Cardiovascular Physiology

Cardiac Output: Stroke Volume & Venous Return

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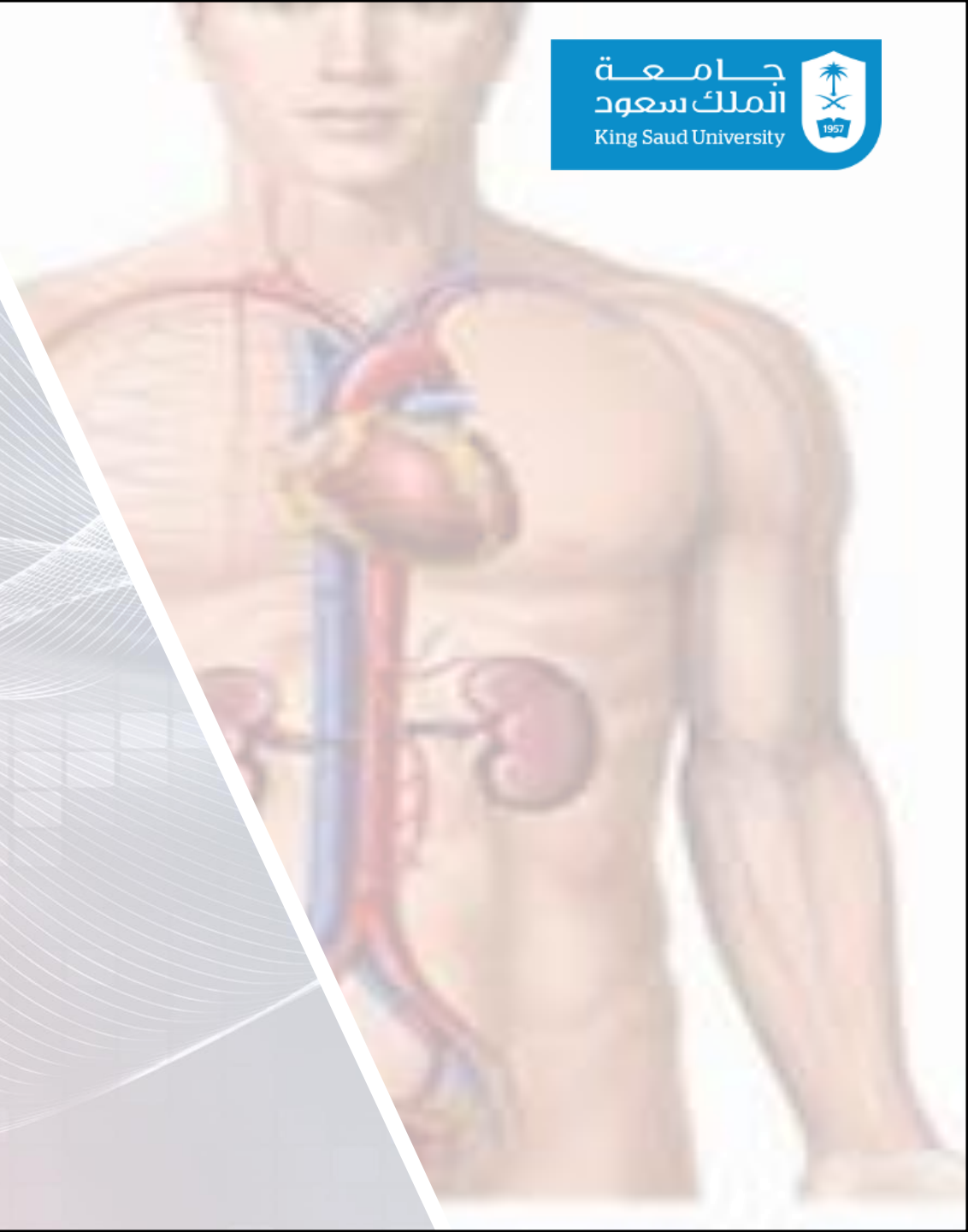


Cardiac Output & Venous Return

Lecture Outcomes

- Define Cardiac Output & its normal values.
- Define stroke volume, End- systolic volume, & end- diastolic volume.
- Identify factors affecting & determining Cardiac Output.
- Role of stroke volume & heart rate regulation on Cardiac Output regulation.
- Understand the role of venous return on Cardiac Output.
- Understand factors affecting the EDV (venous return) & the end- systolic volume (ESV).

Terminologies to Define

- **End-diastolic volume (EDV):**
  - Volume of blood in the ventricles at the end of diastole.
  - ≈ 110-130 mL.
- **Stroke volume (SV):**
  - Amount of blood ejected from ventricles per beat.
  - ≈ 70 - 80 mL/beat.

Terminologies to Define…

- **End-systolic volume (ESV):**
  - Amount of blood left in ventricles at the end of systole.
  - ≈ 40-60 mL.
- **Ejection fraction (EF):**
  - Is the percentage (Fraction) of ventricular EDV which is ejected with each stroke (beat).
  - It is a good index of ventricular function.
  - = SV or (EDV – ESV)/EDV X 100
  - ≈ 60-65 %

Cardiac Output (CO)

Cardiac Output (CO) is the volume of blood pumped by the ventricle per minute

- **CO** is expressed in L/min.
- **At rest, CO is 4-6 L/min (≈ 5 L/min),**
  in healthy adults at rest when the HR = 70 bpm.
- Since normal total adult resting blood volume ≈ 5 L, the blood volume circulates through the body once each minute.

Factors Affecting (CO)

- **Sex** … F (≈ 4.9 L) < M (≈ 5.6 L).
- **Age** … Children < adults.
- **Height & weight**: Body mass index.
- **Body metabolism**.
- **Sympathetic activity**.
- **Exercise** … moderate ↑ CO 2-4 folds of resting (up to 20-25 L/min.)
  athletes ? ↑ CO 7 times of resting (up to 35-40L/min.)
- **Pregnancy**: CO ↑ by > 30% , due to ↑ uterine blood flow.
- **Hyperthyroidism**: Thyrotoxicosis. CO ↑
- ↑ **Body temperature** ↑ CO.

Cardiac Index

- It relates the cardiac output (CO) to the body surface area.
- CO/min/m^2

Cardiac index is relating the heart performance to the size of the individual.

Cardiac Reserve

- During exercise, the CO can increase up to 20-25 L/min, & up to 35-40 L/min in well trained athletes.

The difference between resting CO & maximum volume of blood the heart capable of pumping per minute is known as the cardiac reserve.

Cardiac Output (CO)

- The amount of blood pumped by the ventricle per beat is the Stroke Volume ≈ 70 mL/beat.
- The amount of blood pumped by the ventricle per minute is the CO, & will be equal to:

Cardiac Output = Stroke Volume X Heart Rate

Cardiac Output (CO)	Stroke Volume (SV)	Heart Rate (HR)
= output of ventricle/ minute	= output of ventricle/ beat SV=EDV-ESV	= beats/ minute
≈ 5 L/min (av. 4-6 L/min)	≈ 70 ml/beat (av. 70-80 ml/beat)	≈ 70-75 beats/min (N. 60-100 beats/min)

Cardiac Output (CO)

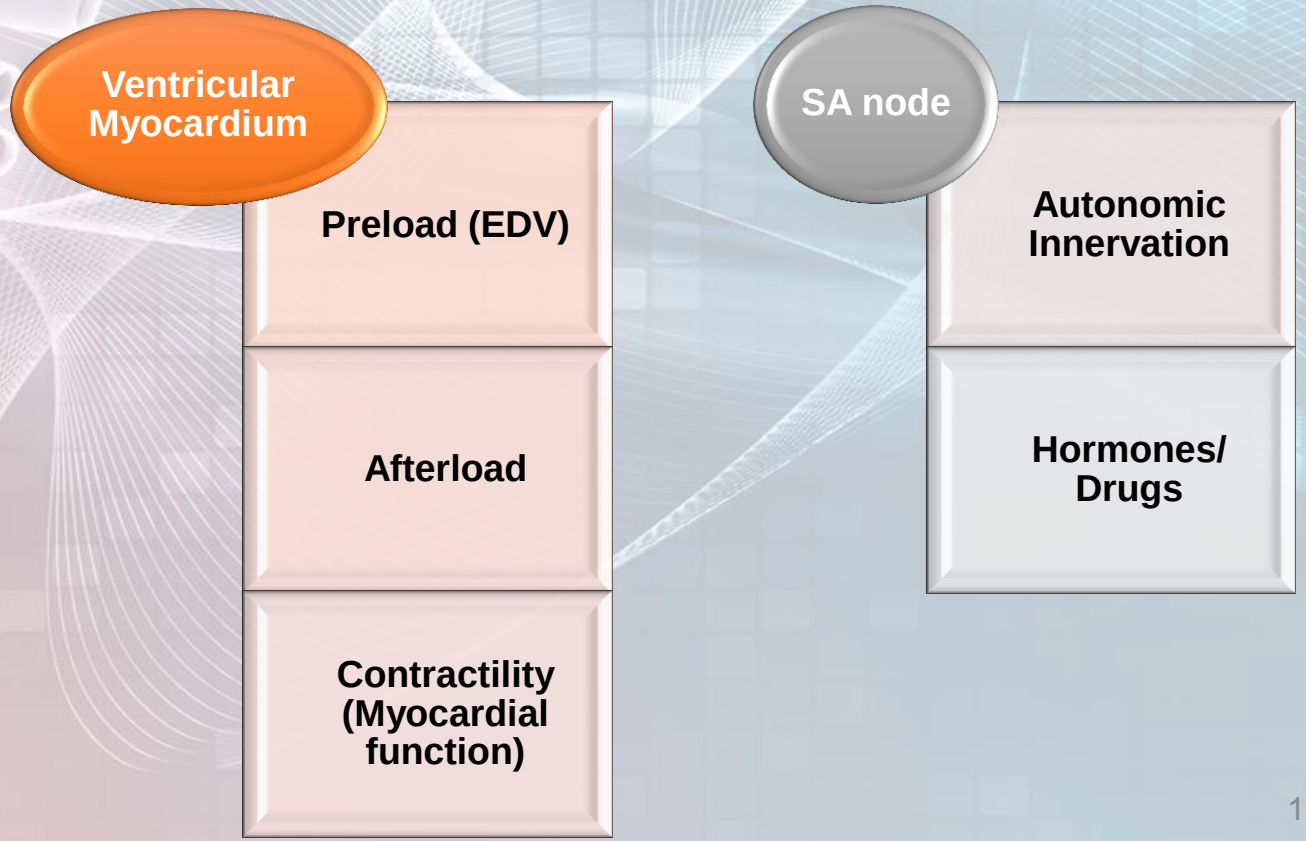
Cardiac Output = Stroke Volume X Heart Rate

- **CO is a function of:**
  - Heart rate.
  - Stroke volume.
- **CO is Determined by:**
  - The heart rate.
  - The stroke volume.

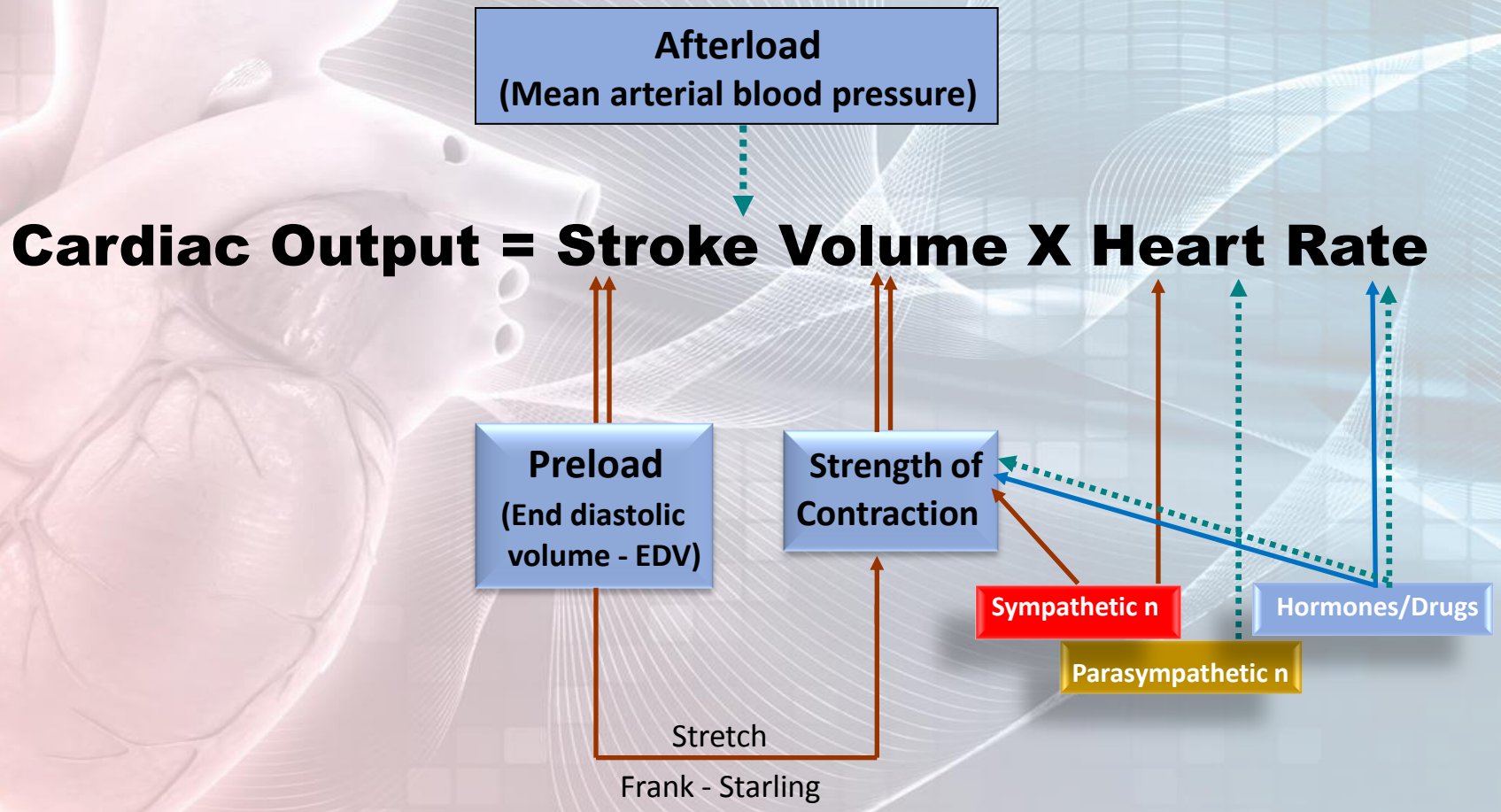
Variables Affecting Stroke Volume & Heart Rate

Cardiac Output = Stroke Volume X Heart Rate

- Any factor that affects these parameters will affect the CO.



Regulation of Cardiac Output

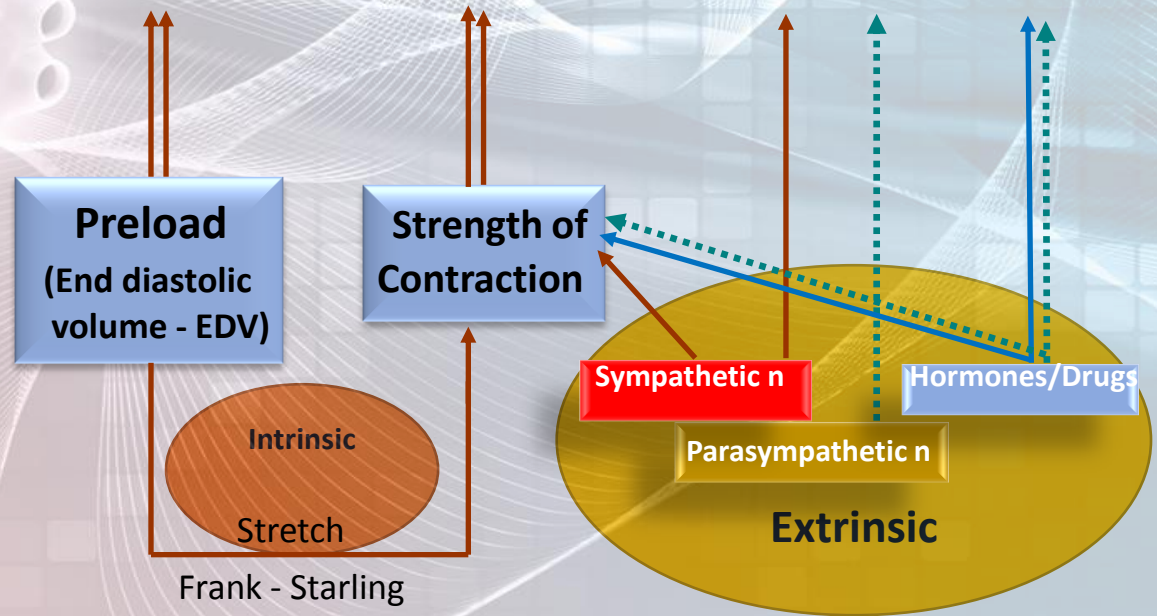


Starling's law of the heart – the more the cardiac muscle is stretched, the stronger the contraction

Regulation of Cardiac Output

Afterload
(Mean arterial blood pressure)

Cardiac Output = Stroke Volume X Heart Rate



Starling's law of the heart – the more the cardiac muscle is stretched, the stronger the contraction

SV

Stroke Volume

Stroke volume (SV) is determined by:

- End- diastolic volume.
- End- systolic 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)

SV

Stroke Volume

- Stroke volume is the amount of blood pumped (ejected) out of ventricle per beat.
- SV = EDV - ESV

CO = Stroke Volume X Heart Rate

CO = (EDV – ESV) X Heart Rate

SV

Stroke Volume: Regulation

- **Stroke volume (SV) is regulated by 3 variables:**

I: **EDV (preload):** Venous Return.

II: **Contractility (Strength of contraction):**

- Starling's law, &
- Sympathetic innervation.

III: **Total peripheral resistance:** afterload.

SV

Preload: (Venous Return)

- **Preload** (**venous return**) is the amount of blood returns to the heart, into the atria, from veins.

End- Diastolic Volume: (EDV)

- **End- diastolic volume** (EDV) is amount of blood presented to the ventricles from the venous return prior to ventricular ejection.
- **When venous return increases, EDV increases & stretches or lengthens the ventricular muscle fibers.**

SV

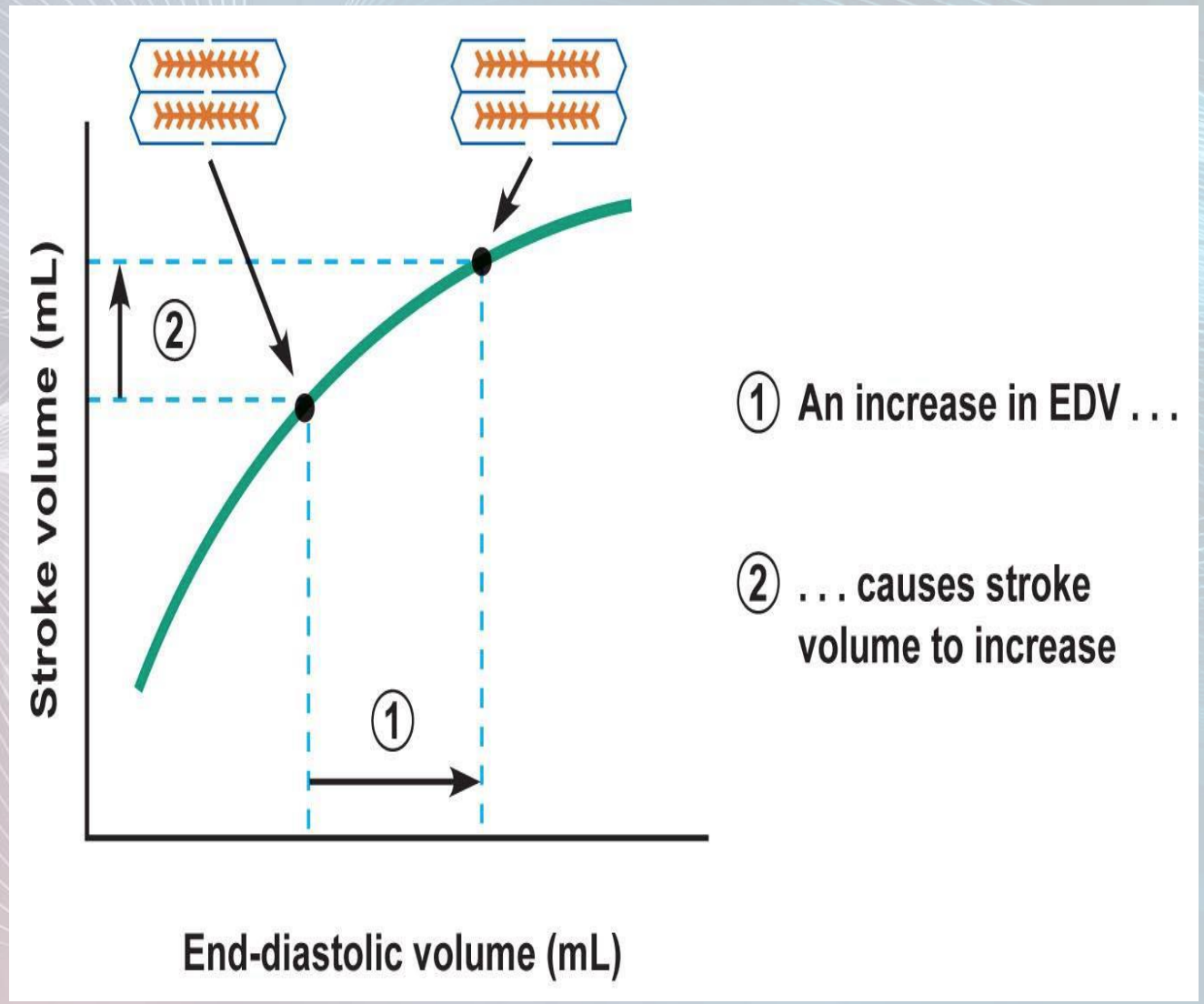
How Does the EDV affect the SV & CO ?
Frank-Starling Mechanism

- **An increase in the End- diastolic volume** (EDV) amount will increase the myocardial fibers stretch, thus increasing the initial fiber length.
- **The increase in the initial fiber length will increase the strength of myocardial contractility.**

SV

Frank-Starling Law

- 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.



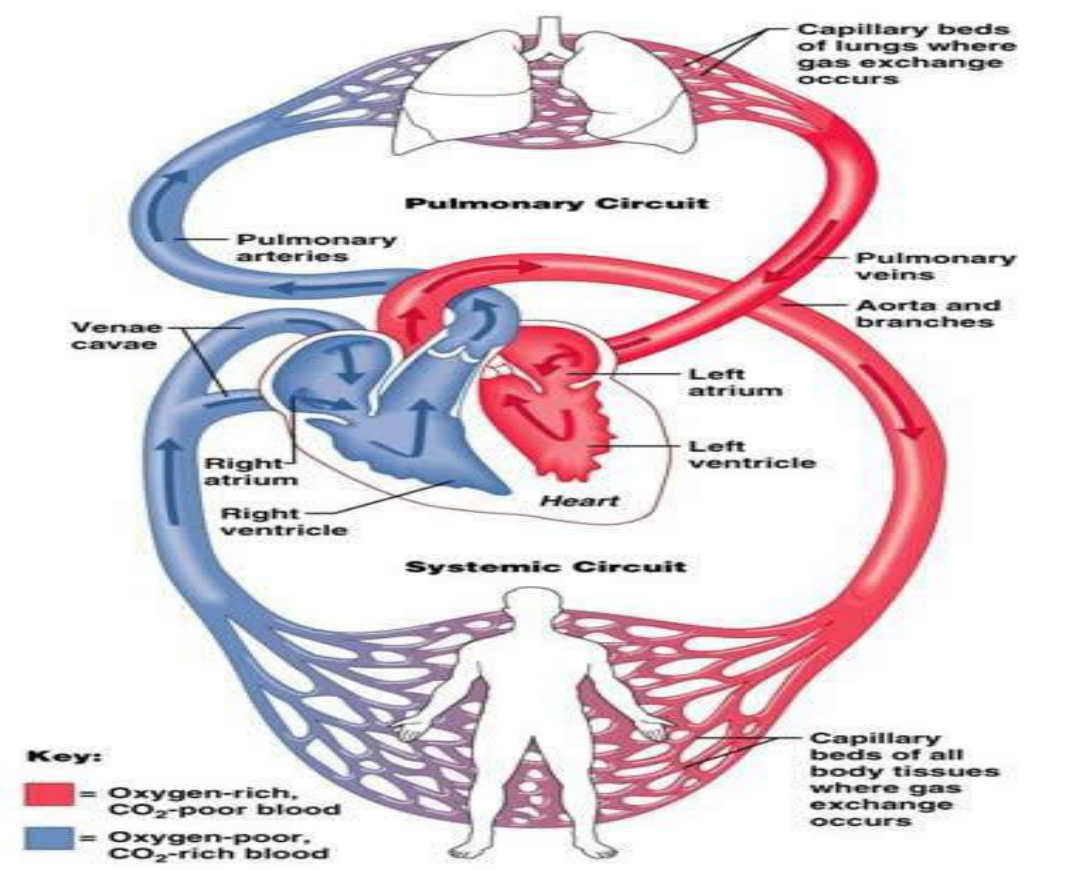
SV

Factors Affecting Venous Return (Preload)

1. Total blood volume.
2. Pressure gradient.
3. Gravity.
4. Veno-constriction: sympathetic nervous system.
5. The presence of valves in the large veins.
6. Skeletal muscle pumps.
7. Respiratory activity (breathing).
8. Right atrial pressure.
9. Muscular contractility of the heart.

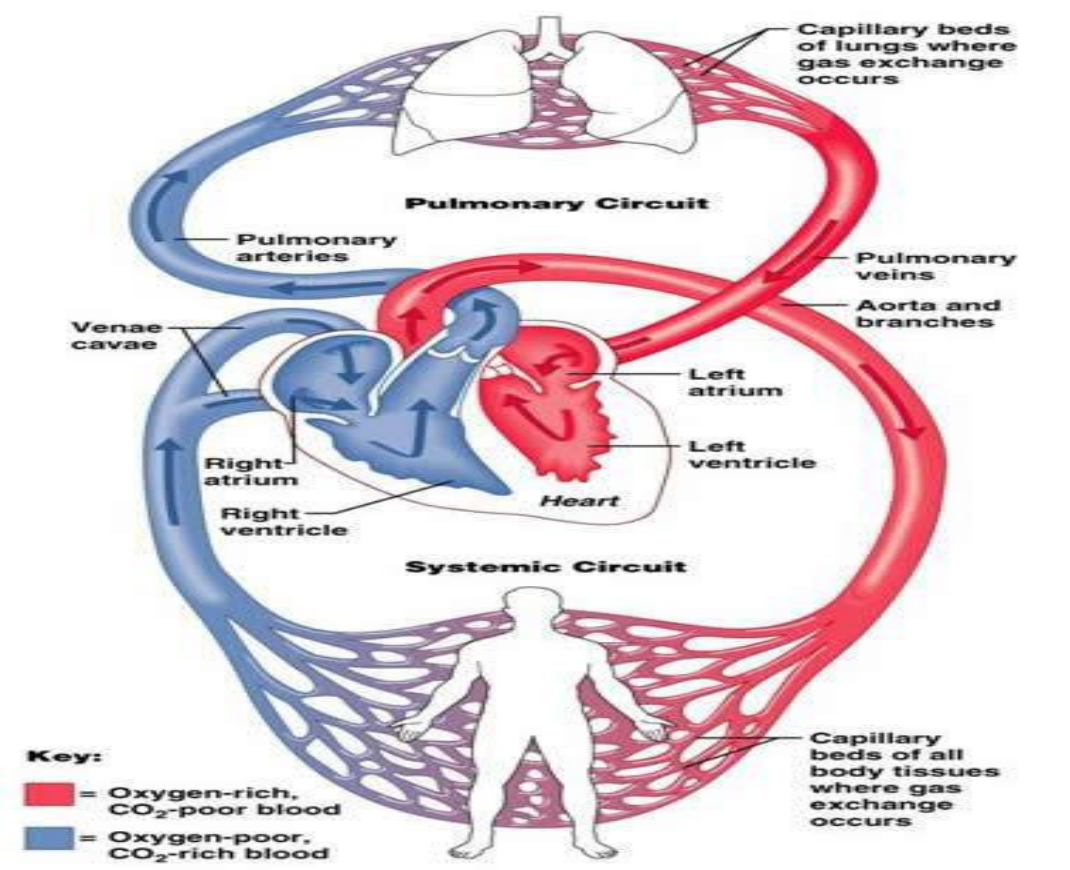
SV

Factors Affecting Venous Return (Preload)



SV

Factors Affecting Venous Return (Preload)

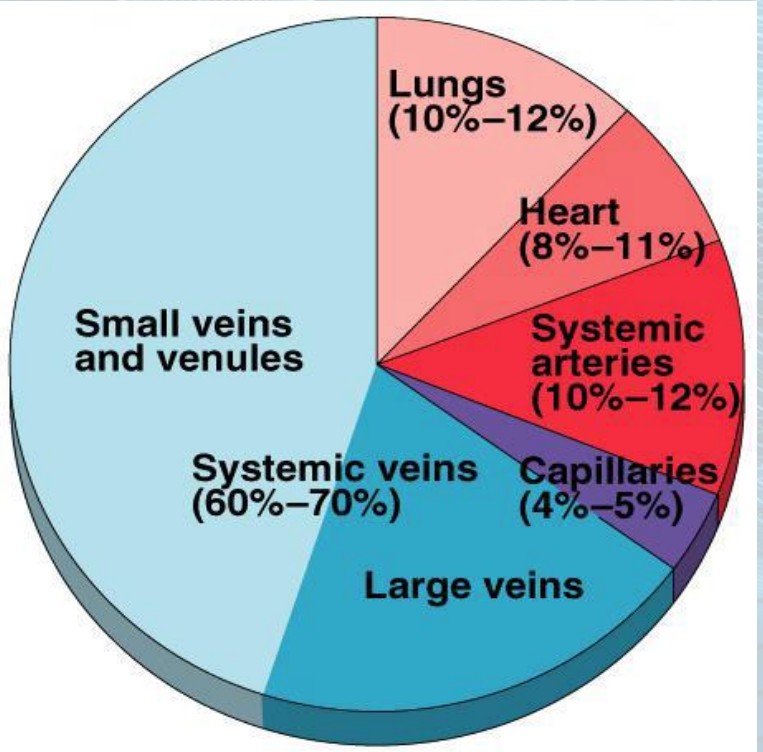


SV

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.



SV

Factors Affecting Venous Return (Preload)

Blood volume:

- At constant venous capacity, as the blood volume ↑→ the Mean Circulatory Pressure ↑→ ↑ VR.
- At constant venous capacity, as the blood volume ↓→ the Mean Circulatory Pressure ↓→ ↓ VR.

- **Venous capacity** is the volume of blood that the veins can accommodate.

SV

Factors Affecting Venous Return (Preload)

Pressure Gradient:

- ↑Pressure gradient → ↑ venous return.
- Since the right atrium is the site of venous blood collection from all around the body → the pressure inside the right atrium i.e. **right atrial pressure (RAP**) is called **central venous pressure (CVP)**
- The pressure is highest in large arteries & continue to drop throughout the pathway, reaching ≈ zero-2 mmHg at **right atrium**.
- The high pressure in the arteries 120 mmHg forces the blood to continually move into areas where the pressure is lower.

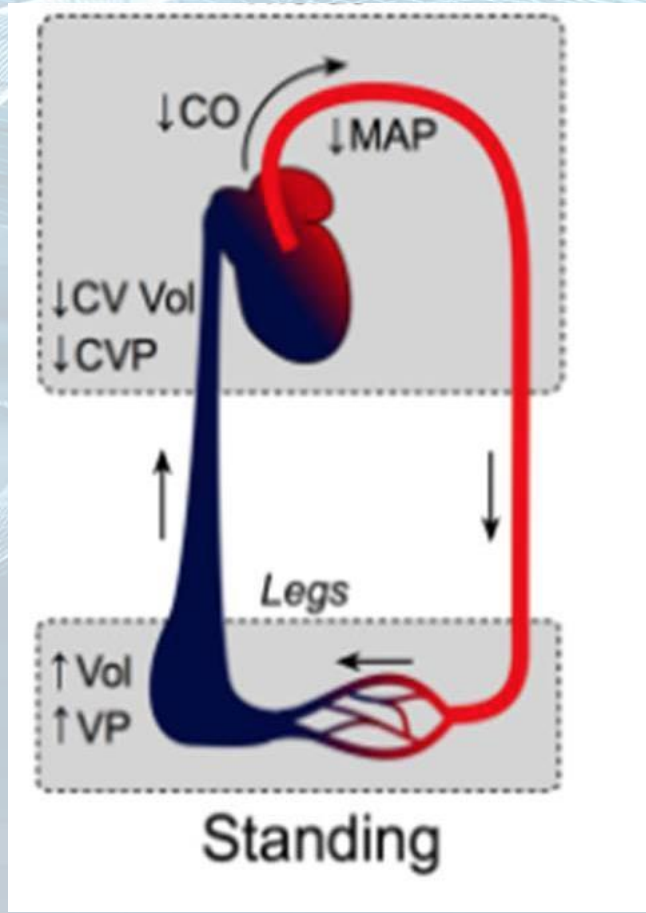
SV

Factors Affecting Venous Return (Preload)

Gravity:

Standing:

- When a person initially stands, right atrial pressure & ventricular EDV falls, which decreases stroke volume by the Frank-Starling mechanism. So, CO & 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.



SV

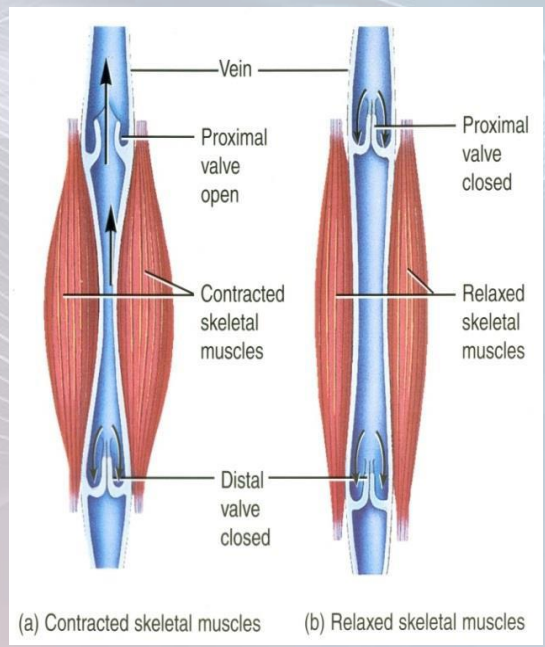
Factors Affecting Venous Return (Preload)

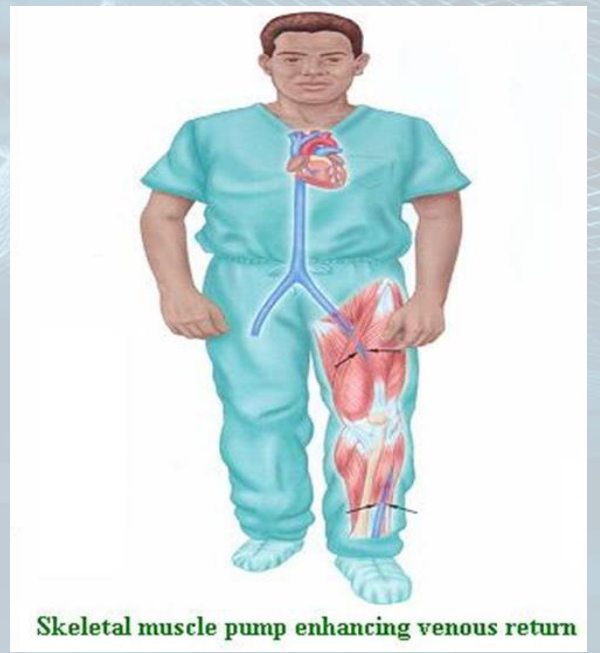
Venoconstriction: By sympathetic stimulation ↑ VR.

The presence of valves: Permit blood to move forward towards the heart & prevent it from moving back toward the tissues.

The skeletal muscle pump:

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



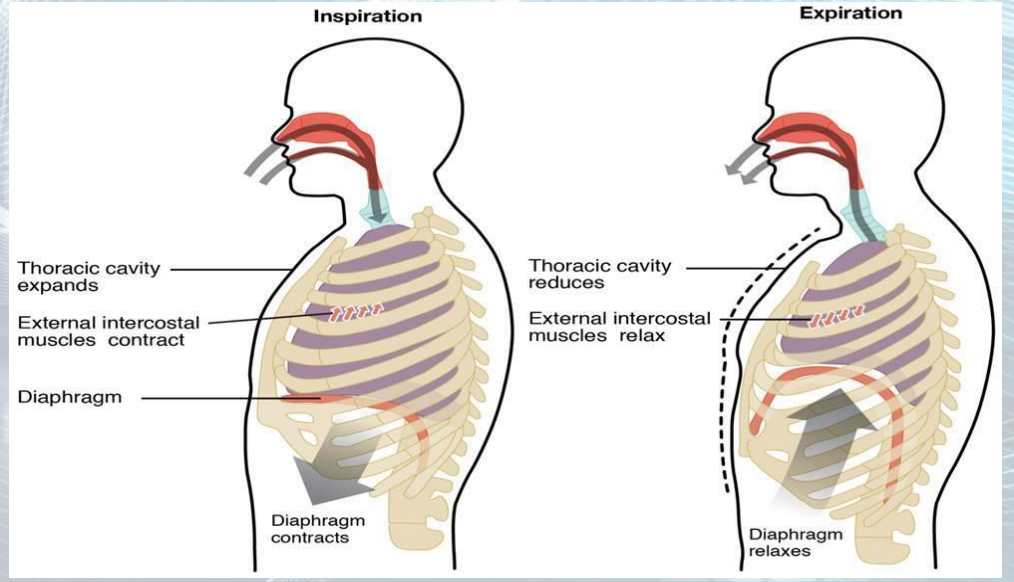

Skeletal muscle pump enhancing venous return

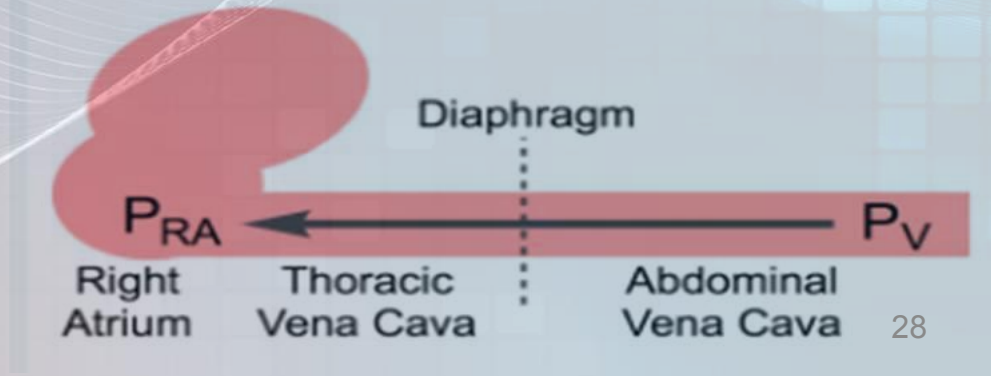
SV

Factors Affecting Venous Return (Preload)

Respiratory activity: Respiratory /thoracic pump.

- Inspiration → ↑ VR 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 → ↓ VR.





SV

Factors Affecting Venous Return (Preload)

Venous return (VR) is decreased when:

1. The right atrial pressure (RAP) is increased.
2. Pumping capability becomes diminished.
3. The nervous circulatory reflexes are absent.

SV

Venous Return Curve

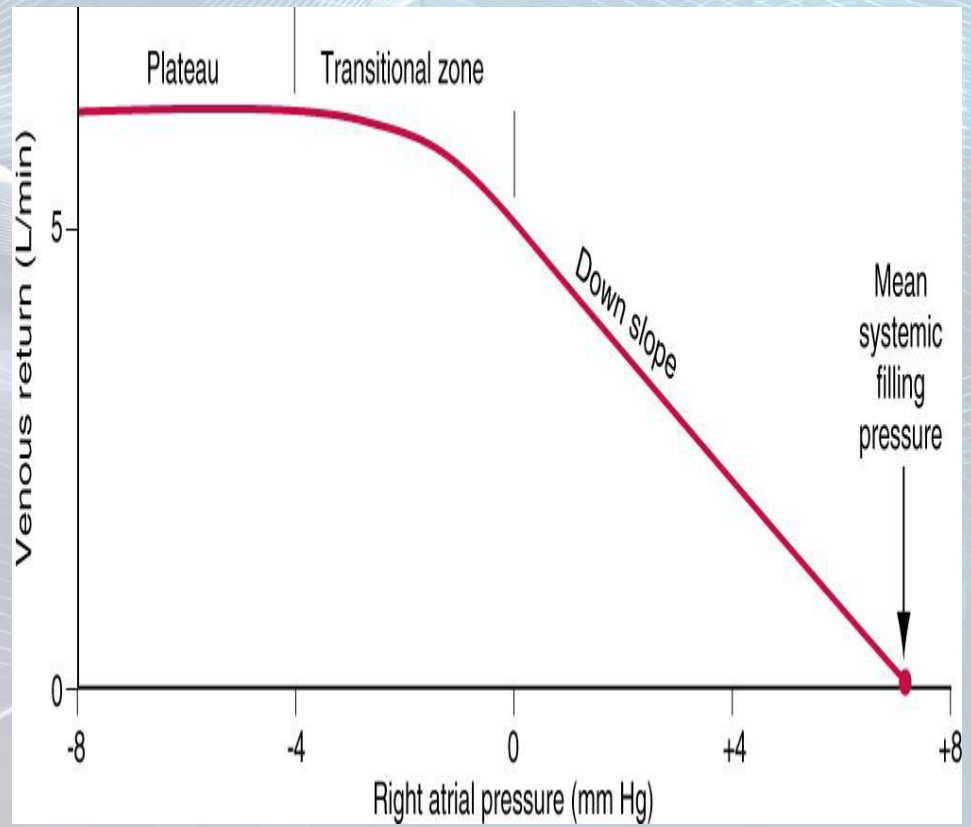
Venous return (VR) curve relates VR to right atrial pressure.

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

  Cause: Collapse of the veins entering the chest.

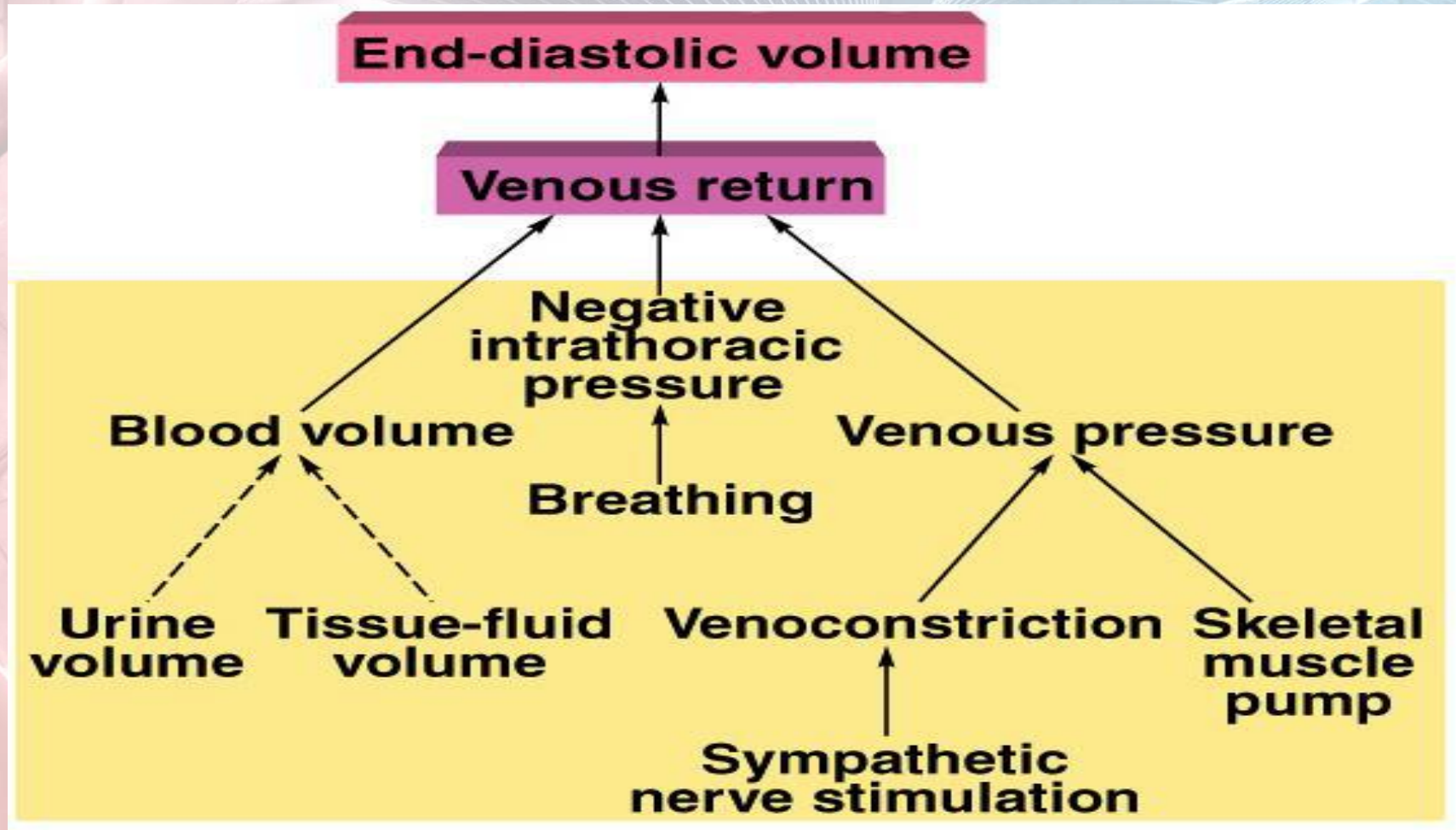
- **Mean Circulatory Filling Pressure is the value for RAP at which venous return is zero.**

  - 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 & is called : Mean Circulatory Filling Pressure (which is pressure of +7 mmHg).



SV

Factors Affecting Venous Return (Preload)



SV

Factors Affecting End- diastolic Volume

EDV is ↑ with:

- Increased total blood volume.
- Increased venous return.
- Increased venous tone.
- Increased skeletal muscle pump (exercise).
- Increased negative intrathoracic pressure.
- Stronger atrial contraction.

EDV is ↓ with:

- Standing.
- Decreased venous return.
- Increased intrapericardial pressure.
- Decreased ventricular compliance.

SV

How Does The End- Diastolic Volume Affect the SV and The CO?

↑ ↑ **EDV**

↓

↑ ↑ Stretch in the myocardium (↑ ↑ Initial fiber length.)

↓

↑ ↑ Myocardial contractility (↑ ↑ Strength of contraction)

[Frank-Starling Mechanism]

SV

End- Systolic Volume (ESV)

End- Systolic volume (ESV) is the volume of blood remaining in the ventricle at the end of systole

↑ ESV → ↓ ESV

↓ ESV → ↑ SV

SV

Factors Affecting End- Systolic Volume

- **End- Systolic volume (ESV) is determined by:**

I: Cardiac contractility:

↑↑ contractility → ↑↑ SV → ↓↓ ESV

↓↓ contractility → ↓↓ SV → ↑↑ ESV

II: Afterload: Vasoconstriction

↑↑ resistance → ↓↓ SV → ↑↑ ESV

↓↓ resistance → ↑↑ SV → ↓↓ ESV

SV

Afterload (Resistance)

- Is the resistance against which the ventricles contract.
- As afterload increases, stroke volume decreases.
- Afterload increases by any factor that restricts arterial blood flow like:

1. Increased arterial blood pressure.
2. Vasoconstriction (peripheral vascular resistance):

↑ ↑ resistance → ↑ ↑ESV

↓ ↓ resistance → ↓ ↓ESV

SV

Afterload (Resistance)

- An increase in aortic pressure will cause an increase in afterload on the left ventricle.

- An increase in pulmonary artery pressure will cause an increase in afterload on the right ventricle.

SV

Total Peripheral Resistance

Afterload & hence ESV is determined by the peripheral vascular resistance

Vasodilatation

↓ resistance

↑ Peripheral blood flow

↑↑ SV

↓↓ ESV

Vasoconstriction

↑ resistance

↓ Peripheral blood Flow

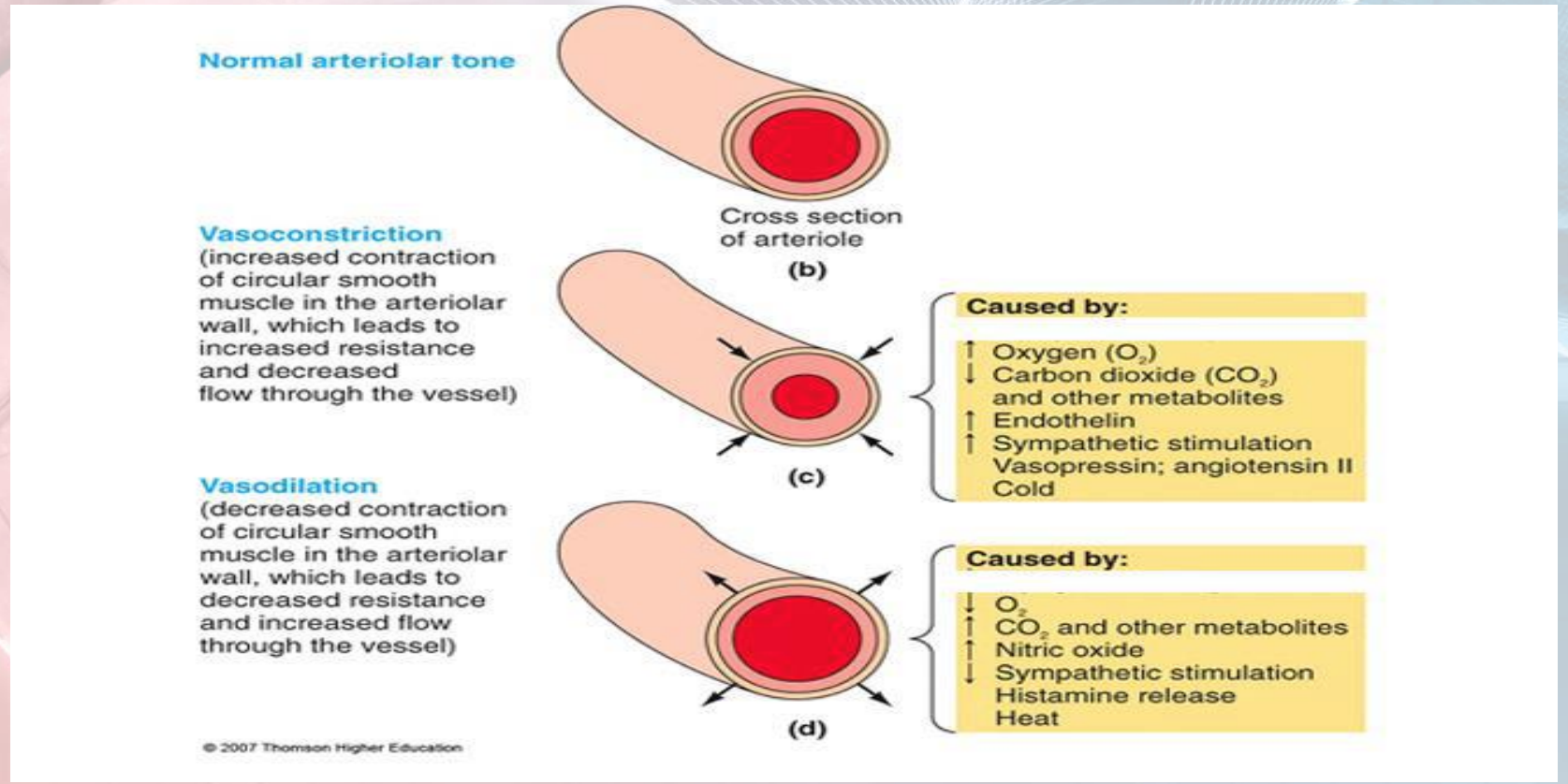
↓↓ SV

↑↑ESV

SV

Total Peripheral Resistance

Afterload & hence ESV is determined by the peripheral vascular resistance



HR

The Heart Rate (HR)

- Normal heart rate = 60-100 beats/min.
  - > 100 beats/min → Tachycardia.
  - < 60 beats/min → Bradycardia.
- **As the HR increases**, the CO increases.
  - ↑ HR up to ≈ 180 bpm, ventricular filling is adequate and CO ↑ .
  - At very high HR, filling may be compromised to a degree that CO falls.
- **As the HR decreases**, the CO decreases.
- **The HR has an influence on cardiac contractility** as well (Frequency-Force Relation.)

HR

Heart Rate: Regulation

Heart Rate (HR) is regulated by:

- **Autonomic nervous system through cardiac control centers in medulla oblongata in the brain stem:**
  - Cardiac-accelerator Center (Vasomotor center) … Sympathetic nerve fibers.
  - Cardiac-inhibitory Center … Parasympathetic nerve fibers.
  - Sympathetic nervous stimulation increases HR and contractility.
  - Parasympathetic nerves (vagus nerve) slow HR.
- **Hormones and Drugs:**
  - Epinephrine & thyroxine increase HR.
  - Increased calcium level in blood causes prolonged contraction.
  - Reduced calcium level in blood decrease HR.

HR

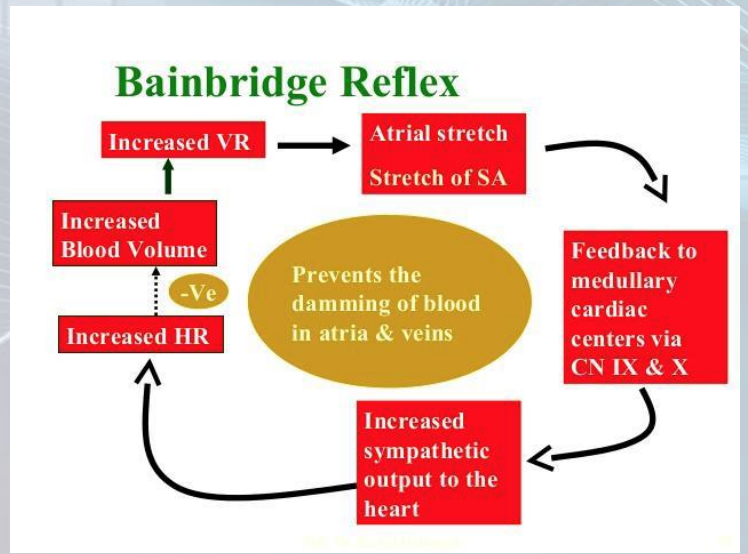
Heart Rate: Regulation

❑ 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.

❑ Blood volume:

- Atrial reflex (Bainbridge reflex).
- This reflex adjusts HR in response to VR.
- Increase blood volume, stimulates stretch receptors in right atrium.
- This triggers increase in HR through increased sympathetic activity.



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