vice versa.
- It is affected by 2 factors:

1- Blood volume.

2- Capacity of the blood vessels mainly veins.

1. Mean systemic filling pressure (MSFP):

MSFP is increased:

- Blood volume increases
- Venoconstriction (decreased capacity of the circulation) e.g. sympathetic stimulation

MSFP is decreased:

- Blood volume decreased (hemorrhage)
- Veno-dilatation (increased capacity of the circulation)

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2- Central venous pressure:

- the right atrial pressure (RAP) and is normally 0 – 2 mmHg.
- It is the main sucking force of the ventricular filling
- As, the difference between CVP and MSFP (the pressure gradient **ΔP**) that regulates VR (blood flow to the heart).
- So, the less the RAP is, the more will be the venous return and vice versa.
- If right atrial pressure reaches 8 mmHg (= MSFP) → **ΔP** is zero (no VR)

2- Central venous pressure:

- If RAP is below -1mmHg, VR couldn't be more increased (VR remains constant) ⟹ because at this pressure (-1 mmHg) the intrathoracic veins opening in right atrium start to collapse due to compression by intrathoracic pressure
- It is also affected by 2 factors:

1- Blood volume.

2- Venous compliance.

3. Resistance to VR:

- Increase RVR ⟹ VR decreases and vice versa

➢ *It is of low value 1.4 mmHg/L/min.*

✓ *It can be calculated by Ohm's law*

VR = ΔP (MSFP – RAP) / RVR

So, RVR = ΔP / VR = 7 - 0 mmHg / 5 L/min= 1.4 mmHg/L/min.

✓ *So, the more the RVR is, the less the venous return will be.*

Mechanisms that help VR in the standing position against the effect of gravity

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1.Cardiac suction:

- **Atrial suction effect**

that occurs during the 1st part of rapid ejection phase of the cardiac cycle due to drop of RAP (CVP) leading to sucking of the blood from large veins to the atria.

- **Ventricular suction effect**

that occurs during the rapid filling phase sucking blood from the atria and consequently the large veins due to sharp drop of ventricular pressure

2- Thoracic pump:

- It is the effect of intrapleural (intrathoracic) pressure [the negative pressure measured in the space between the visceral and parietal pleura] → suction of blood from abdominal veins to thoracic veins.
- During expiration, there is less venous return because the intrapleural pressure is average -4mmHg and is transmitted to the intrathoracic veins and the intrabdominal pressure is +5mmHg transmitted to the intraabdominal veins. So , the pressure gradient will be 5 – (- 4) = 9mmHg leading to increased VR.

2- Thoracic pump:

- During inspiration the reverse occur, intrapleural pressure decreases to -8mmHg and the intraabdominal pressure increases to 6mmHg due to descent of the diaphragm. So the pressure gradient will be 6 – (-8) = 14mmHg . Hence venous return will be increased more.
- During Valsalva's manoeuvre → +ve intrathoracic pressure → --VR
- During Muller's manoeuvre → -ve intrathoracic pressure → ++VR

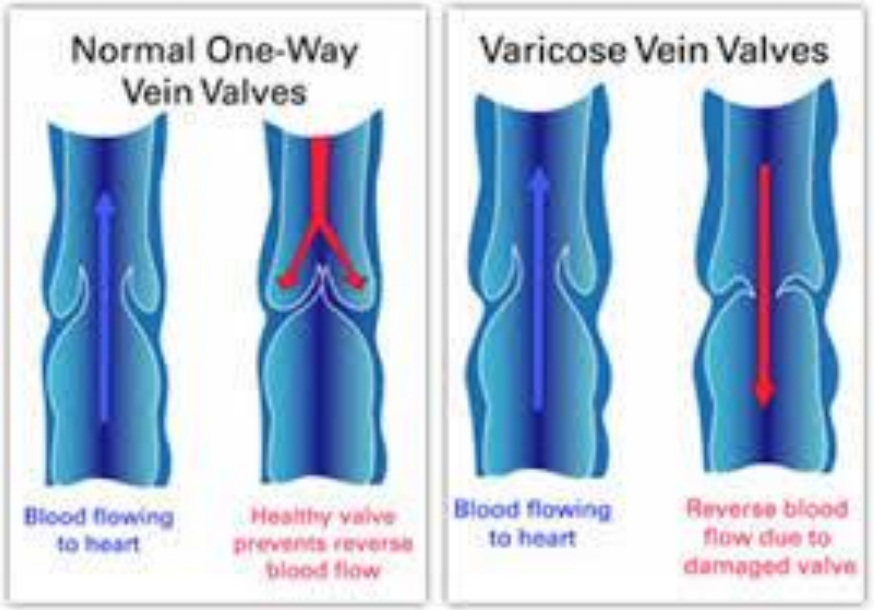
3. Muscular contraction:

- Compresses veins pushing blood upwards towards the heart

4. The venous valves:

One–way valves that allow blood flow towards the heart and prevent its retrograde flow.

In case of incompetent valves as in case of varicose veins, the blood will pool in lower limb veins due to gravitational force decreasing VR.



5.Sympathetic venoconstricion

- Sympathetic tone increases during standing position → venoconstriction → pushing blood upwards.

Cardiac efficiency

- Ratio between mechanical work of the heart and total cardiac energy consumption.
- Normally, Cardiac efficiency is 20 - 25 % i.e.. 25% of the total energy consumption of the heart is converted to mechanical work.
- Mechanical work is pressure – volume work (pumping) and kinetic work (to give velocity to the blood)

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