بسم الله الرحمن الرحيم

السلام عليكم ورحمة الله وبركاته

Cardiovascular System Block
Stroke volume & Cardiac Output (Physiology)

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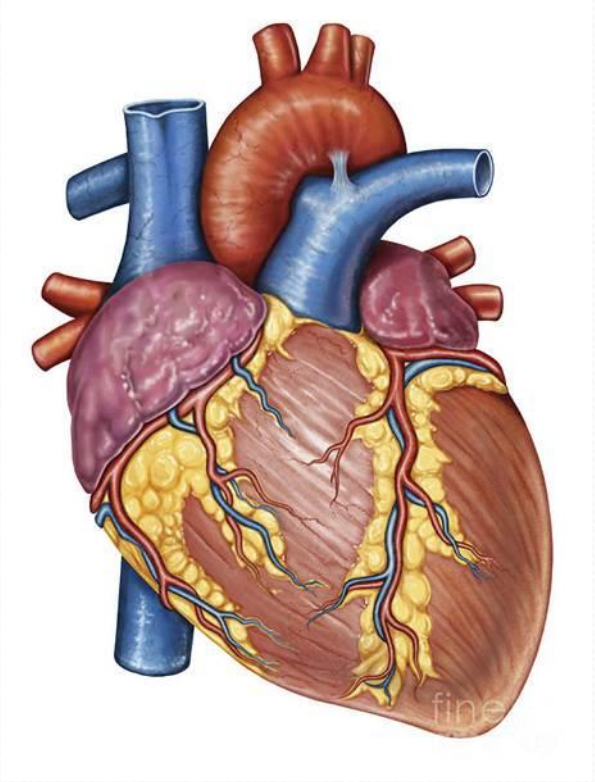


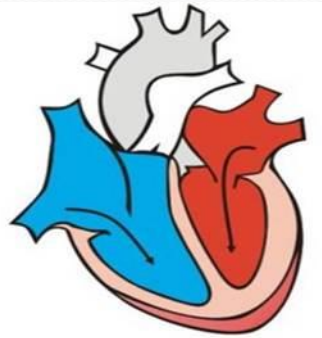
Learning Objectives

- Define stroke volume, end-systolic volume, and end-diastolic volume
- Define cardiac output, its normal values and factors affecting it
- Understand the determinants of CO and how CO is regulated
- Understand the factors affecting the EDV (the venous return)
- Understand the factors affecting the ESV (cardiac contractility & afterload)
- Know how heart rate changes affect CO
- Identify the factors that affect heart rate
- Know the method for measurement of CO (The direct Fick's method)

Remember What Are ?

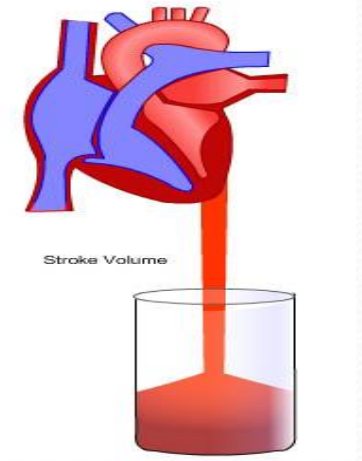
- **End-diastolic volume (EDV):**

  Volume of blood in ventricles at the end of diastole = **110-130 mL.**



- **Stroke volume (SV):**

  Amount of blood ejected from ventricles during systole = **70-80 mL/beat.**



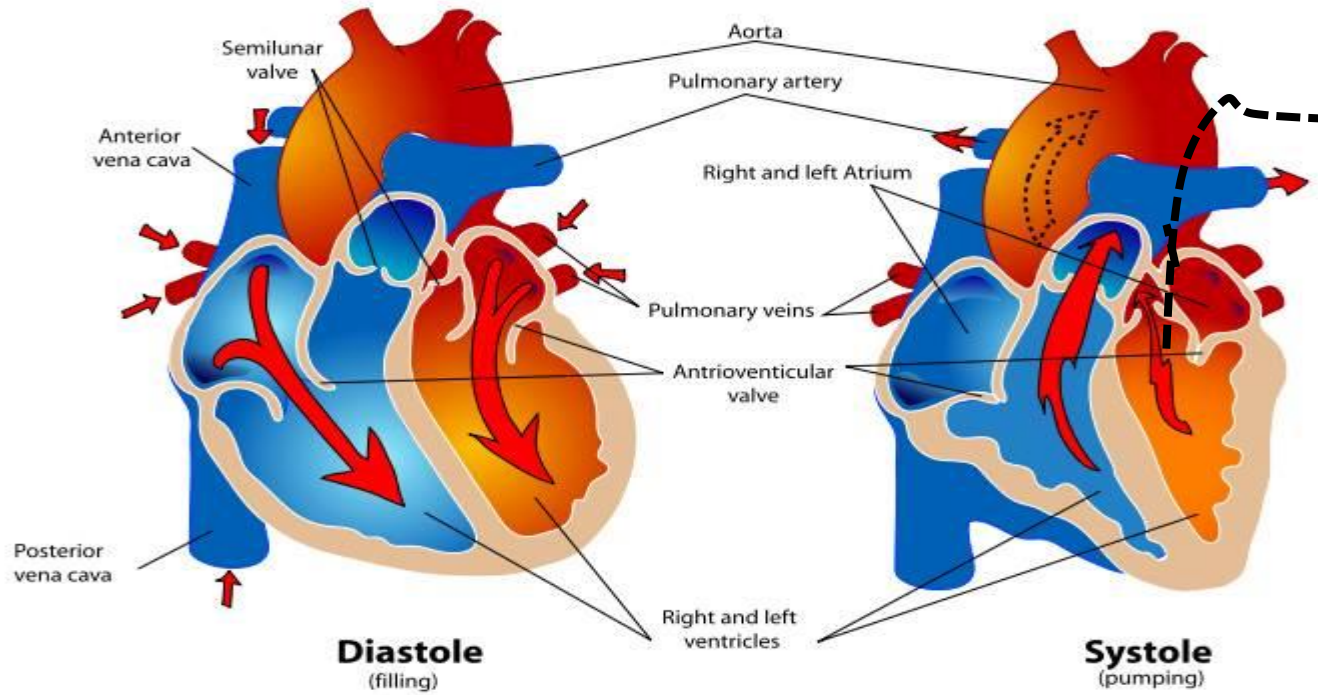
- **End-systolic volume (ESV):**

  Amount of blood left in ventricles at the end of systole = **40-60 mL.**

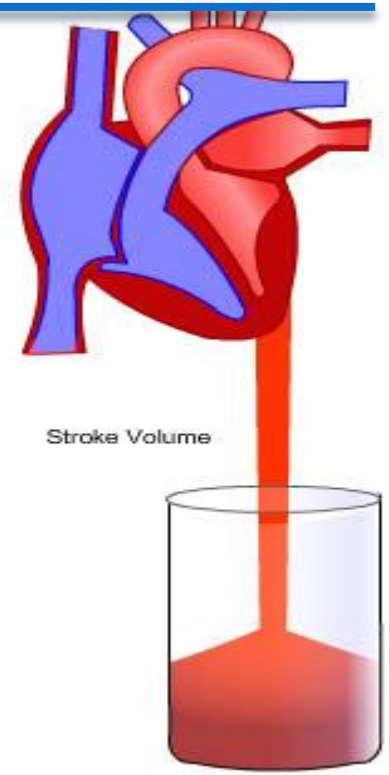
- **Ejection fraction (EF):**

  Fraction of end-diastolic volume ejected during a heart beat =stroke volume/end diastolic volume = **60-65 %.**

The Stroke Volume



Amount of blood ejected from ventricles during systole (per beat) = ***The stroke volume***



Volume of blood in ventricles at the end of diastole (***End-diastolic volume***) ≈110-130 ml

−

Amount of blood left in ventricles at the end of systole (***End-systolic volume***) ≈40-60 ml

=

The stroke volume (70-80 ml)

What determines the stroke volume?

The Cardiac Output

- If the amount of blood pumped by the left ventricle ***per beat*** = ***stroke volume (≈70ml).***

- ***What is the amount of blood pumped by the left ventricle per minute?***

  = Stroke volume (SV) X heart rate (HR)

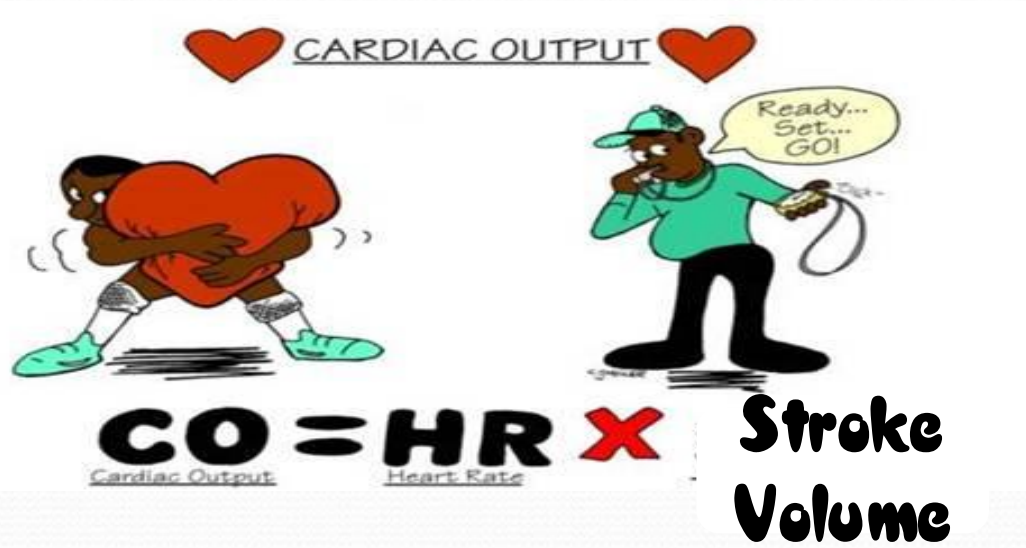
  = Cardiac output (CO)

  ≈ 5L/min

The Cardiac output (CO)

- It is the volume of blood pumped by each ventricle per minute **≈ 5L/min**.

- Since the normal adult blood volume is about ***5 L***, the entire blood supply passes through the body once each minute.



CO= 70 x 70 ≈ 5L/min.

Normal Values of Cardiac Output at Rest

- **Resting cardiac output:**
  - The average cardiac output for the resting adult is 5L/min.
  - CO vary with size of individual.
  - Children have smaller CO than adults.
  - Women have smaller CO than men.
  - For men, CO ≈ **5.6L/min.**
  - For women, **≈ 4.9L/min.**

- **The factors affecting cardiac output are:**
  1. Body metabolism
  2. Exercise
  3. Hyperthyroidism
  4. Pregnancy
  5. Increase body temperature.

What is the Cardiac index ?

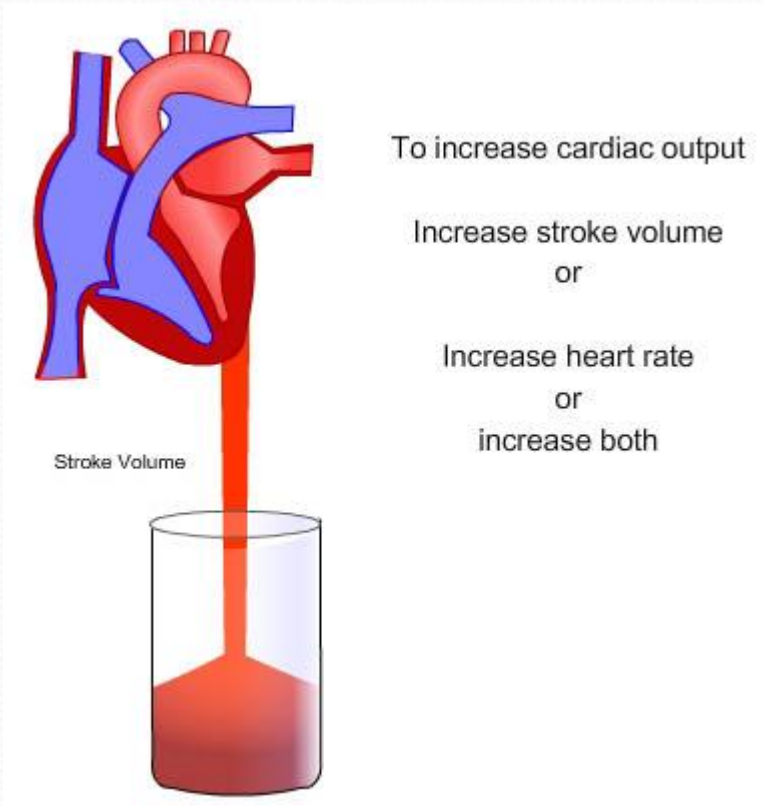
- It relates the cardiac output to body surface area. (CO/min/m^2 of body surface area
- Thus relating heart performance to the size of the individual.
- Normal Cardiac index= (3.2 Liters/min/m^2 body surface area).

What is the Cardiac reserve ?

- It is the difference between cardiac output at rest and the maximum volume of blood that the heart can pump per minute.

Regulation of CO

- CO is crucial since it is also the amount of blood that flows into the circulation and is responsible for transporting substances to and from the tissues.
- Thus, the body has strict control mechanisms that maintain adequate CO.
- There are 2 major factors which determine CO.
- These are the SV and HR.



Determinants of the CO

CO = SV X HR

{End-diastolic volume (EDV) – end-systolic volume (ESV)}

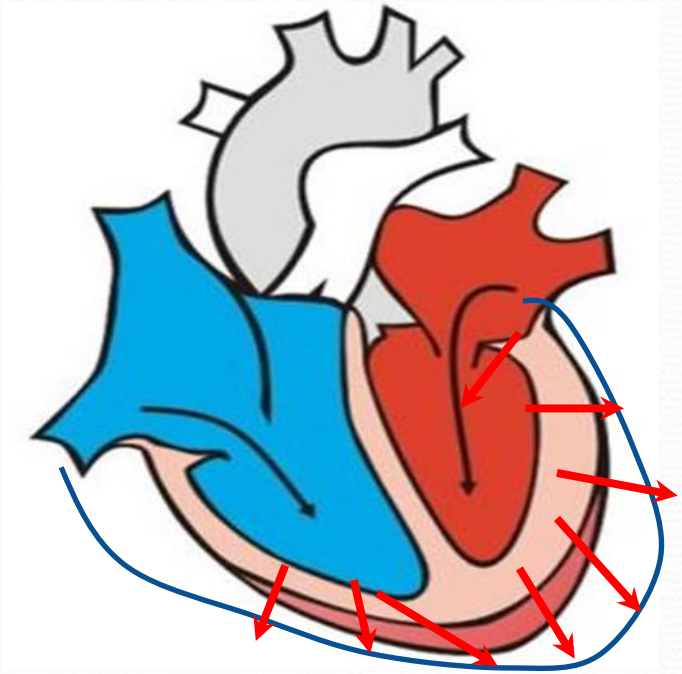
?

CO = {EDV-ESV} X HR

Any factor that affects these parameters will affect the CO

The End-Diastolic Volume (EDV)

- ***EDV*** = is the volume of blood in the ventricles prior to ventricular ejection.
- ***How does the EDV affect the SV and hence the CO?***



↑↑ EDV

↓

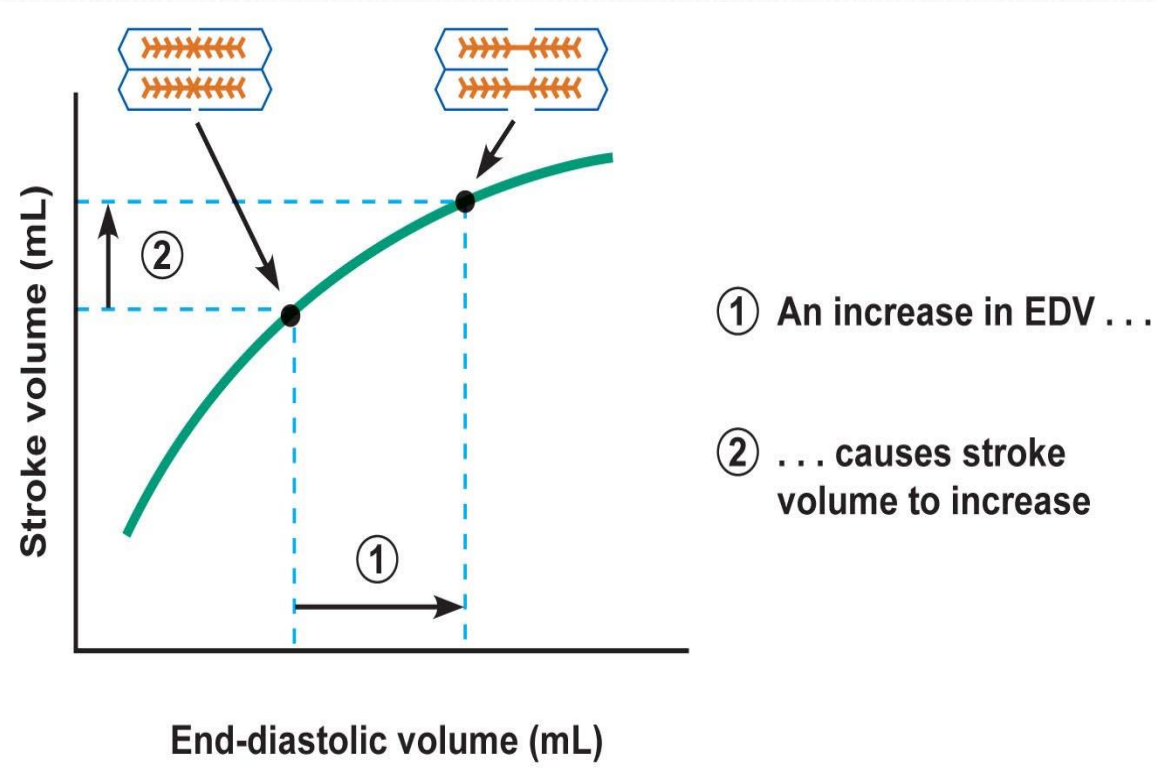
↑↑ The stretch in the myocardium
(↑↑ initial fiber length)

↓

↑↑ Myocardial contractility
(↑↑ Strength of contraction)
Frank-Starling mechanism

Frank-Starling Law

- The Frank Starling principle is based on the length-tension relationship within the ventricle.
- If EDV (preload) is increased, the ventricular fiber length is also increased, resulting in an increased 'tension' of the muscle (i.e **the stroke volume increases in response to increase of the end diastolic volume).**
- This is called the Frank-Starling mechanism (or Starling's Law of the heart)
- Within physiological limits, the heart pump all blood comes to it without allowing stasis of blood in veins.

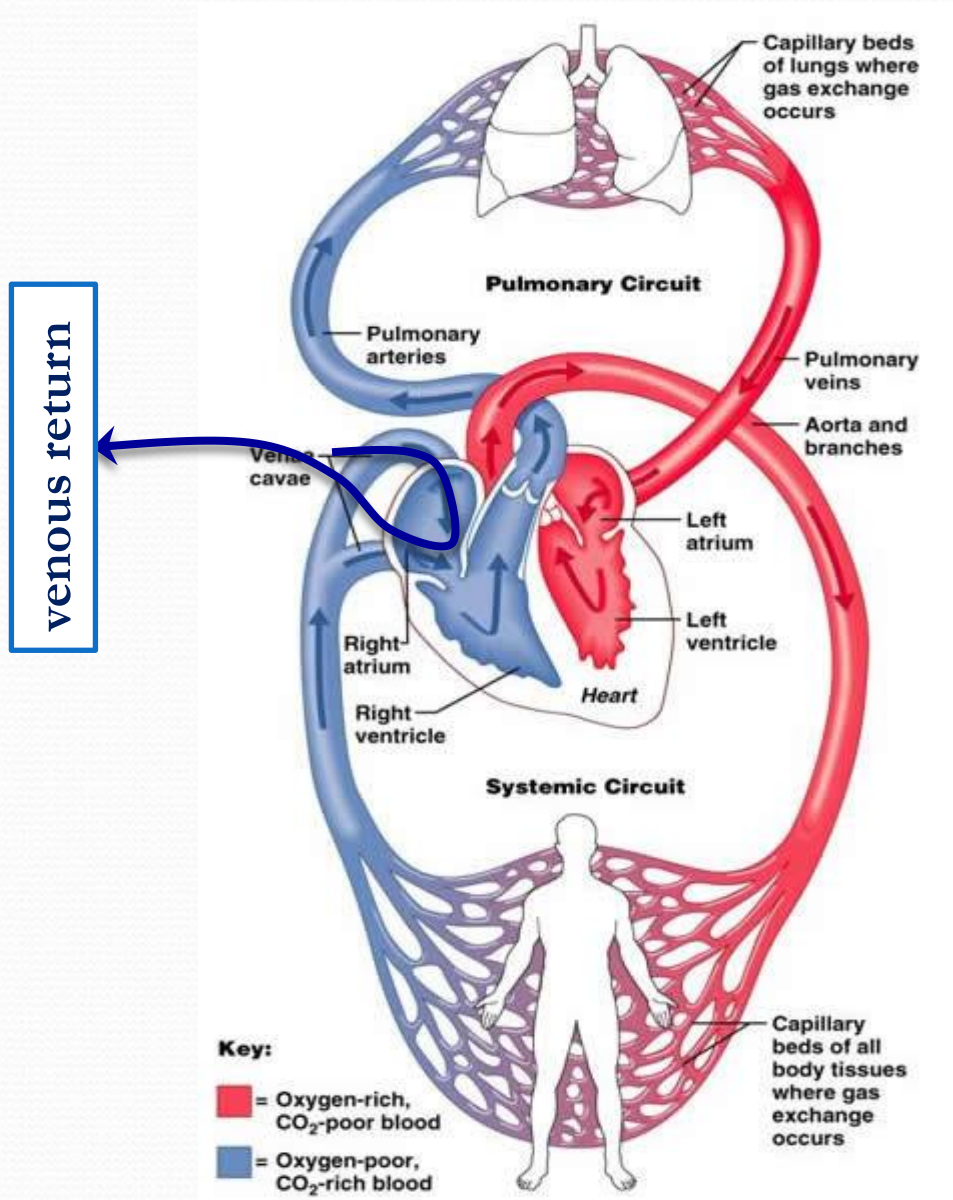


Factors Affecting the EDV

- ***What determines the EDV?***

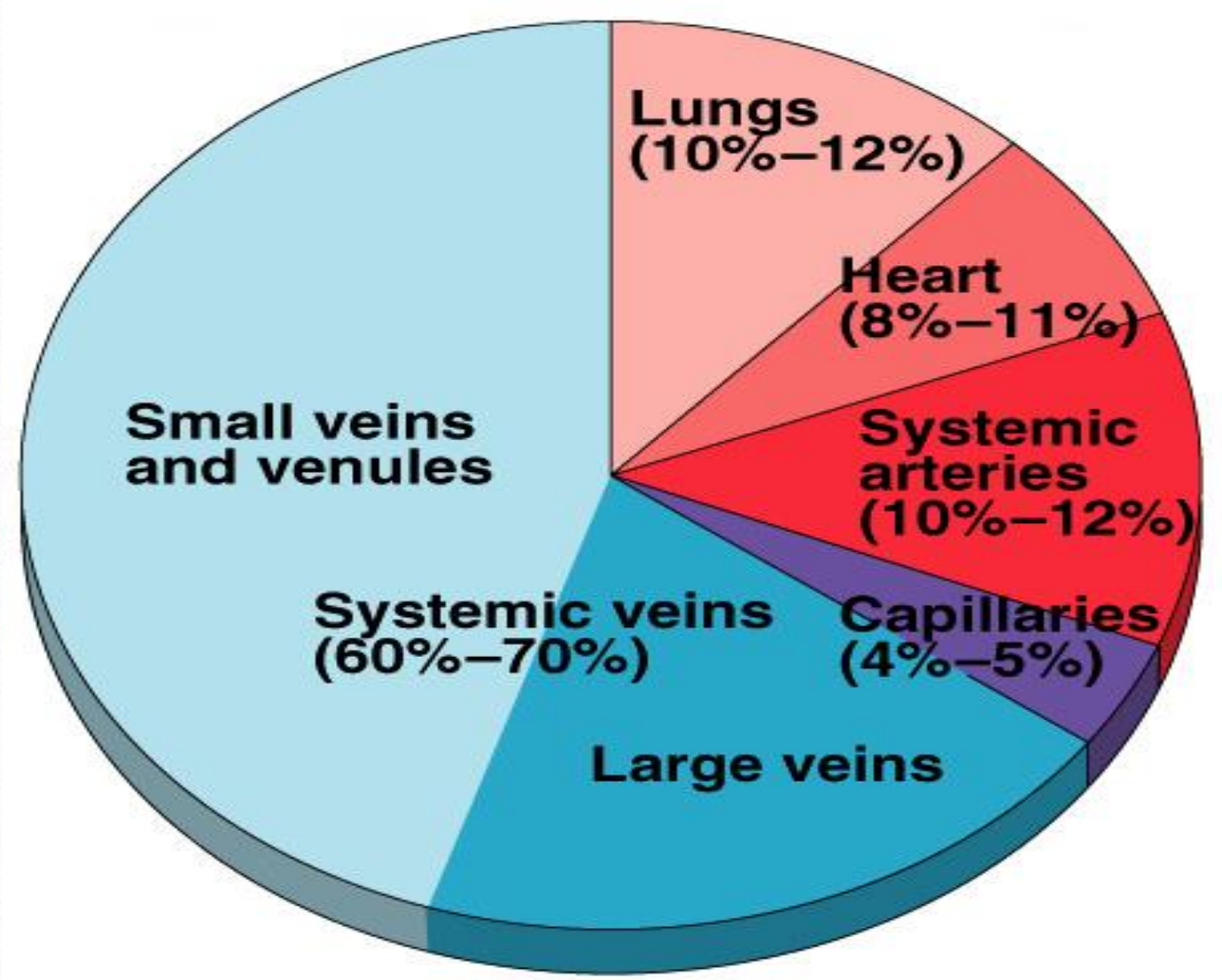
  The venous return (VR).

  - Venous return is the quantity of venous blood flowing from large veins into the right atrium each min.



What is about the veins?

- Veins hold most of blood in body (70%).
- They are called capacitance vessels
- They have thin walls & stretch easily to accommodate more blood without increased pressure (= higher compliance)
- They have only 0 -10 mm Hg Pressure.



Venous return curve

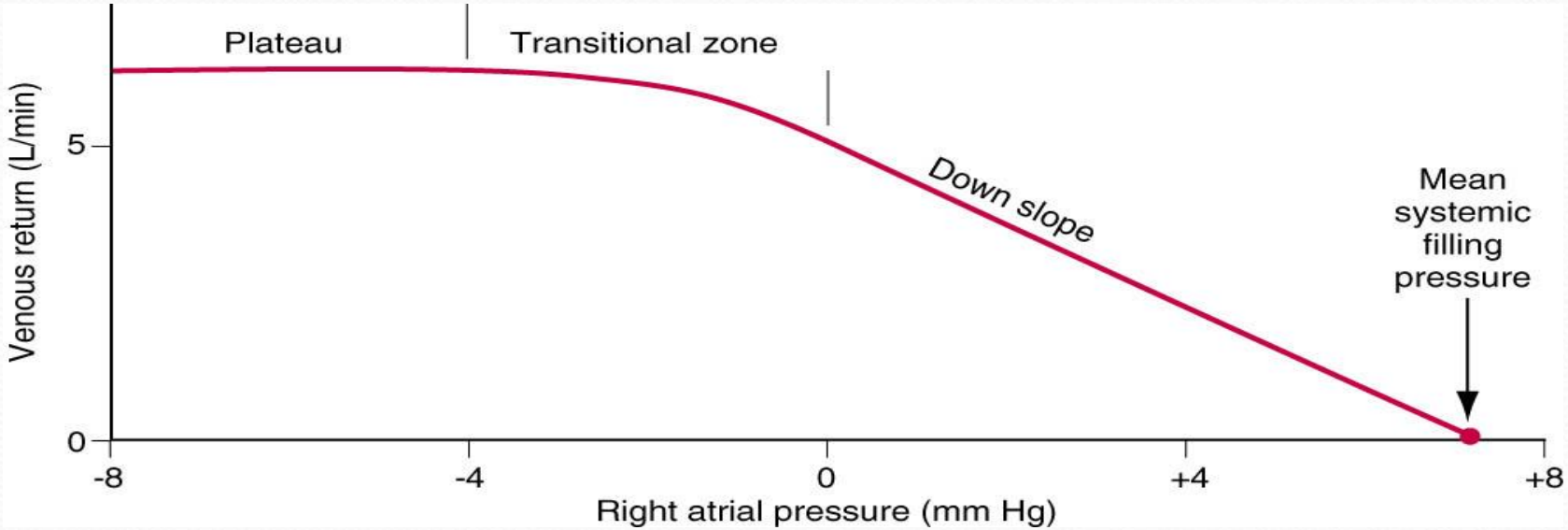
Venous return (VR) curve relates VR to right atrial pressure.

VR is decreased when:

1- The right atrial pressure (RAP) is increased,

2- Pumping capability becomes diminished.

3- The nervous circulatory reflexes are absent.

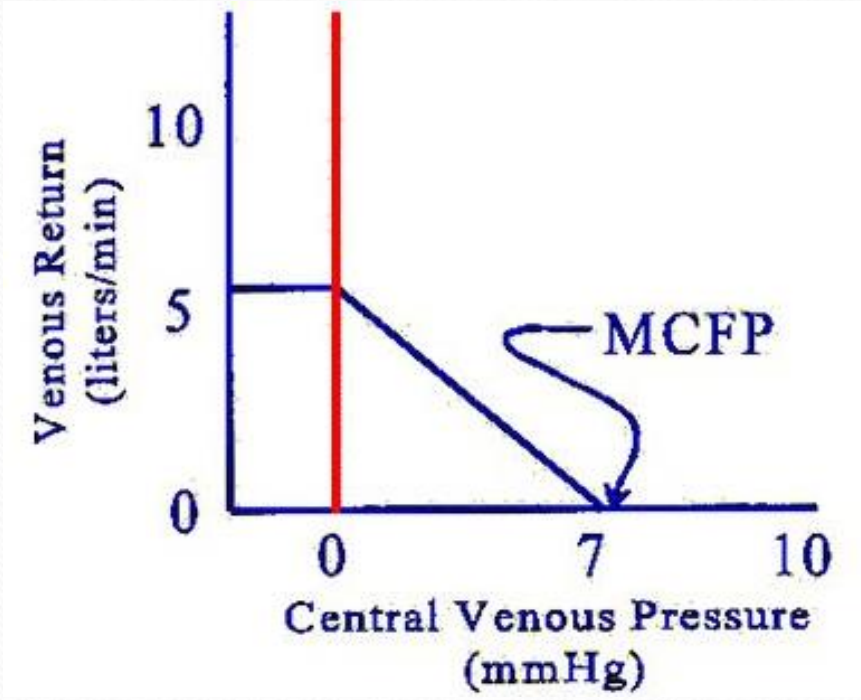


Venous return curve…..Cont.

- When the RAP falls below zero, no further increase in VR and a plateau is reached

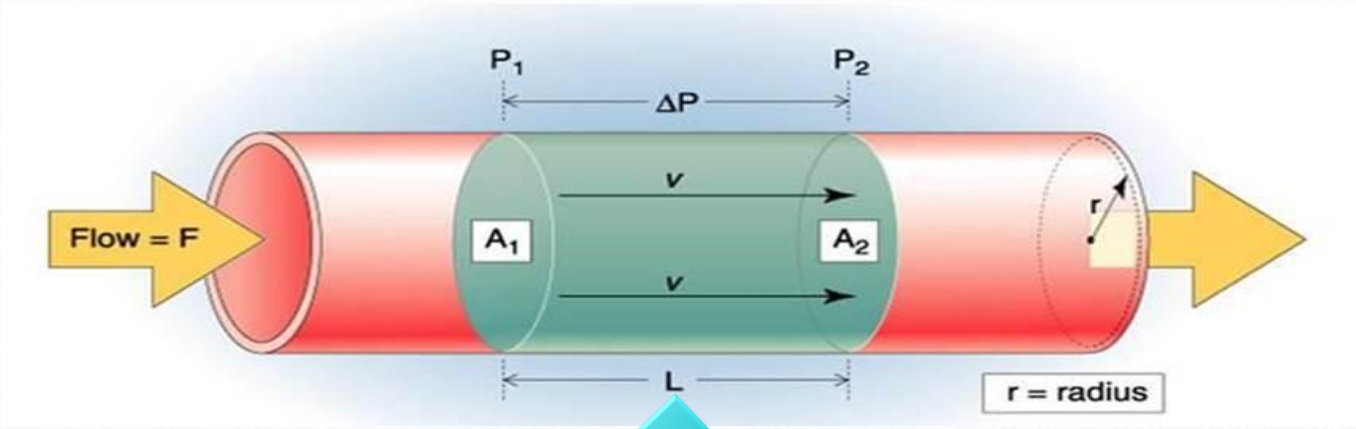
  Cause: collapse of the veins entering the chest.

- Mean Circulatory Filling Pressure is the value for right atrial pressure at which venous return is zero
  - When the heart is stopped by shocking the heart with electricity or any reason, flow of blood cease in the circulation.
  - Without blood flow, the pressures everywhere in the circulation become equal and is called : Mean Circulatory Filling Pressure (which is pressure of +7 mmHg).



Basic Principles

Flow of any fluid (blood) through a tube (vessel) depends on:-



- The pressure difference between the two ends *(Pressure gradient)*
- Blood flows from *high* pressure to *low* pressure

- The resistance to blood flow through the vessel
- Controlled by the diameter of the vessel

Factors Affecting the Venous Return

1- Blood volume & pressure gradient.

2- Gravity

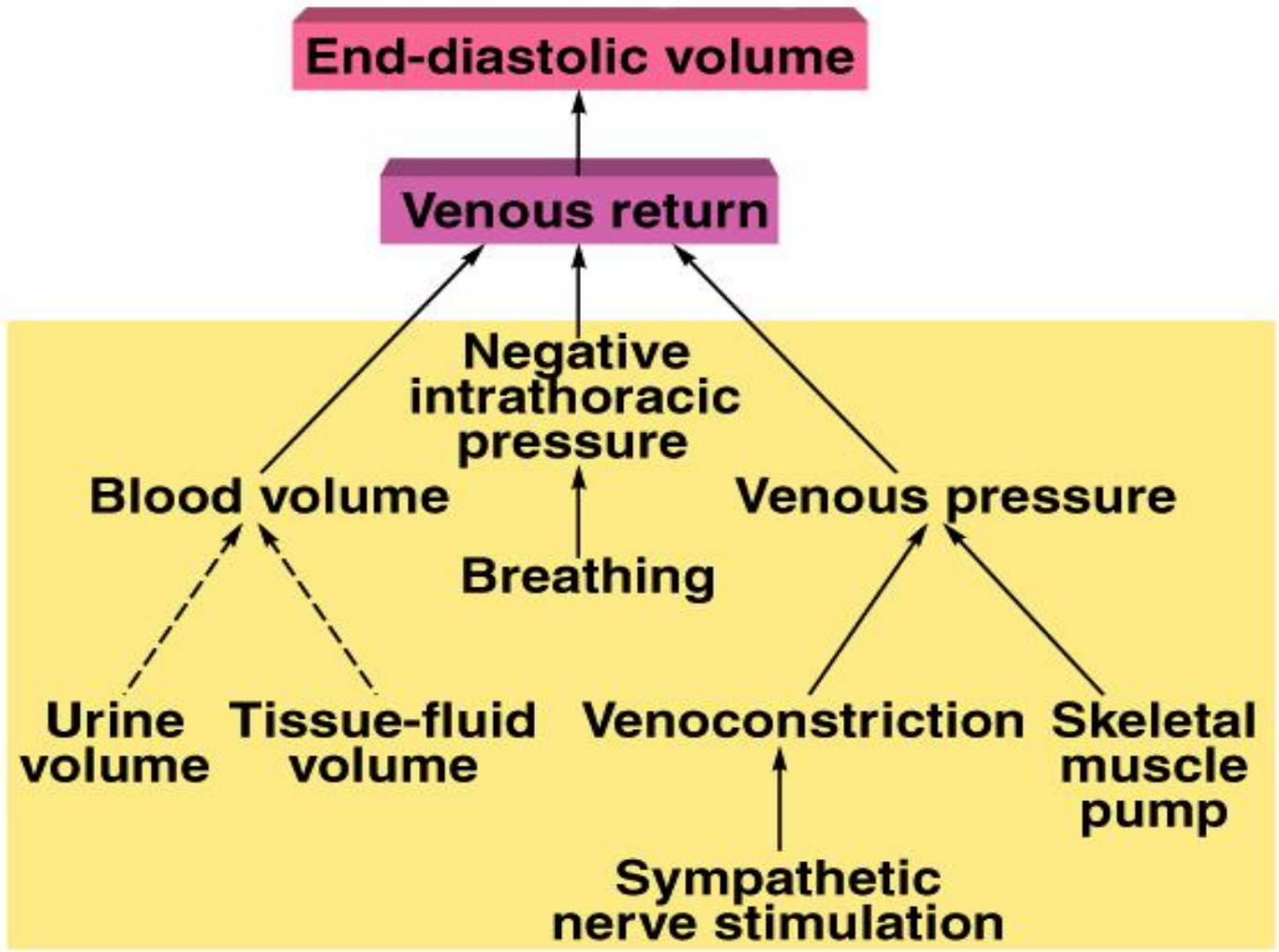
3- Venoconstriction

caused by sympathetic NS

4-The presence of valves in the large veins

5- Skeletal muscle pumps.

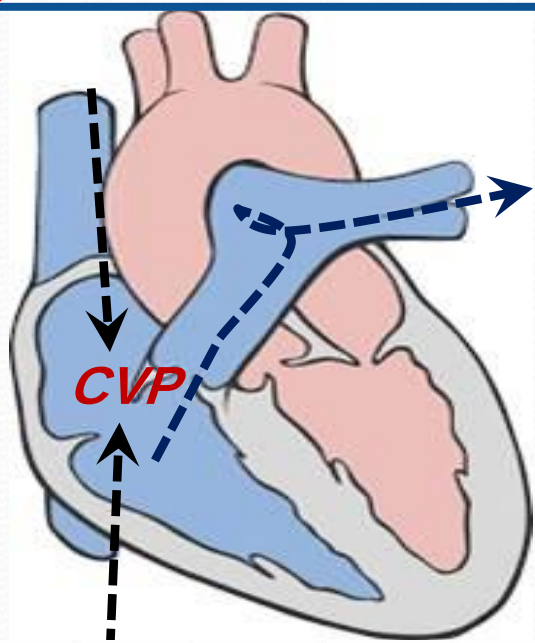
6- Respiratory activity.

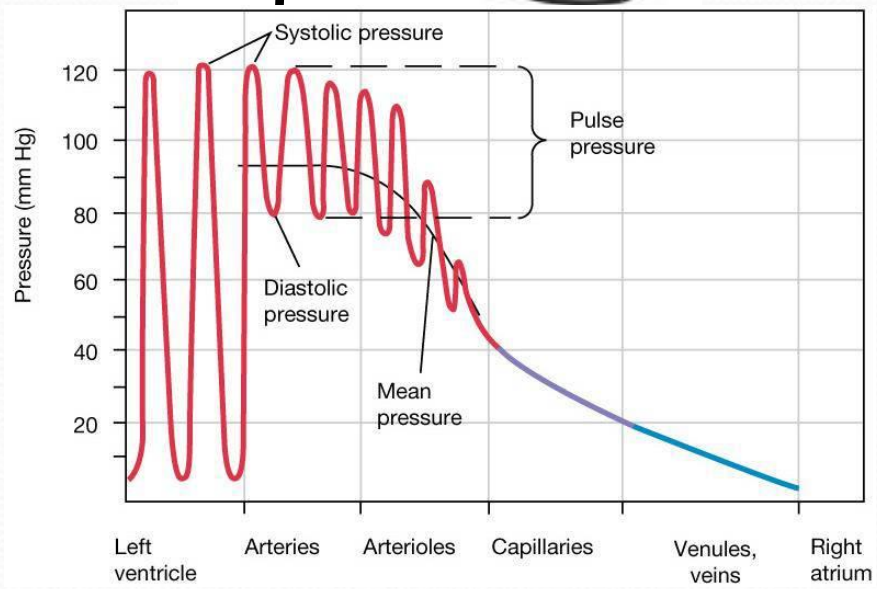


Factors Affecting Venous Return

1- Pressure gradient.

- ↑Pressure gradient → ↑ venous return.
- Since the Rt atrium is the site of venous blood collection from all around the body → the pressure inside the Rt atrium i.e. ***right atrial pressure (RAP)*** is called ***central venous pressure (CVP)***
- The pressure is highest in large arteries and continue to drop throughout the pathway, reaching ≈ zero at ***right atrium***.
- The high pressure in the arteries force the blood to continually move into areas where the pressure is lower.

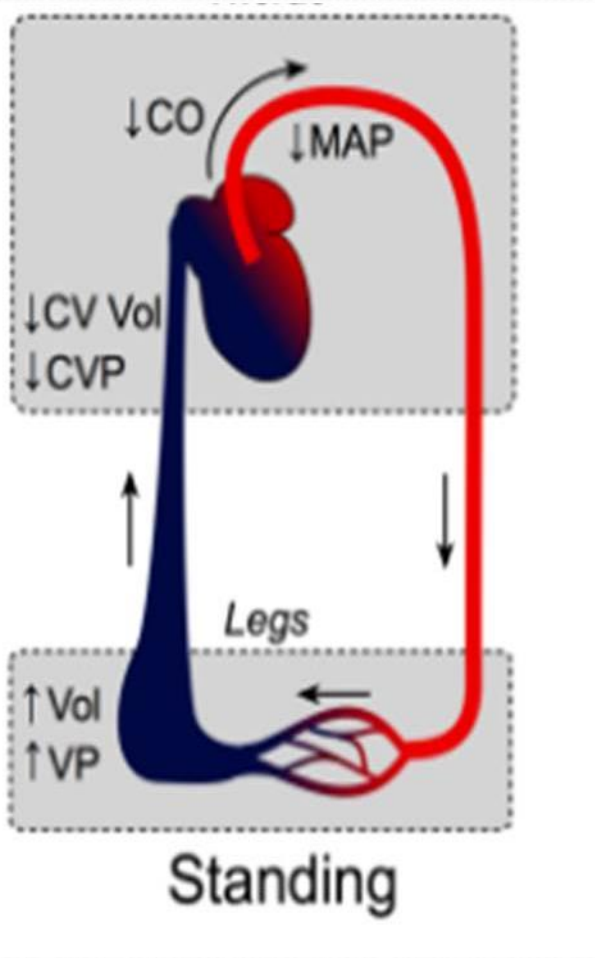




Factors Affecting Venous Return. . . (Cont.)

2- Gravity → ↓ venous return.

- Standing:
  - When a person initially stands, right atrial pressure and ventricular EDV falls, which decreases stroke volume by the Frank-Starling mechanism. So, cardiac output and arterial pressure decrease.
  - The flow through the entire systemic circulation falls because arterial pressure falls, therefore the pressure gradient driving flow throughout the entire circulatory system is decreased.



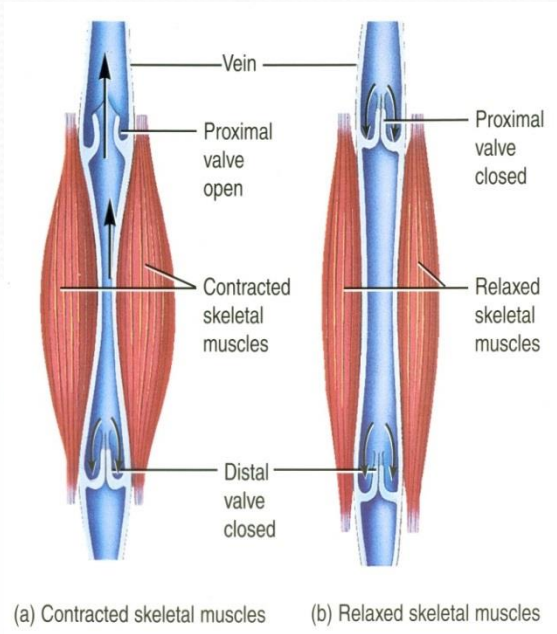
Factors Affecting Venous Return. . . (Cont.)

3- **Venoconstrcition** by sympathetic stimulation ↑ venous return

4- **The presence of valves** in the large veins.

5- **The skeletal muscle pump**:

Rhythmical contraction of limb muscles (as occurs during walking, running or swimming) → ↑ venous return by the muscle pump mechanism that squeeze the blood vessels between muscle fibers.

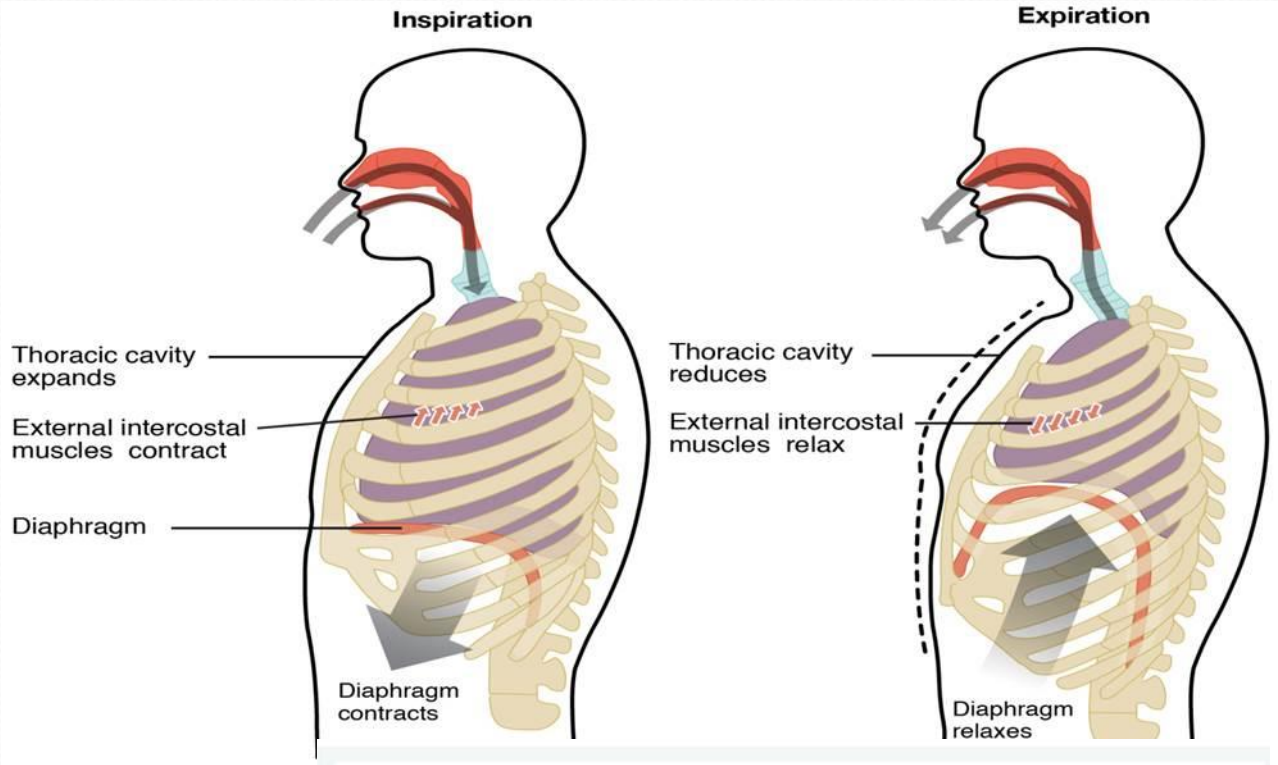


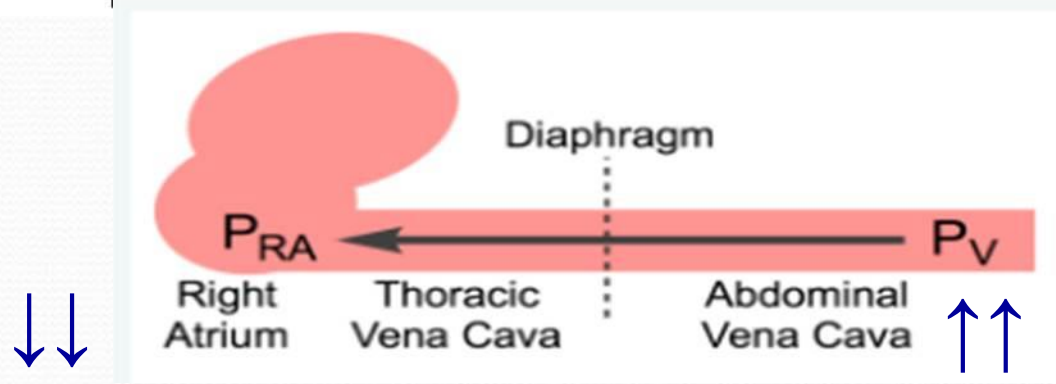
Skeletal muscle pump enhancing venous return

Factors Affecting Venous Return…(Cont.)

6- ***Respiratory activity:***

- Inspiration → ↑ venous return because of a decrease in right atrial pressure.
- In Valsalva maneuver (forceful expiration against a closed glottis), intrapleural pressure become positive which is transmitted to the large veins in the chest → ↓ venous return.





Determinants of the CO…..Cont.

CO = SV X HR

{End-diastolic volume (EDV) – end-systolic volume (ESV)}

CO = {EDV-ESV} X HR

Any factor that affects these parameters will affect the CO

End-Systolic Volume (ESV)

- ***ESV*** = The volume of blood remaining in the ventricle at the end of systole.
- ↑ End-Systolic Volume (ESV) → ↓ stroke volume
- ↓ End-Systolic Volume (ESV) → ↑ stroke volume
- ***ESV is determined by:***
  1. **Cardiac contractility**
  2. ***Afterload***

Determinants of ESV

1- Cardiac contractility:

- ↑↑ contractility → ↓↓ ESV → ↑↑ SV
- ↓↓ contractility → ↑↑ ESV → ↓↓ SV

- **N.B** ***Revise Factors affecting Cardiac Contractility (Inotropic Effectors) in the lectures : "Contractile mechanism in cardiac muscle "(Slide 27-31)***

Determinants of ESV….Cont.

2- Afterload:

- It is expressed as tension which must be developed in the wall of ventricles during systole, i.e the load the heart needs to overcome to open the semilunar valves and eject blood to aorta /pulmunary artery.

- As afterload increases, stroke volume decreases.
- Afterload increases by any factor that restricts arterial blood flow like:

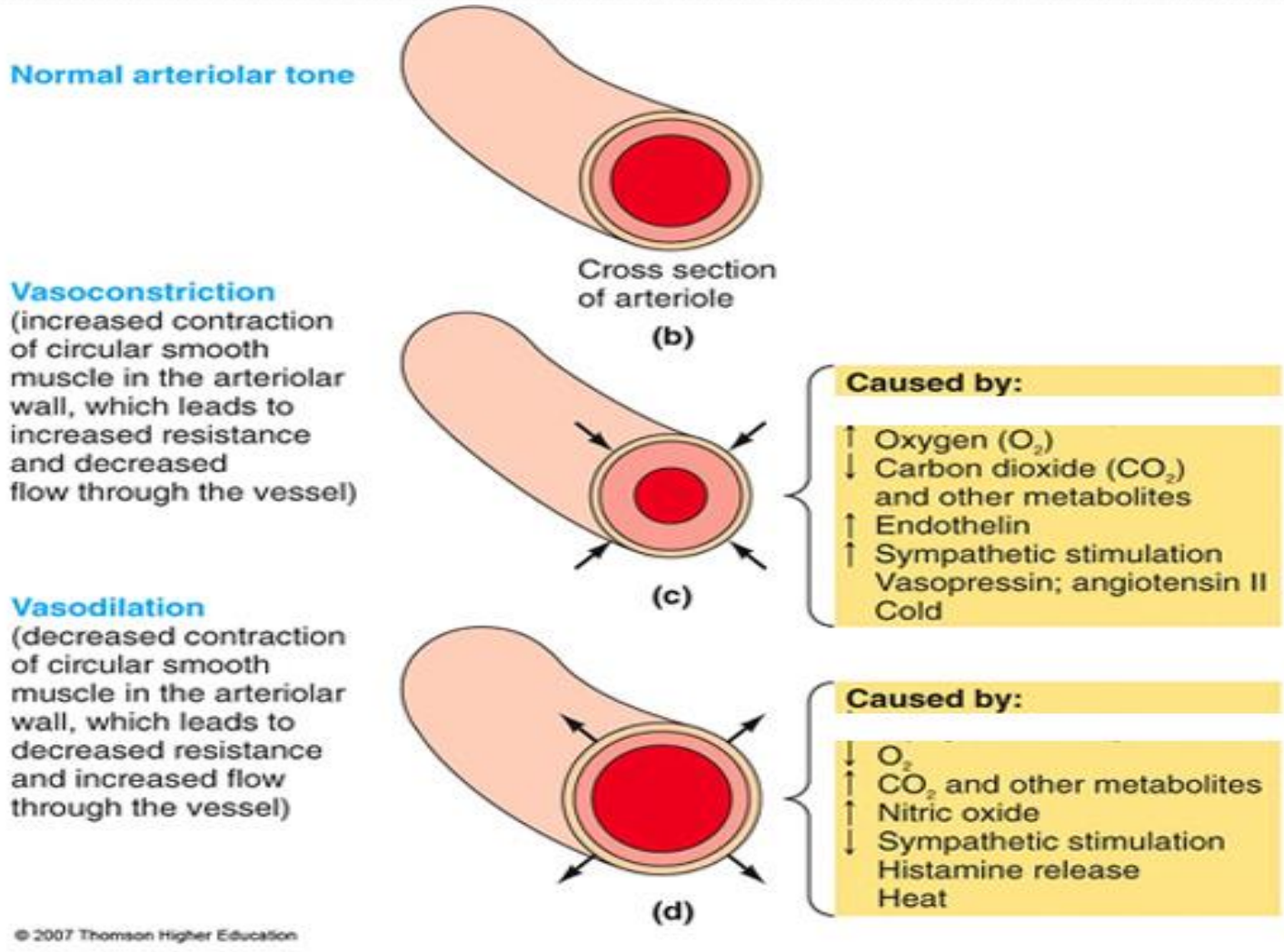
1- Increased arterial blood pressure.

2- Vasoconstriction (the peripheral vascular resistance).

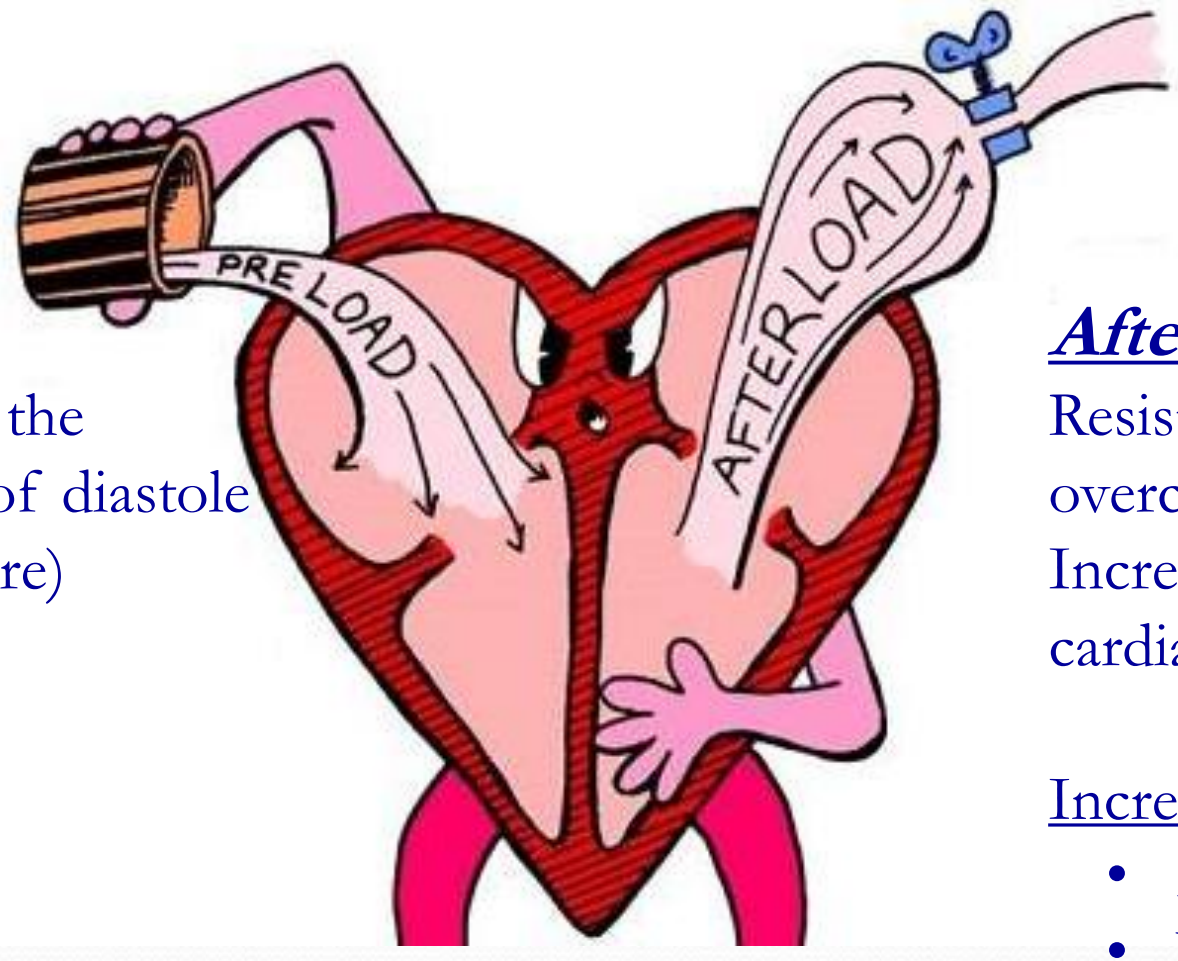
- ↑↑ resistance → ↑↑ ESV
- ↓↓ resistance → ↓↓ ESV

Determinants of ESV (…Cont.)

Afterload and hence ESV **is determined by the peripheral vascular resistance**



What Are Preload and Afterload



Preload

Volume of blood in the ventricle at the end of diastole (end diastolic pressure)

Increased in:

- Hypervolemia
- Heart failure

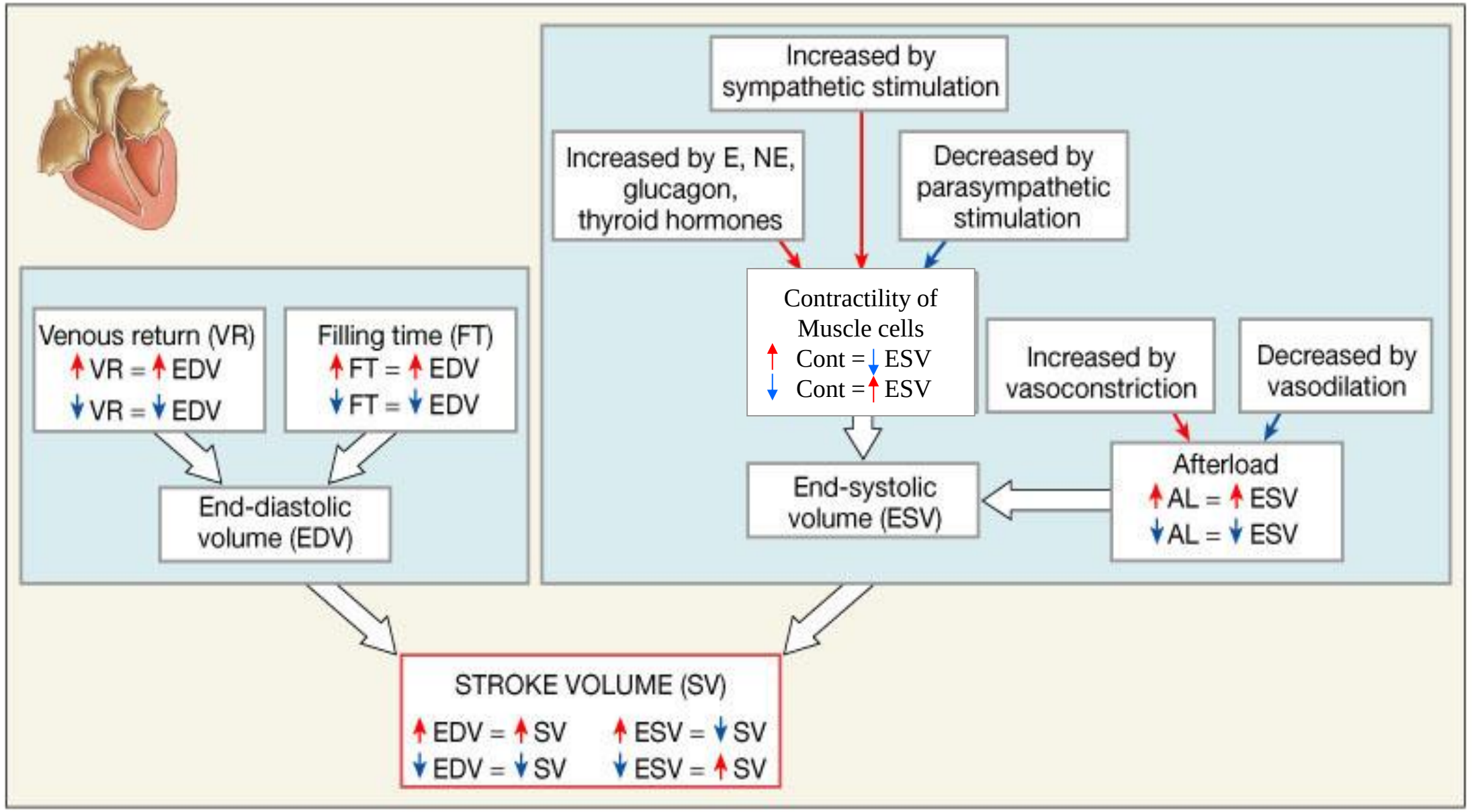
Afterload

Resistance left ventricle must overcome to circulate blood. Increased afterload=Increased cardiac workload

Increased in:

- Hypertension
- Vasoconstriction

Summary of the Factors Affecting Stroke Volume



Determinants of the CO…..Cont

CO = SV X HR

{End-diastolic volume (EDV) – end-systolic volume (ESV)}

CO = {EDV-ESV} X HR

Any factor that affects these parameters will affect the CO

The Heart Rate (HR)

- Normal heart rate = 60-100 beats/min

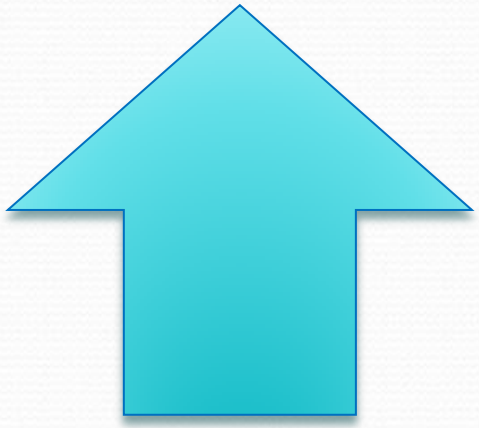
- > 100 beats/min → Tachycardia
- < 60 beats/min → Bradycardia

- Normal heart rate is regular sinus rhythm.

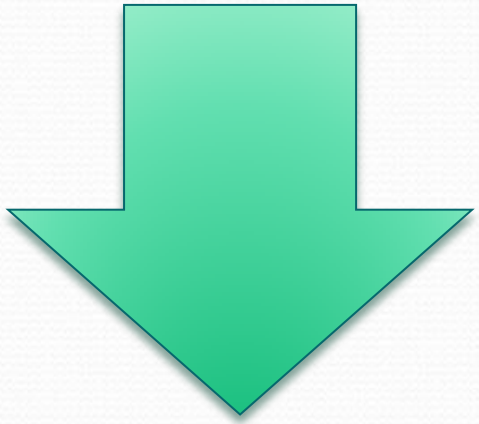
Regulation of heart rate

1- **Autonomic nervous system**

- Sympathetic nerves increase HR as occurs during stress, crisis or low blood pressure



Sympathetic Stimulation



Vagal (Parasympathetic) Stimulation

- Parasympathetic nerves (vagus nerve) slow HR.

Under normal conditions the SA node is under continuous vagal tone causing its basal firing rate to decrease (Vagal Tone)

Regulation of heart rate…..Cont.

2- **Physical factors:**

- Age: Resting HR is faster in fetus and then gradually decreases throughout life.
- Gender: HR is faster in females (72-80 beats/min) than in males (64-72 beats/min).
- Temperature: Heat increases HR as occurs in high fever. Cold has the opposite effect.
- Exercise: Increases HR through sympathetic nervous system.

3- **Hormones and drugs**

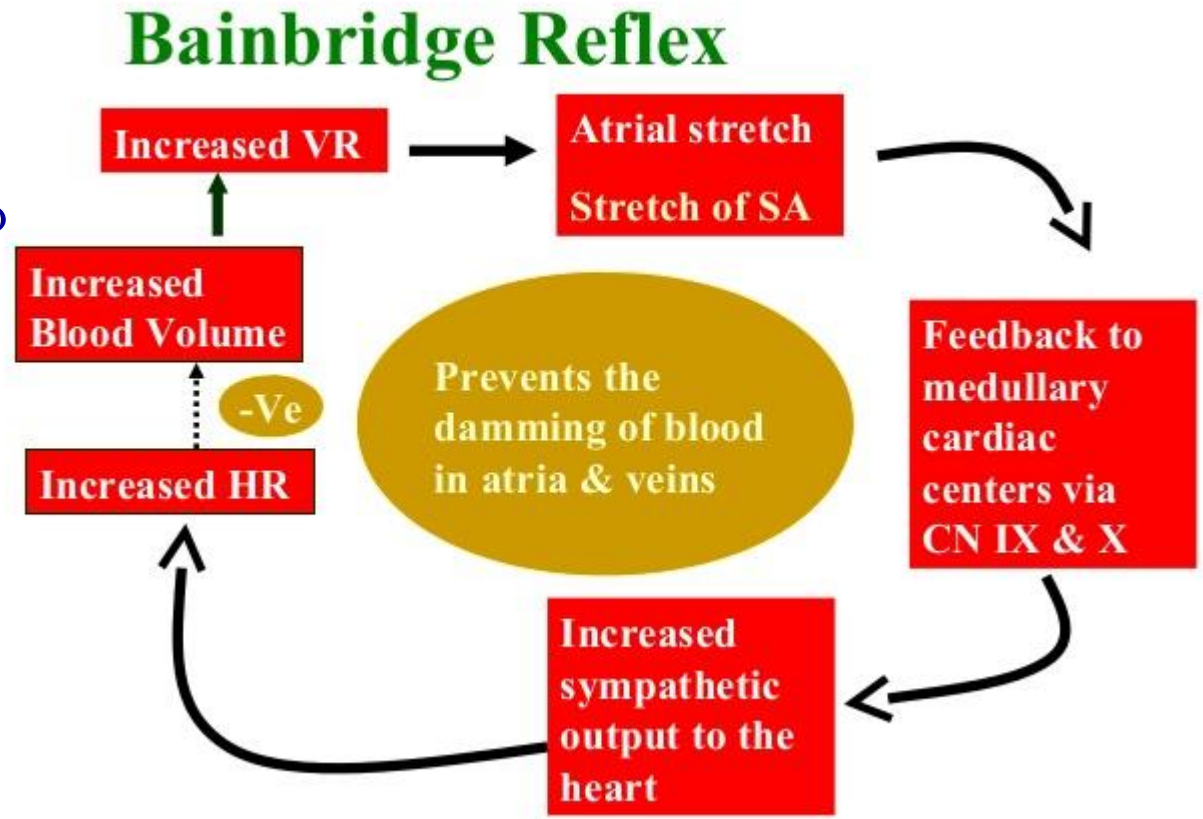
- Epinephrine and thyroxin increase HR.
- Increased calcium level in blood causes prolonged contraction
- Reduced calcium level in blood depress the heart.

Regulation of heart rate…..Cont.

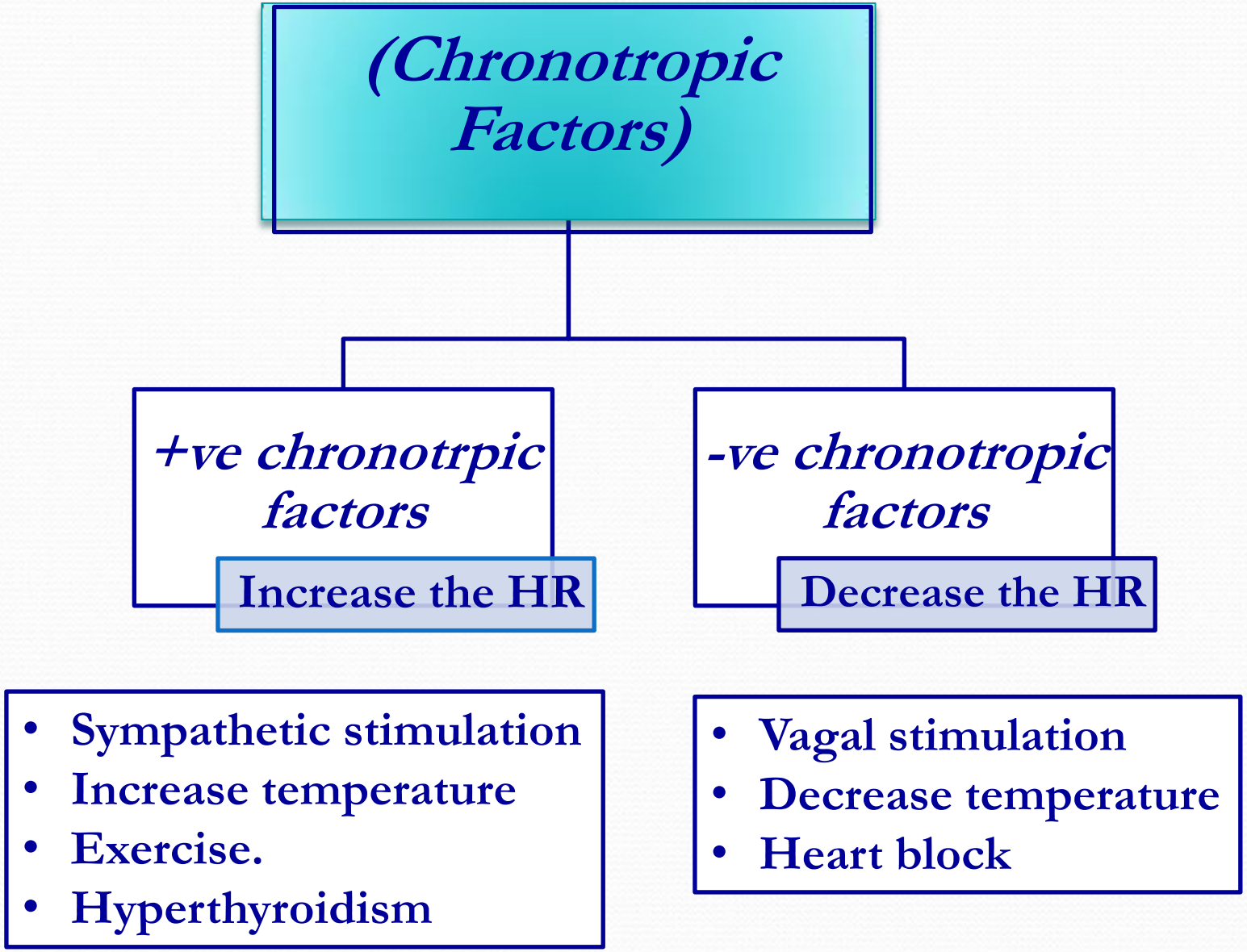
4- Blood volume

Atrial Reflex (Bainbridge reflex)

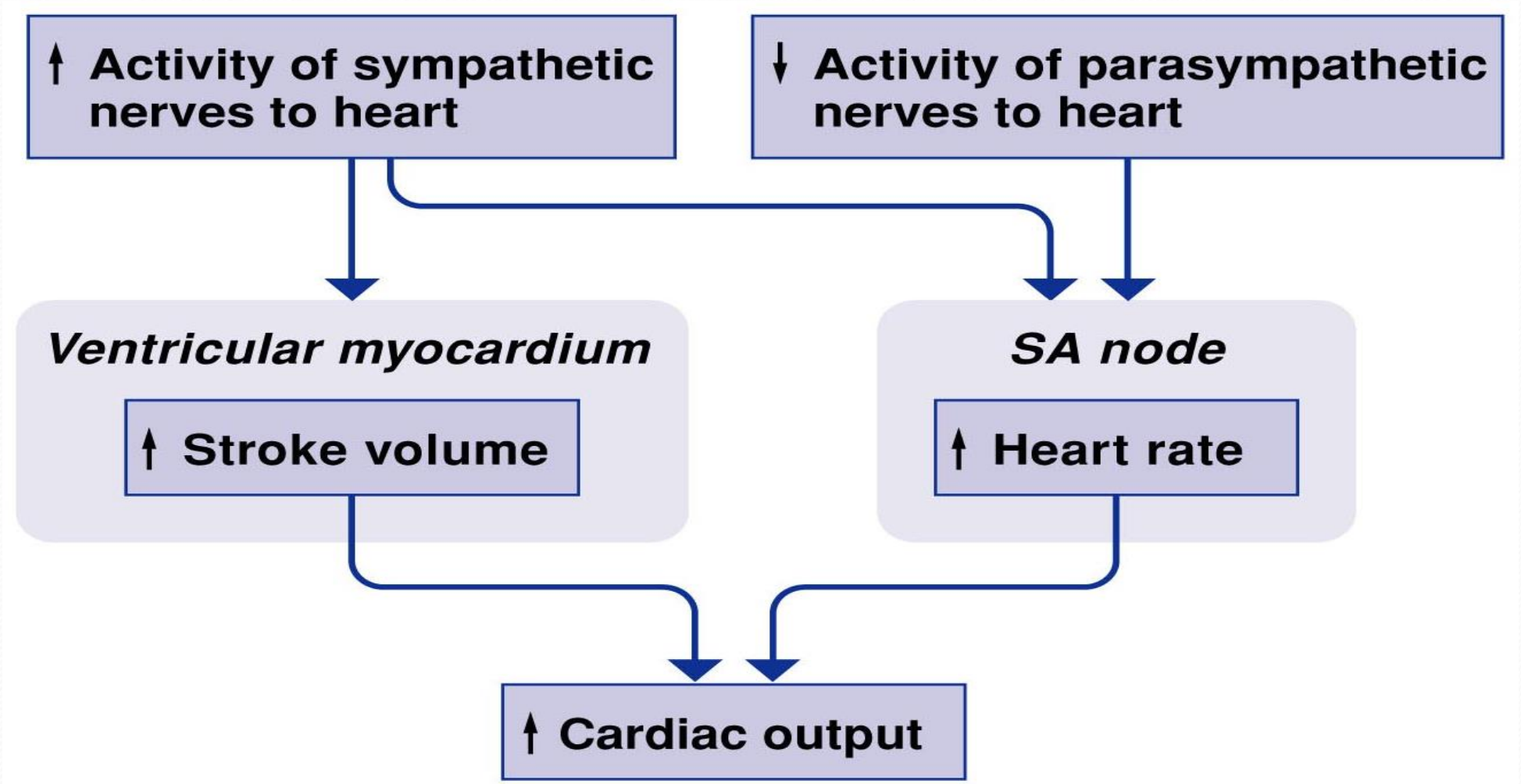
- This reflex adjusts heart rate in response to venous return.
- Increase blood volume, stimulates stretch receptors in right atrium.
- This triggers increase in heart rate through increased sympathetic activity.



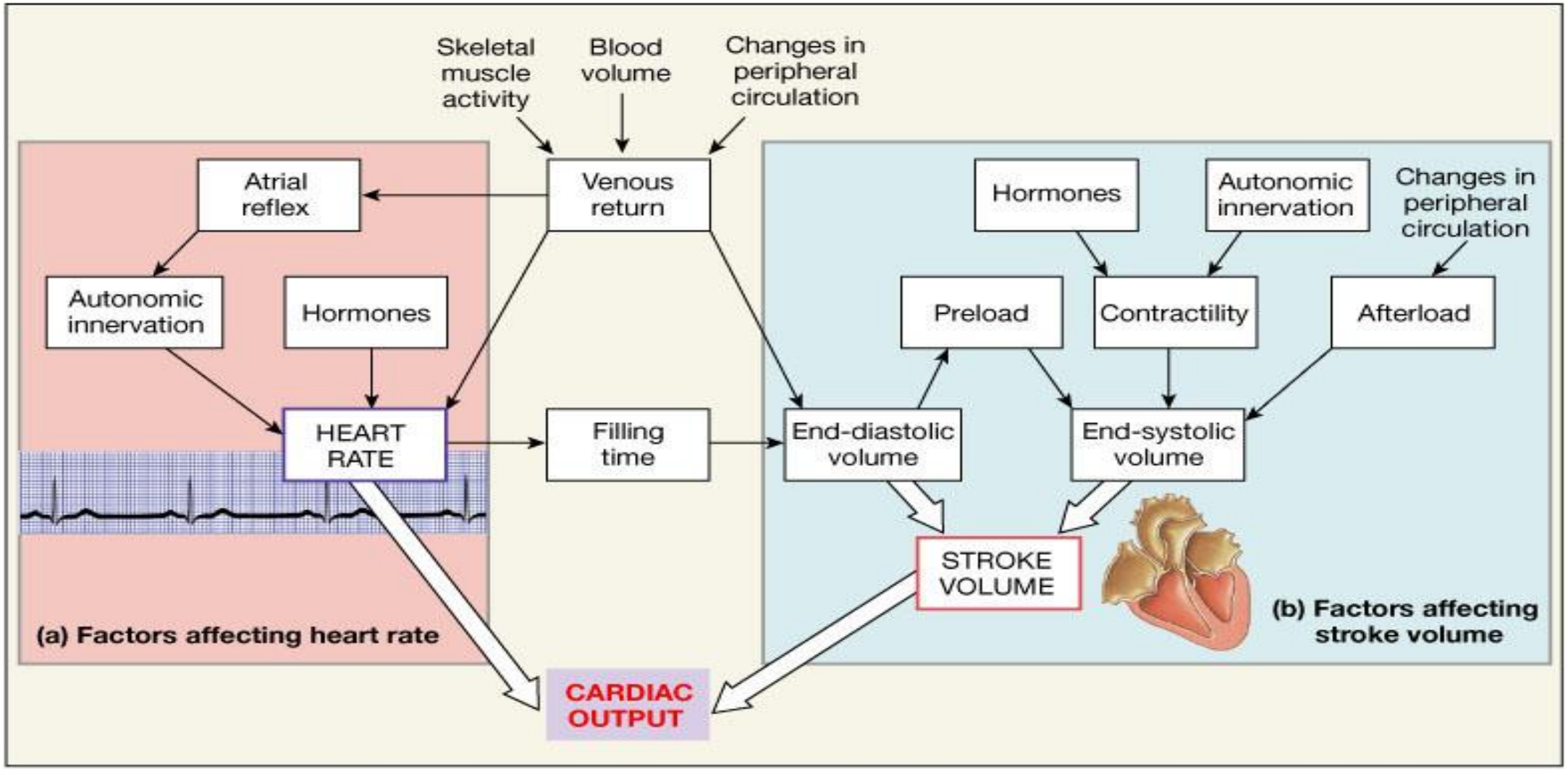
Factors Affecting the HR (Chronotropic Factors)



Regulation of Cardiac Output by Autonomic Nerves



Summary of the Factors Affecting Cardiac Output



Measurement Of C.O.

The Direct Fick's Method:

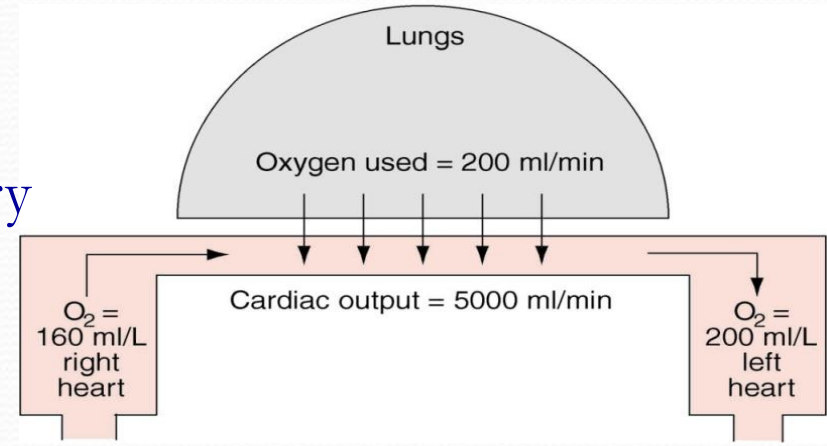
It states that, the amount or volume of any substance taken up by an organ or by the whole body is equal to:

(The arterial level of the substance – its the venous level) X blood flow.

$$\text{Blood flow (CO)} = \frac{\text{Amount of the substance } (O_2 \text{ consumption ml/min})}{\text{(Arteriovenous O2 difference ml/L)}}$$

Methods for measuring cardiac output (The Fick's principle)

- In the steady state, the rate of O_2 consumption by the body must equal the amount of O_2 leaving the lungs in the pulmonary vein minus the amount of O_2 returning to the lungs in the pulmonary artery.
- The total O_2 consumption, is simply the difference between the inspired and expired O_2. It can be measured with an exhaled gas collection bag using any oxygen meter.
- The amount of O_2 in the pulmonary veins= pulmonary blood flow (CO) X the O_2 content of pulmonary venous blood.
- Likewise, the amount of O_2 returned to the lungs via the pulmonary artery = pulmonary blood flow (CO) X the O_2 content of pulmonary arterial blood.
- O_2 consumption = CO× [O_2] pulmonary vein – CO× [O_2] pulmonary artery
- O_2 consumption = CO X [O2] pulmonary vein- [O2] pulmonary artery
- CO= O_2 consumption per minute/arteriovenous O_2 difference




Thank You!