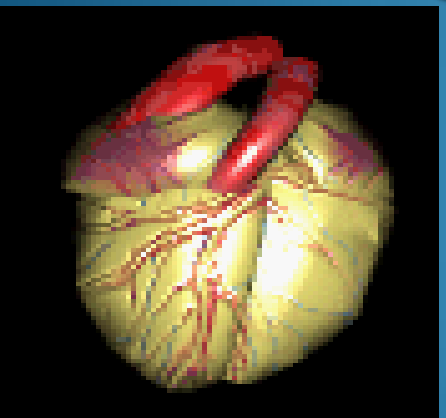
MEDICAL UNIVERSITY – PLEVEN
FACULTY OF MEDICINE
DISTANCE LEARNING CENTER

Lecture № 7

Cardiac cycle. Function of the valves. Work output of the heart. Heard sounds. Intrinsic and extrinsic regulation of myocardial performance



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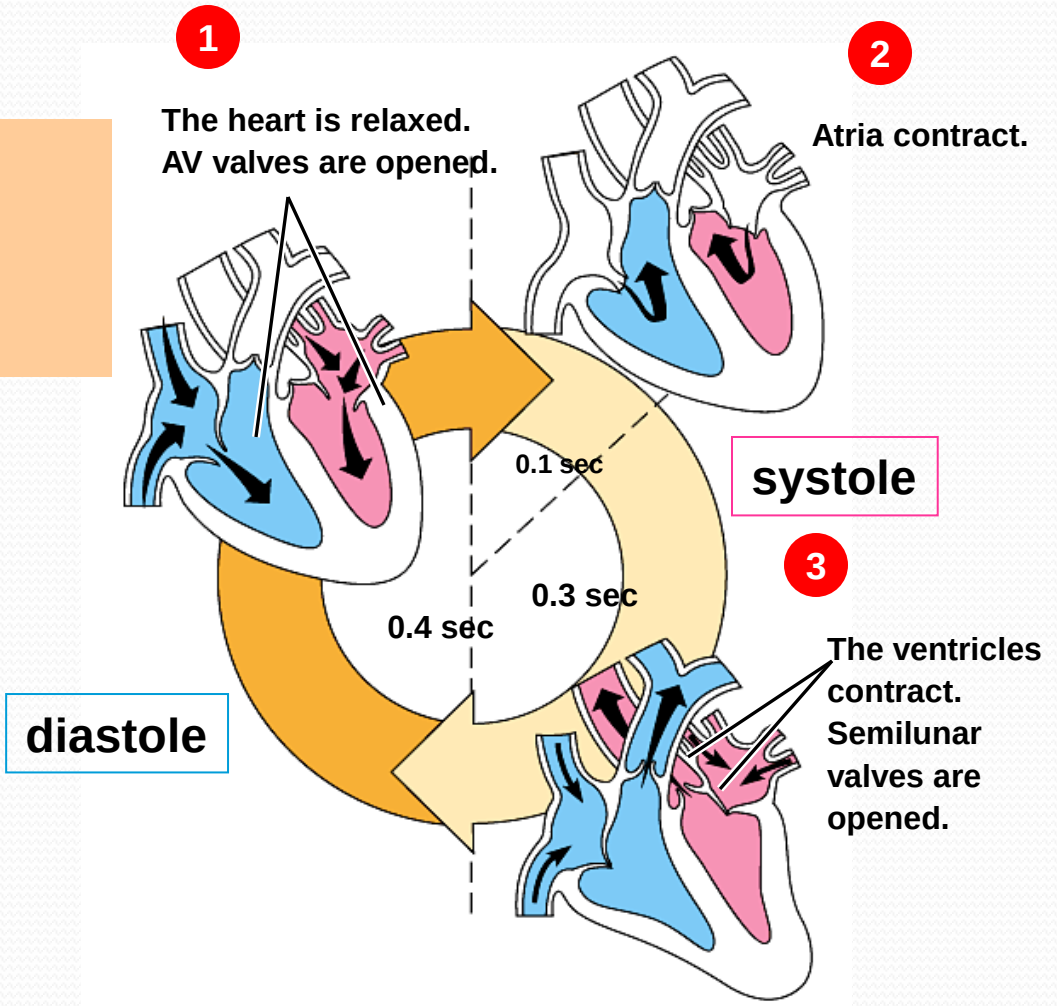
The heart contracts and relaxes rhythmically.

Cardiac cycle is the time for performing of one systole and one diastole.

HR=75/min; CC=0,8s

As=0,1s | Ad=0,7s

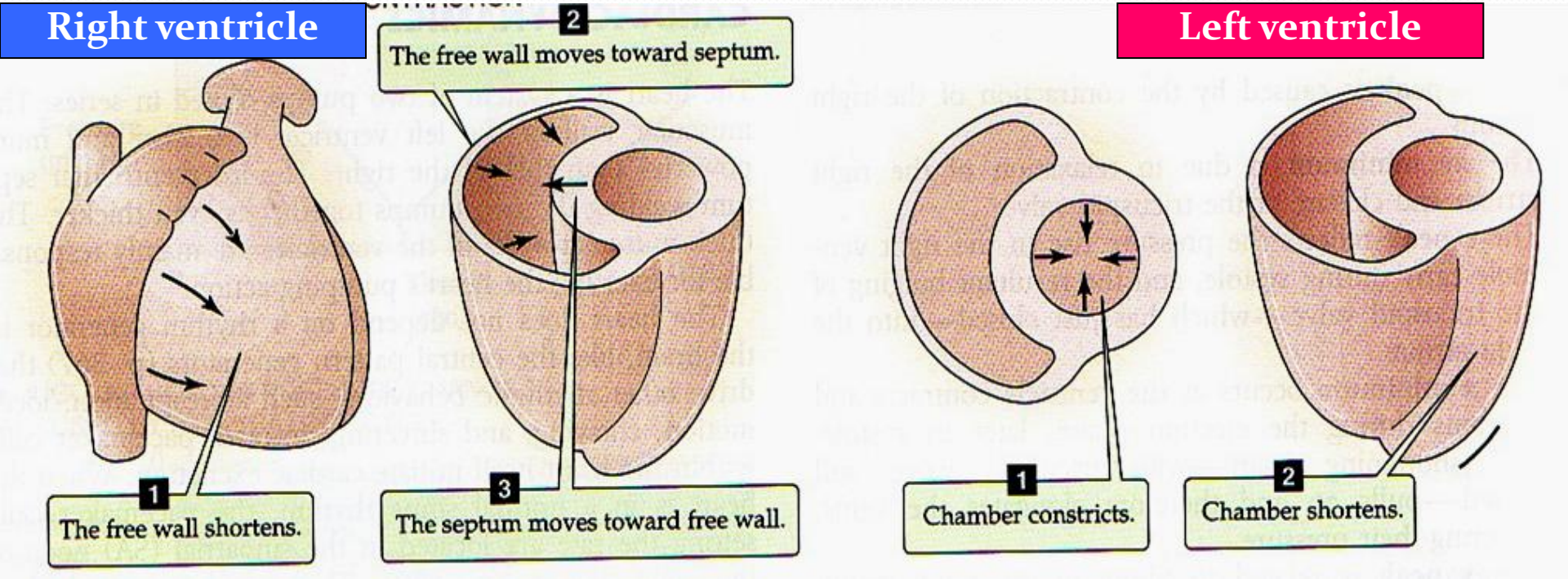
Vs=0,3s | Vd=0,5s



1 systole + 1 diastole = cardiac cycle

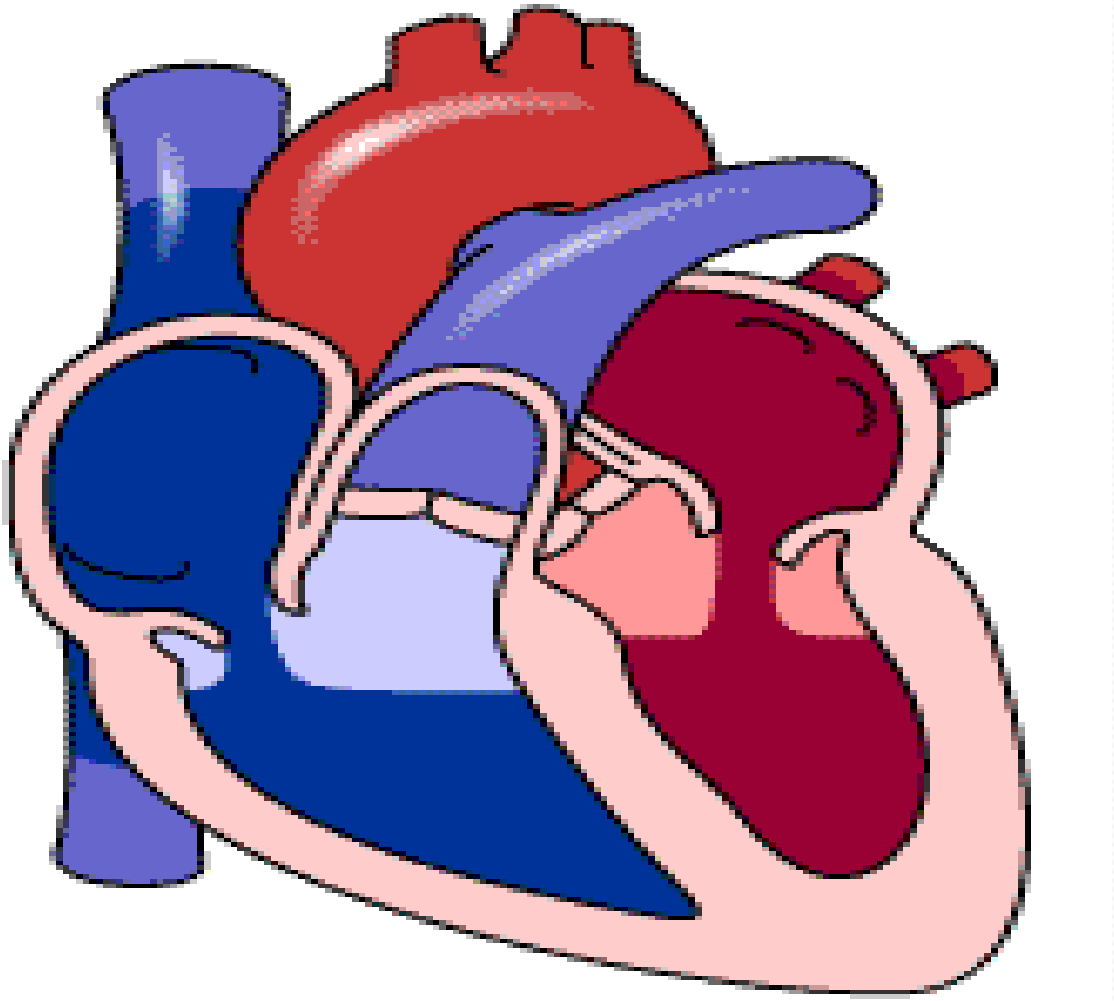
Contraction of ventricles

- ventricles have circular and spiral muscle fiber layers
  - ✓contraction of spiral layer causes shortening of their length
  - ✓contraction of circular layer causes shortening of their diameter
  - ❖ contraction of both ventricles begins from the apex and goes to the basis that ensures movement of the blood to semilunar valves
  - ❖ relaxation occurs from the basis to the apex of ventricles ensuring rapid blood flow entering from atria



The pump function of the heart

- the heart consists of two successively connected pumps
  - right ventricle pumps venous blood into the lungs
  - left ventricle pumps oxygenated blood into systemic circulation

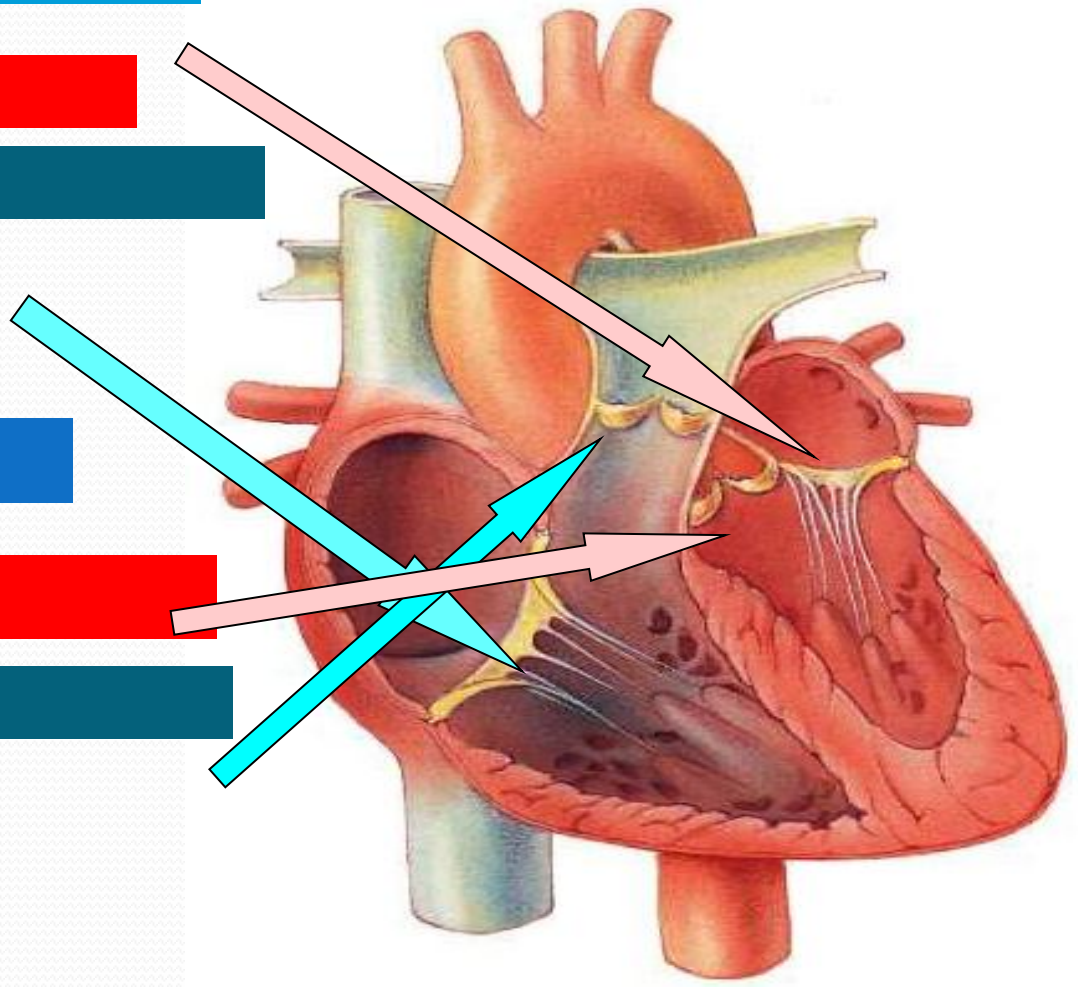


The valves of the heart

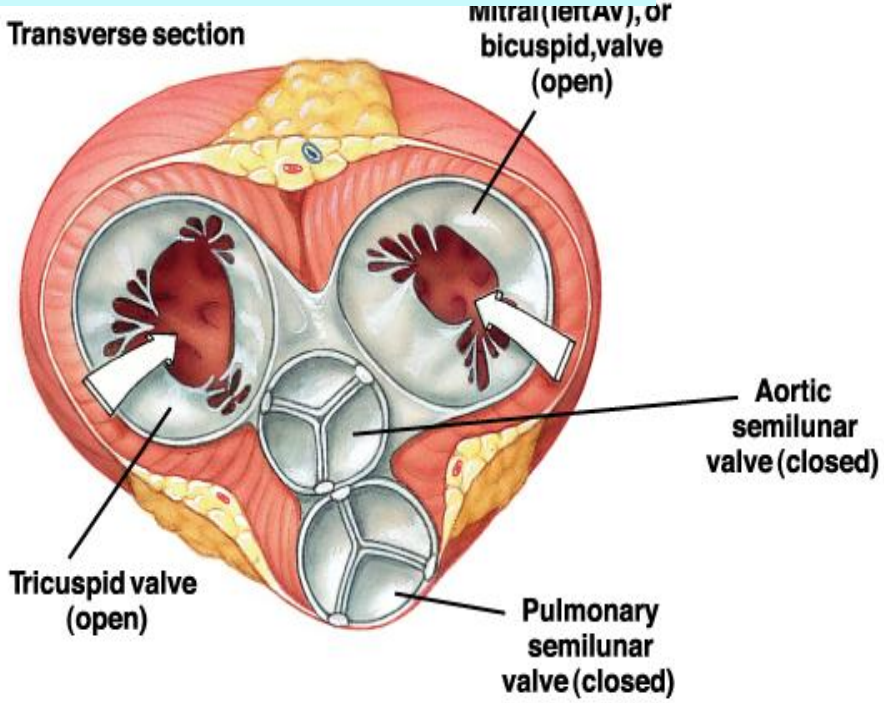
- the function of the valves is to ensure one way successive blood flow
  - they open or close under influence of the pressure on the both side

- atrio-ventricular valves:
  - mitral (bicuspid)
  - tricuspid

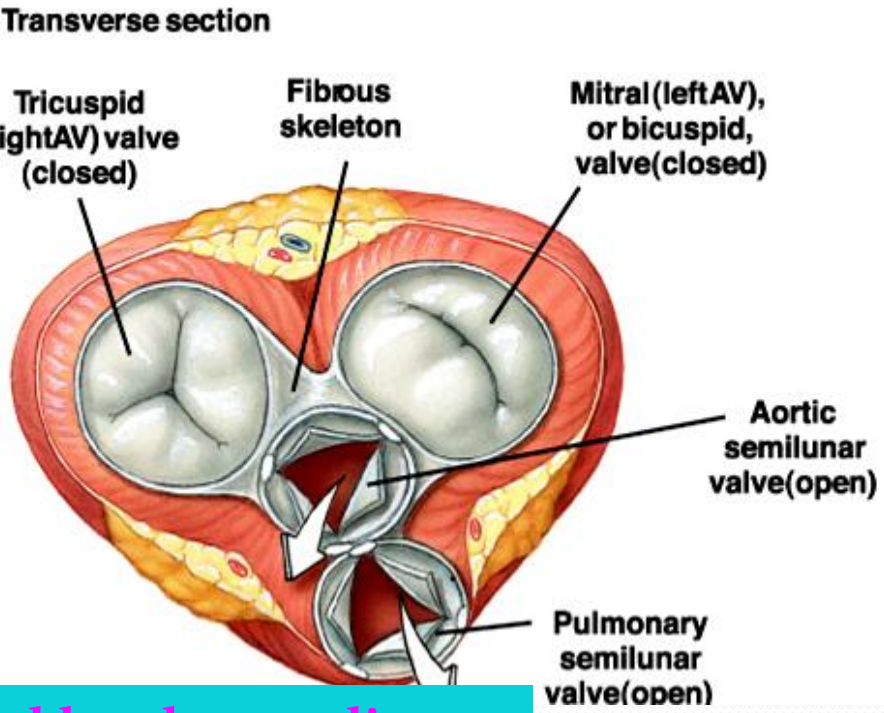
- semilunar valves:
  - aortic
  - pulmonal



The valves during the diastole

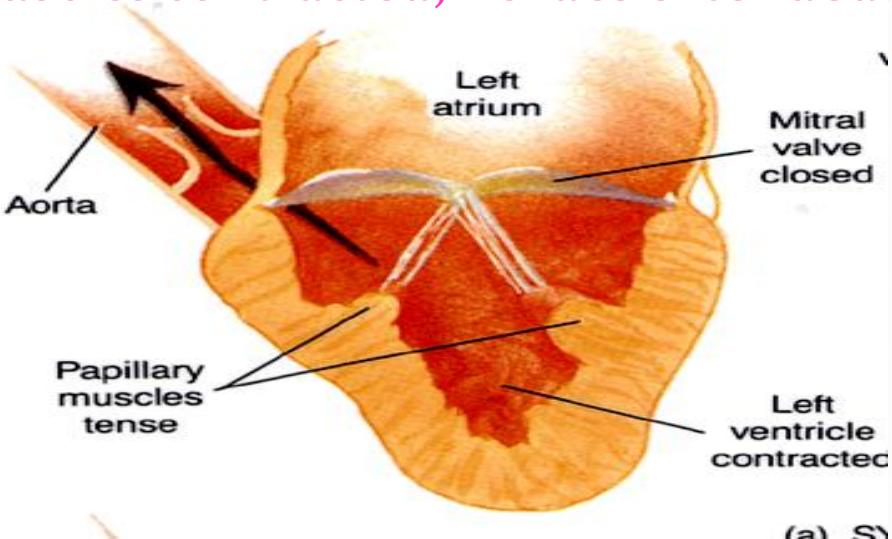


The valves during the systole

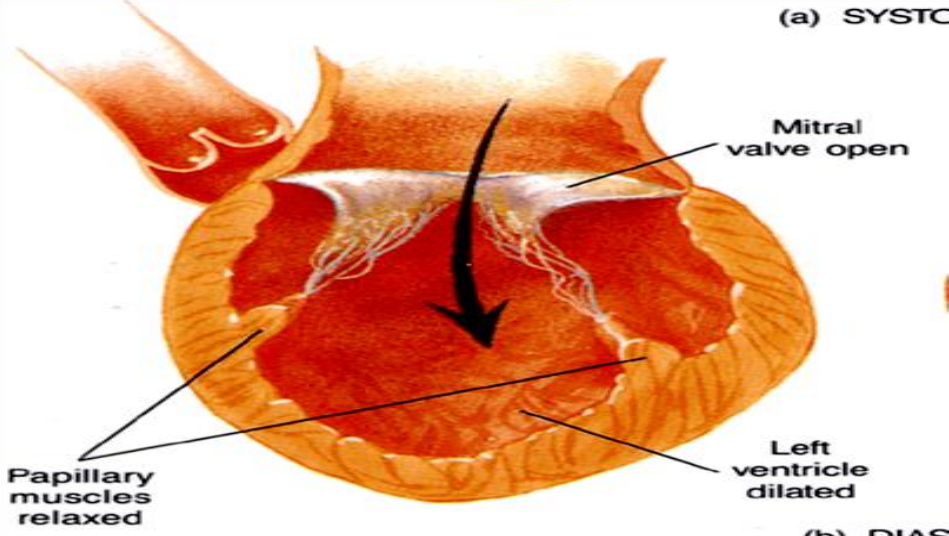


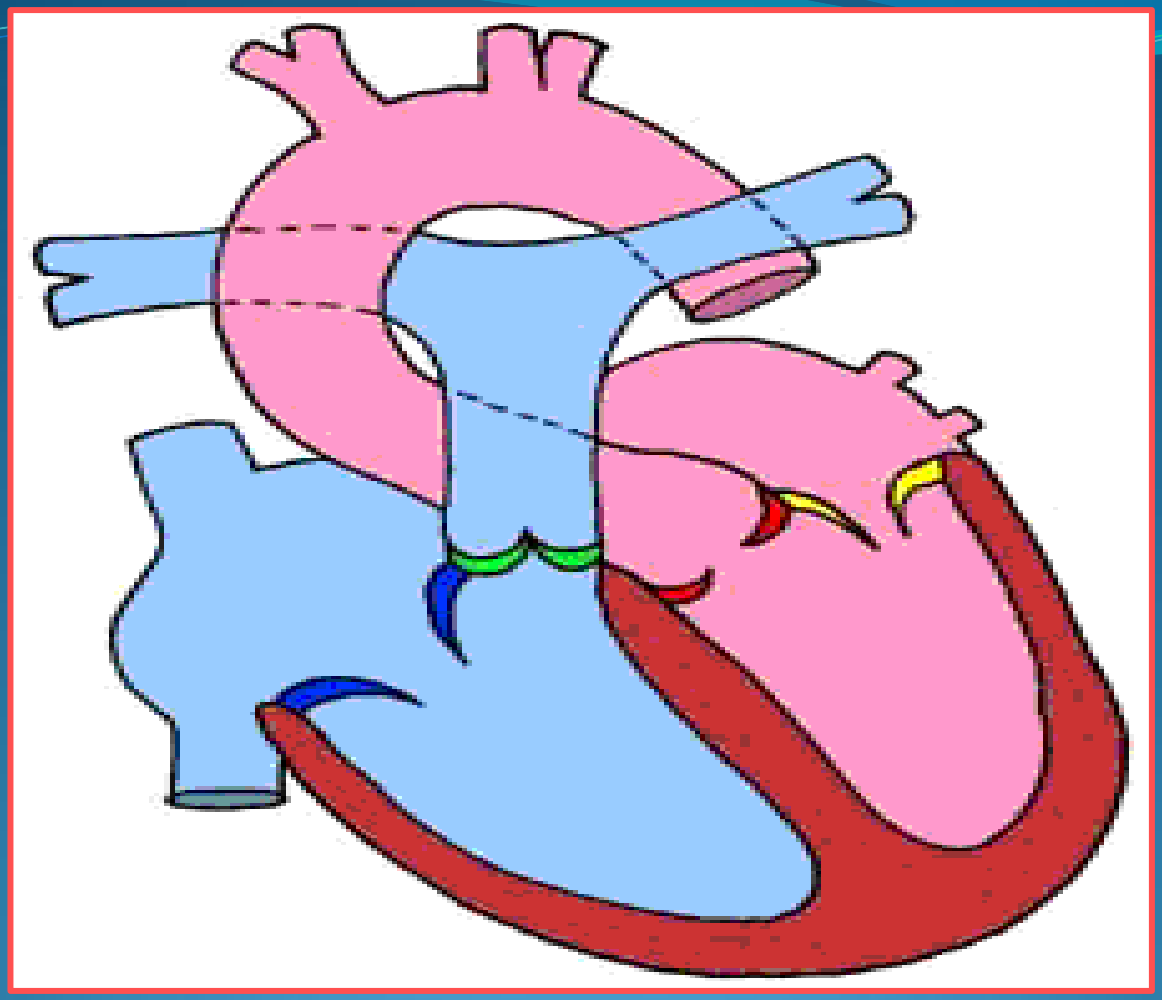
Function of papillary muscles and hordae tendineae

muscles contracted, hordes extended



muscles and hordes relaxed





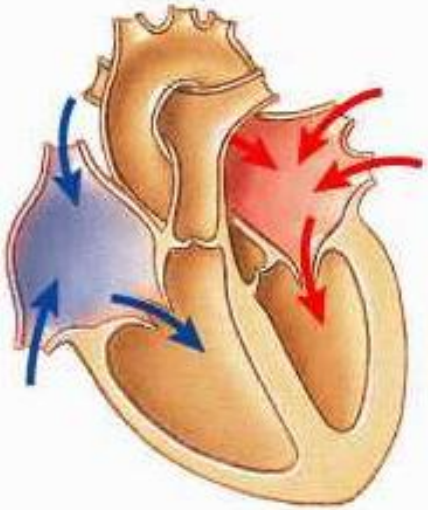
- Papillary muscles and hordae tendineae prevent turn of cusps of A-V valves to atria during ventricular systole.

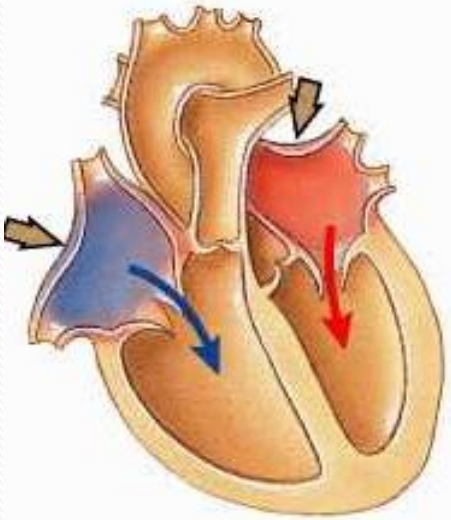
Phases of cardiac cycle

phase 1. ***diastole***: Atria and ventricles are relaxed. The blood from big veins flows into the ventricles through atria and fills them. The pressure and the volume of the ventricles increase.

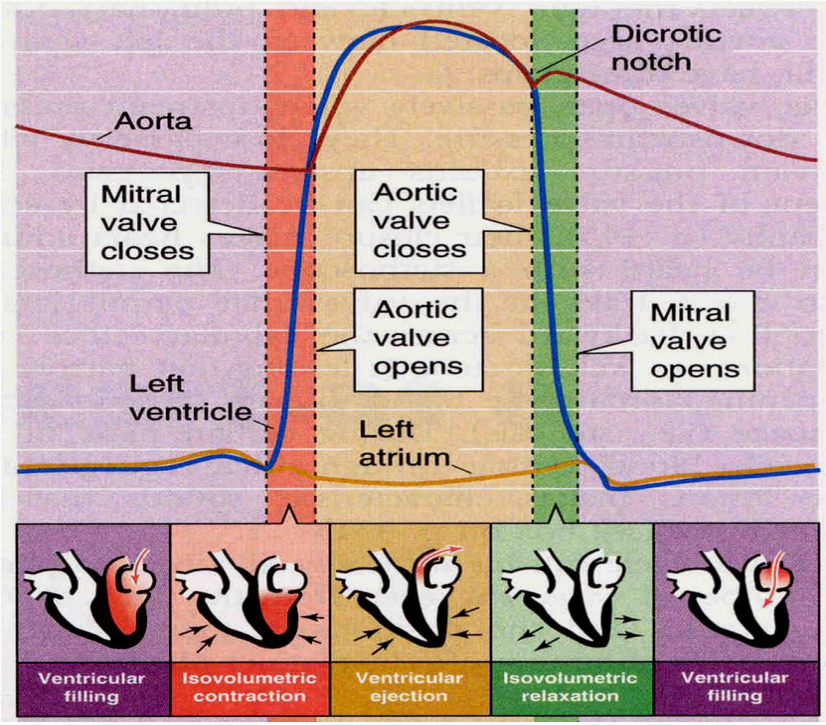
phase 2. ***atrial contraction***: the ventricles are additionally filed with blood (20-30%) and their volume riches maximal value.

phase 1





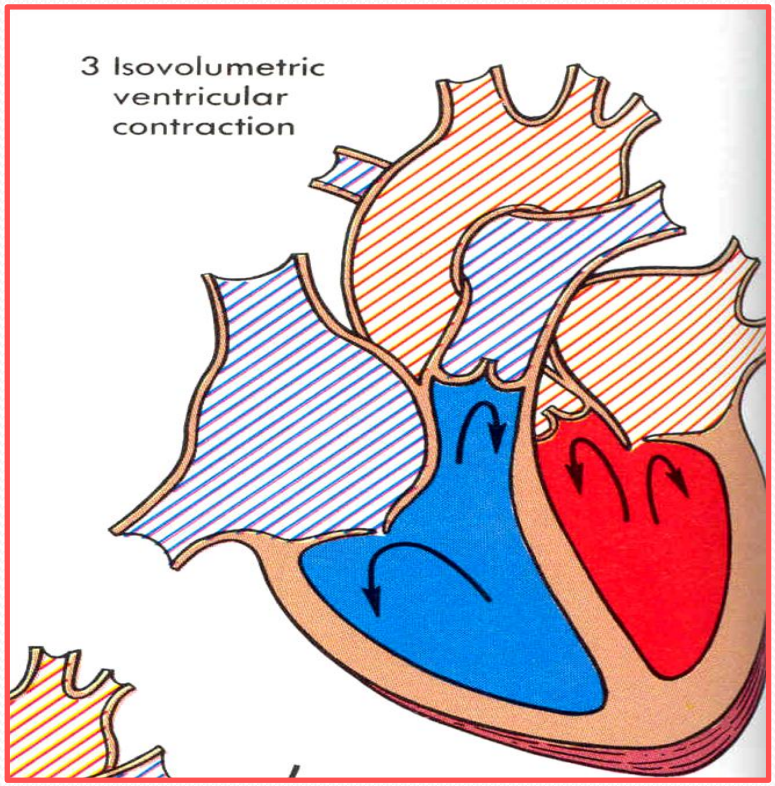
phase 2

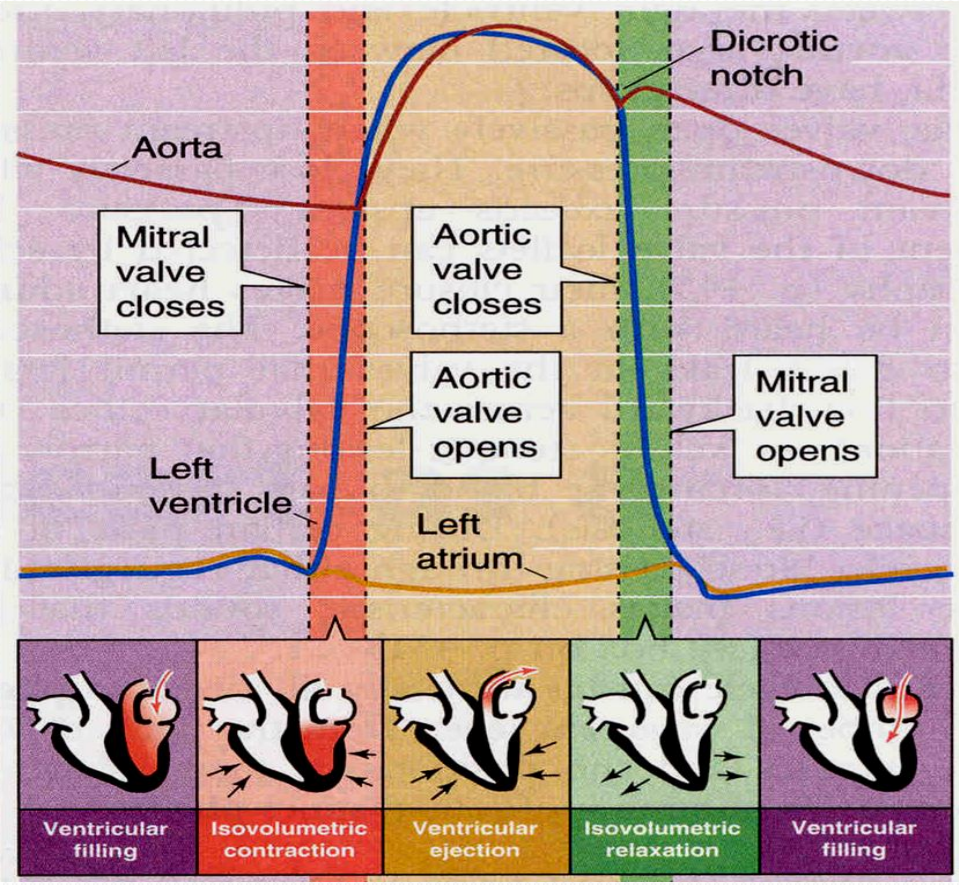


During these 2 phases of ventricular filling:
$P_v < P_a$ - AV valves are opened
$P_v < P_{art}$ – semilunar valves are closed.

Phase 3. isovolumetric contraction of ventricles

- It begins with closer of AV valves - $P_v > P_a$
- Semilunar valves also are closed, because $P_v < P_{art}$ and the volume of ventricles remains the same independently of the pressure change.
- It lasts to the moment when $P_v > P_{art}$ and semilunar valves open.



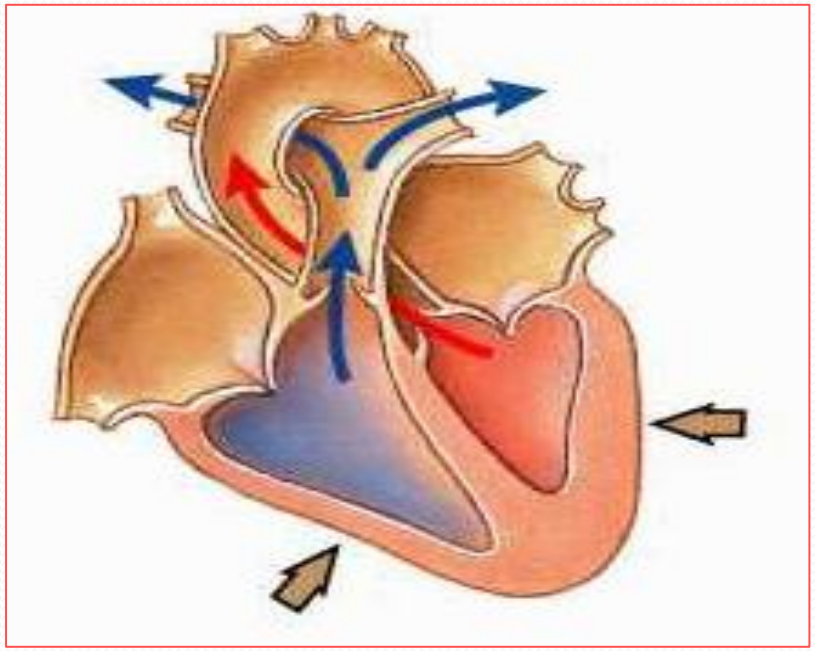


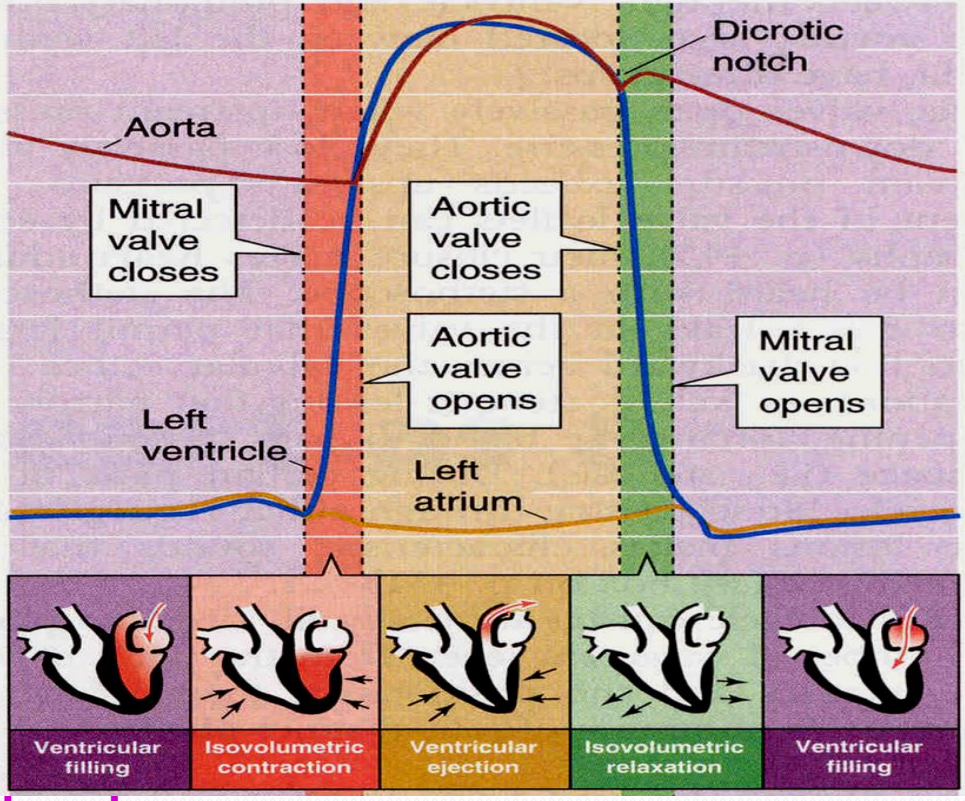
Phase4. ventricular ejection

Two sub phases:

✓**rapid ejection**– $P_v > P_{art}$ and the blood enters arteries with high velocity. The pressure of ventricles and large arteries increases, reaching the maximal value to the end of this period. The volume of the ventricles suddenly decreases.

✓**slow ejection**– the pressure of ventricles and arteries begins to decrease and $P_{art} > P_v$, but the blood flows under inertion.



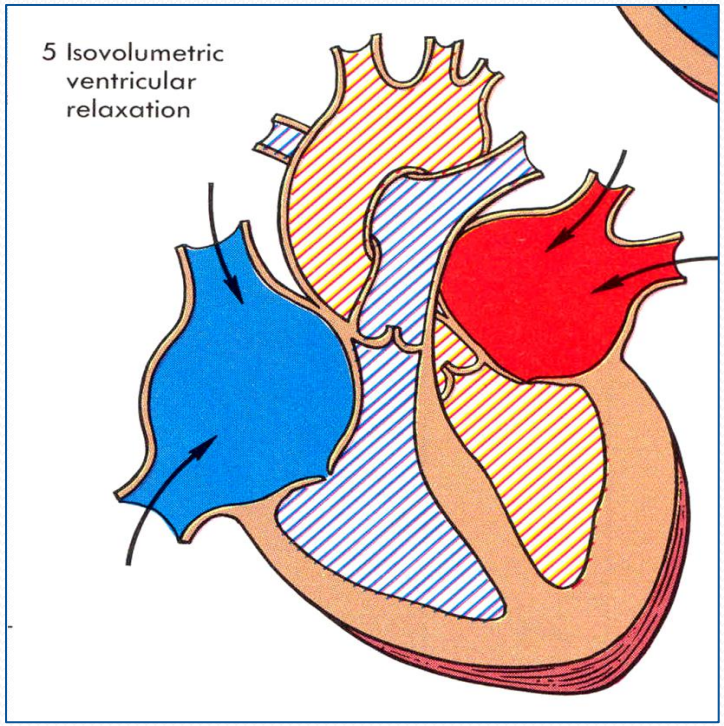


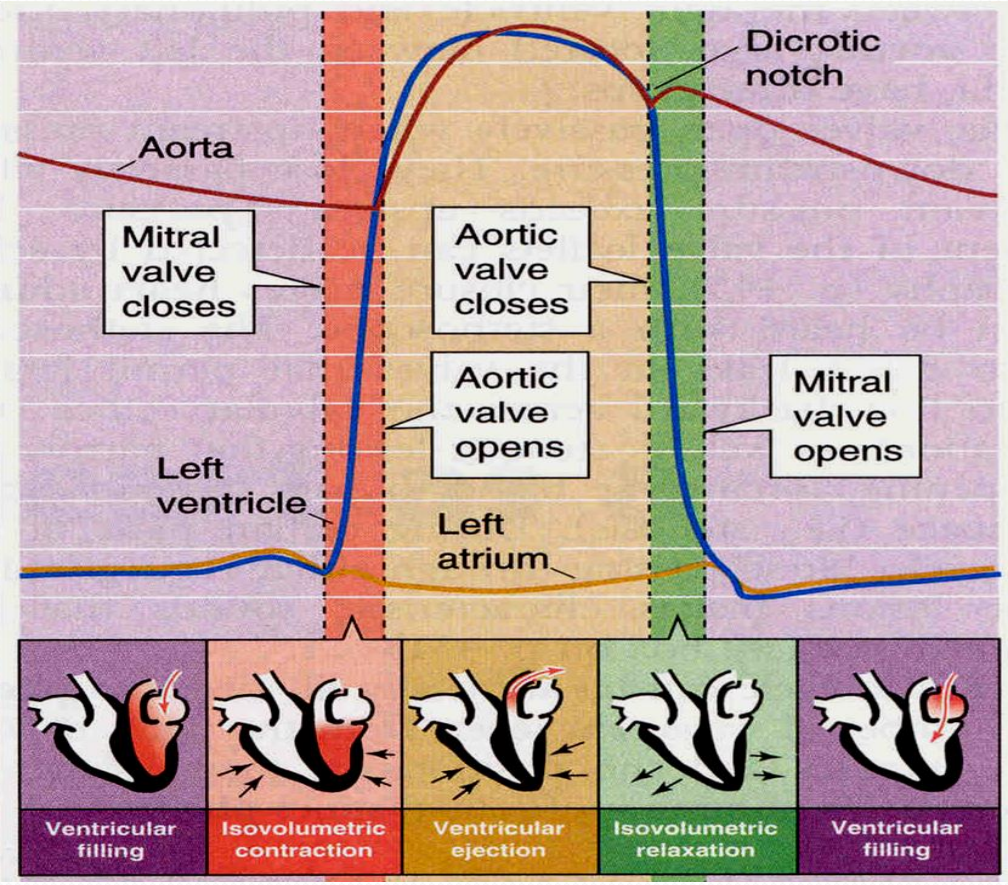
During this phase: $P_v > P_a$ – AV valves are closed
$P_v > P_{art}$ – semilunar valves are opened, but at the end gradient turns

Phase 5. Isovolumetric relaxation

- the ventricular pressure suddenly decreases, because of relaxation of the walls P_v<
-> closer of semilunar valves

- AV valves also are closed and the volume of ventricles remains constant





$P_V > P_A$ - AV valves are closed

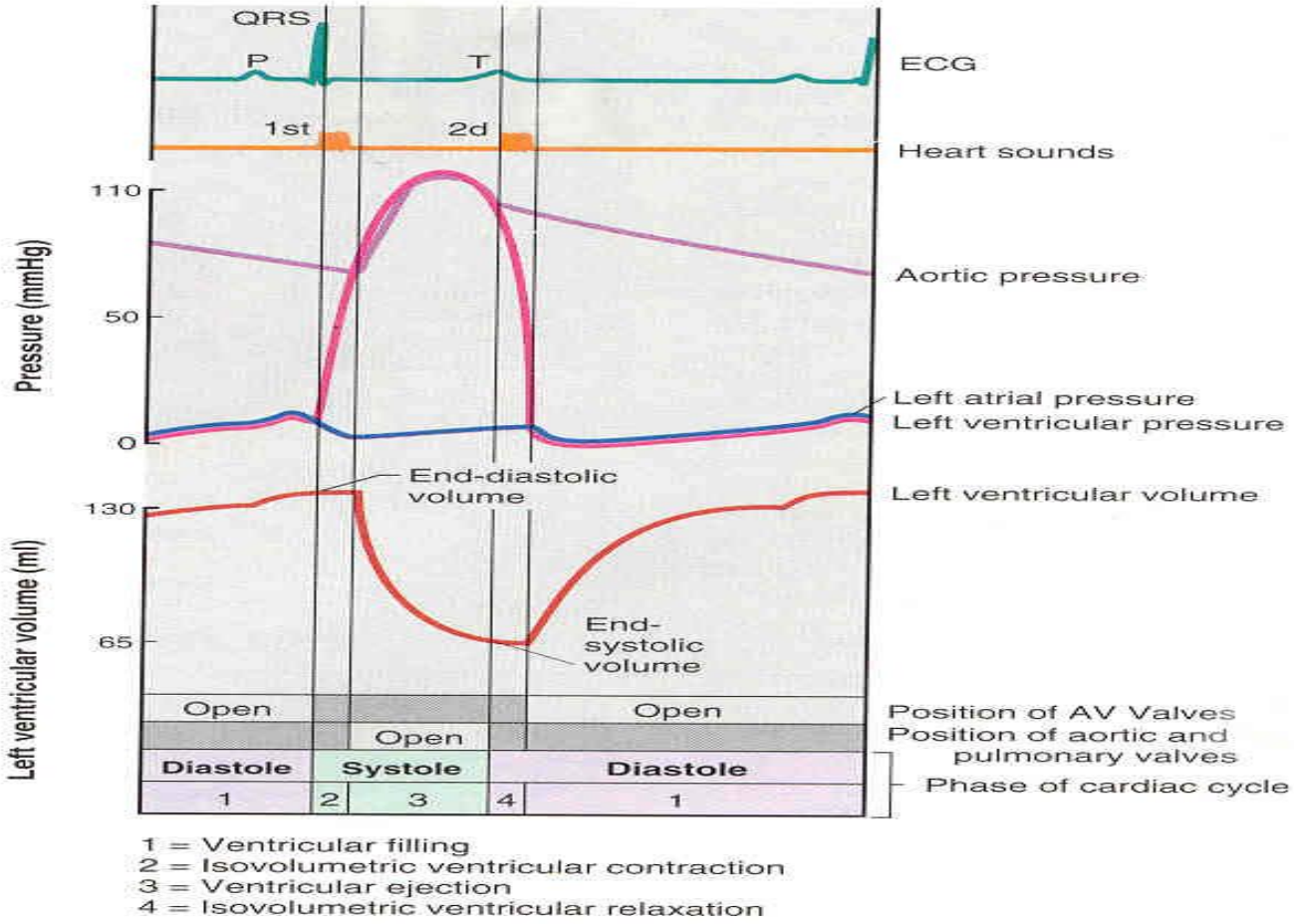
$P_V < P_{ART}$ - semilunar valves also are closed

Phase 1. Ventricular filling

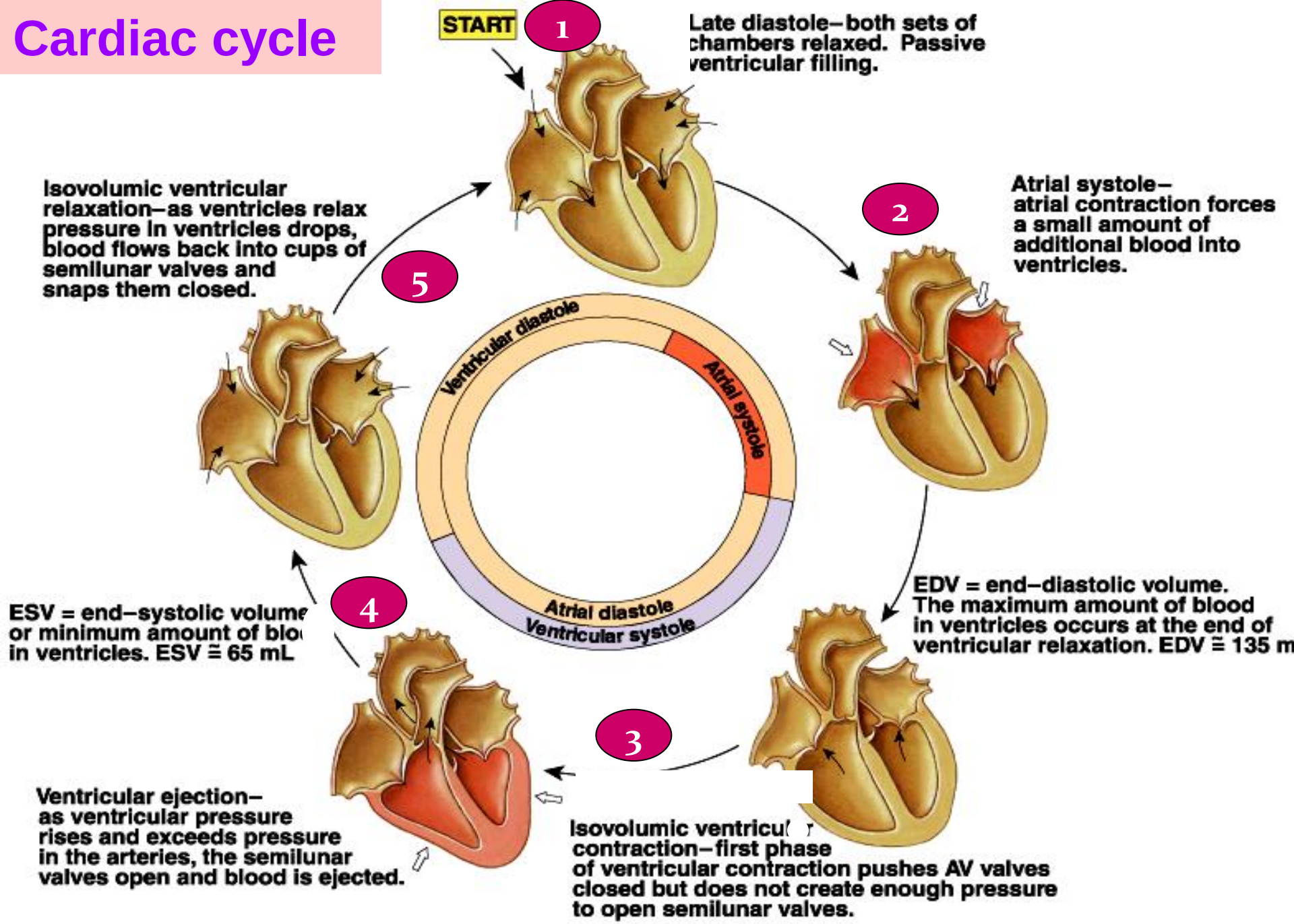
Two sub phases:

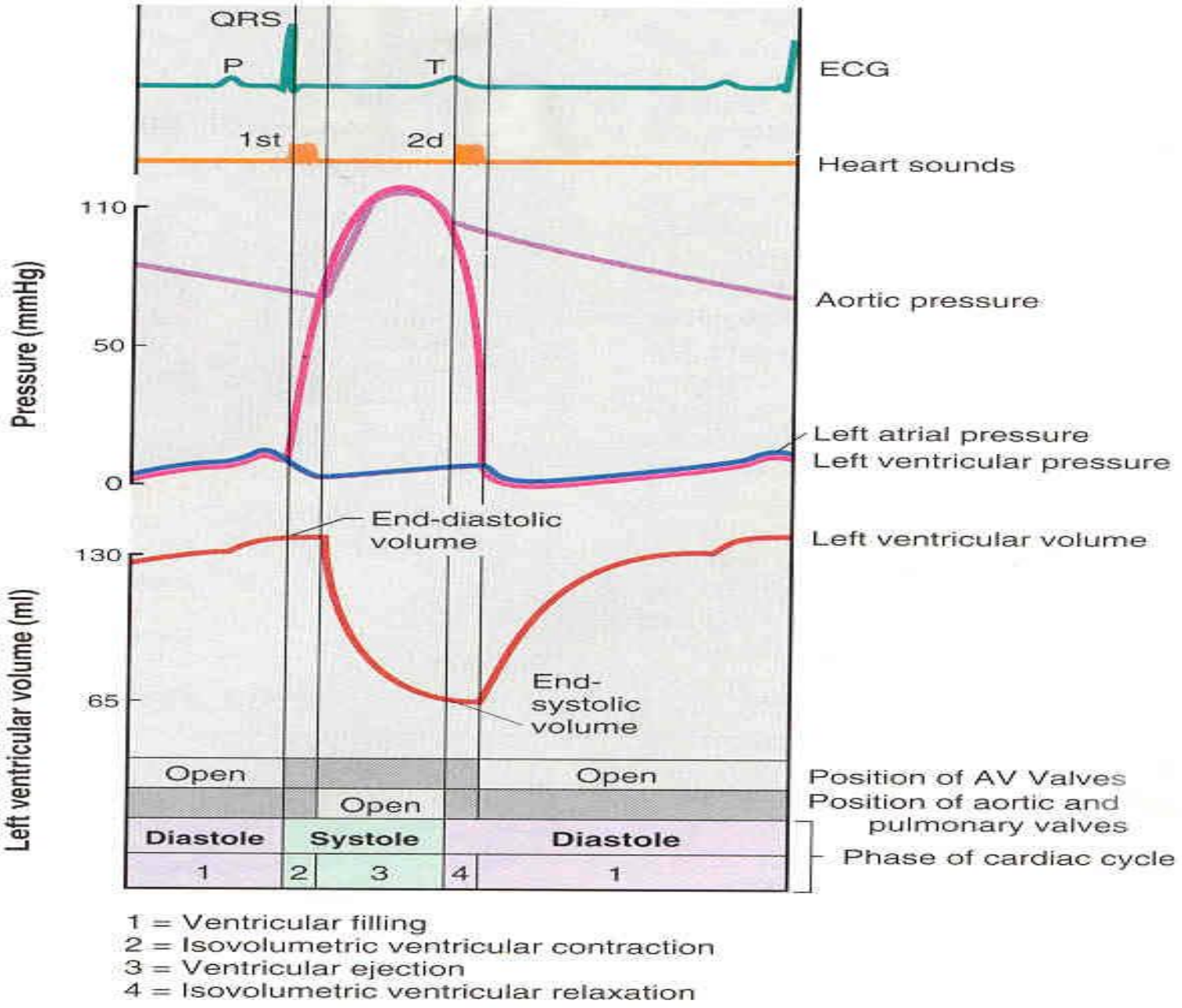
✓**rapid filling**– begins with opening of AV, when $P_v < P_a$. The volume of the ventricles increases rapidly, but their pressure lasts to drop, because of lasting relaxation of the muscles

✓**slow filling**– the pressure of the ventricles increases, because of decreased compliance with increase of the volume



Cardiac cycle

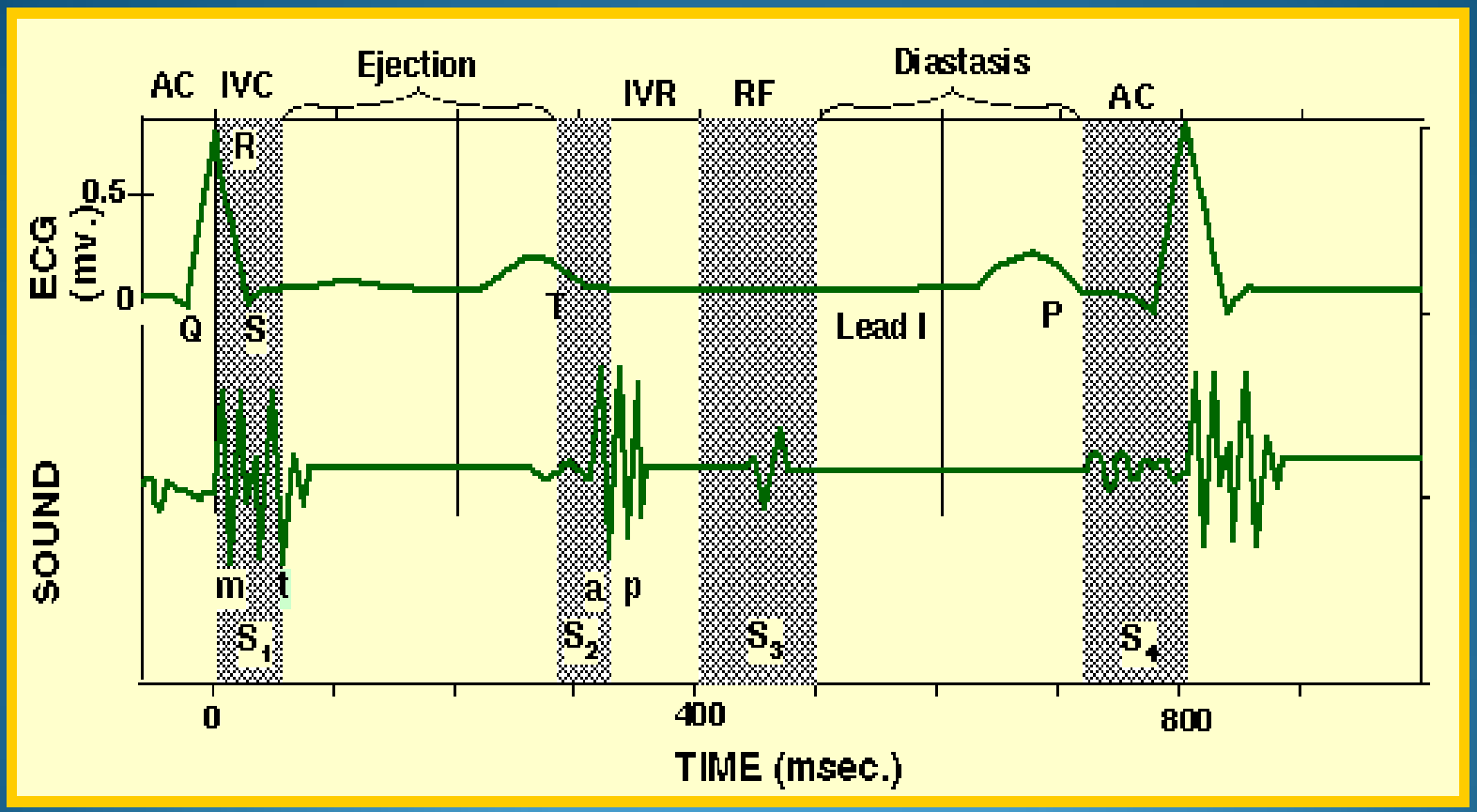



QRS
P
T
ECG
1st
2d
Heart sounds
110
50
0
Pressure (mmHg)
Aortic pressure
Left atrial pressure
Left ventricular pressure
End-diastolic volume
Left ventricular volume
130
65
Left ventricular volume (ml)
End-systolic volume
Open
Open
Open
Position of AV Valves
Position of aortic and pulmonary valves
Diastole
Systole
Diastole
1
2
3
4
1
Phase of cardiac cycle
1 = Ventricular filling
2 = Isovolumetric ventricular contraction
3 = Ventricular ejection
4 = Isovolumetric ventricular relaxation

Heart sounds

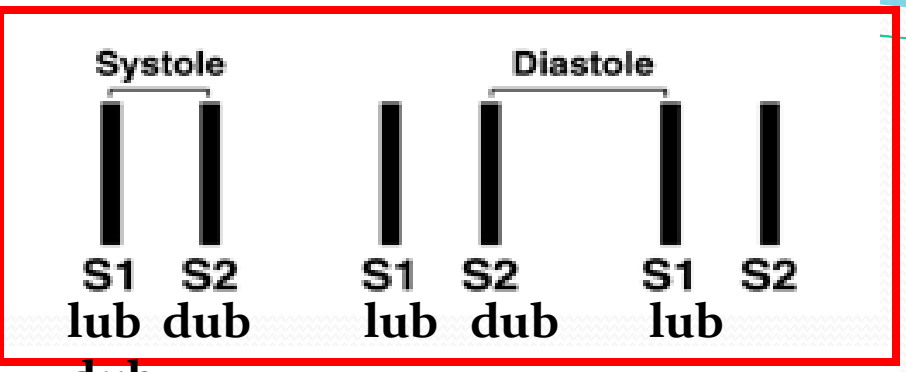
We can hear always 2 heart sounds.

- First (S1) – systolic
- Second (S2) – diastolic

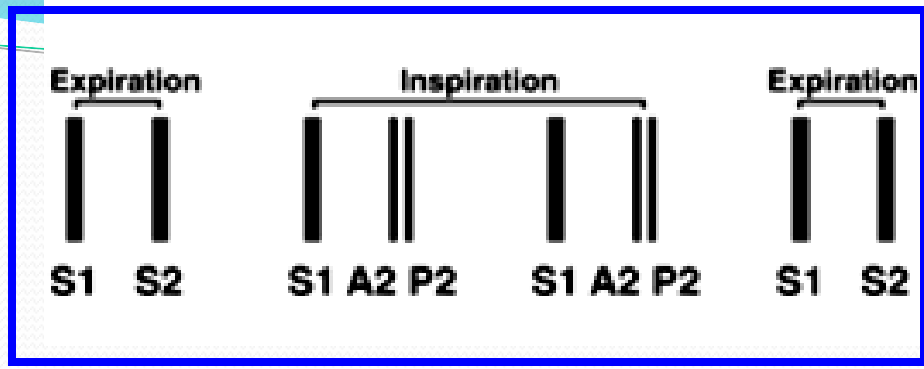


We may register S3 or S4 heart sounds.

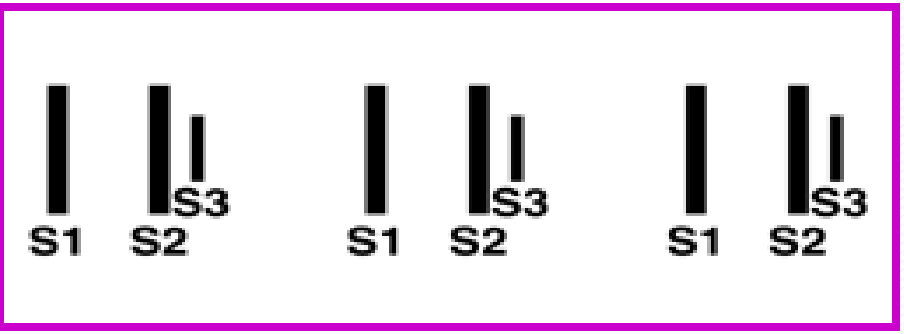
NORMAL CARDIAC CYCLE



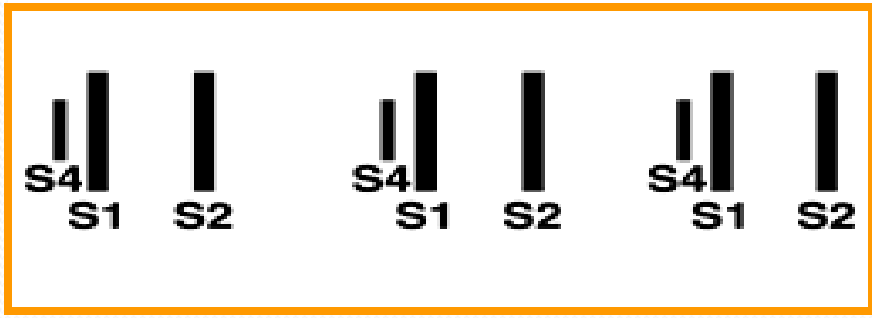
PHYSIOLOGIC SPLITTING OF S2



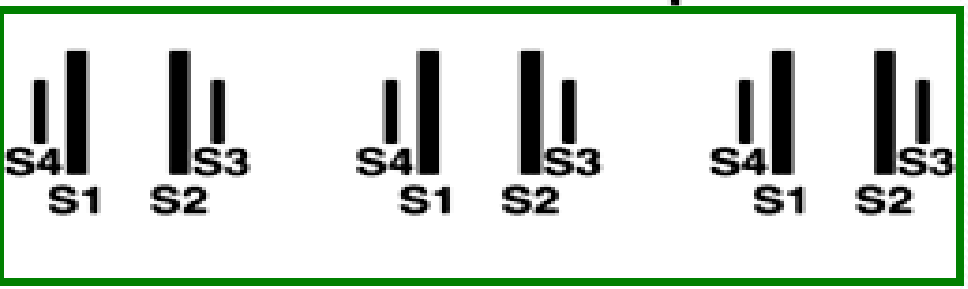
EXTRA HEART SOUNDS - S3

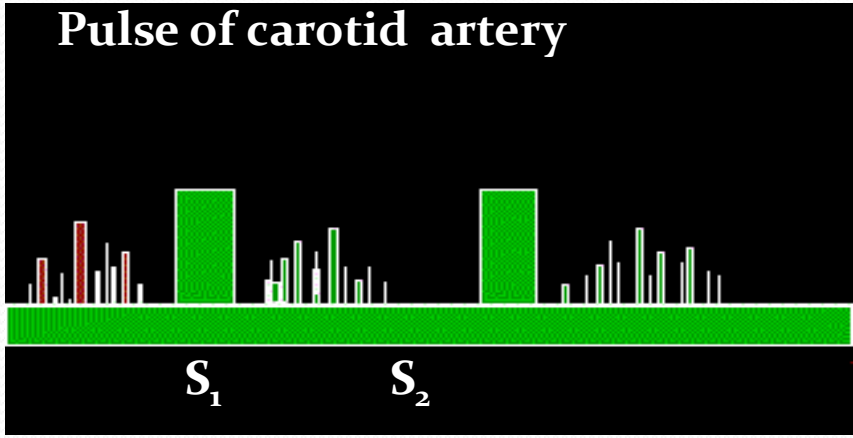


EXTRA HEART SOUNDS - S4

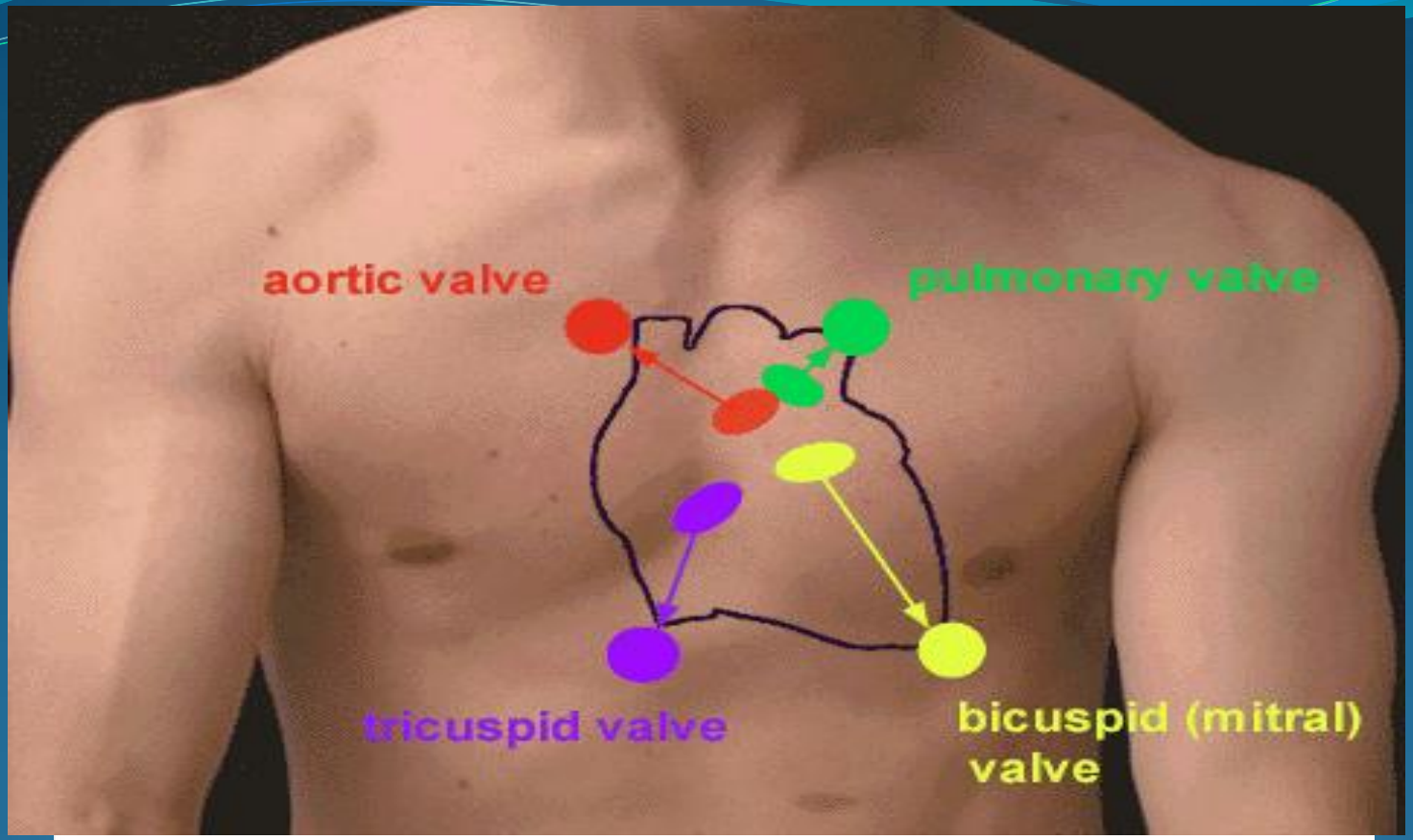


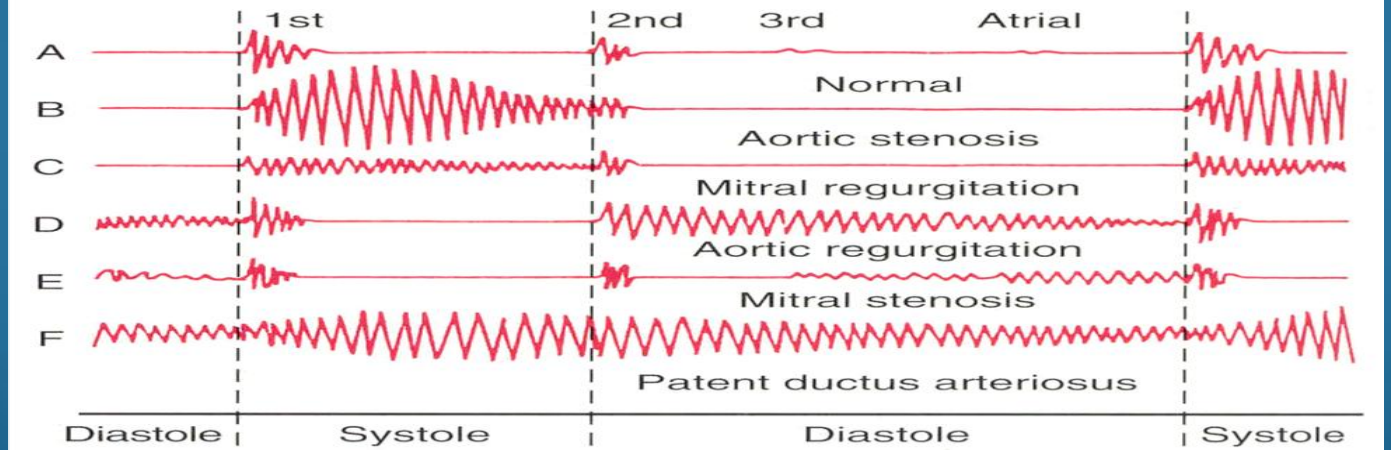
EXTRA HEART SOUNDS - S3 AND S4
Summation Gallop





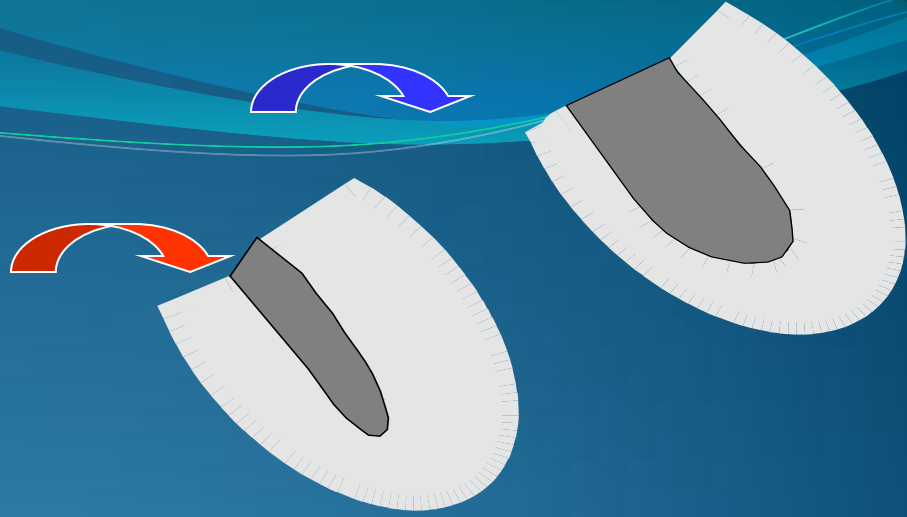
Auscultatory sites of the heart valves



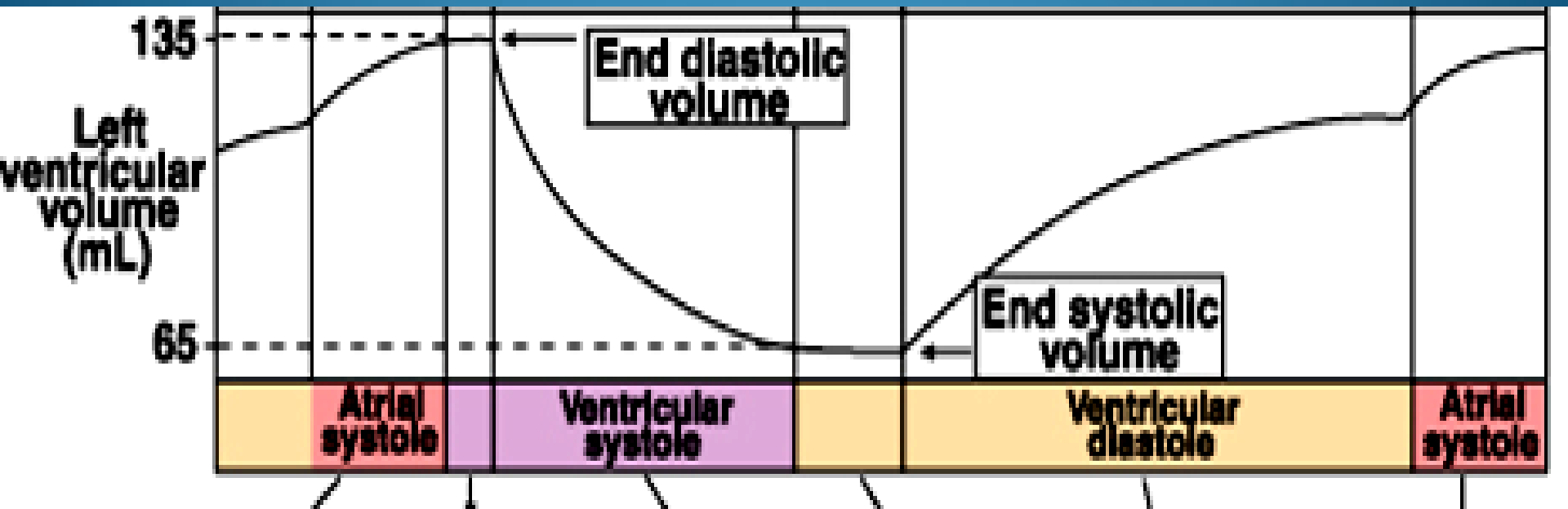


Heart volumes

- ✓**end diastolic volume =** 120-140 ml
- ✓**end systolic volume** = 50-65 ml
- ✓**stroke volume =** 70 ml
- ✓**cardiac output = 4-6 l/min; av. 5,25 l/min**



- ❑ **The stroke volume of both ventricles must equal!!!**

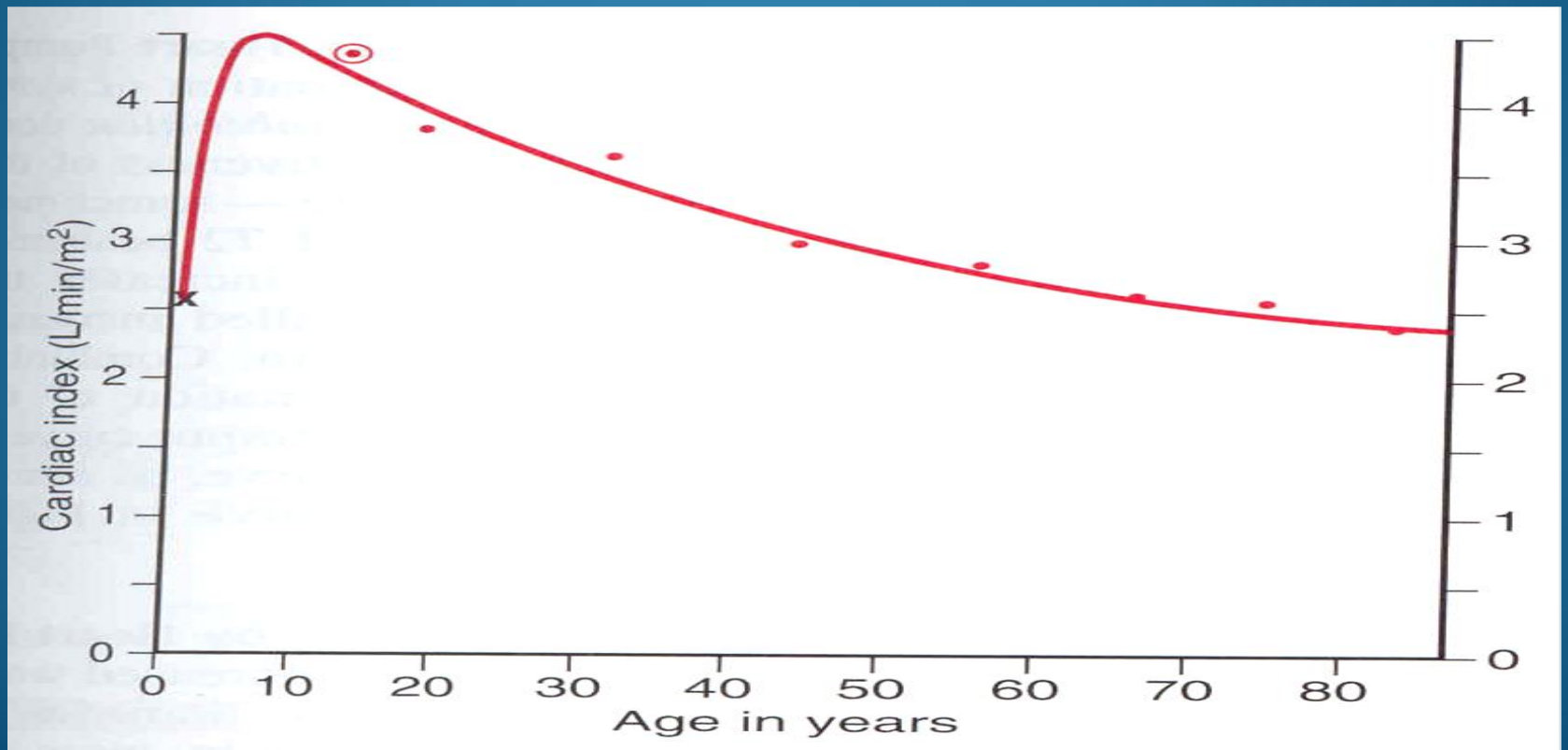


Cardiac output depends on:

- ✓ age
- ✓ sex
- ✓ body surface area
- ✓ physical activity

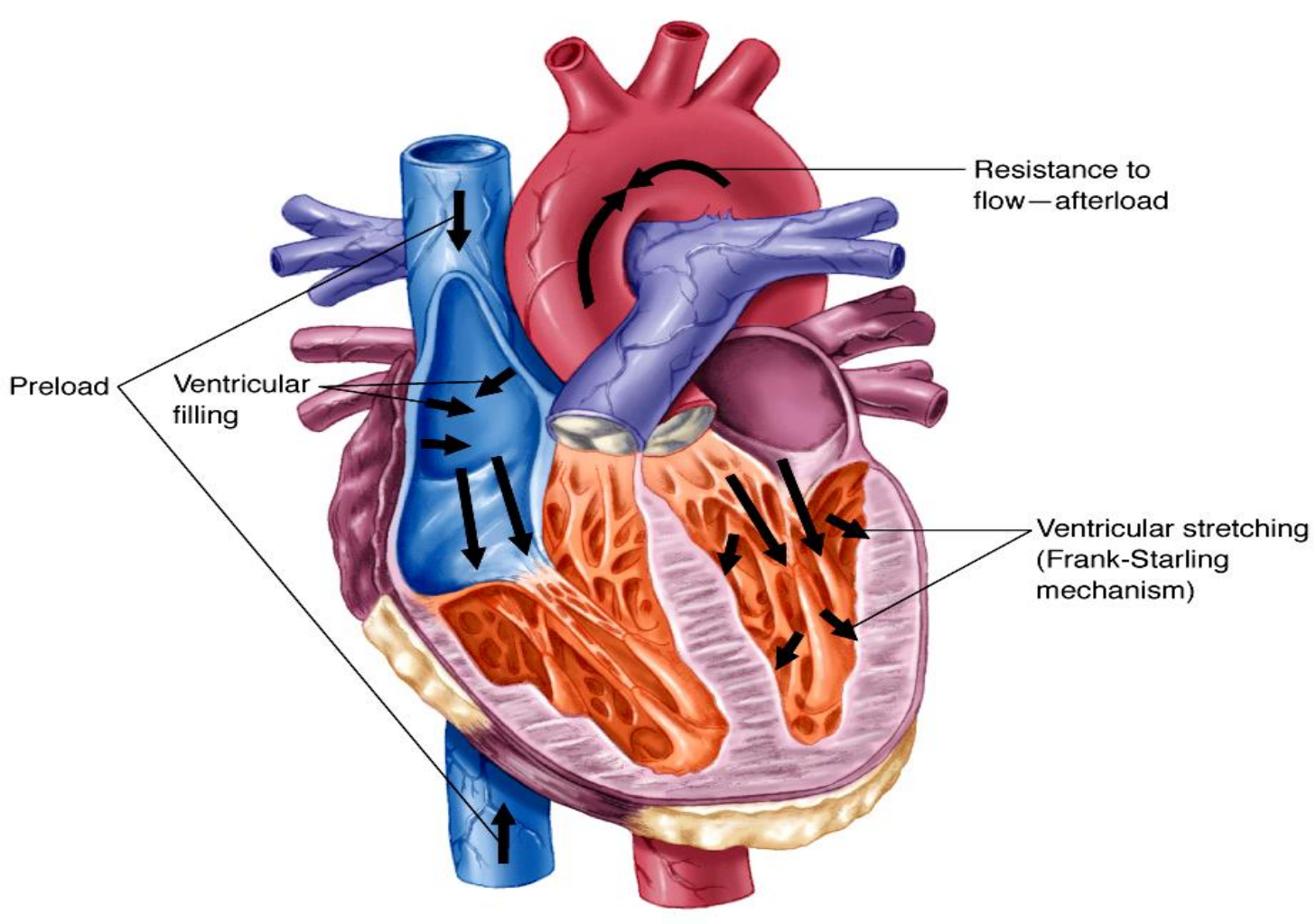
➢ cardiac index = CO : BSA ~ 3 l /min / m^2

❖ CI decreases with aging after 10 years of age.

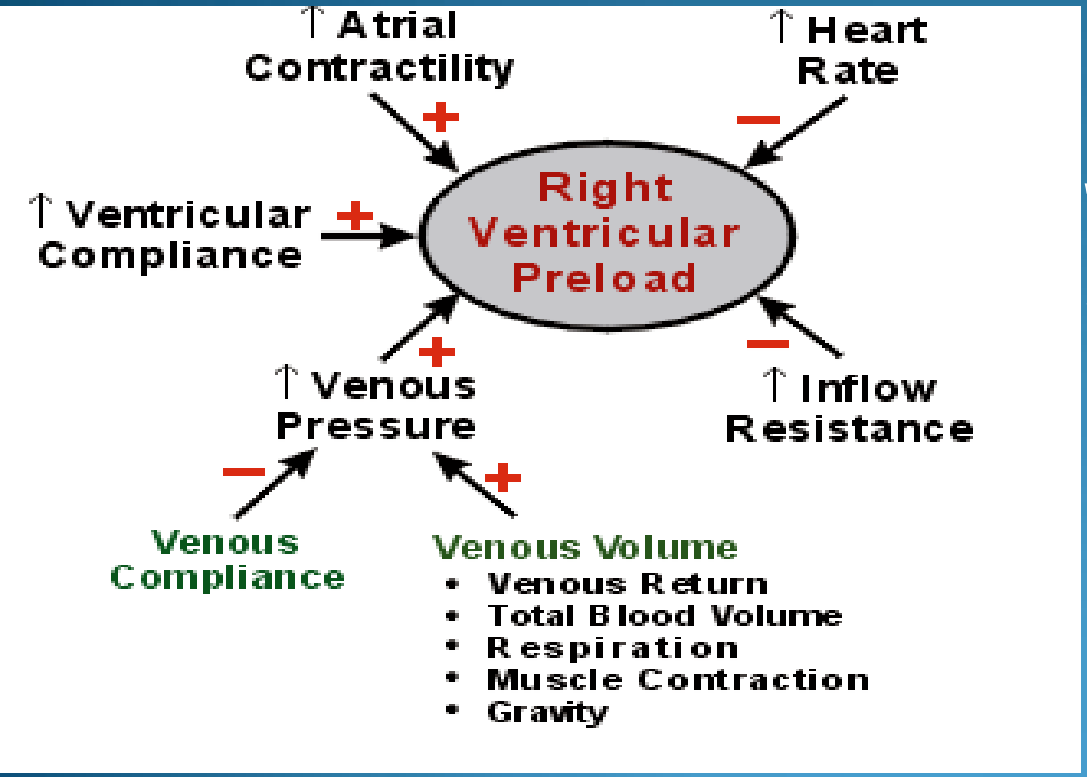


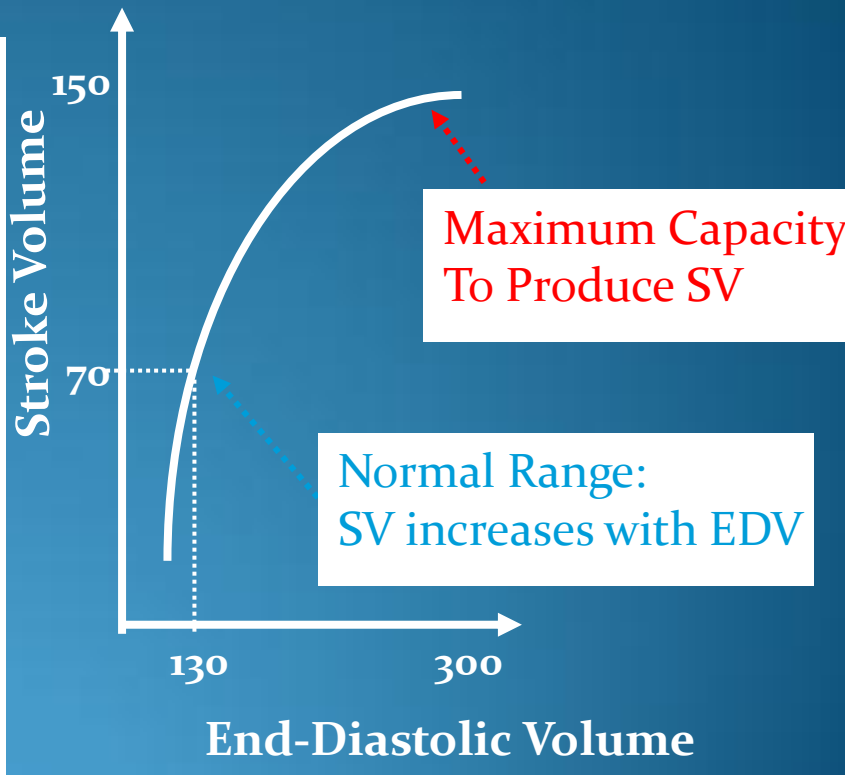
Factors on which stroke volume depends :

- ✓ preload – ventricular filling
- ✓ afterload – resistance of big vessels
- ✓ ventricular contractility

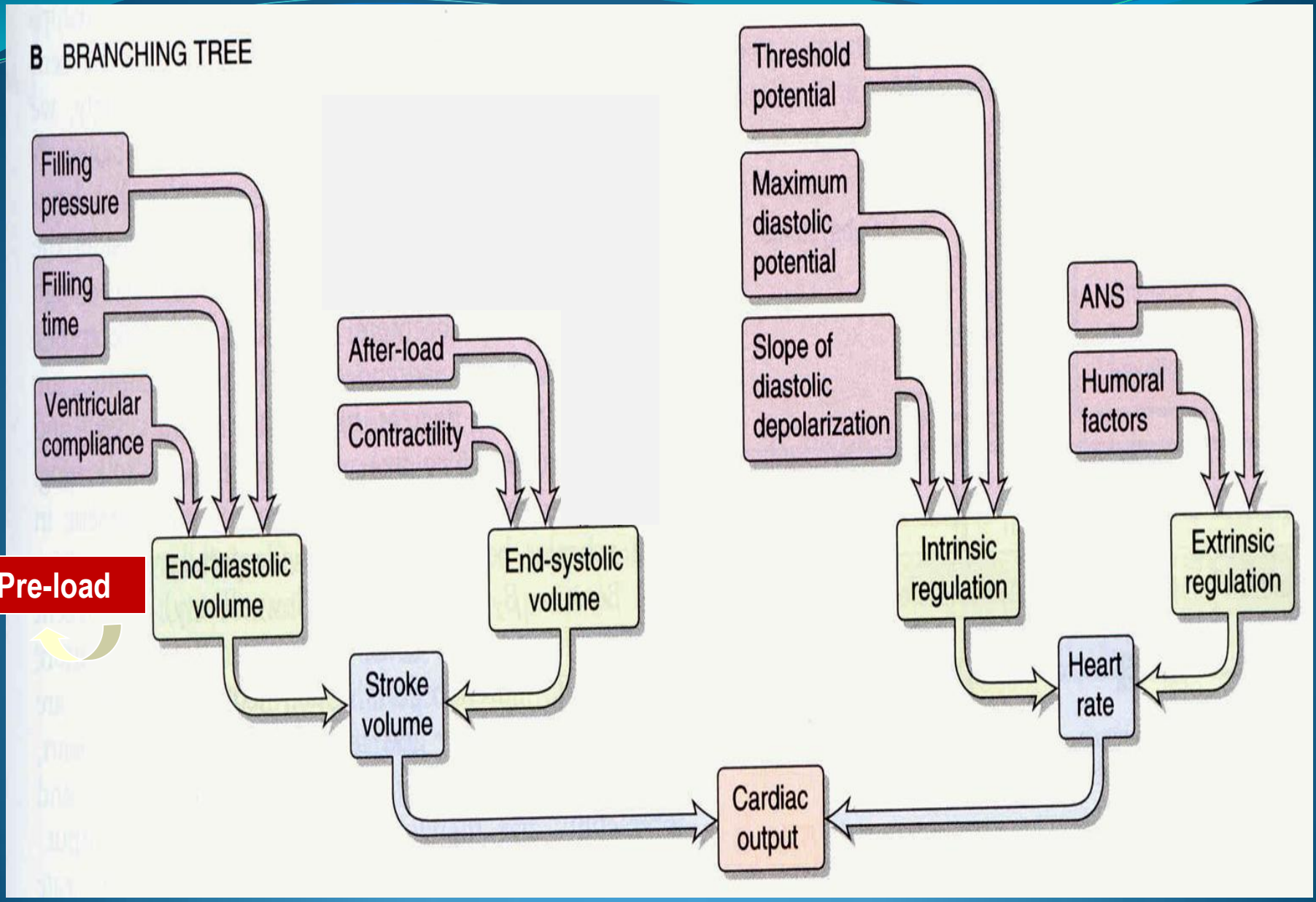


❖Preload depends on:

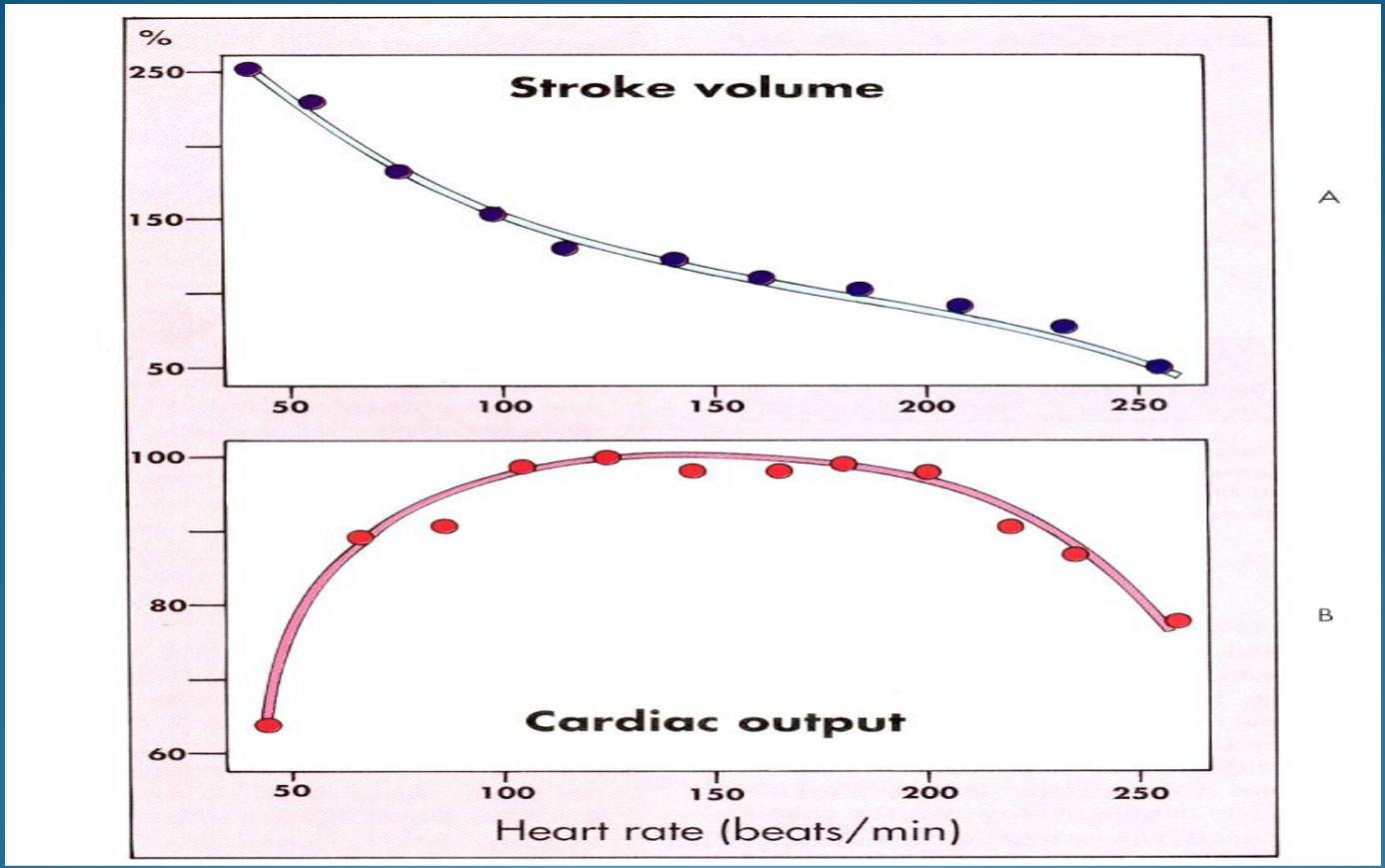




Factors on which cardiac output depends



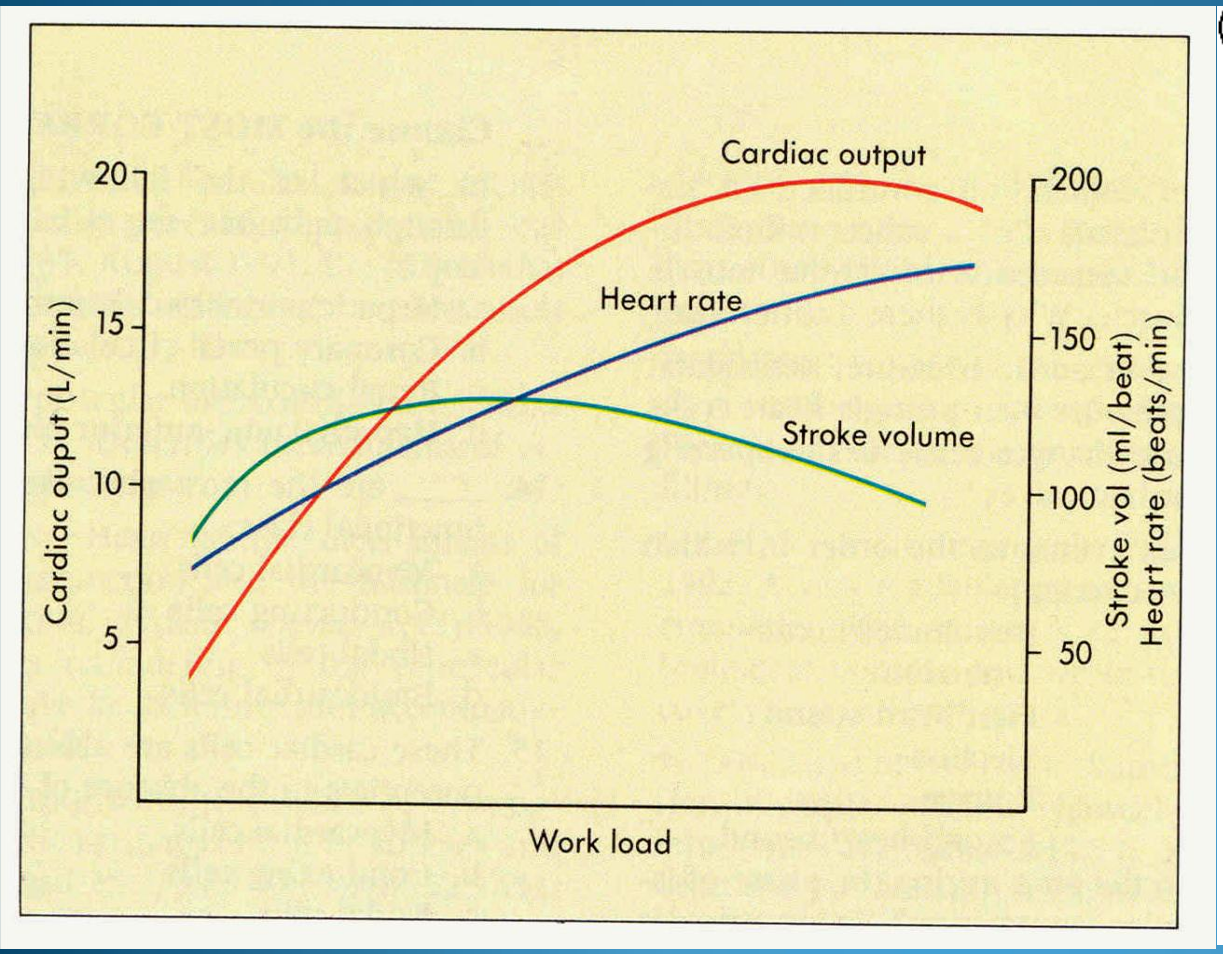
Cardiac output	=	Stroke volume	x	Heart rate

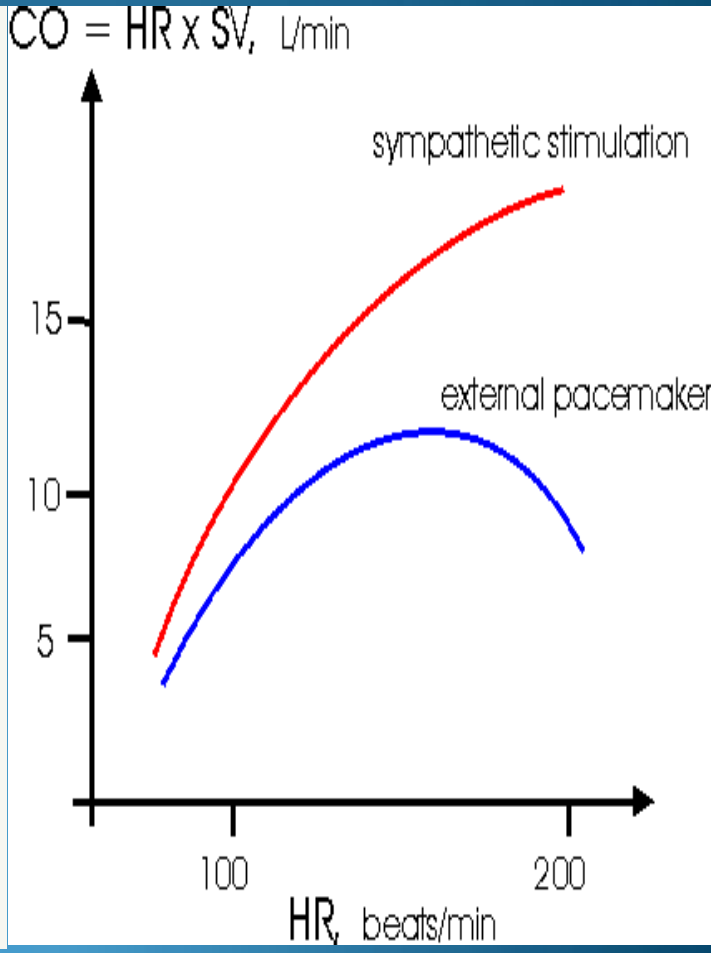


- Stroke volume decreases during increased heart rate.
- Cardiac output increases during increase of heart rate from 50 to 100 b/min.
- After that remains relatively constant and decreases when HR is higher than 200 b/min.

❑ During exercises cardiac output increases 5 (7) times:

- ✓ ↑HR ; max HR= 220 – age
- ✓ ↑contractility

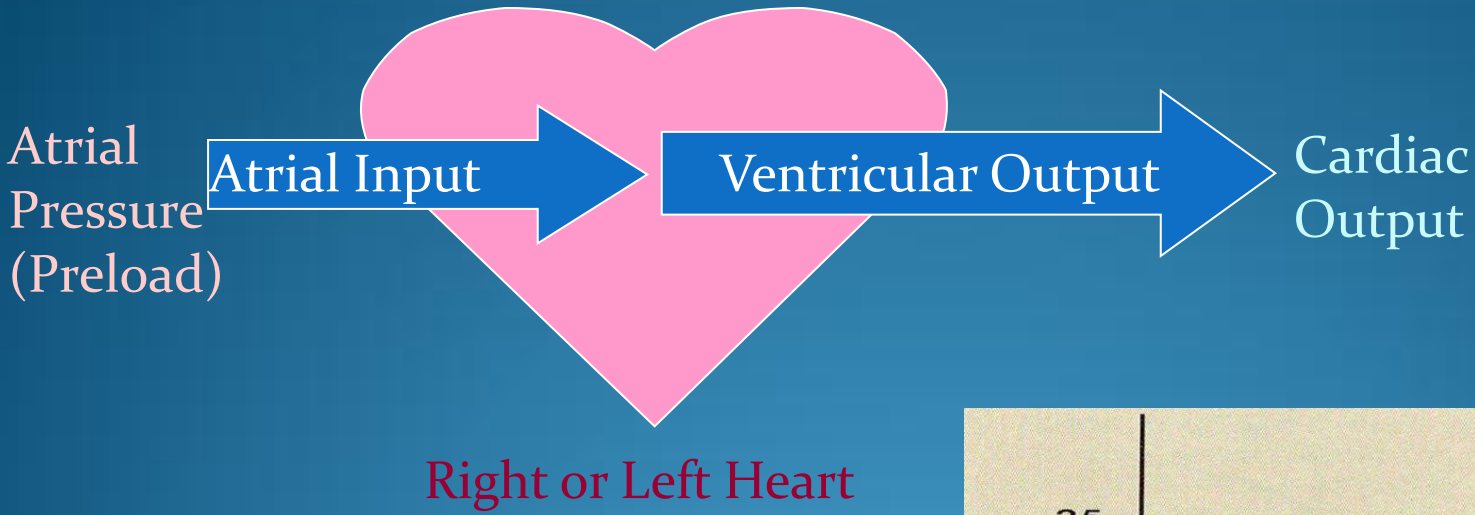


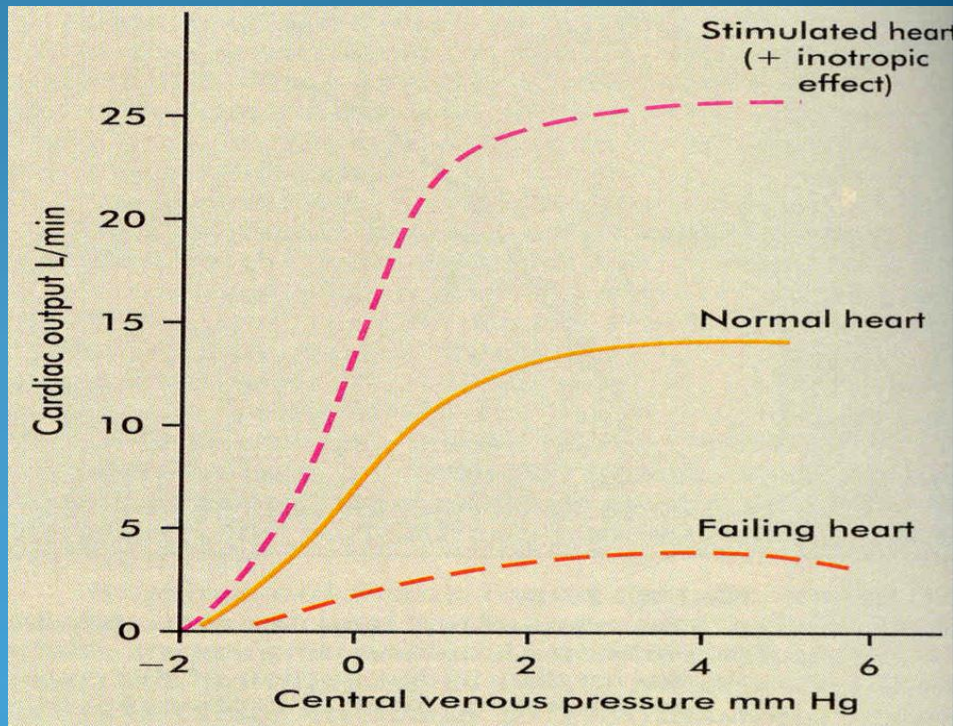


Functional curves of the heart

- They connect pump function of the heart with the peripheral factors determining the return of the blood to the heart.

- The venous return is the main factor that determines EDV!!!





The heart work

❖ The heart performs external work to eject stroke volume into the two rings of circulation and to give acceleration of blood to flow:

✓ejection of the stroke volume against the pressure (Ws)

$Ws = P.V$

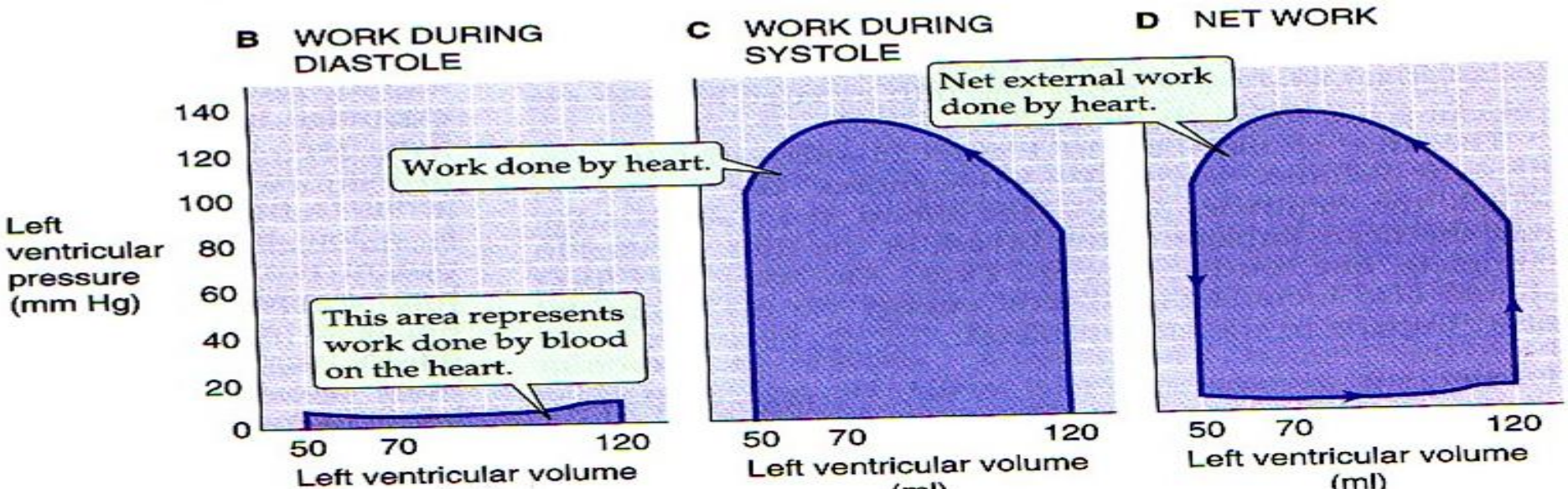
✓Kinetic work(Wk)

$Wk = \frac{1}{2} mV^2$

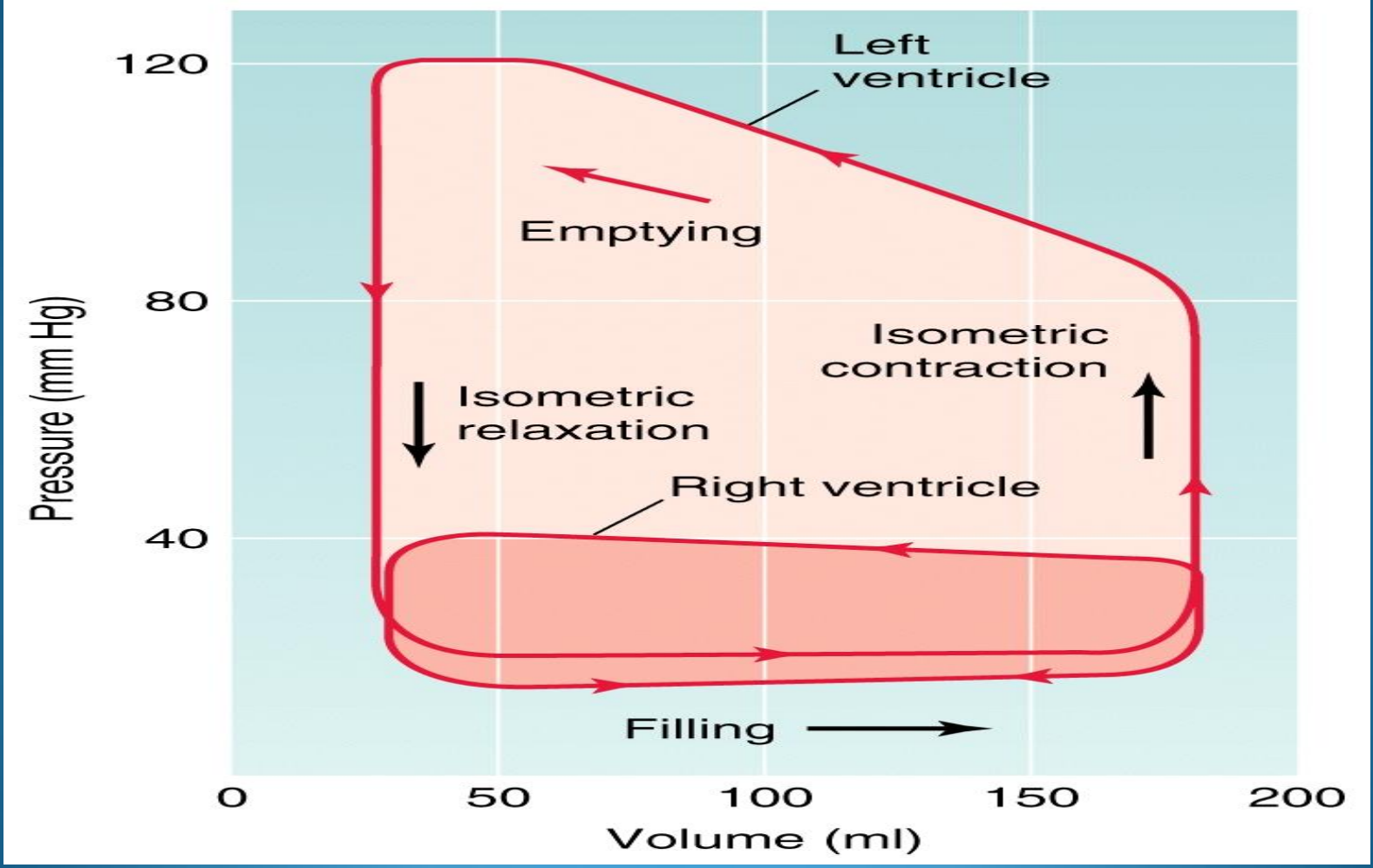
$E = P.V + \frac{1}{2} mv^2 + k.T. \Delta t$

$k.T. \Delta t$ –the heat during isovolumic contraction

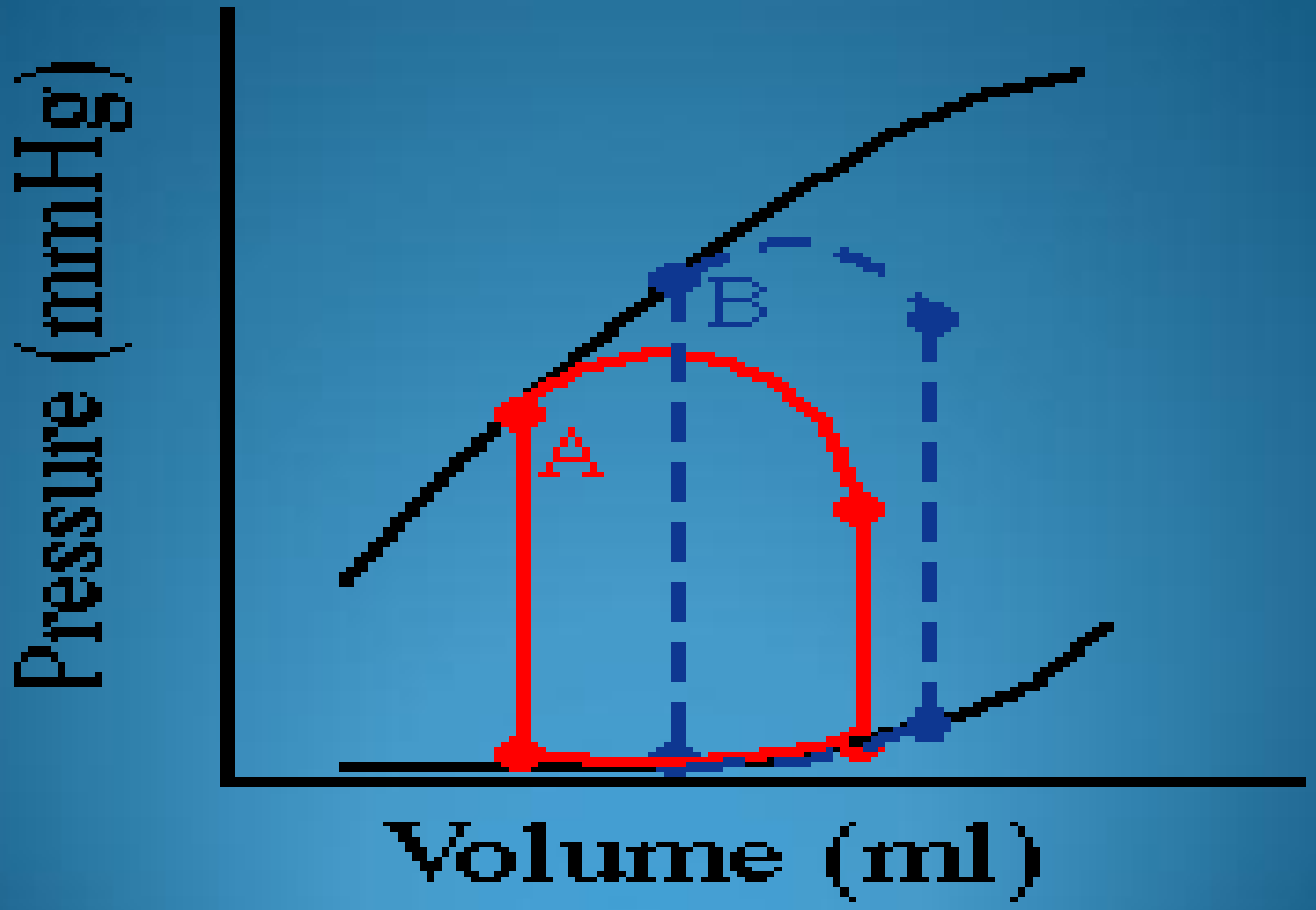
$W_{total} = P. V + \frac{1}{2} mV^2$



- The external work of left ventricle is 5 times greater than this of right ventricle, because 5 time greater is its tension during the systole.



❑ During one the same external work the heart uses much energy when works at the condition of increased afterload (B), than the situation of increased preload (A).



Myocardial metabolism

- ❖ oxidative phosphorilation of:
  - ✓fatty acids 60%
  - ✓glucose, lactic acid - 35-40%
- ➢the heart has high oxygen consumption(OC)

Cardiac State	MVO_2 (ml O_2/min per 100g)
Arrested heart	2
Resting heart rate	8
Heavy exercise	70

Organ	O_2 Consumption (ml O_2/min per 100g)
Brain	3
Kidney	5
Skin	0.2
Resting muscle	1
Contracting muscle	50

Autoregulation

- It ensures adaptation of the heart to the changes of hemodynamic conditions without participation of extracardial factors.

2 mechanisms:

➢ heterometric (law of *Frank – Starling)*

- It triggers when the length of myocytes is changed before start of contractio (↑EDV)

❖ It ensures stronger than normally contraction to decrease EDV.

➢homeometric

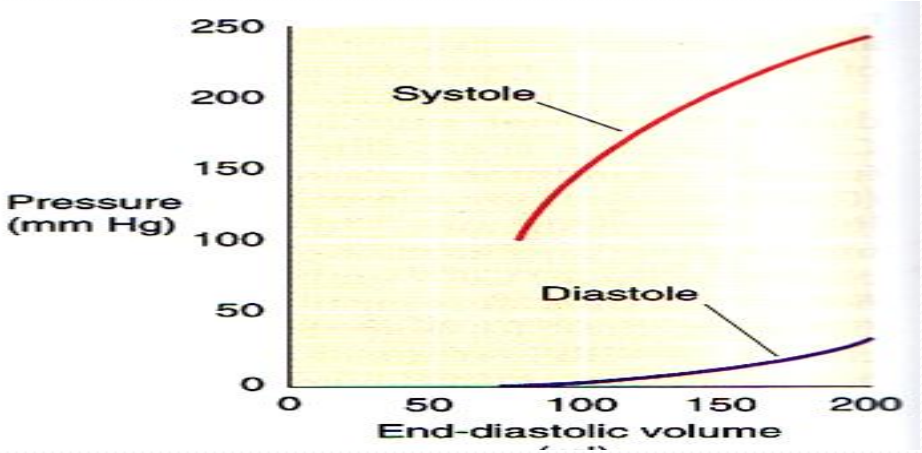
- It triggers without change of the length of myocytes:

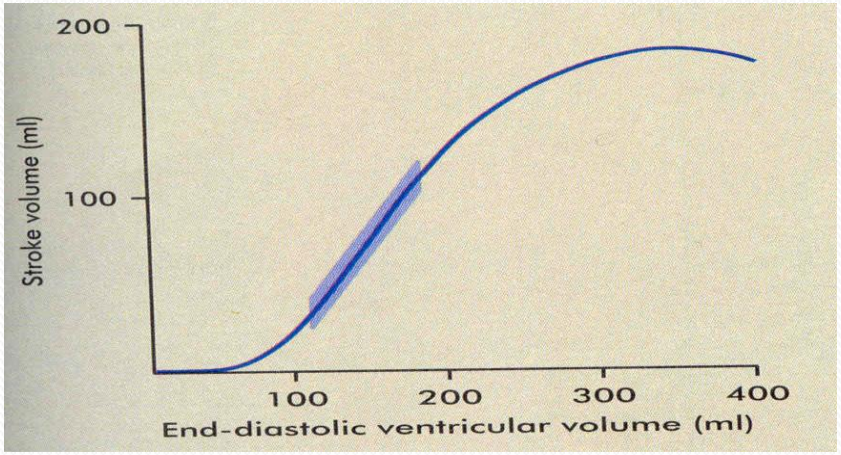
✓change of HR- effect of *Bowditch*

✓increased afterload effect of *Anrep*

❖ It ensures stronger than normally contraction during increased HR and arterial pressure

Heterometric autoregulation





- Increased EDV lengthens the cardiomyocytes and this leads to:

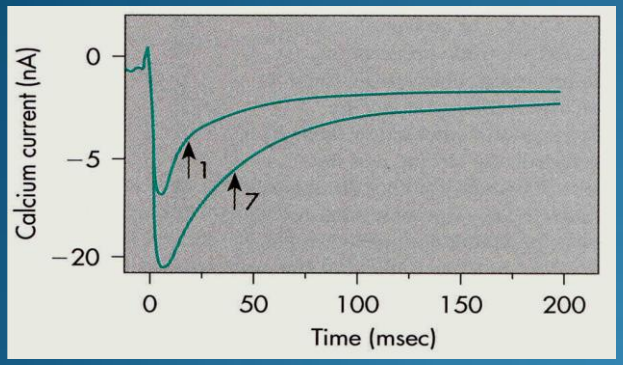
✓ increased sensitivity of troponin C to Ca^{2+} and increased rate of formation and splitting of cross bridges between myosin and actin.

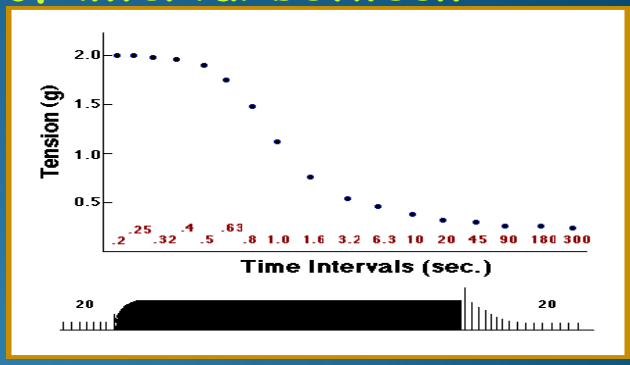
Homeometric autoregulation

❑ effect of Bowditch

❖ Myocardium develops higher tension with shortening of interval between stimuli.

- it is due to increase of $[Ca^{2+}]_i$





❑ effect of Anrep

❖ Sudden increase of aortic pressure causes:

✓decrease of SV, ESV and EDV increase.

❑ Mechanism of Frank and Starling makes improvement of SV.

Extracardial control on heart performance

- It is nervous and humoral.

Nervous regulation

- Parasympathicus –n.vagus

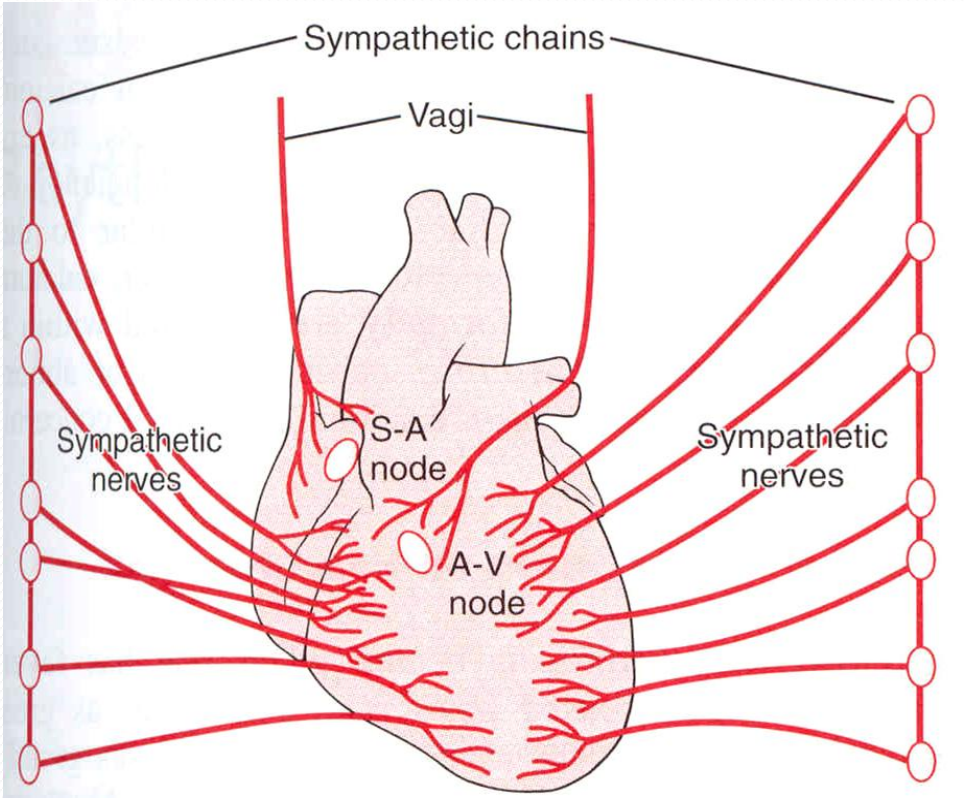
✓right n.vagus -> SA node

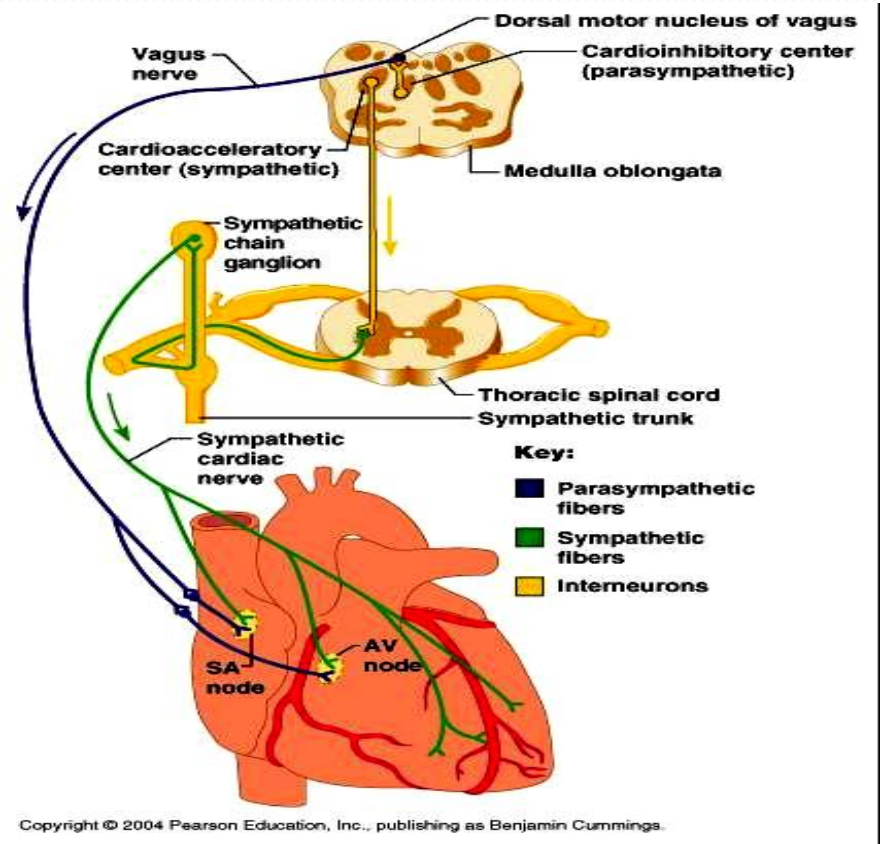
✓left n.vagus – AV node

- atria are innervated by the n. vagus, but ventricles are not

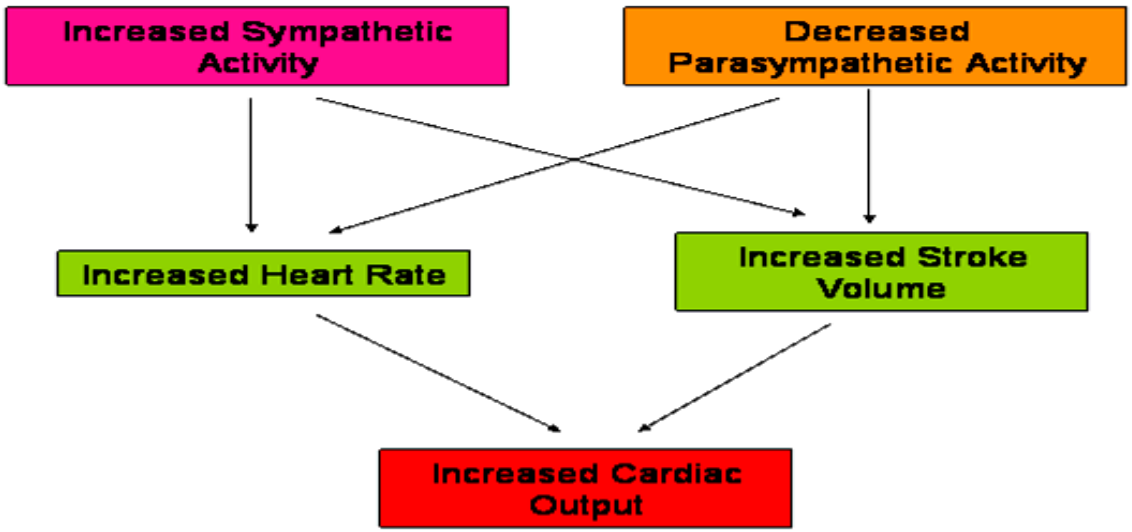
- sympathicus – from upper thoracic segments of spinal cord

- sympathicus innervates whole heart



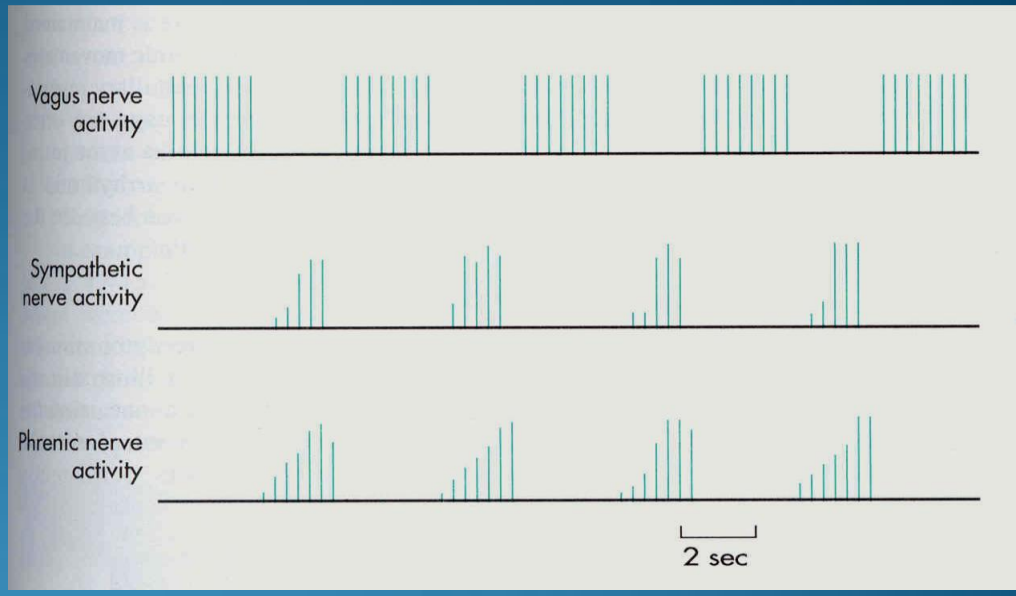


Effects of parasympathicus		Effects of sympathicus
rapid Short time lasting		slow long time lasting
Negative	Chronotropic effect	positive
Negative	Dromotropic effect	positive
Negative : on the atria	Inotropic	positive: on the atria and venticules
Negative	Lusitropic	positive

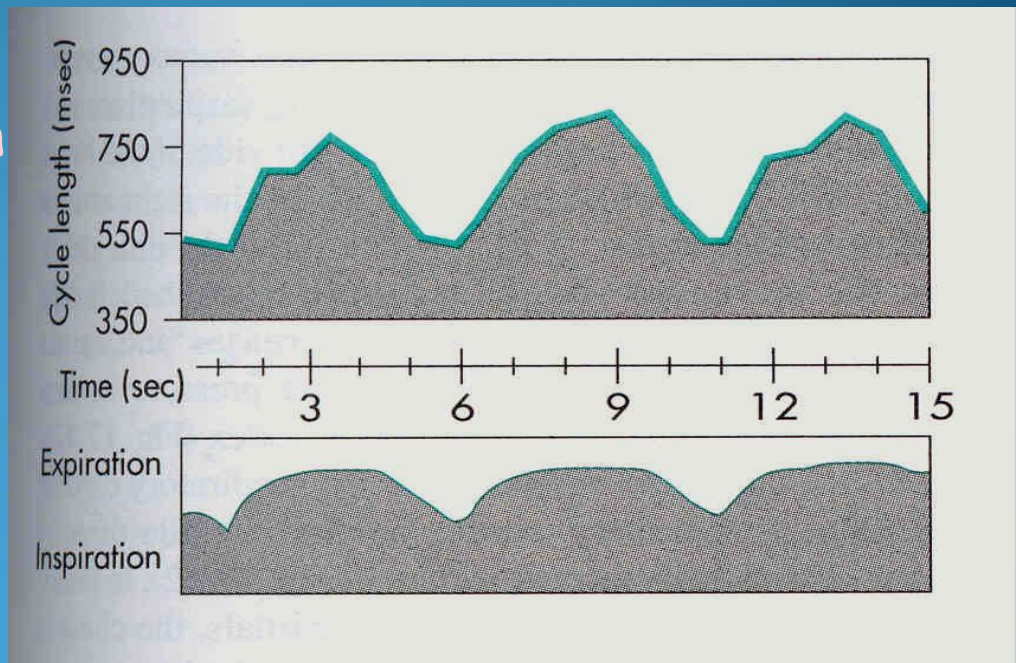


❑ Cyclic changes of the tone of the nerves of autonomic nervous system during respiration cause respiratory arrhythmia.

- The tone of vagus decreases during inspiration and sympathicus tone increases.



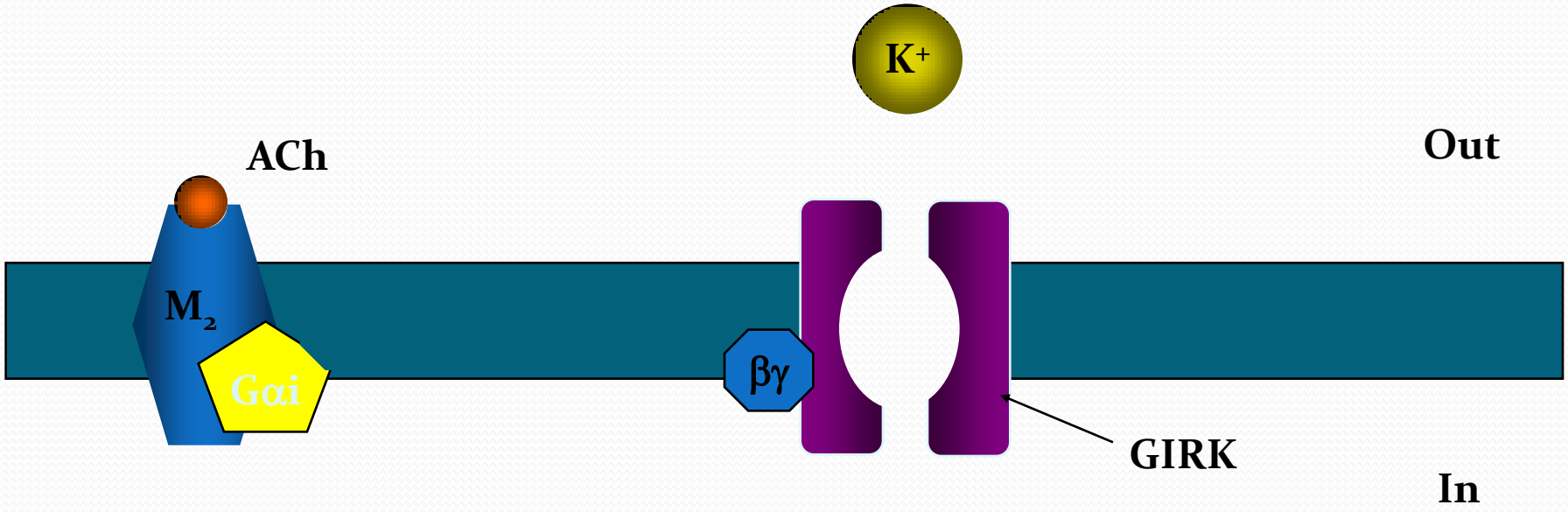
- HR increases during inspiration and decreases during expiration.



❑ Mechanism of action of Acetylcholine on SA node

✓Acetylcholine binds to M_2 choline receptors.

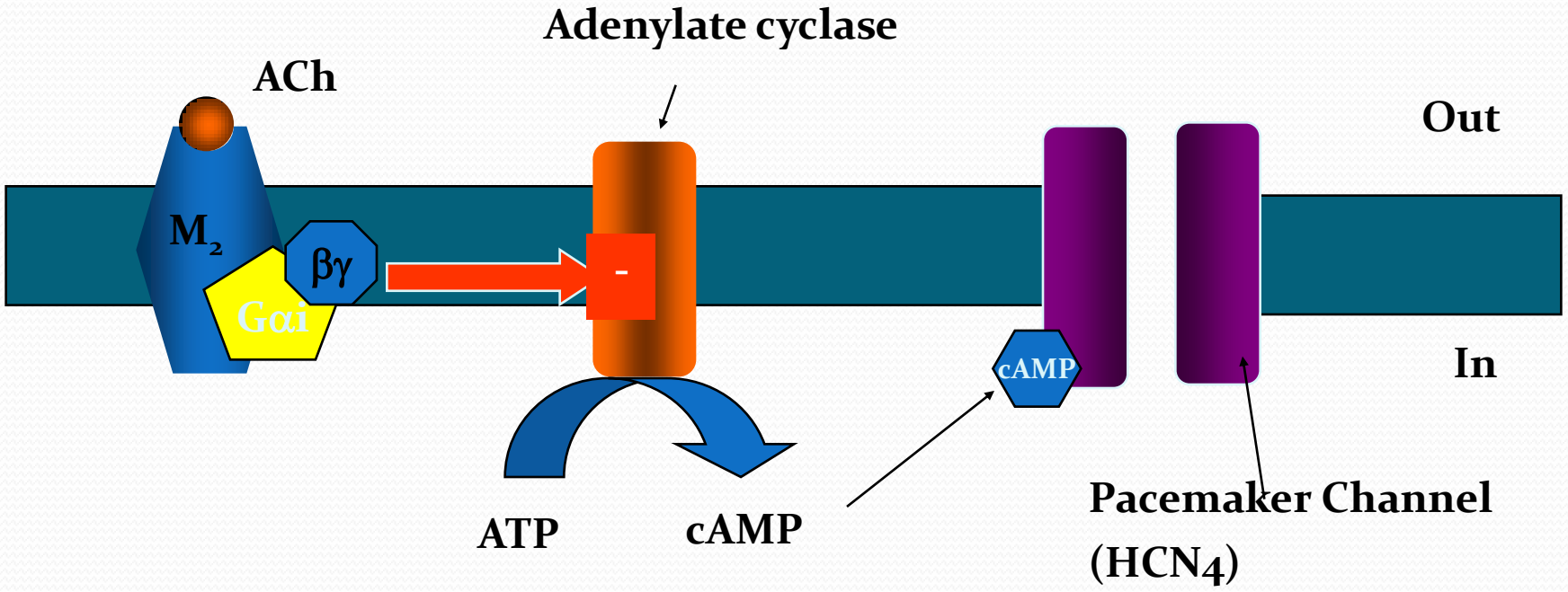
✓M_2 receptors are connected with G protein, its βγ-subunits open K channels (GIRK).



❖ Hyperpolarization of cell membrane slows down diastolic depolarization.

❑ Mechanism of action of Acetylcholine on SA node

➢ M_2 receptors through $G\alpha_i$ inhibit adenylate cyclase -> ↓cAMP -> ↓activity of non selective cation channels, responsible for I_f.

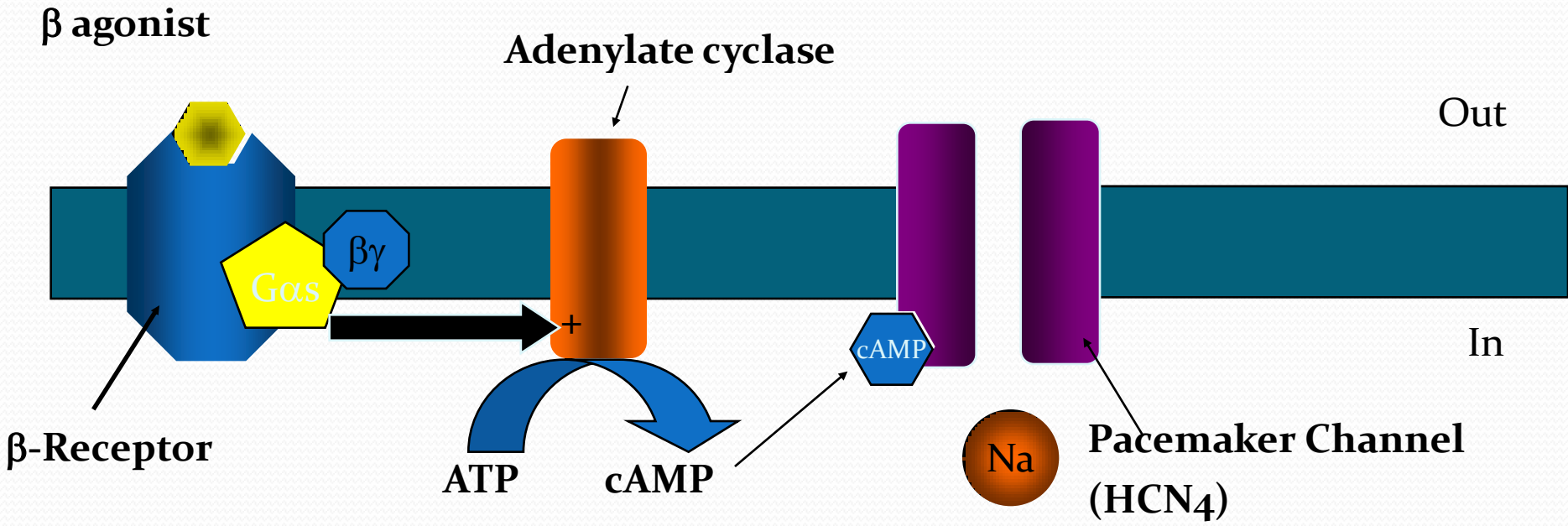


✓This slows down depolarization and decreases HR

❖ Strong vagus stimulation can completely stop generation of AP by SA node!!!

❑ Chronotropic effect of Sympathicus

➢ NA and A bound with β_1 adrenoreceptors of SA node



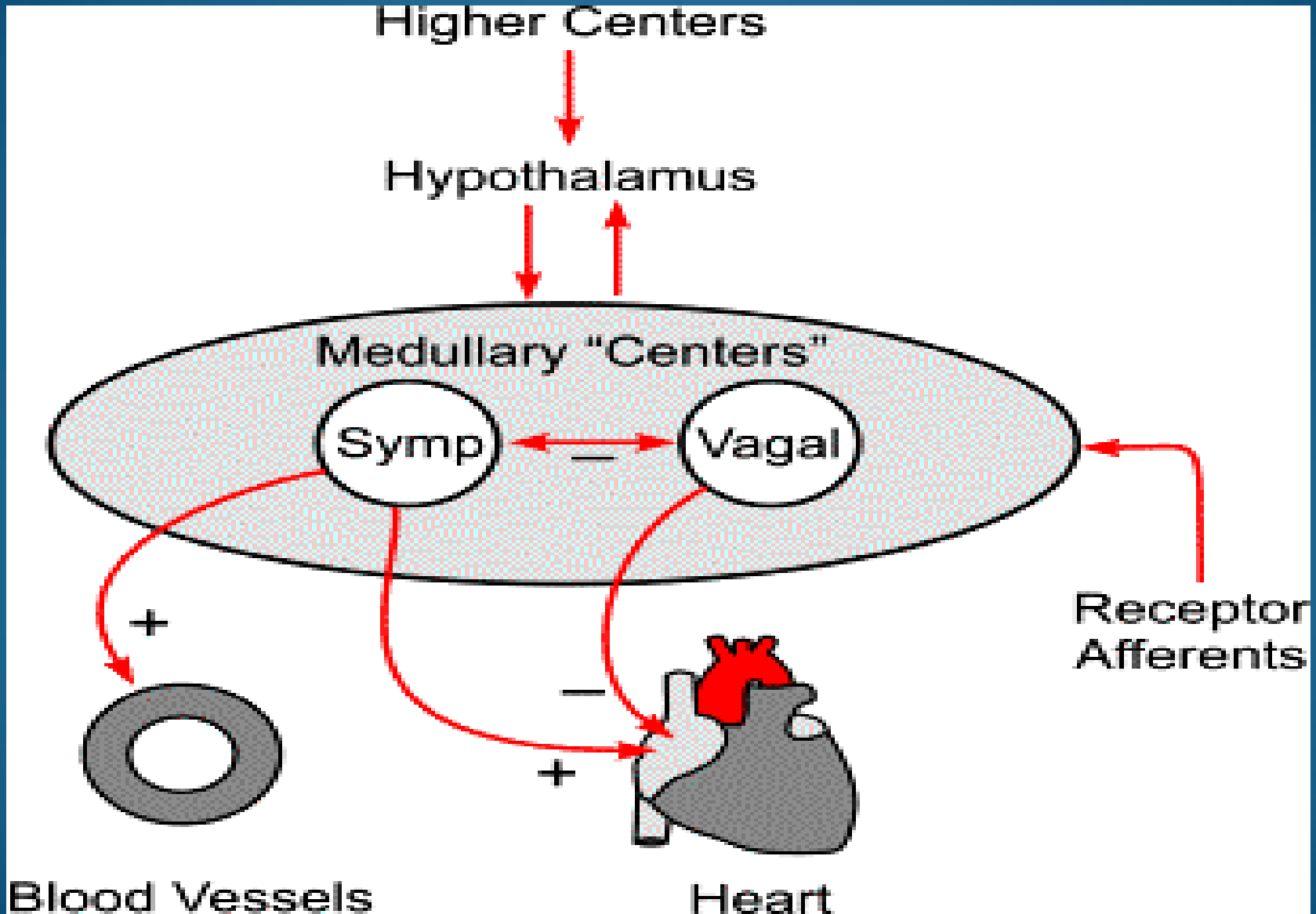
✓ activation of G protein stimulates adenylate cyclase

✓ cAMP activates non selective cation channels responsible for If

✓ cAMP activates PKA -> phosphorilation of Ca channels, participating in generation of AP

❖ this accelerates depolarization and HR increases

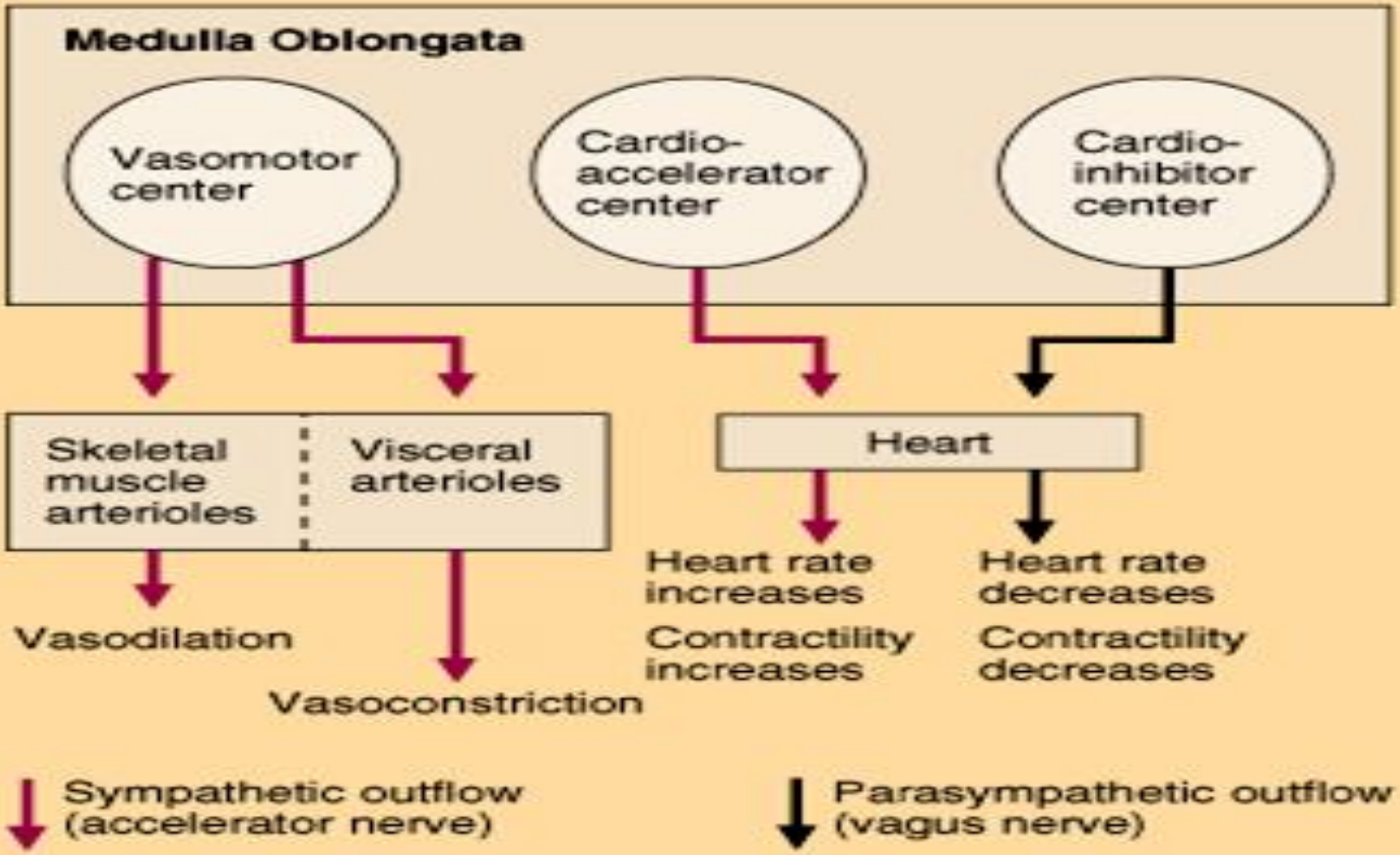
The tone of autonomic nervous system nerves on the heart is changed under the influence of the other NC, situated at medulla oblongata, hypothalamus and brain cortex.



❖Into medulla oblongata are situated two centers, controlling the heart performance.

➢Cardio-accelerator center – through sympathicus performs positive effects on heart performance

➢Cardio-inhibitor center - through vagus nerve performs negative effects on heart performance



Reflex control of heart performance by mechanoreceptors

- The volume receptors excite by the increased volume.
  - These receptors are situated at:

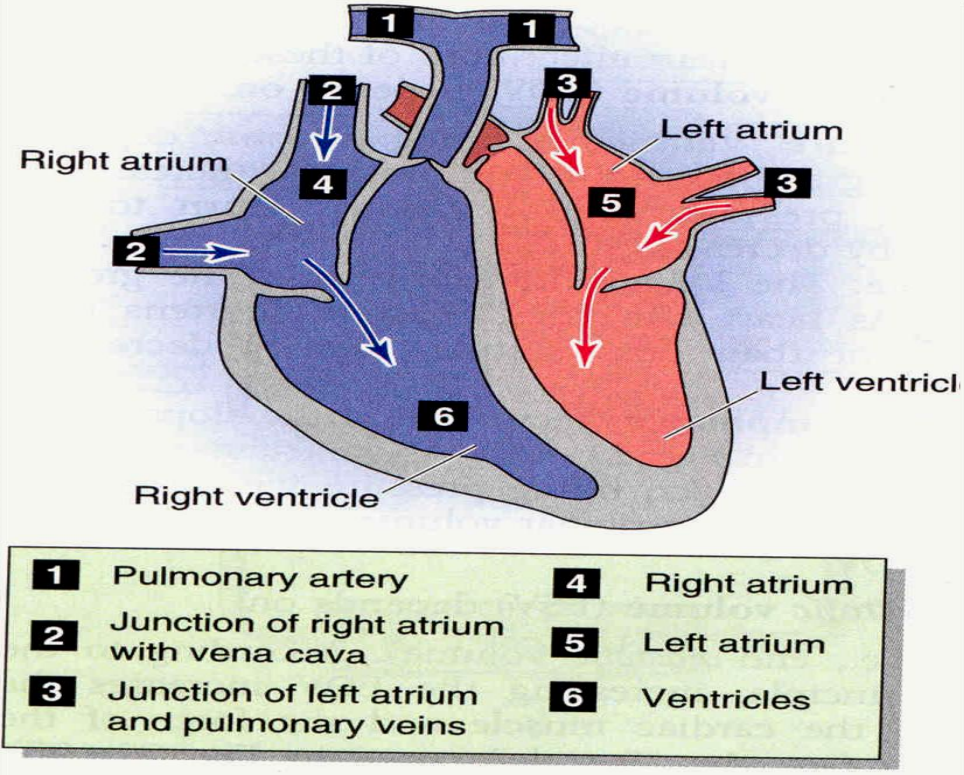
✓Pulmonary artery ✓atria ✓ventricles

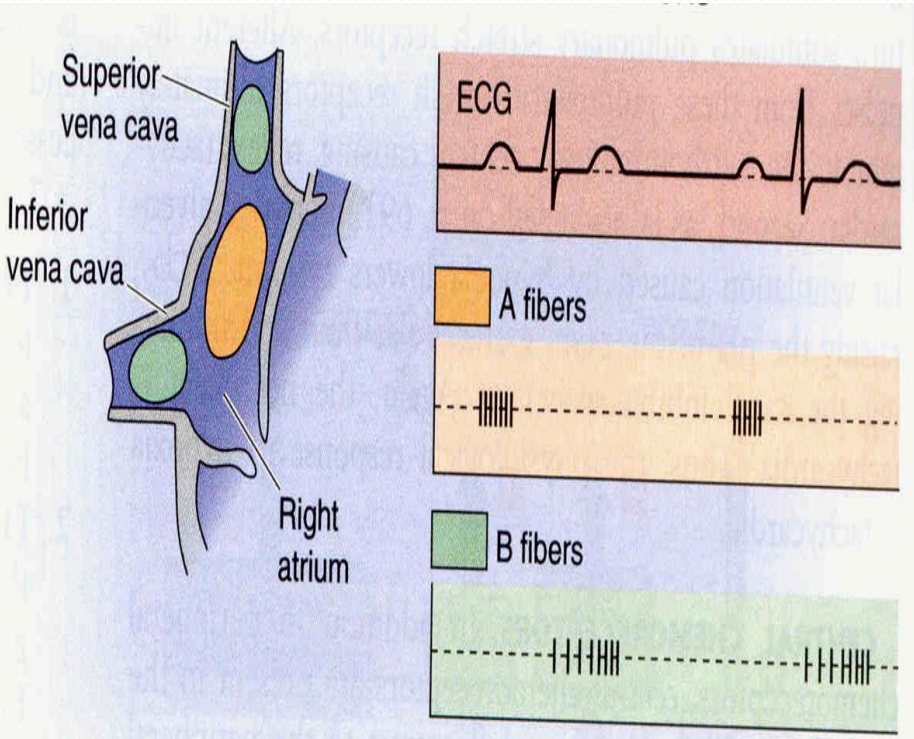
✓V. cava superior and
v. cava inferior

- into the atria are situated 2 types of receptors:

✓ type A – they excite during atrial contraction

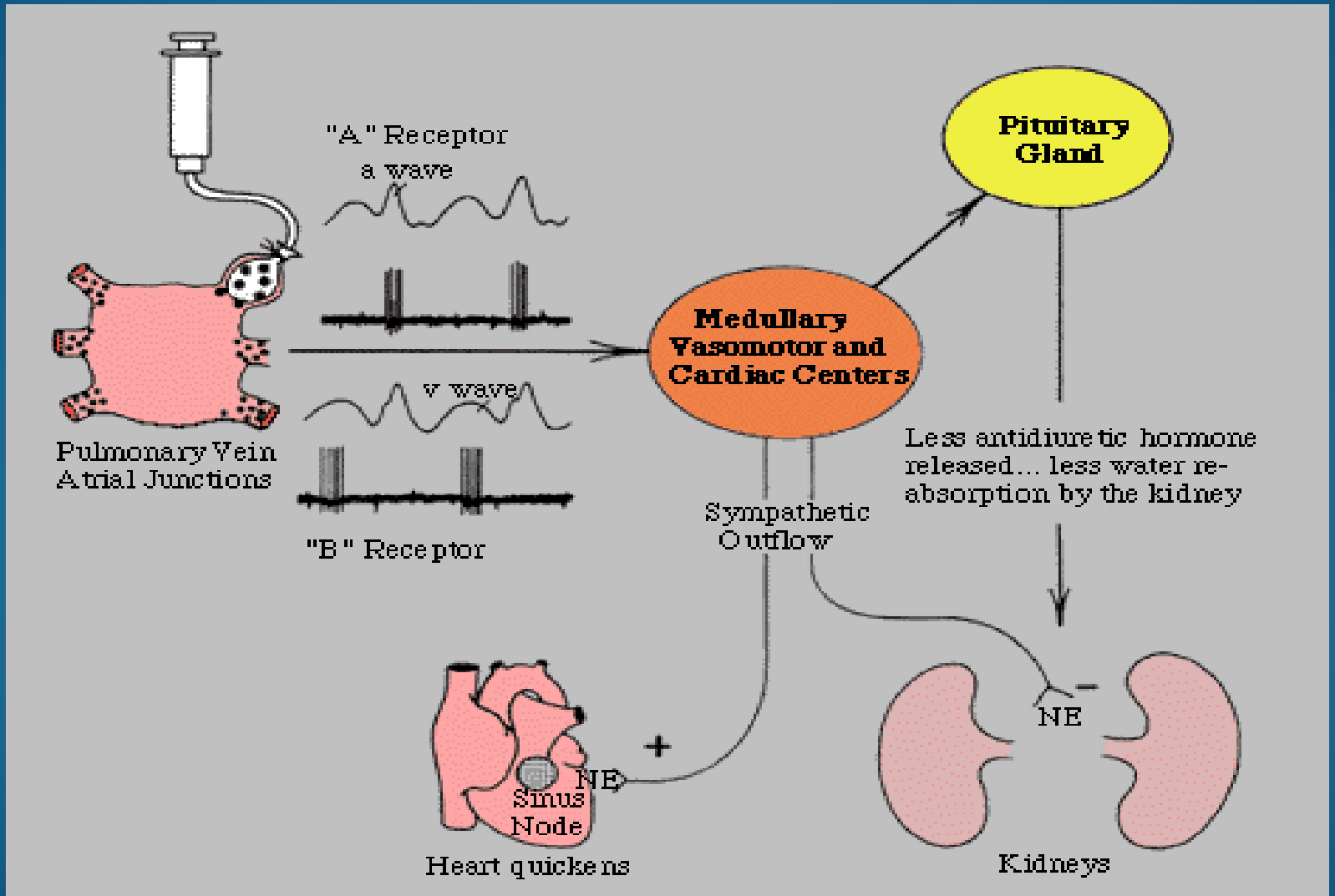
✓ type B – they excite during atrial filling



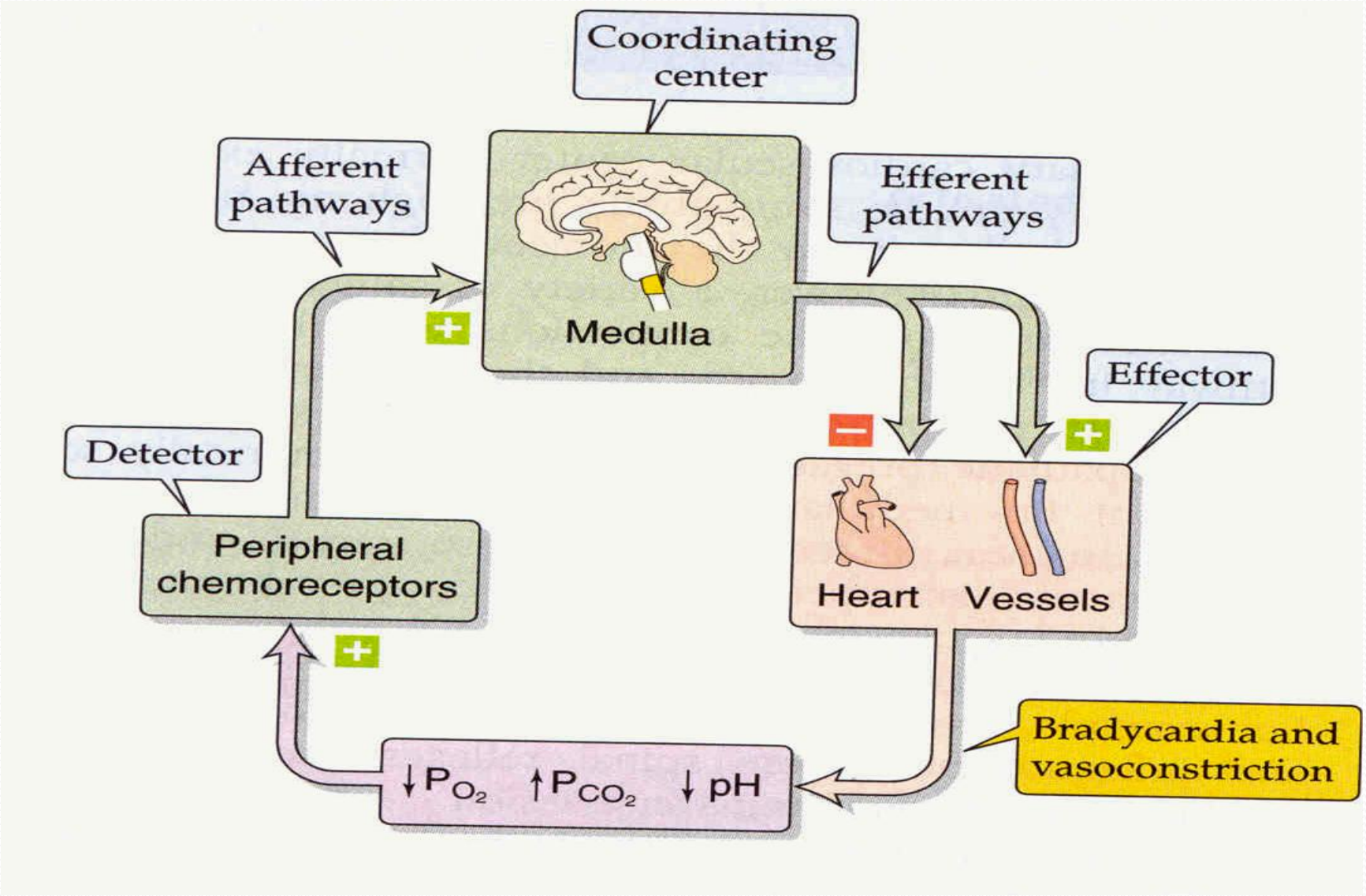


Increased blood volume stimulates atrial mechanoreceptors type B:

- HR increases because increased tone of sympathicus and stimulation of SA node and decreased tone of vagus nerve – reflex of **Bainbridge**



Reflex control of heart performance by chemoreceptors



❑ Humoral control of heart performance

❖ **substances with positive inotropic action**

1. Epinephrine and Norepinephrine
2. Angiotensin II
3. Glucagon
4. Thyroid hormones
5. increased $[Ca^{2+}]_o$

❖ **substances with negative inotropic action**

1. increased $[K^+]_o$
2. Intracellular acidosis
3. Adenosine
4. Acetylcholine

Thanks for your attention!