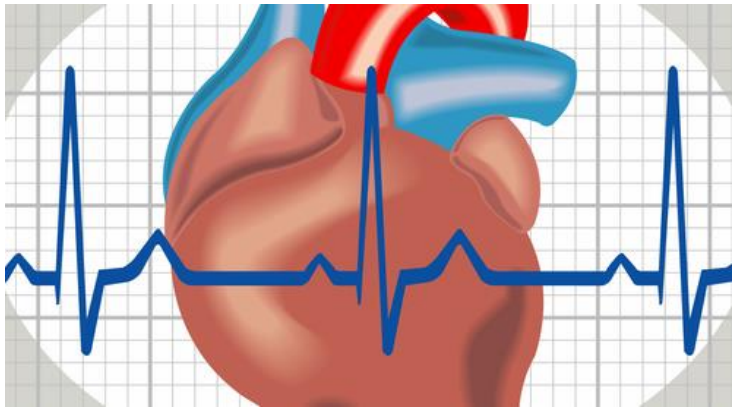
Ashur University
College of Medicine

Grade 1

Cardiovascular Module
Cardiovascular Physiology
Lec.5

Heart Rate & Stroke Volume



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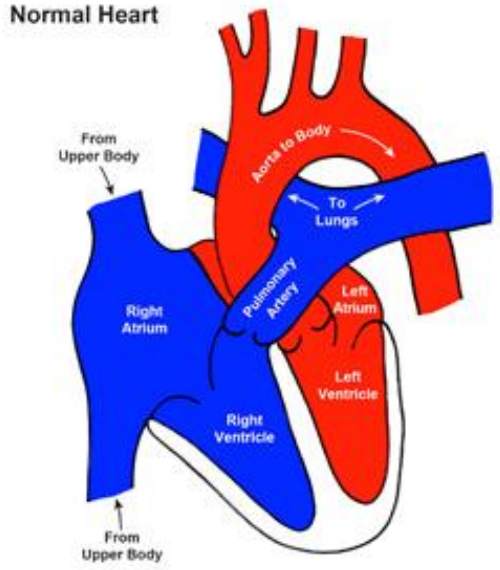
14/4/2025

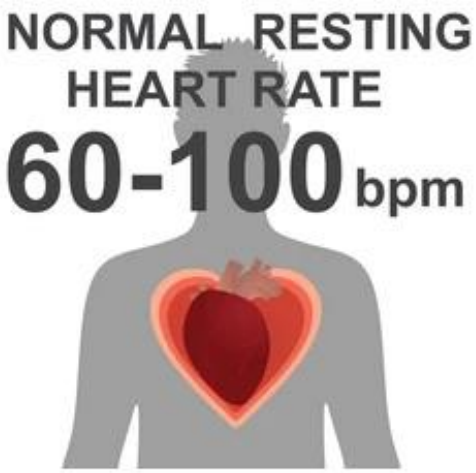


Objectives:

- Define heart rate (**HR**) and Stroke volume (**SV**).
- Summarize the factors that regulate heart rate and Stroke volume.
- Describe how End-diastolic volume (**EDV**), total peripheral resistance (**TPR**)interact to control stroke volume.

EDV is the amount of blood in a ventricle immediately before the contraction, at the end of diastole.

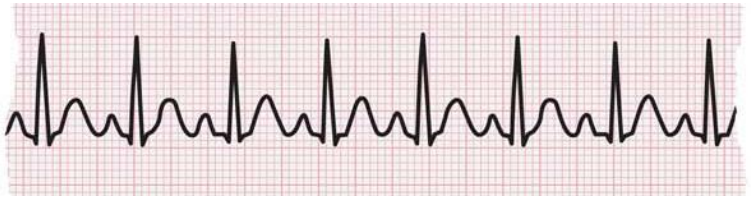
Heart rate



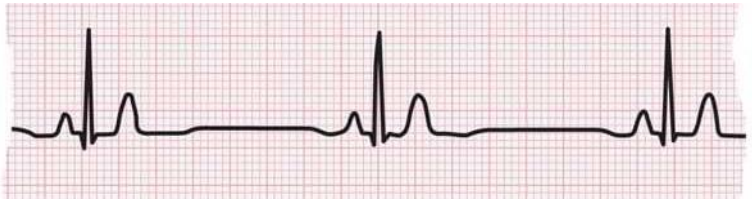
- The heart rate refers to the ventricular rate of beating per minute.
- Normally, averages 72 beats/minute (**B**eats **p**er **m**inute= **Bpm**) (range 60-100 beats/minute) →



- Tachycardia..higher than 100 beats/min→



- Bradycardia… lower than 60 beats/min →

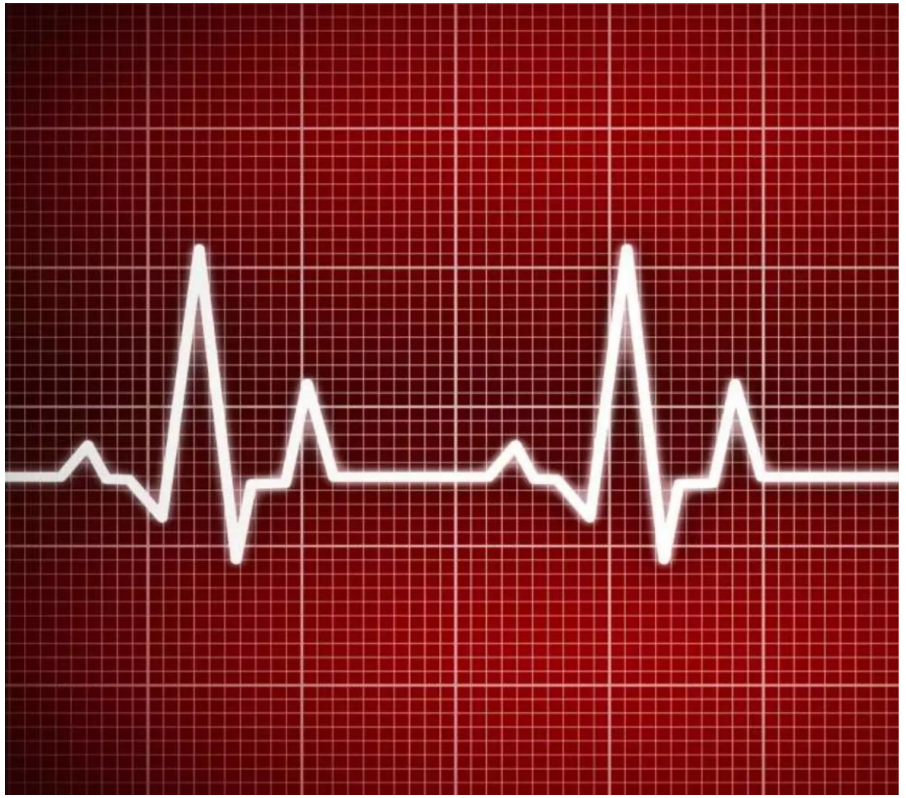


Regulation of Heart Rate

- Positive chronotropic factors increase heart rate

  (chrono = time)
- Negative chrontropic factors decrease heart rate.
- Can be neural , chemical or physical factors.





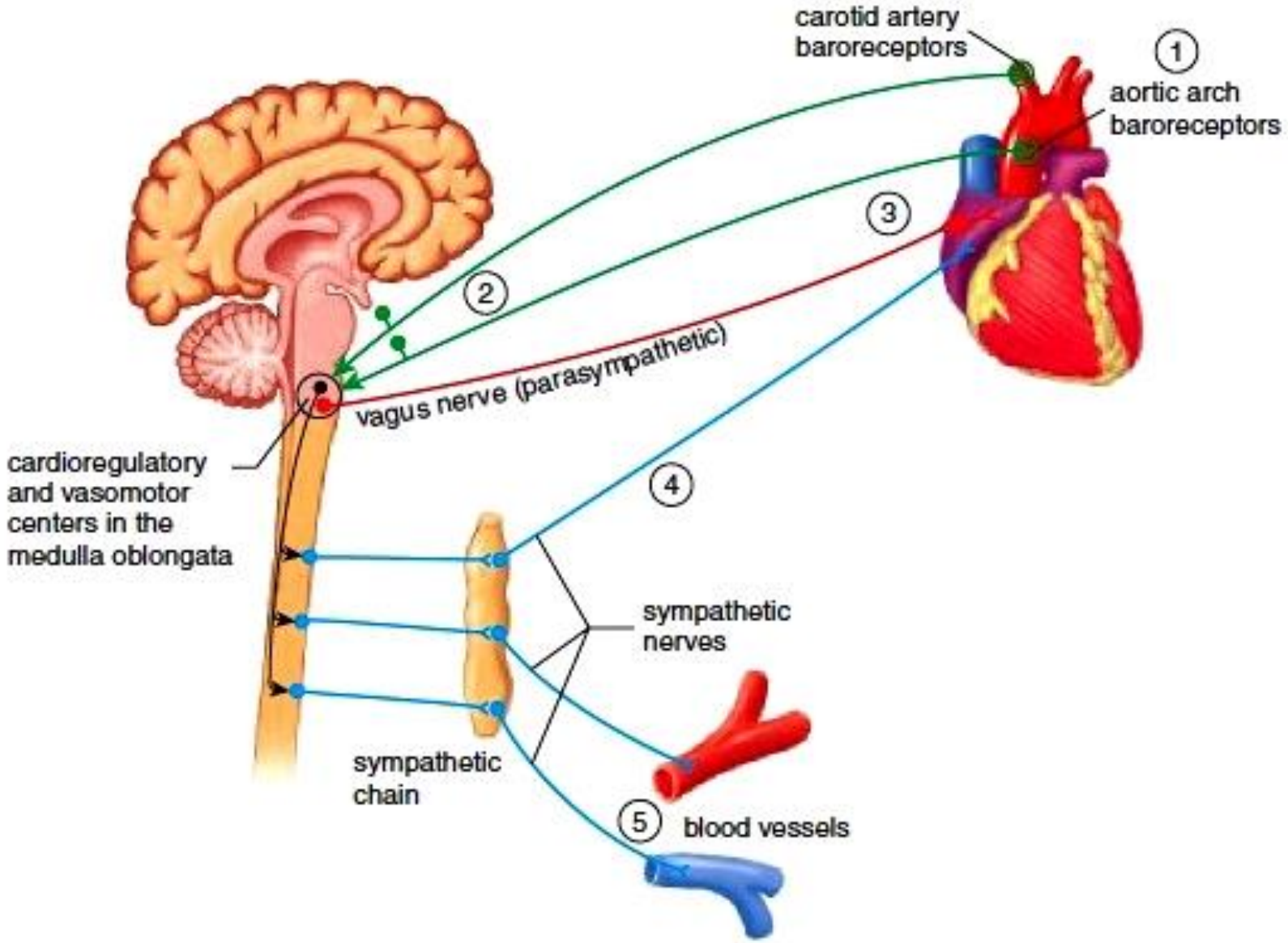
Regulation of Heart Rate

Regulation of heart rate:

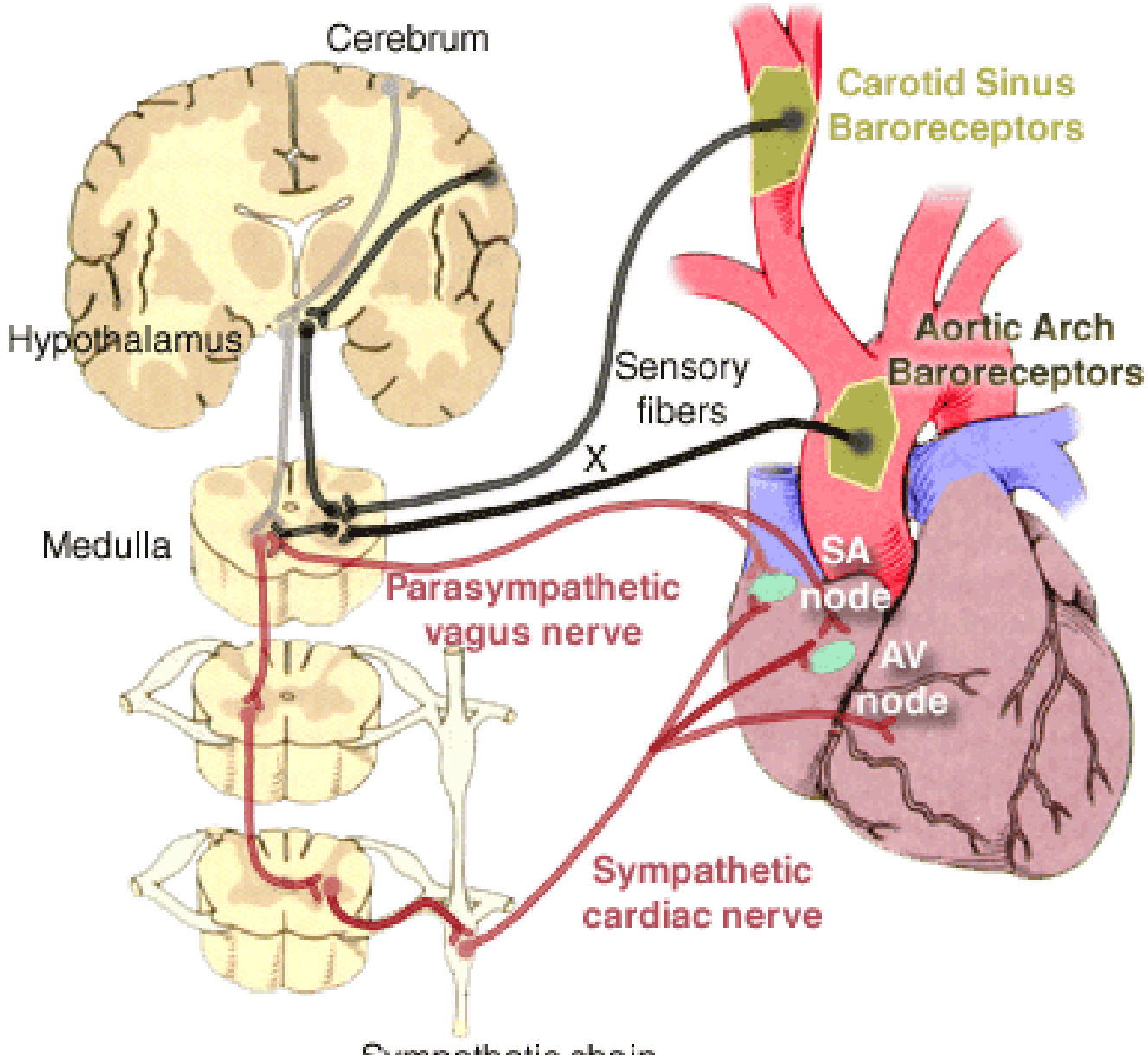
1. Baroreceptors in the aortic arch and carotid arteries monitor blood pressure.
2. Nerve impulses from the baroreceptors signal the cardioregulatory center.
3. Increased parasympathetic impulses decrease heart rate.
4. Increased sympathetic impulses increase heart rate.

Regulation of blood pressure:

5. Increased sympathetic impulses cause blood vessels to constrict.



Regulation of Heart Rate



Cardiovascular centers

1- Vasomotor center (VMC):

A- Vasoconstrictor center (VCC):

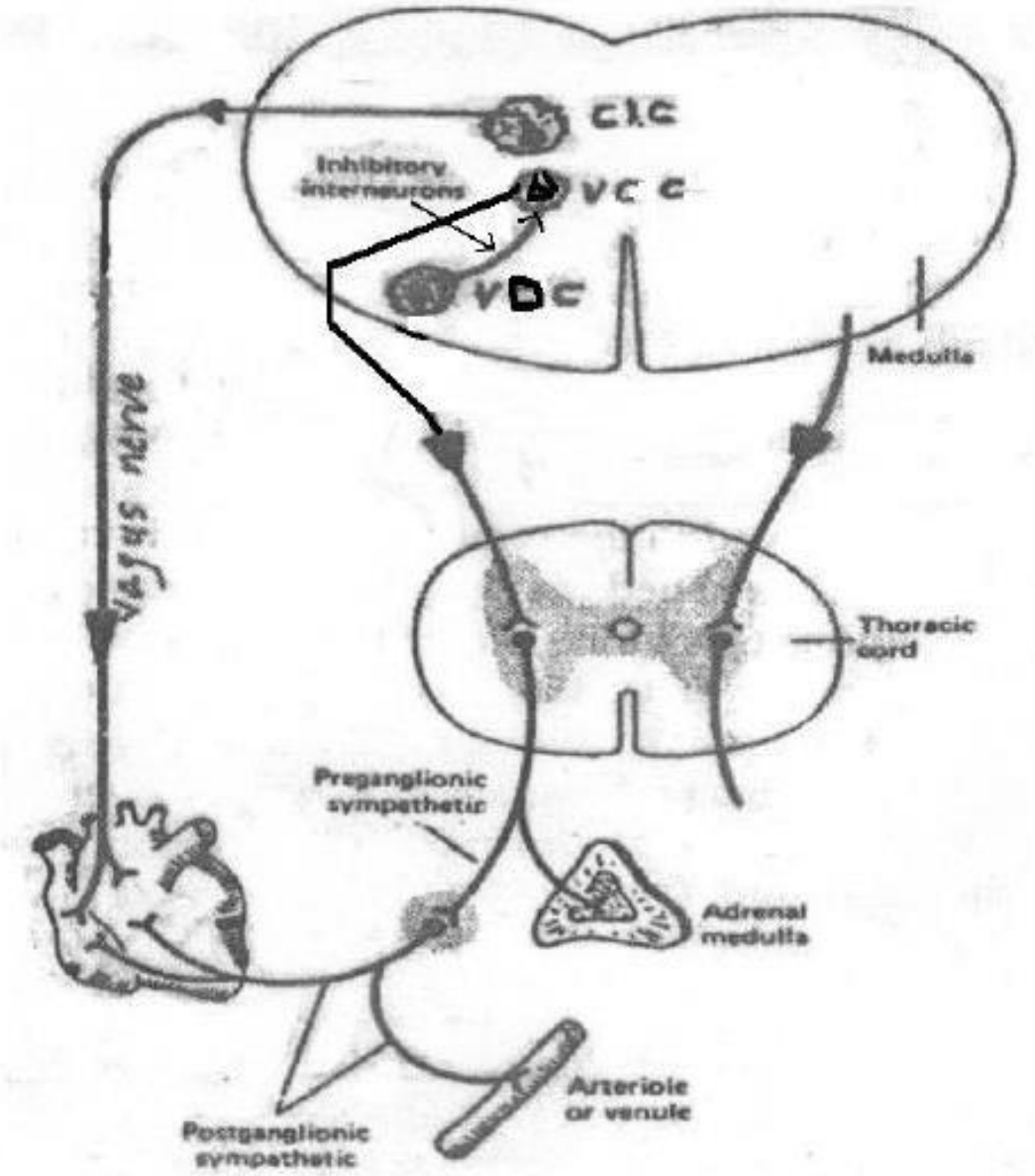
- The blood vessels (leading to generalized vasoconstriction (VC)).
- The adrenal medulla (leading to secretion of Catecholamines).
- The heart (increasing heart rate).

B- Vasodilator center (VDC):

inhibiting the activity of VCC causing generalized vasodilatation.

2- Cardioinhibitory center (CIC):

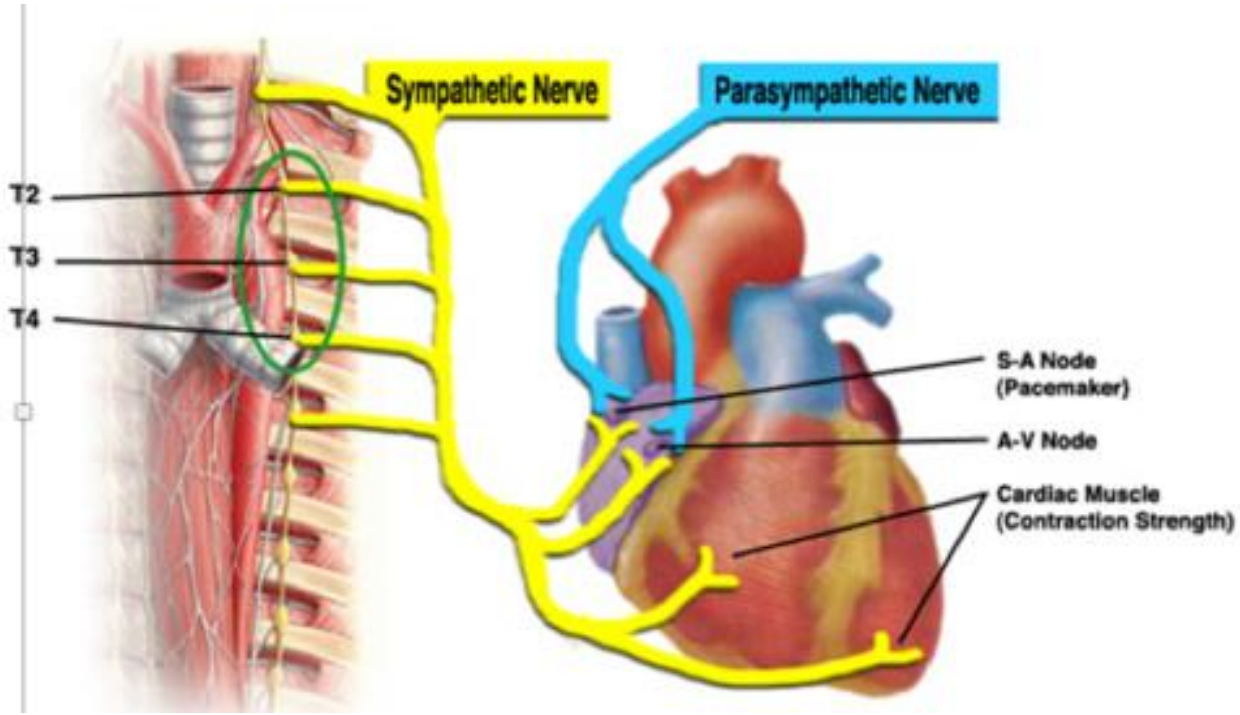
Decreasing heart rate



Neural regulation of the heart rate

- **Sympathetic nerves**: supply all parts of the heart (atria, ventricles, and conduction system). When activated they **increase Heart rate (+ve chronotropic effect).**
- **Parasympathetic nerves**: supply atria, SA & AV nodes but not the ventricles.

  When activated, they **decrease Heart rate (-ve chronotropic effect).**



Reflexes

- **Bainbridge reflex (atrial stretch reflex)**

right atrial pressure or distention leads to heart acceleration through medullary VCC.

- **Baroreceptor reflex**

stretch receptors, carotid sinus and aortic arch, glossopharyngeal and vagus nerves, inhibit the VCC and excite the vagal center (CIC) resulting in vasodilation, and decreased heart rate.

- **The carotid sinus reflex**

pressure on carotid sinus area, reflex slowing of heart rate and vasodilation. reflex increase in the vagal tone (bradycardia) and decrease in the sympathetic vasoconstrictor tone (vasodilation).

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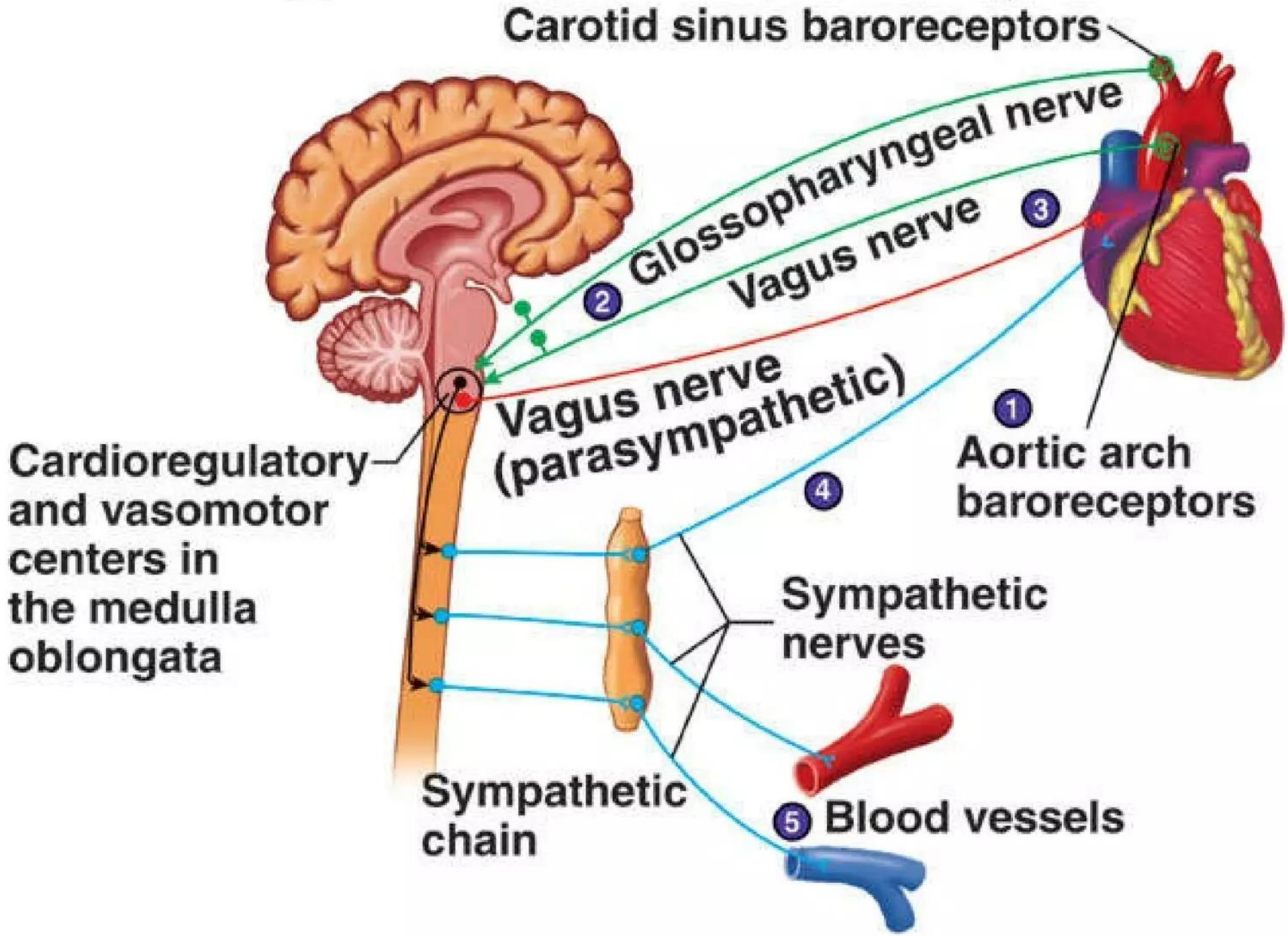


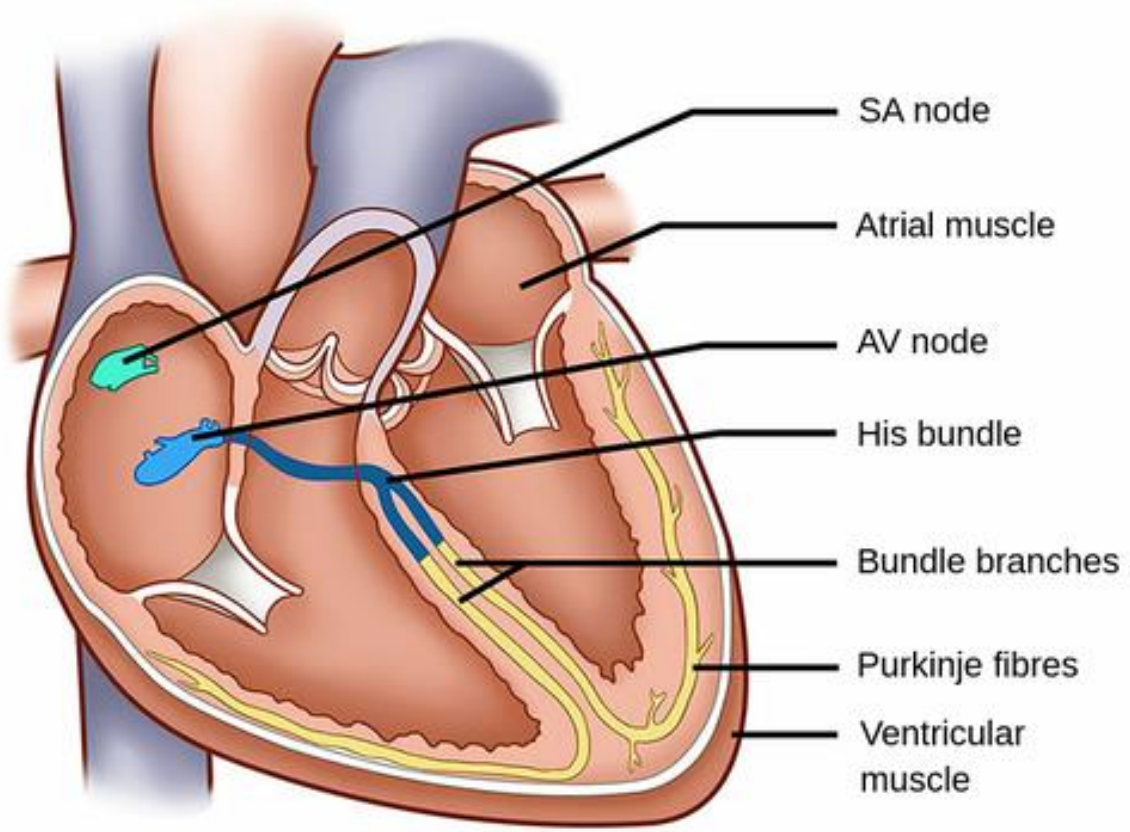
SA node activity

- **Factors directly affect SA node activity:**
- **Physical factors:**

Body temperature; 1 °C increases the heart rate by 10-20 beats/minute.

- **Mechanical factors:**

Right atrial distension may directly excite the SA node leading to tachycardia



SA node activity

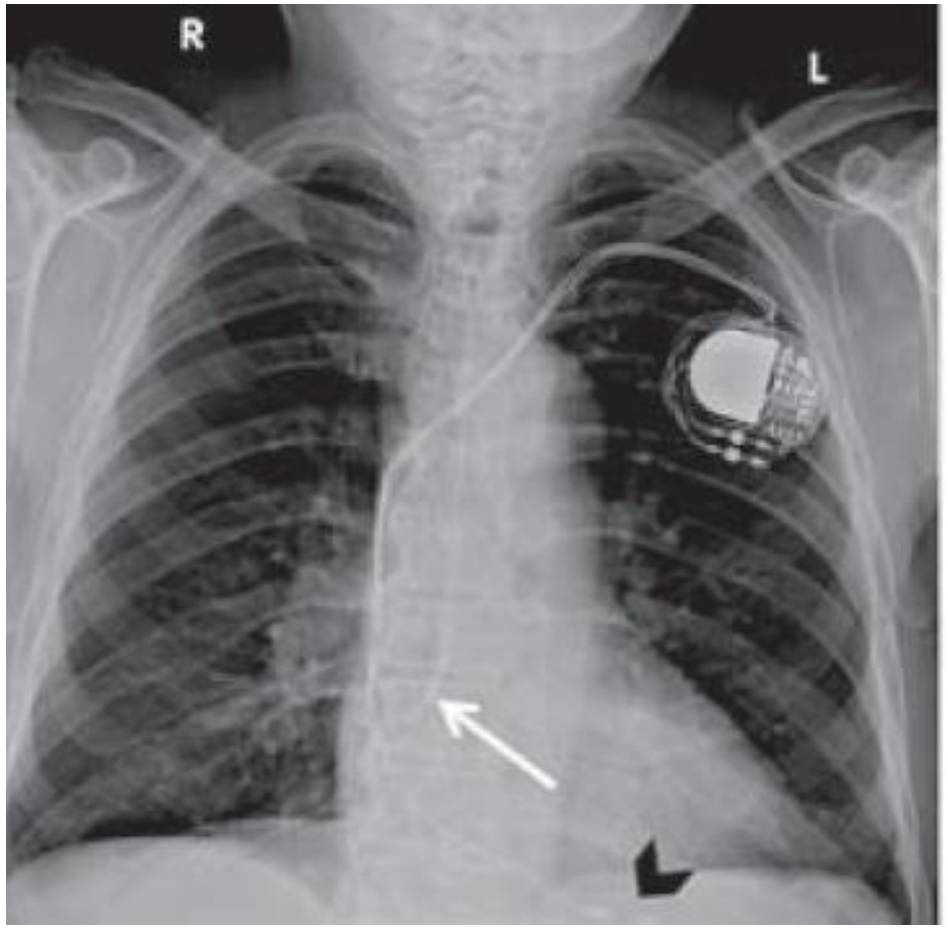
Effects of hormones, drugs & chemicals:

- **Adrenaline**; Small doses **↑ Heart Rate (HR)** by a direct action on (β1) receptors in SA node.
- **Thyroxine**: **↑HR** by direct stimulation of SA node & ↑ its sensitivity to catecholamines.
- **Atropine**: **↑HR** by blocking parasympathetic activity.
- **Histamine**: a potent vasodilator substance which → marked drop of arterial Blood pressure, resulting in **↑HR** .
- **Autonomic drugs**: Sympathomimetic drugs (**amphetamine**) cause tachycardia (**↑HR**) while

parasympathomimetic drugs (**acetylcholine**) cause bradycardia.

Cardiac pacemaker





Stroke Volume (SV)

- **Stroke volume (SV)** is defined as Volume of blood ejected by each ventricle per one beat.
- It is about **70 ml/beat** at rest but may increase to 150 ml/beat with exercise.
- The ventricles don't completely empty themselves of blood during contraction.
- **2/3** of blood is ejected **(ejection fraction)**,1/3 is left there. (in the ventricles)
- Compensatory mechanism : **↓ HR → ↑ SV**

Stroke Volume (SV)

- **SV = EDV – ESV**

SV=Stroke Volume EDV =End Diastolic Volume ESV =End Systolic Volume

- **EDV:** volume of blood in ventricles at end of diastole , Normal (110 to 120 ml).
- **ESV:** volume of blood in ventricles at end of systole, Normal (40 ml).
- **SV = 70 to 80 ml**.
- **SV = End-diastolic volume - End-systolic volume**

130 ml - 60 ml = 70 ml

Regulation of Stroke Volume

SV = EDV - ESV

Determined by 3 variables:

Preload: affects mainly **EDV.**

Afterload: affects mainly **ESV.**

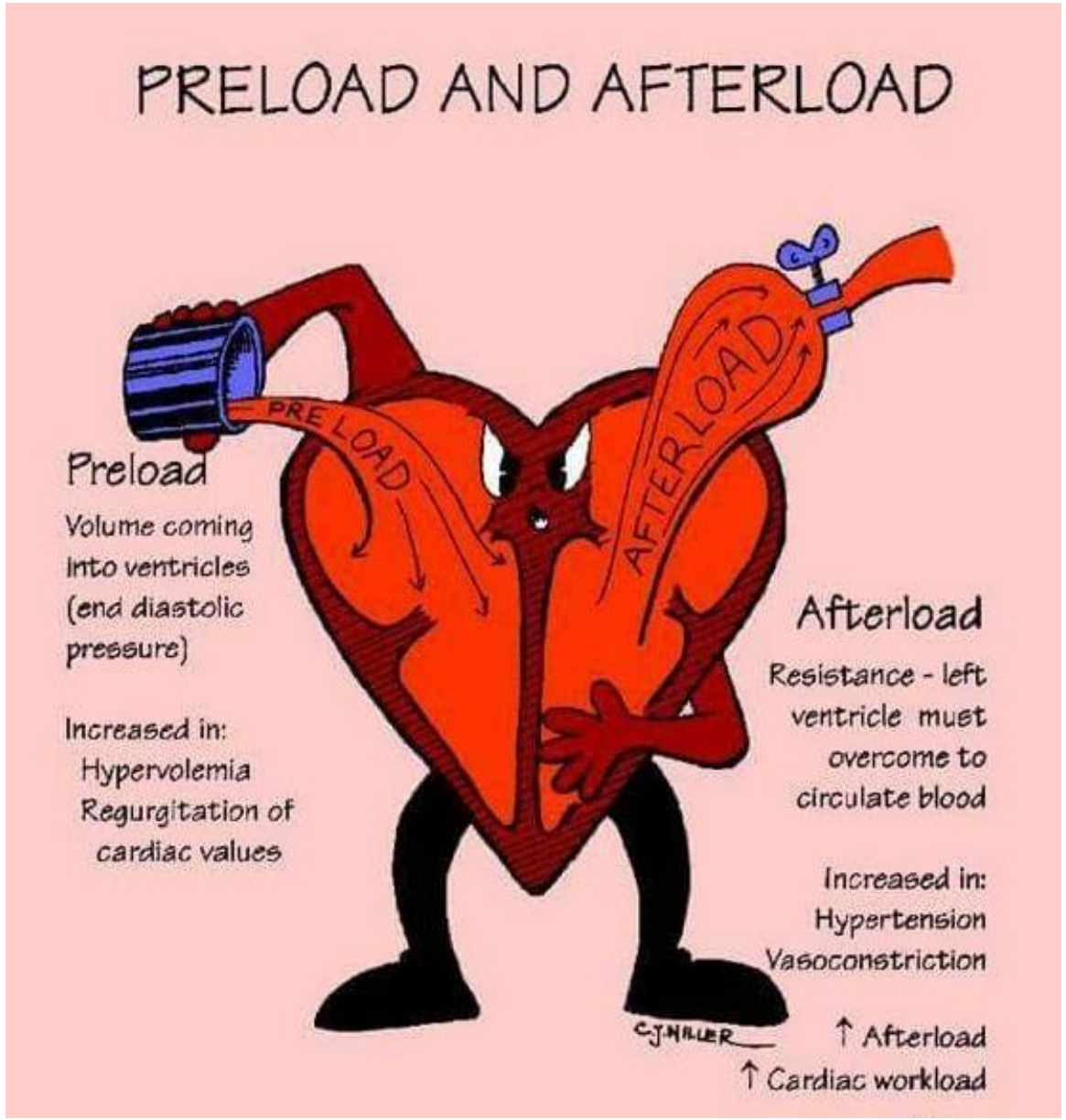
Inotropy: affects mainly **ESV.**

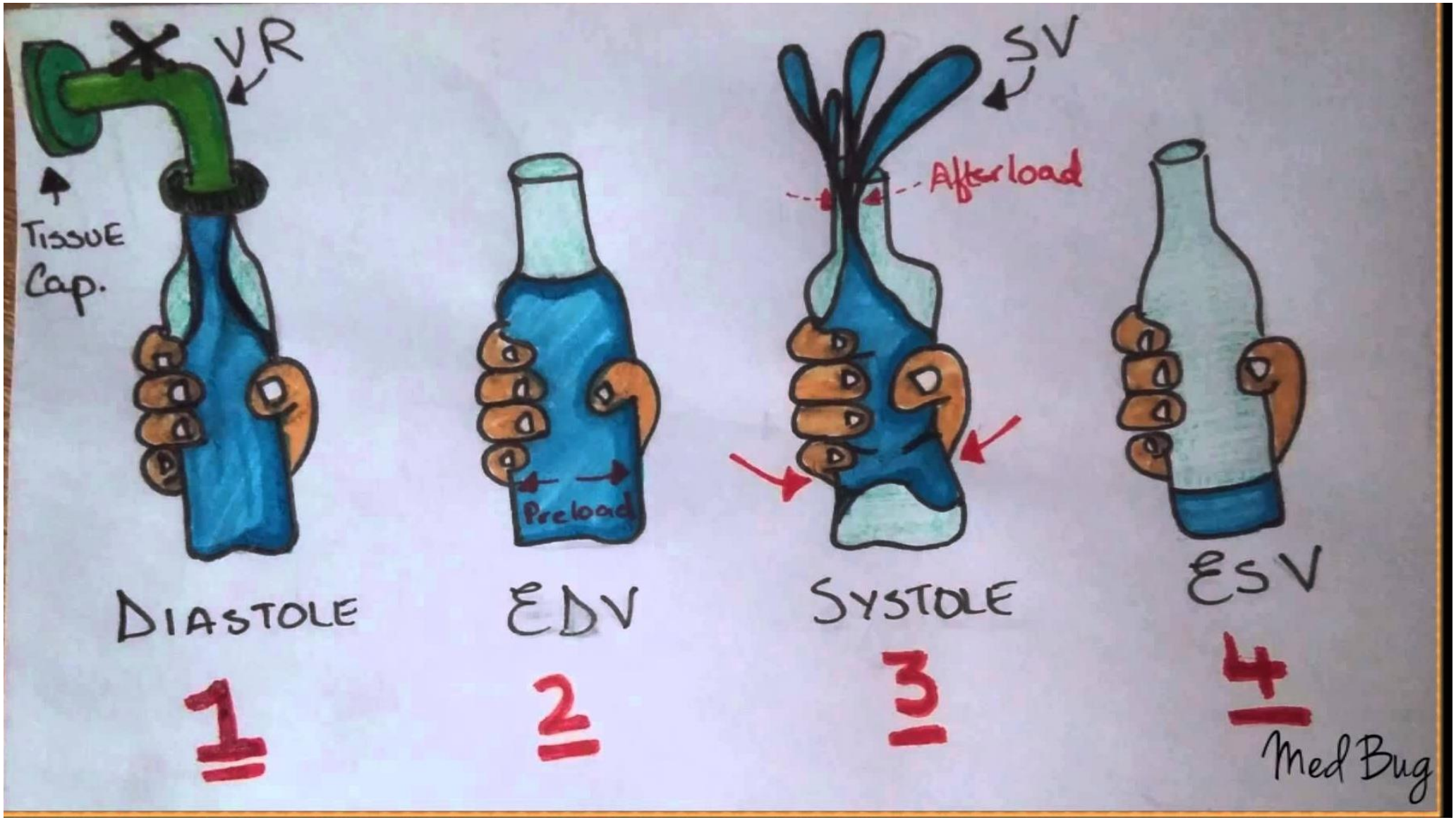
Regulation of Stroke Volume

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

  It is workload on heart prior to contraction **(preload)**.

- **Total peripheral resistance (TPR)** = which impedes ejection from ventricle; impedance to blood flow in arteries **(afterload)**.



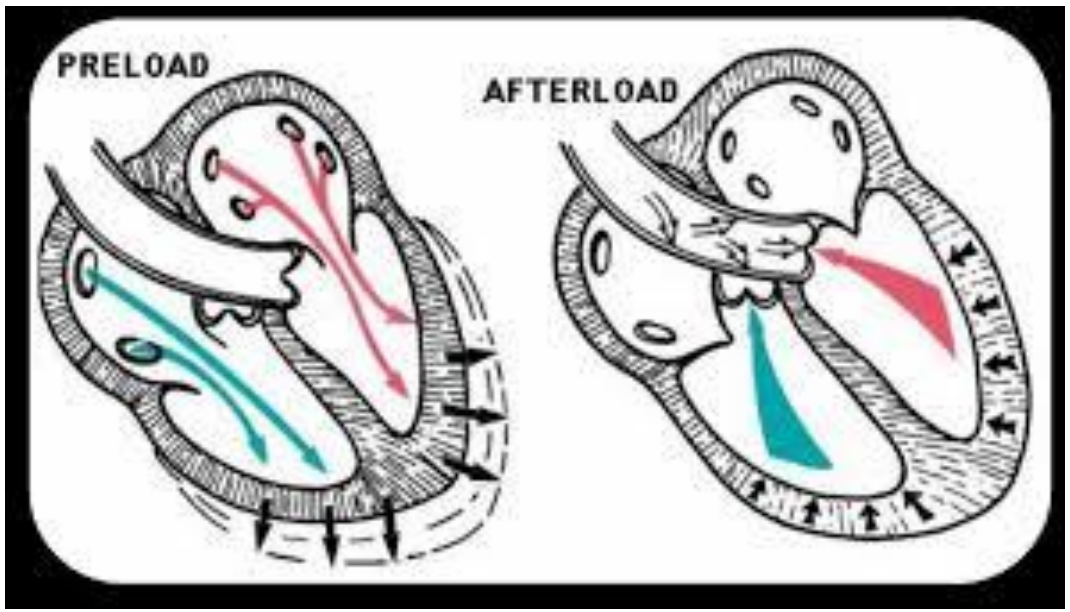


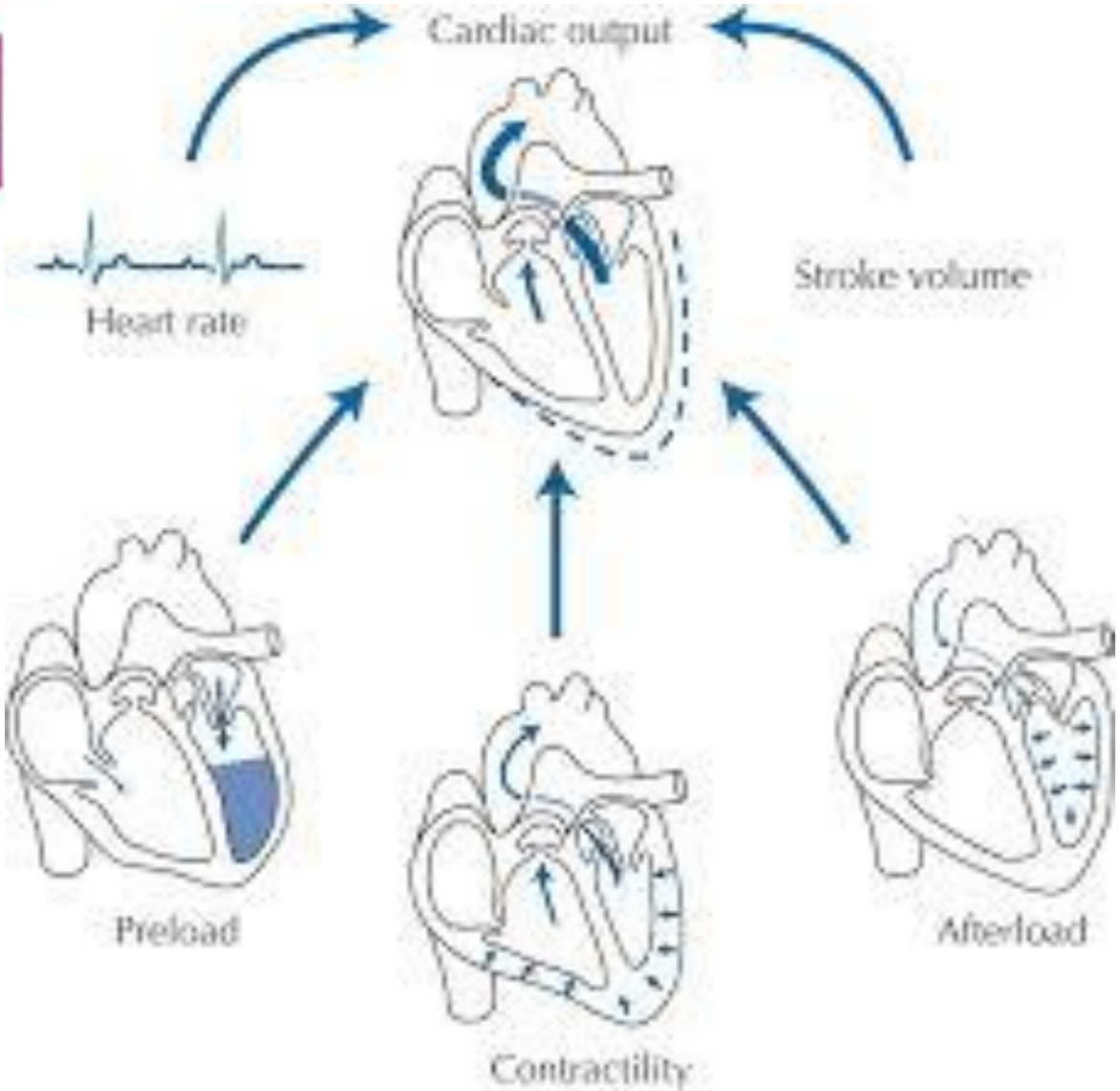
Preload Afterload

Regulation of Stroke Volume

- **End-diastolic volume (EDV):** SV is directly proportional to preload; an increase in EDV results in an increase in stroke volume **(Frank-Starling law).**
- **The total peripheral resistance:** SV is inversely proportional to **TPR** (afterload); it is the aortic impedance; higher peripheral resistance; higher arterial pressure that reduce the stroke volume.
- **Sympathetic input (Inotropy):** Strength of contraction varies directly with EDV; myocardial contractility is directly proportional with SV.

Preload | **Heart Contraction** | **Afterload**



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