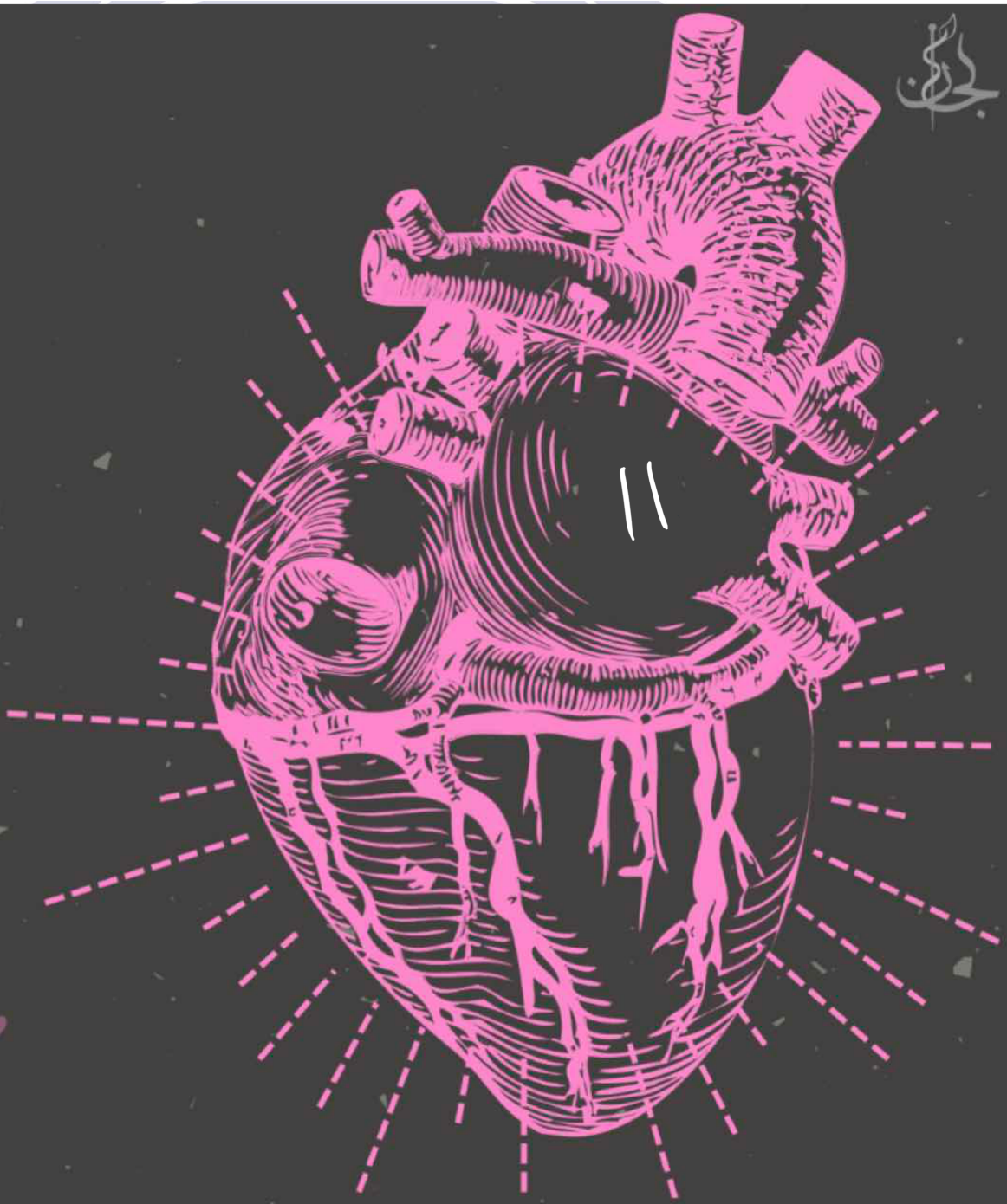
CVS



PHYSIOLOGY

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Cardiac Output Control Concepts:

- ***Cardiac output is the sum of all tissue blood flow*** and it's affected by their regulation (since the cardiac output is distributed to tissues then an increase in blood flow to tissues is due to increased Cardiac output [***CO***]).

$$CO \ \alpha \ \text{Tissue } O_2 \text{ use}$$

- The higher tissue demand for O_2 the higher the blood flow = Higher CO
- ***Usually CO is not mentioned when we talk about variations between human beings since the average cardiac outputs are constant from one person to another.***
- Instead we use cardiac index? surface area varies from one person to another

$$Cardiac\ index = \frac{Cardiac\ output}{Surface\ area\ m^2} = \frac{5L/min}{1.7m^2} = \frac{\frac{3L}{min}}{m^2}$$

- Cardiac index = 3L/min/m^2 [Range ***(2.6-4.2)*** L/min/m^2] .

Diastolic pressure contributes by 2/3 of the mean systemic pressure
systolic contributes by 1/3 of " "

Right atrial p. = zero

ohm's law

$$Cardiac\ output\ (flow) = \frac{\Delta p}{resistance\ of\ circulation}$$

$$\frac{mean\ Arterial\ pressure - Right\ Atrial\ Pressure}{Resistance\ of\ whole\ circulation\ from\ aorta\ to\ right\ artium} = \frac{MAP - 0}{TPR} = \frac{MAP}{TAP}$$

Total peripheral resistance

Why is Right atrial Pressure = ZERO?

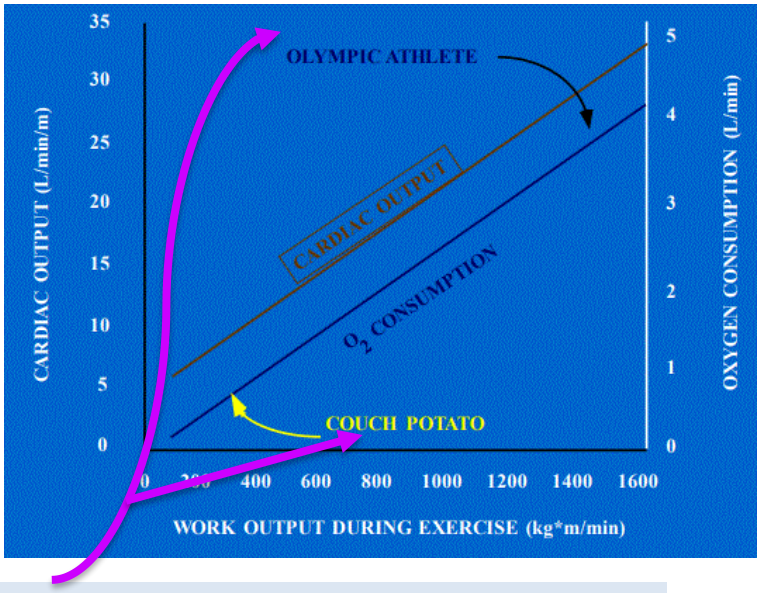
This allows the venous return from area of higher blood P. like superior vena cava to area of lower blood P. Right atrium.

$$CO = \frac{MAP}{TAP} \rightarrow MAP = CO \times TAP$$

CO= Stroke volume X Heart rate

- ***Stroke volume*** is the volume of blood pumped by ***one ventricle*** per beat which is normally = 70ml/beat
- ***Normal heart rate = 75 beat/min***
- Normal resting Cardiac output = 70ml * 75 beat/min = ***5L/min***

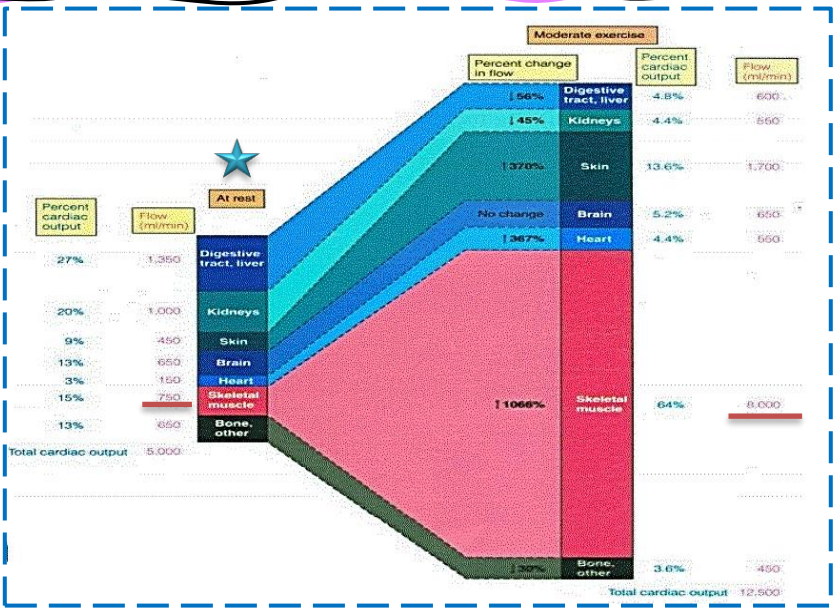
- *This tells you again that Cardiac output is proportional to tissue oxygen consumption.*
- ***The higher the workout intensity the higher the CO***
- ***Note that CO and O_2 consumption are parallel to each other → they are directly proportional.***



An Olympic athlete has a very high level of O_2 consumption since he/she can do high intensity workouts, compared to a couch potato or a lazy person

Distribution of CO during rest and moderate exercise:

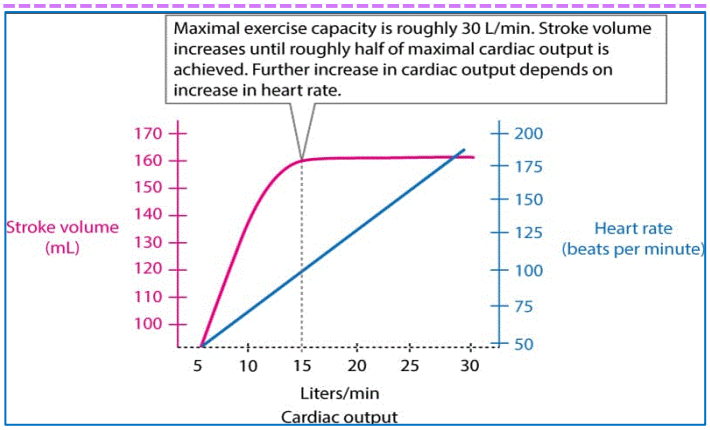
- At ***resting cardiac output*** (5L/min) skeletal muscle receives blood flow that's lesser than 1L although skeletal muscles make up 40% of our body mass.
- But during moderate exercise the ***blood flow to skeletal muscles increases from 750ml/min to 8L/min***, this is due to increase in CO from 5L/min to 15L/min.



- Also, ***Skin Blood flow*** ↑ ? Because skin is important for temperature regulation during exercise ,since contraction of muscles increases, so we have faster metabolism → but only 25% of generated ATP is used for work → the rest ***75%*** is converted to heat →that's why we need to control this temperature by increasing blood flow to skin.
- On the other hand, blood flow to GIT ↓ ? It's more important to supply skeletal muscles, skin and heart [↑ exercise intensity = ↑ ***tissue demand for O_2*** =↑ **CO** = ↑ ***Heart work***, so the heart itself needs more O_2 → ↑***Coronary flow***] during exercise.

Note :

*Since **CO= Stroke volume x Heart rate**, CO increases by the increase of SV and HR. During exercise both SV and HR increase, but when we reach half of CO_{max} (15L/min) SV can no more increase and **the further increase of CO is dependent on ↑ HR [Beats faster get blood flow out faster, heart pumps same volume of blood in less time, instead of pumping 5L each min, ↑HR = pump 5L each 20 second, so after one min heart pumped 15L.***



Variations in Tissue Blood flow:

	Per cent	ml/min	ml/min/ 100 gm
Brain	14	700	50
Heart	4	200	70
Bronchi	2	100	25
Kidneys	22	1100	360
Liver	27	1350	95
Portal	(21)	(1050)	
Arterial	(6)	(300)	
Muscle (inactive state)	15	750	4
Bone	5	250	3
Skin (cool weather)	6	300	3
Thyroid gland	1	50	160
Adrenal glands	0.5	25	300
Other tissues	3.5	175	1.3
Total	100.0	5000	---

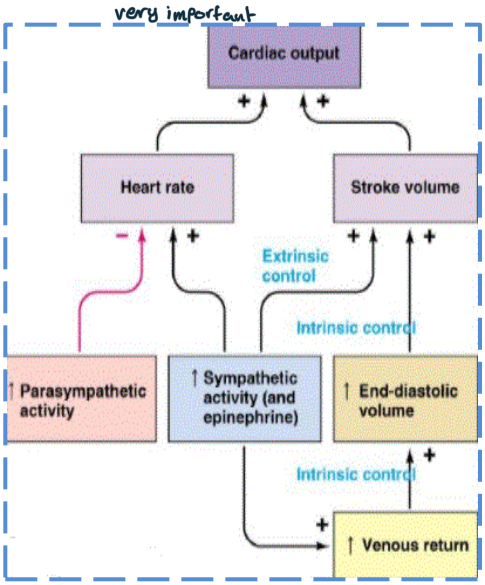
not as blood supply, but for urine formation → blood filtration

1. ***The Heart has one of the highest blood flow and high O_2 consumption.***
2. Although ***kidneys*** blood flow (BF) is ***360 ml/min***, but this BF goes for filtration and not because kidneys need more O_2. Same concept applies to the liver.
3. ***Adrenal and thyroid gland***: Tiny organs, BF to them is much higher than what they need so again high BF is not due to high O_2 demand.

Control of Cardiac Output:

- ***Parasympathetic stimulation*** only affects heart rate and has little effect on SV → its innervation is mainly to Atria
- ***Sympathetic stimulation***:
  - ↑HR
  - ↑SV (+ve inotropy, ↑force of ventricular contraction →Ventricles squeeze → pumps more than 70ml per beat)
  - ↑Venous return to atrium this means ↑amount of blood in atrium so ventricular volume will increase= ↑End-diastolic P. = ↑Preload = ↑ stretching of ventricle before contraction = FORCEFUL CONTRACTION = ↑SV

very important

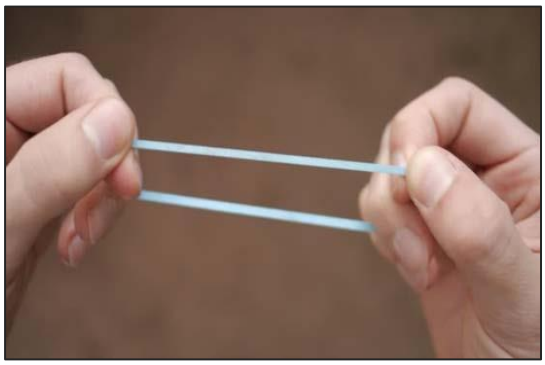


Frank-Starling Law

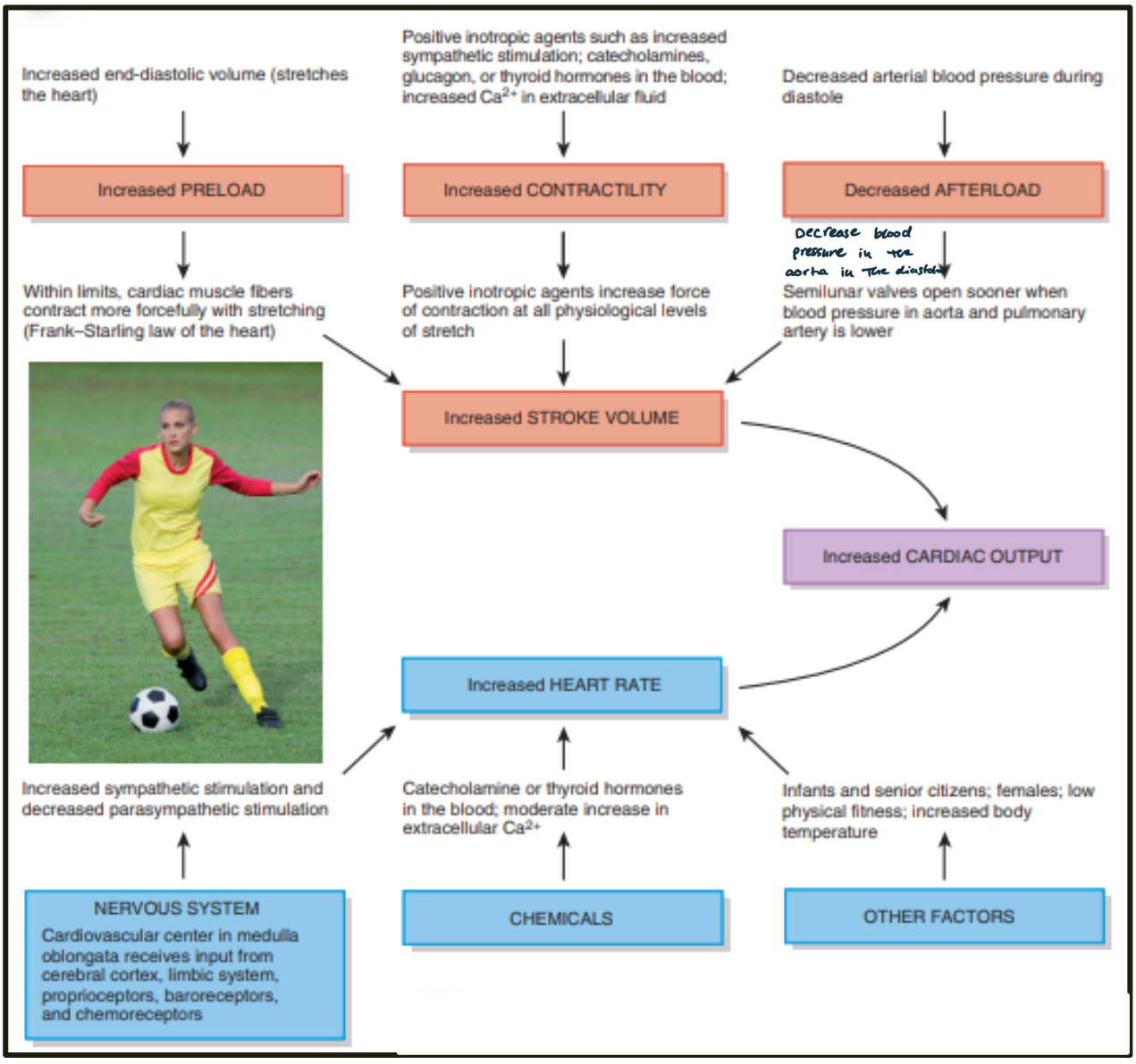
EXTRA:

It's like holding a rubber band, the more you stretch it , the higher the force generated than if the stretching was low.

Now imagine that rubber band is the ventricle and as you stretch it by adding more and more blood (↑volume) the ventricle will contract, it would be a powerful contraction, ventricle will squeeze more than usual thus releasing higher volume of blood = ↑SV.



Control of Cardiac Output:



Factors ↑ SV:

- ↑Preload= ↑Stretch = ↑force of contraction
- ↓Afterload =↓ Aortic pressure → lower aortic pressure means that ventricle can pump more blood because low pressure means lower resistance to blood flow →also ventricle needs to develop lower pressure to overcome this low aortic pressure → valve opens earlier than usual

  On the other hand, in cases of hypertension ventricle needs to develop higher pressure to open valves → lowering the amount of blood that is pumped → this also increases the ventricle demand for energy and increases work that must be done to be able to pump blood in order to maintain a constant amount of SV.

Factors ↑ HR:

- Sympathetic stimulation
- Chemicals like catecholamines, thyroid hormone, moderate increase in extracellular Ca^{+2}
- Infants have higher HR can reach up to 120 beats/min
- Senior citizens (Elderly) ? as you age your body is more susceptible to stiff and clogged arteries that obstruct blood flow from heart →this makes heart pump blood more often thus adding more heart beats to your resting rate to overcome this block.
- Adult female has slightly higher resting HR than males.
- Athletes [High physical fitness] have lower resting HR (30-40) (not to worry about) → that's likely because exercise strengthens heart muscle, and this allows it to pump a greater amount of blood with each heartbeat [HIGH SV]. So, a person with low physical fitness is the opposite (he has higher heart rate)
- Higher temperature ↑ permeability to ions → faster depolarization → SAN discharge impulses faster → ↑HR

Autonomic effect on heart:

- ***Cardiac output only increases if BOTH SV and HR increase together.***
- But in ***moderate*** changes in HR, the ***SV can compensate*** to keep a ***constant CO*** [e.g. If heart rate increases and we mean here a moderate increase→ SV will decrease → CO is kept constant and if HR decrease then SV will increase also and CO is again constant]

$$CO = \uparrow HR \times \downarrow SV$$

$$CO = \downarrow HR \times \uparrow SV$$

- But if ***HR increases significantly*** then ***SV can no more compensate*** because there's not enough time to fill during diastole and CO is decreased.
- If ***HR is significantly decreased,*** even if there's more time for filling during diastole →this filling is not enough to keep CO constant → CO is also decreased.
- ***Since sympathetic stimulation causes increased HR [180-200] and increased contractility (↑SV), then CO is increased = 15-20L/min***

- ***Parasympathetic effect is mainly on HR*** ? vagal fibers mainly goes to atria so it's effect on ventricles is slight and cause minimal decrease in contractility [little ↓SV] so CO is decreased here.

- While in ***tachycardia*** [very fast heart rate] although ***HR is high***, but CO is decreased → as I said before because ventricle has less time to fill during diastole.

Cardiac contractility:

- Measured ventricular pressure provides an indication of the contractility or relaxation of the ventricles of a heart. The derivative of pressure over time (dP/dt) can be used to measure cardiac contractility, but since this measure increases by increasing afterload and preload we consider it as ***inaccurate measure***
- **Instead we use : $\frac{dp/dt}{p.vent.}$ where p ventricle is instantaneous ventricular pressure**.
- ↑K^+ → ↓ contractility ? it's likely because increased k^+ level leads to more positive resting membrane potential to a level that is even higher than threshold which means that once muscle depolarizes and contracts it can't repolarizes to allow another contraction.
- ↑Ca^{+2}= ↑ contraction, and excess Ca^{+2} = spastic contraction. ***↓Ca^{+2}= cardiac dilation***

Work of ventricles: ↑EDV → ↑ atrial pressure

- *During the latter part of the ejection phase blood can still leave ventricle even if the aortic pressure is higher in aorta and that's because of* ***momentum of blood flow***.
- ***Total energy of blood*** = $(Volume \times pressure) + \frac{mass \times v^2}{2}$

  $= v \times p + Kinetic\ energy$

The main energy that's spent for moving blood in circulation

The heart performs a small amount of kinetic work when it imparts velocity to the blood as it leaves the ventricles. But kinetic energy is minimal in left ventricle so we can neglect it

- ***Kinetic energy*** is usually ignored and only considered when we talk about ***abnormalities like aortic stenosis***. In aortic stenosis we must push blood though a narrow orifice → Ventricle needs higher amount of energy to move the same amount of blood → and that energy is ***spent*** as kinetic work, so it increases and may reach up to 50% of total energy→ no more neglected in this condition.
- ***Although left and right ventricles pump the same amount of blood*** [5L/min], the work that's done by left ventricles is much higher? Because left ventricle will pump blood to aorta which has much higher pressure that pulmonary artery → ***total energy is higher in left ventricles.***

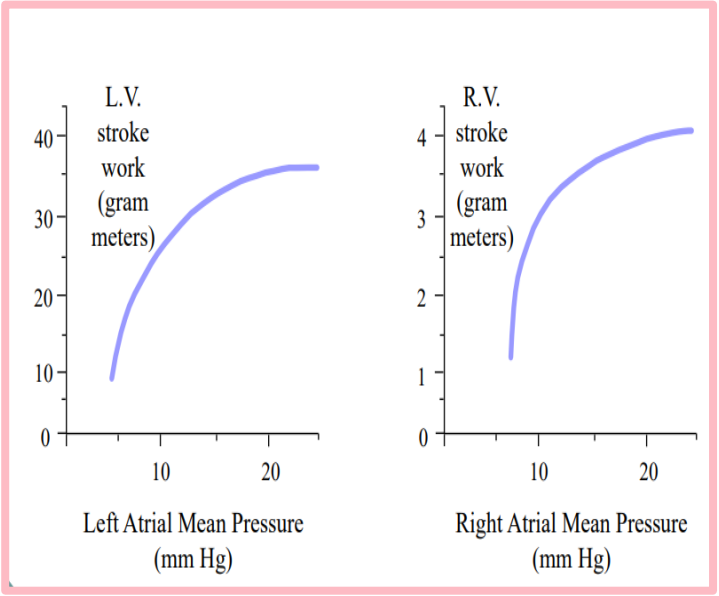
Total energy of blood = $v \times p + Kinetic\ energy$

- Higher in left vent.
- Constant in right and left
- Higher in aorta
- Neglected

Ventricular stroke work output

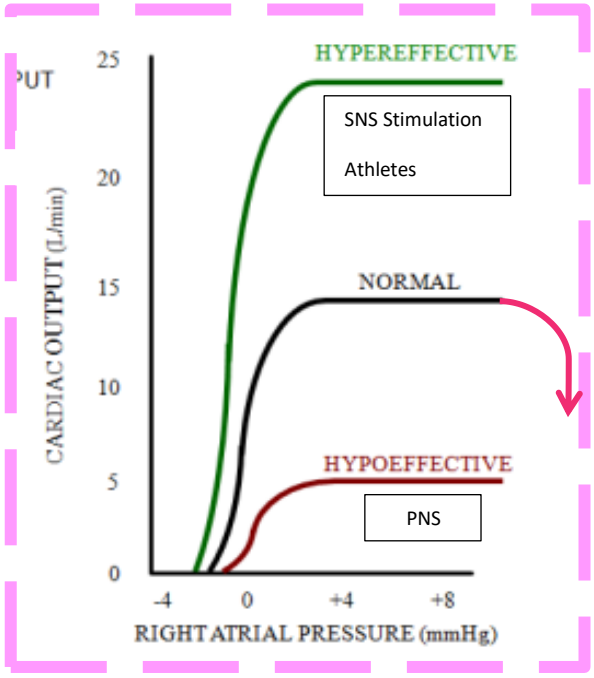
Note that stroke work is different:

40 in left vent. And 4 in right vent. indicates that left vent does 10 time more work than right vent.



Cardiac output curves:

- *Why do we use Right atrial pressure with cardiac output instead of ventricular volume which is responsible of cardiac output?*
  I. It's much easier to measure RAP than measuring right ventricular volume.
  II. ***RAP is the reflection of volume in right ventricle***? ↑ vent. Pressure = ↑volume in ventricle which means that atrial volume and pressure is also high because same amount of blood will flow from atrium to ventricle.
  III. RAP ***α*** right vent. Volume.
- Again, according to frank-starling law:



↑Venous return = ↑RAP=↑Vent. Volume (preload)= ↑stretch= powerful contraction= ↑Cardiac output

- ***Frank-Starling law is correct within physiological limits***, if we exceed the limit the CO will be decreased. The limit in the curve is the plateau. The ***pink arrow*** illustrates the drop in CO if we exceed the limit.
- Why athletes have hyper-effective heart? their heart is ***hypertrophic*** → higher force of contraction = ↑ CO.
- PNS mainly reduces CO by reducing HR and it has minimal effect on force of contraction (SV). However, it still reduces the SV minimally.

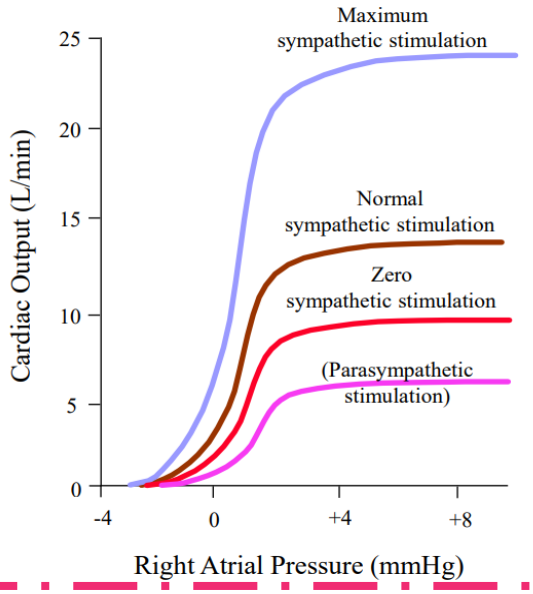
Note that as we ↑ sympathetic tone ↑↑ in Cardiac output.

$$\uparrow CO = \uparrow HR \times \uparrow SV$$

Also note how PNS lowers CO (↓HR)

$$\downarrow CO = \downarrow HR \times \downarrow SV$$

Epinephrine, dopamine and glucagon (+ve inotropy)

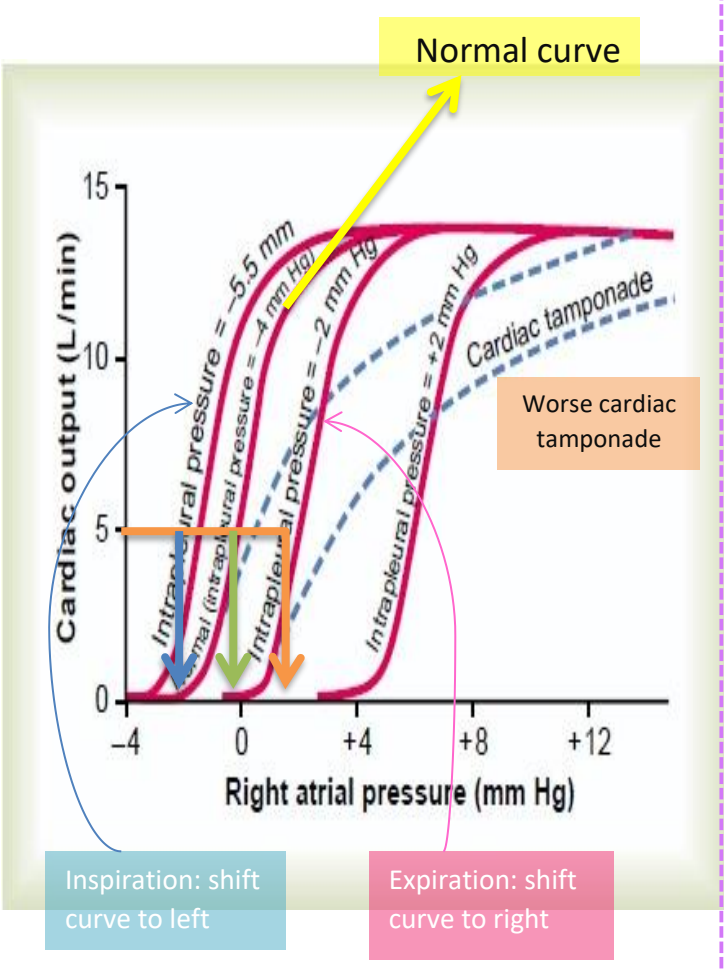


- ***Intrapleural pressure [IPP]*** is the pressure in the *intrathoracic space in between the vital organs in thorax*.
- The pressure inside the right atrium and the vena cava has relationship with IPP ?

During inspiration the diaphragm moves downward expanding the thoracic cavity→ this leads to drop in the intrathoracic pressure → ↓ IPP

This change in pressure is transmitted to the heart lowering the right atrial pressure, which facilitates venous return (↓ RAP = more blood can move from area of high p. (vena cava) to area of low (p. Right atrium) =↑ ventricular volume

The opposite happens during expiration.



- Normal IPP= ***-4mmHg***, note that when the cardiac output is 5L/min the RAP= zero, but as IPP decrease the curve will be shifted to left by the same amount and now at the same CO→ RAP= -ve pressure.
- If IPP is ↑, RAP also ↑, the pressure gradient is lesser here and the flow is also less. To have the same flow the right atrial pressure must increase.

As IPP increases → RAP must increase too ? → to have the same amount of Blood flow → notice that at CO= 5L/min as we move to the right, we have ↑ RAP

- Pathological condition that increase IPP→ ***pleural effusion*** (accumulation of fluid).
- An increase in IPP ***doesn't LIMIT*** the filling of the heart? In case of pleural effusion although pleura is filled with fluid but it's away from the heart.
- On the other hand, in ***cardiac tamponade***, fluid accumulate in pericardium→ in this case *filling of heart is limited* (resistance) , and you can observe that by reduction in maximal cardiac output, unlike pleural effusion→ max CO is kept constant.
- Also, in cardiac tamponade we cannot reach the maximum CO easily since you have much higher pressure to reach. ***And in case of worse cardiac tamponade CO max can not be reached.***

Pts with cardiac tamponade suffer from low CO→ low tissue blood flow ,they might complain of suffocation.

Treatment of cardiac tamponade is by removing the accumulated fluid.

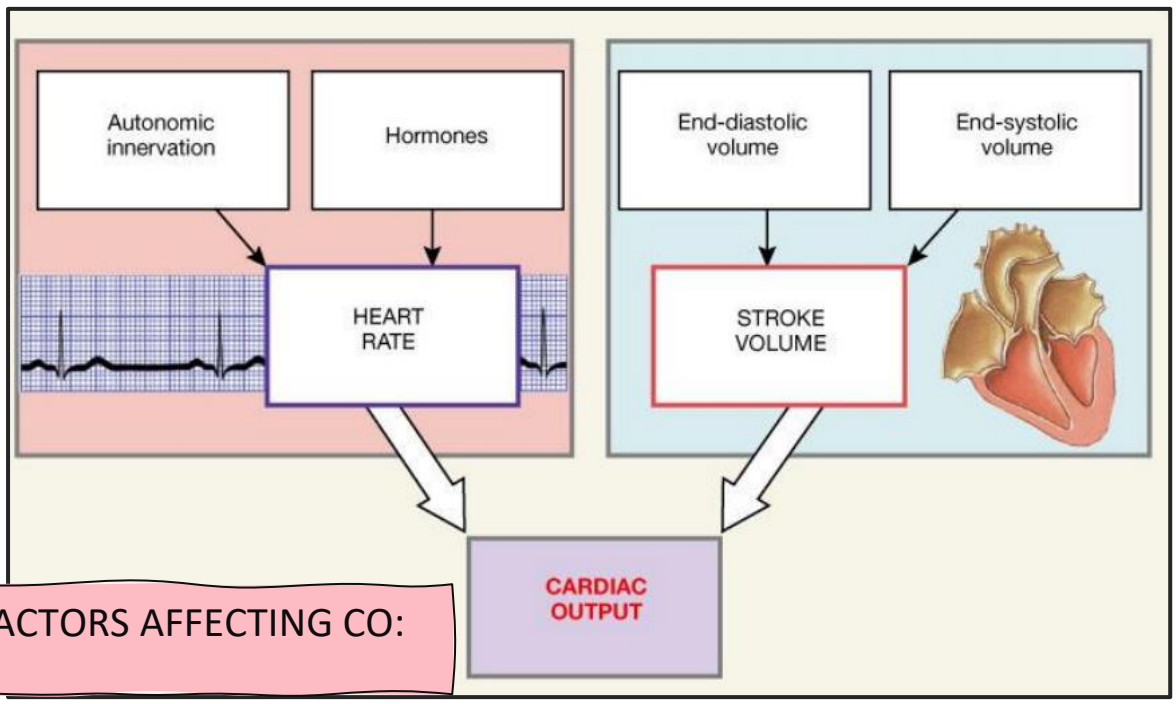
❖ ***Plateau of CO is determined by contractility and heart rate:***

Shift Plateau upward	Shift Plateau downward
SNS	PNS (↓HR)
Heart Hypertrophy (athletes)	Myocardial infarction
	Valvular diseases: Stenosis/regurgitation
	Myocarditis
	Cardiac tamponade
	Metabolic damage

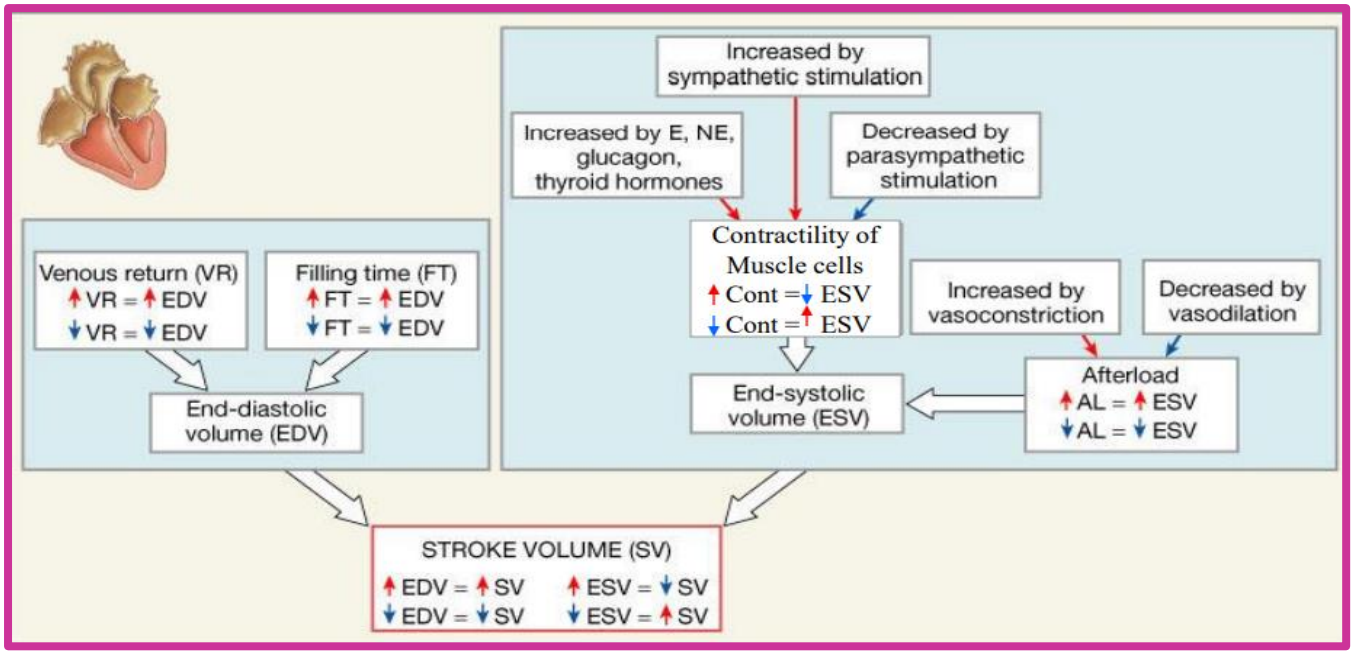
In stenosis it's hard to move blood against narrow orifice

And in regurgitation the blood flow back to ventricle instead of flowing to aorta.

In myocardial infarction and metabolic damage → decrease in heart muscle mass→ decreased CO



FACTORS AFFECTING CO:



SV= EDV-ESV → ↑SV=↑EDV-↓ESV

EDV : volume at the end of diastole (at the end of filling phase, how much did we fill?)

EDV increases :

1. ↑Venous return
2. ↑Filling time

ESV: volume at the end of systole (after end of contraction, how much blood is left in ventricle?)

ESV increases:

1. By ↓ in contractility (lower amount of blood is pumped→ higher blood is left in ventricle at end of systole)
2. Vasoconstriction in aorta (higher aortic pressure =↑afterload → reduced ability of vent. To pump blood so more blood left in vent).

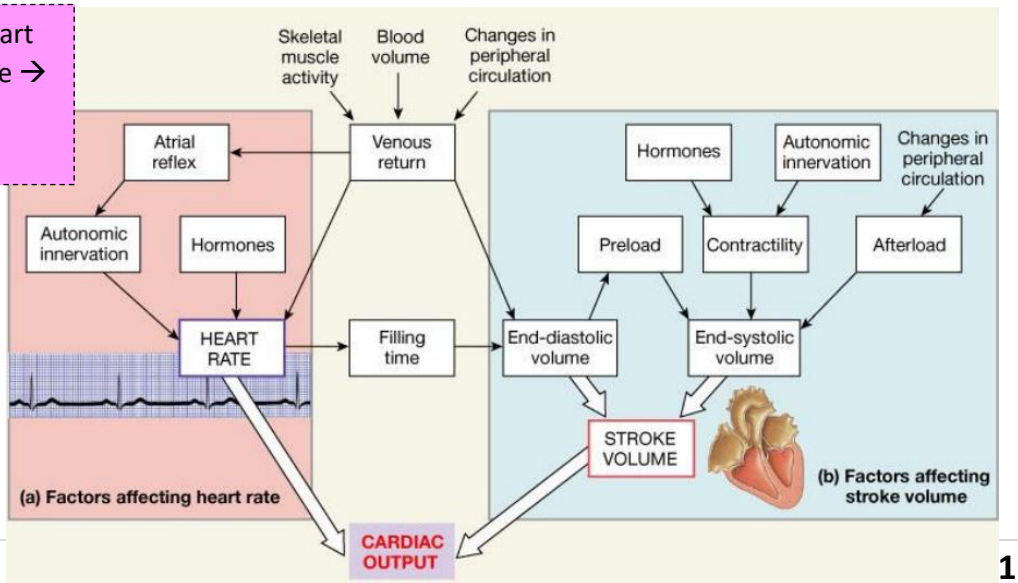
Summary:

Atrial reflex increases heart rate → reduce filling time → reduce stroke volume → reduce CO

↑Venous return:

1. ↑Skeletal muscle contraction ? push blood faster
2. Vasodilation
3. High blood volume

↑Venous return will increase EDV



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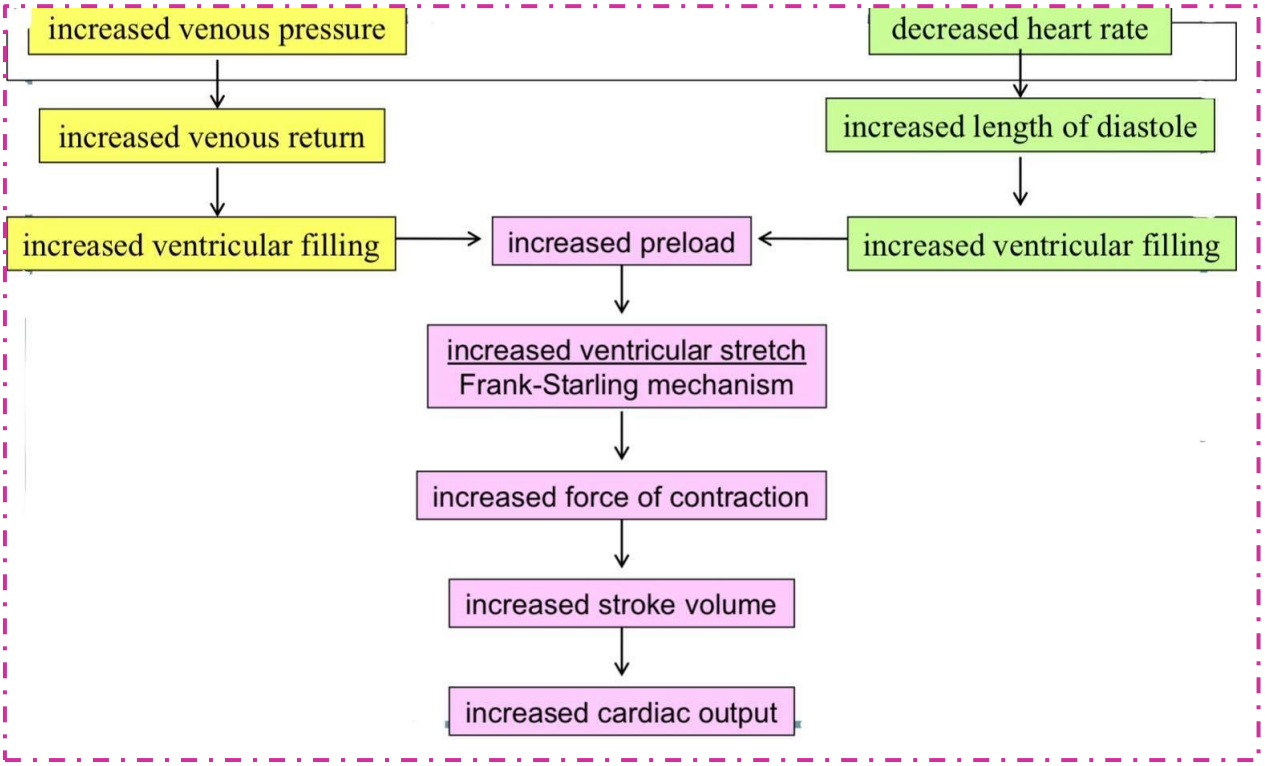

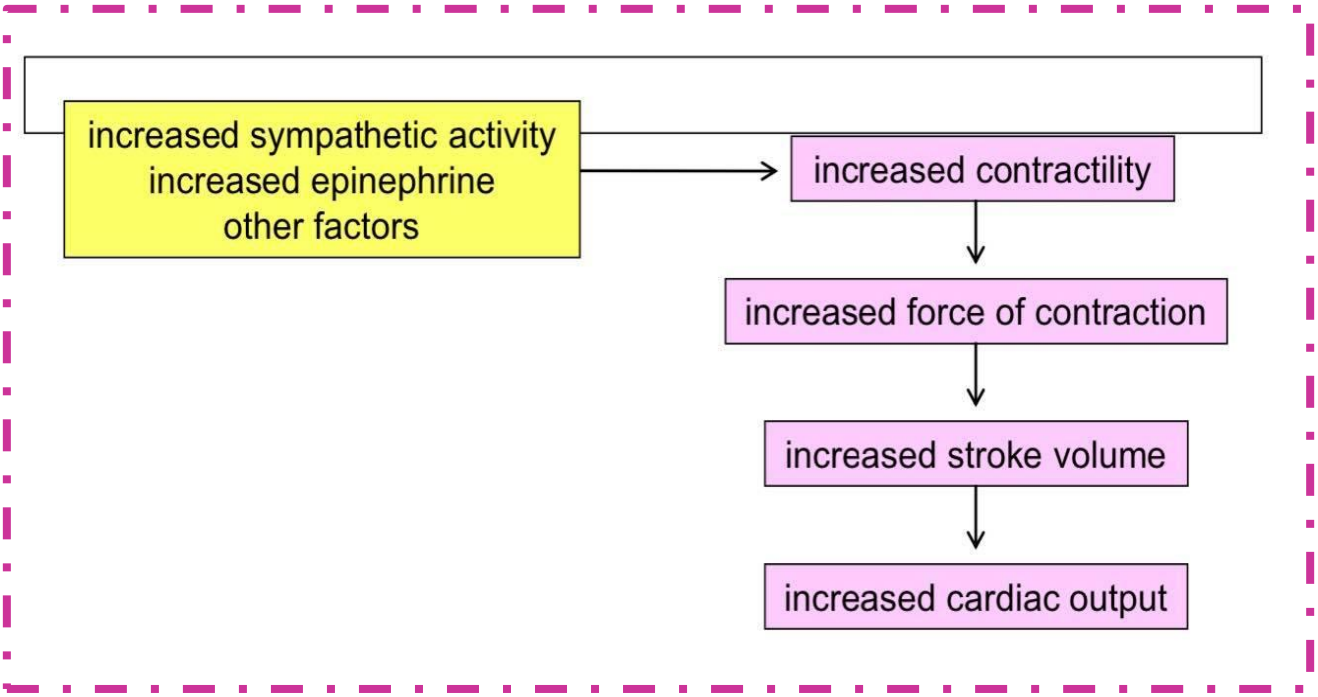

GOOD LUCK