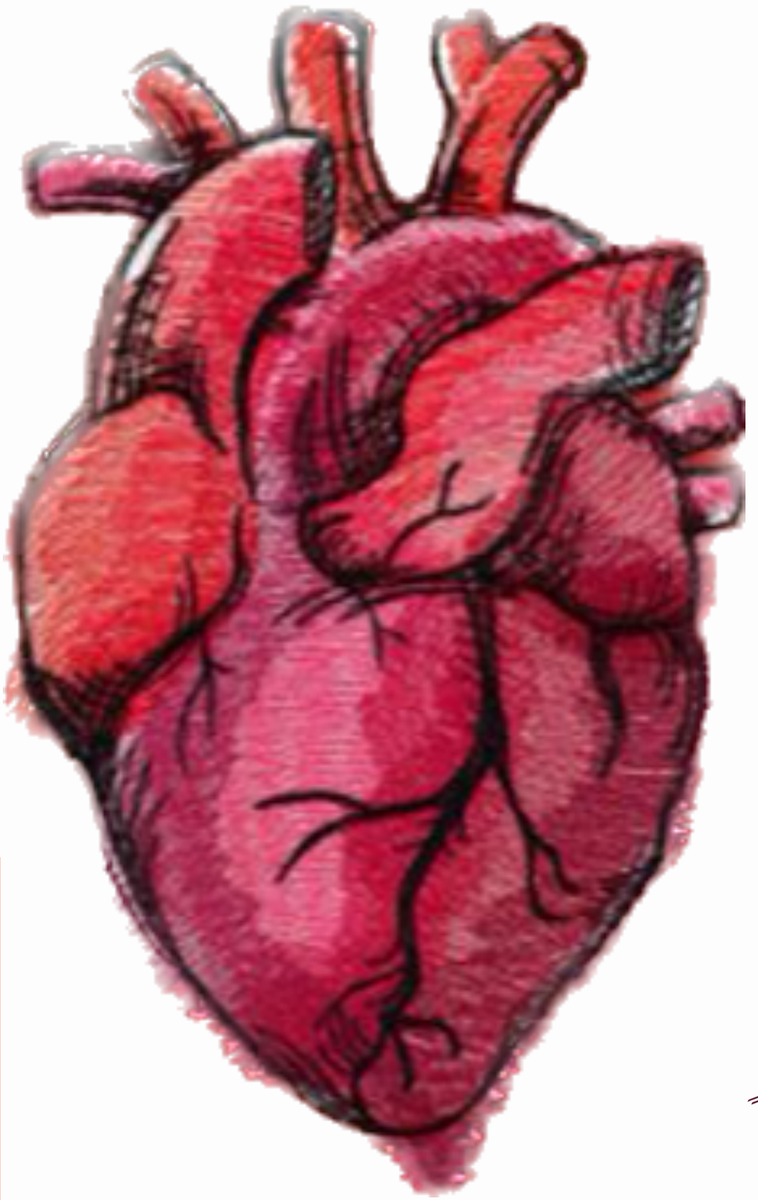

النادي الطبي

CARDIOVASCULAR SYSTEM

SUBJECT : physiolog

LEC NO. : 8 & 9

DONE BY : Abdulmuhsin Bani Mustafa

وَقُل رَّبِّ زِدْنِي عِلْمًا

SCAN ME!

The Cardiac Output And Its Regulation I & II

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Lecture Objectives:

1. Define the cardiac output and cardiac index.
2. Describe the relationship between cardiac output, stroke volume and the heart rate.
3. Describe how stimulation of the sympathetic and parasympathetic affect the cardiac output.
4. Explain the relationship between EDV and ventricular performance (Frank-Starling curve).
5. Describe factors affecting EDV and define cardiac reserve.
6. Describe the effect of autonomic nervous system, and ions on the pumping activity of the heart.
7. Describe the energy expended and O_2 utilization by the heart.
8. Understand methods of determination of cardiac output.

The Cardiac Output (CO)

↳ only from one side

- (def.) The CO is the volume of blood pumped by each ventricle per minute. It is ***not*** the total amount of blood pumped by the heart.
- The CO is about 5-6 L/min at rest. As blood volume is about 5-6 L, hence the whole blood volume circulates in ***one*** minute.
- During exercise the CO can increase to 20-25 L/min and even to as high as 40 L/min in well-trained athletes.
- The CO is ***equal*** to aortic blood flow, venous return, or pulmonary blood flow.
- **CO = SV X HR**

all blood in our body is 5-6 L so all blood move around the body within a minute

it is not fixed number, it changes depend on the bodies metabolism needs

↳ amount of blood pumped by each ventricle

cardiac output of the LV = the cardiac output of RV because it's a close circuit.

- **The Stroke Volume (SV):** the amount of blood pumped out of each ventricle per beat. It is about 70 ml in a resting man in supine position

  SV = EDV-ESV

  the extra amount of blood that could be carried by the ventricle in case in exercise

- **The Cardiac Reserve:** the difference between the cardiac output at rest and the maximal volume of blood the heart is capable of pumping per minute

- **The Cardiac Index (CI):** It is the output per minute per square meter of body surface area. CI is about 3.2 L/min.m^2

  is there a relation between cardiac output & body weight?
  yes it increase but not in a linear relation ship, but, it's more proportional with the surface area of the body than body weight its usted

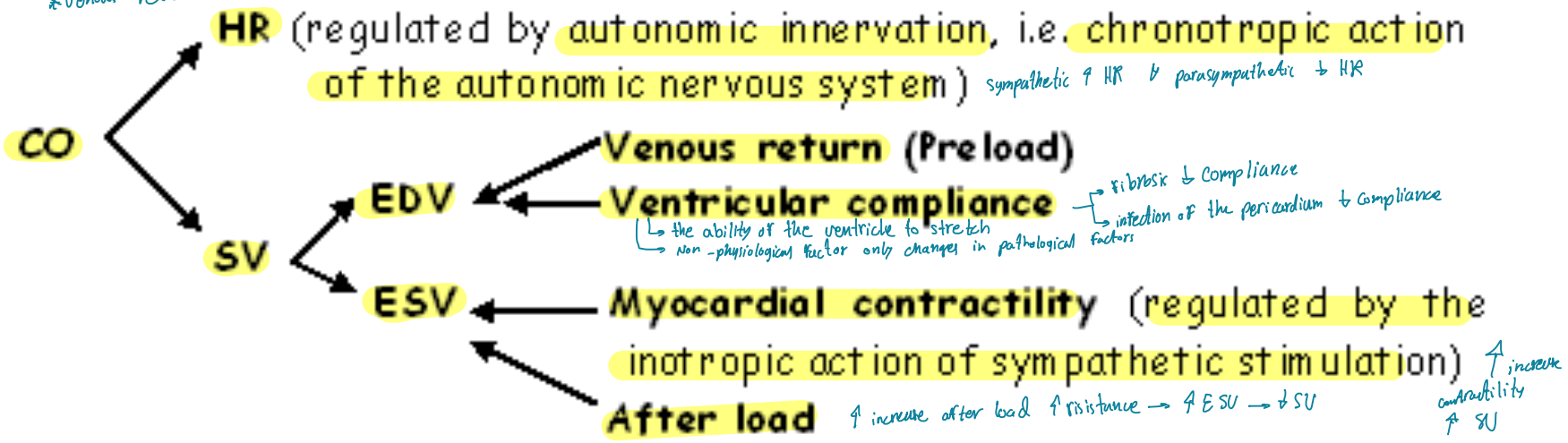
- **The Ejection Fraction % (EF %):** the percent of the end diastolic volume that is ejected with each stroke **(EF = SV/EDV %)**, it is about 65%. EF is an index for the resting myocardial contractility.

  it pump 2/3 of the blood in the ventricles
  this is used to access the contractility of the heart → ↑ EF ↑ contractility
  → ↓ EF ↓ contractility
  is measured by eco
  < 55% → begning of heart failure

Factors controlling Cardiac Output

The two determinants of CO are the ***Heart rate (HR)*** and the ***Stroke Volume (SV)***. However, no rise or fall in CO can take place unless it is preceded by an increase or a decrease in venous return respectively

* the cardiac output is the responsibility of the vascular system
↑ in contractility won't increase CO unless there is more blood available
*venous return control cardiac output → so what increase or decrease Venous return



Definitions

1. The Preload – is the degree to which the myocardium is stretched before it contracts

2. The after load – is the resistance against which blood is expelled from the ventricle

3. The myocardial contractility – is the strength (or power) of contraction at any given end diastolic volume time + strength

The Heart rate

- The normal range is 60-90 beats/min. The effective range in changing CO is between (40) - (180-200) beats/min. larger heart result longer HR
- Heart rate > 110 = Tachycardia increase heart rate more than that the cardiac output will decrease this is due to decrease the filling time, there will be no enough blood
- Heart rate < 50 = Bradycardia
- The rate is determined primarily by autonomic influences on the SA node.
- The parasympathetic and sympathetic effects on heart rate are antagonistic. ↳ stronger
- The inherent rate of the SA node's spontaneous discharge is about 110 beats/min, however, because of vagal tone the average rate is close to 70 beats/min.
- The heart rate can be regulated by other factors such as body temperature, circulating hormones (such as adrenaline and noradrenaline, thyroxin, glucagon, etc.), and stretching of the right atrium (Bainbridge reflex). ↳ in respiration
- If heart rate increases without an increase in venous return, **SV will decrease in a paradoxical fashion** so that the CO remains constant.

→ Prof. Guyton cut the IVC in laboratory animal and then put a pump between the two areas so the blood must pass through the pump before the IVC then to the R.A. so the venous return is controlled by the pump, he made the venous return constant & at the same time he stimulated the heart to ↑ HR while the venous return is constant, the cardiac output was constant so we can conclude the the ↑ HR decrease the SV → this experimentally but in our bodies when HR ↑ the SV ↓ with it.

we only say the preload increase only if the venous return ↑ & the ventricle stretch more, starling-Frank law of the heart: ↑ venous return ↑ EDV ↑ stretch the heart will contract more strongly & pump the extra blood leading to increase in CO this is the main difference between the myocardium & skeletal muscle, so they conclude that when the blood volume increase the myocardium fibers increase in length before contraction thus increase the contractility of the heart

*he increased the weight the fibre stretch more creating a preload before contraction, so when it's stimulated electrically the strength of contraction increase.

the greater the stretch of myocardium
the greater the force of the contraction
the greater the cardiac output

this lead to the ability of heart transplant

" The heart pumps what receives "

" The heart can auto regulate its cardiac output without the need of nerve controlled "

The Preload (EDV)

- If heart rate remains stable and more blood returns to the heart (i.e. more venous return) the EDV will increase. This results in a greater force of contraction, and consequently, a greater stroke volume and a greater CO (**Frank-Starling law of the heart**). This fact is applied to both sides of heart.
- Venous return increases if the gradient between the heart and the peripheral veins increases. This happens in response to;
  1. Increased blood volume (↑ mean systemic filling pressure).
  2. Venoconstriction (i.e. ↑ sympathetic tone).
  3. Dilation of arterioles.
  4. Skeletal muscle pump.
  5. Respiratory pump.
  6. Gravitational effect.
  7. Reduced right atrial pressure (i.e. ↑ contractility)

eating salty food → water → ↑ blood volume → ↑ venous return → ↑ cardiac output

→ veinous in the body contract it will force the blood in it towards the heart ↑ venous return

increase the blood going to the venous side

pressure the blood vessels and force the blood in it towards the heart
so when we walk the venous return increase

→ deep inspiration increase venous return while deep expiration decrease venous return

→ increase venous return from the upper part while decrease the venous return from the lower side

that is why when someone faint we lay them in the ground & raise their leg to increase their venous return

eg. in summer → blood vessel in the foot dilate which cause them to accomidate more blood result to decrease in venous return

blood moves from high pressure to low pressure

↳ reduce atrial pressure ∴ difference in pressure between atrium & ventricle increase leading the blood moves to ventricles → why does happen?

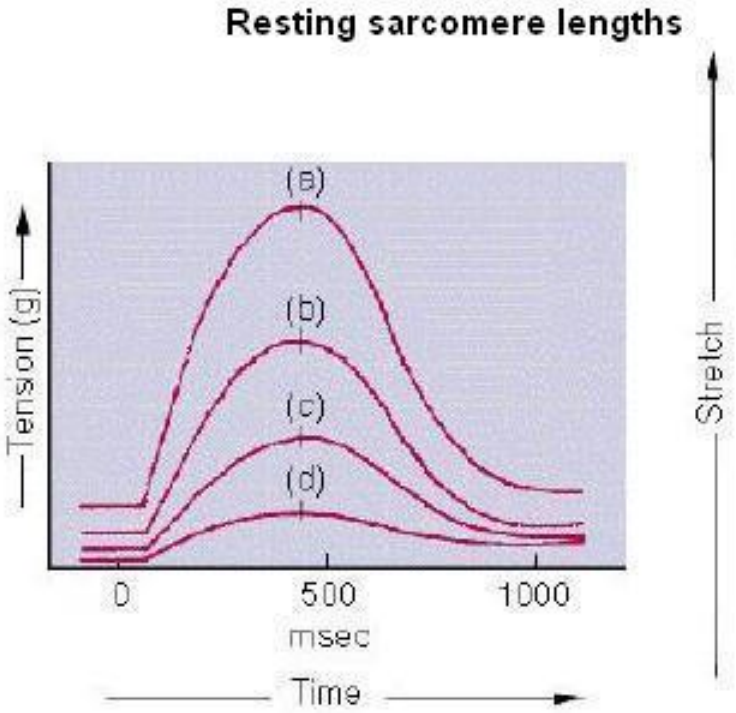
the reduce right atrial pressure is due to contractility taking to decrease SV → so ventricle take more blood from atria → ↓ pressure in atria → ↓ venous return

Intrinsic Regulation of Heart Pumping - The Frank-Starling Law and Mechanism

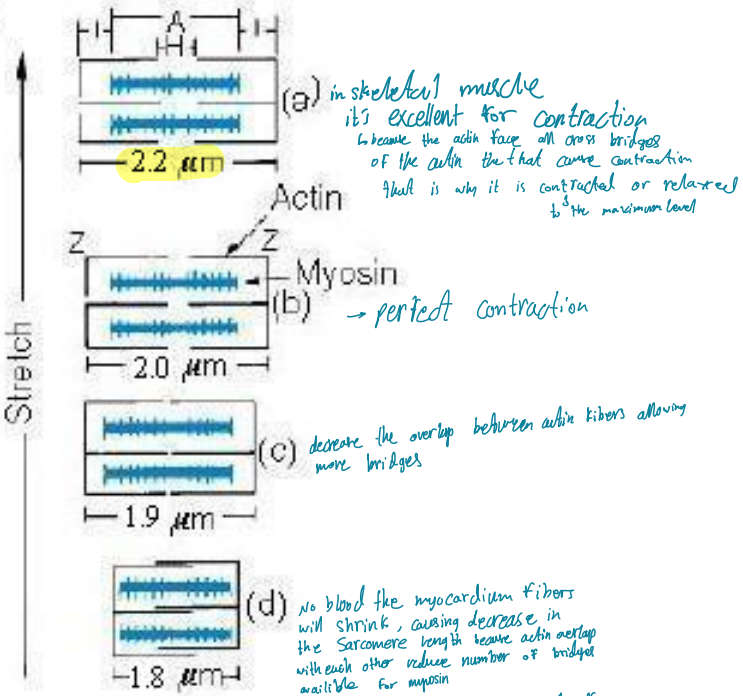
- The amount of blood pumped by the heart each minute is determined almost entirely by the **venous return**.
- This intrinsic ability of the heart to adapt to increasing volumes of inflowing blood is called the ***Frank-Starling mechanism of the heart.***
- (def.) The energy of contraction is proportional to the initial length of the cardiac muscle fiber (i.e. the preload) which is proportionate to the EDV.
- This means that (within physiological limits, the heart pumps all the blood that returns to it by way of the veins). In other words ***the heart pumps what it receives.***
- Mechanism: stretching of ventricles brings actin and myosin filaments to a more optimal degree of interdigitation for force generation. This law describes the length-tension relationship of the myocardial fiber.
- In addition, Stretch of the right atrial wall directly increases the heart rate by 10 to 20 percent, which also helps increase the amount of blood pumped each minute.

Frank-Starling Law

Increases in end-diastolic ventricular volume produce increases in stroke volume (Compare to ascending limb of length-tension plot). The force of heart muscle varies with wall fibre tension, which is a function of length.



Resting sarcomere lengths



in skeletal muscle it's excellent for contraction because the actin have all cross bridges of the actin that cause contraction that is why it is contracted or relaxed to the maximum level

→ perfect contraction

decrease the overlap between actin fibers allowing more bridges

No blood the myocardium fibers will shrink, causing decrease in the Sarcomere length because actin overlap with each other reduce number of bridges available for myosin

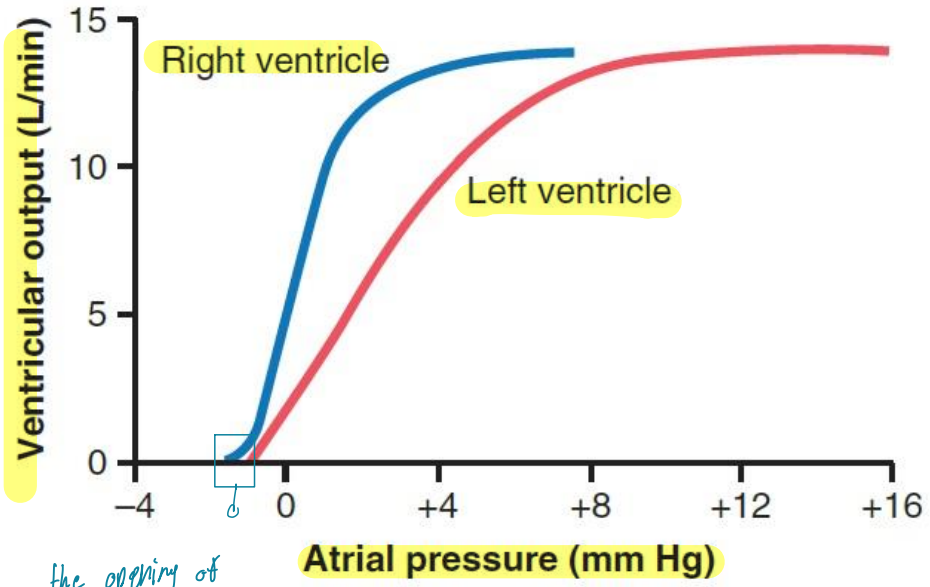
↓

poor contraction because part of the actin will pulled in the same force in opposite direction

b > c > d

Ventricular Function Curve

The two curves of this figure represent function of the two ventricles of the human heart. As the right and left atrial pressures increase, each ventricular volume and strength of cardiac muscle contraction increase, causing the heart to pump increased quantities of blood into the arteries per minute.



the opening of SVC & IVC is almost zero -1

why you had to say good bye?

Effect of autonomic nervous system on cardiac pumping

- Sympathetic stimulation can increase pumping (output) by 100%. By contrast, the output can be decreased to as zero by vagal (parasympathetic) stimulation.
- Sympathetic stimulation can increase cardiac pumping by (1) increasing the heart rate from a mean of 70 to 180-200 beats/min. Also, sympathetic stimulation (2) increases myocardial contractility, therefore increasing the pumping rate and the ejection pressure.
- Conversely, inhibition of the sympathetic nerves to the heart eliminates the sympathetic tone. This inhibition can decrease cardiac pumping to a moderate extent (about 30% below normal).

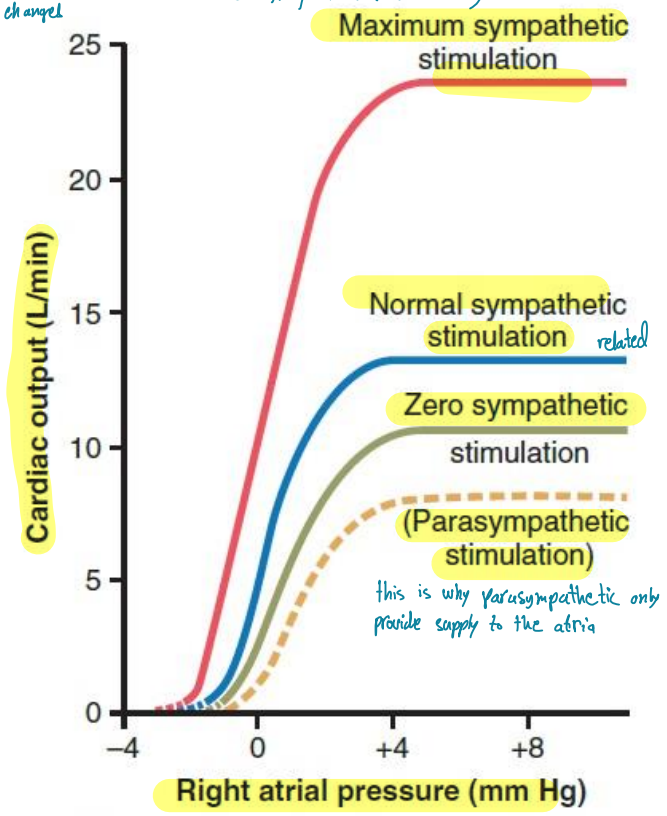


Figure Effect on the cardiac output curve of different degrees of sympathetic or parasympathetic stimulation.

Effect of autonomic nervous system on cardiac pumping (cont.)

this slide talk about the effect of parasympathetic on heart → it effects heart more than myocardium / contractility

- Parasympathetic (vagal) stimulation, if continuous, can stop heartbeat for few seconds, then heart "escapes" and start beating at 20-40 beats/min. This will reduce the pumping rate greatly.

"escape from Parasympathetic effect" → not completely but it would be bradycardia

- The effect of vagal stimulation is mainly to decrease the heart rate rather than to decrease greatly the myocardial contractility. Nevertheless, the great decrease in heart rate combined with a slight decrease in heart contraction strength can decrease ventricular pumping by 50 percent or more.

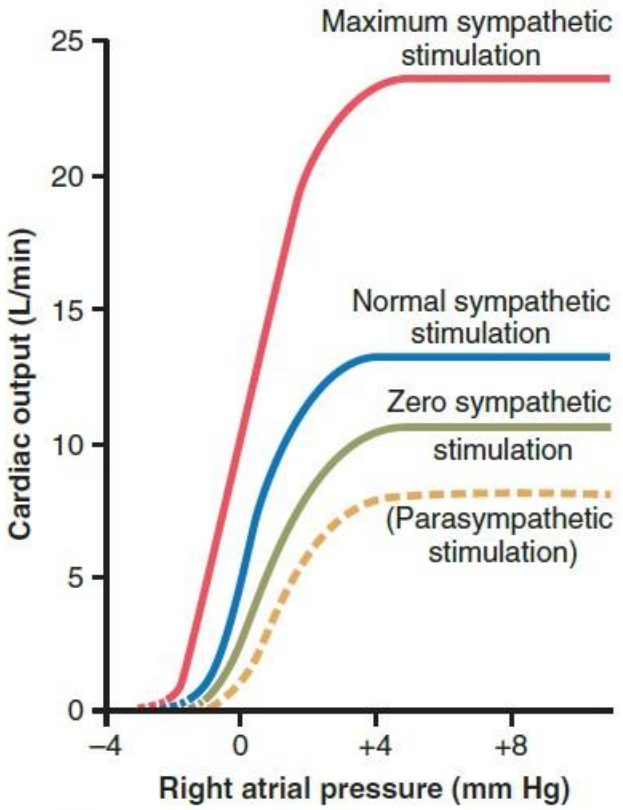
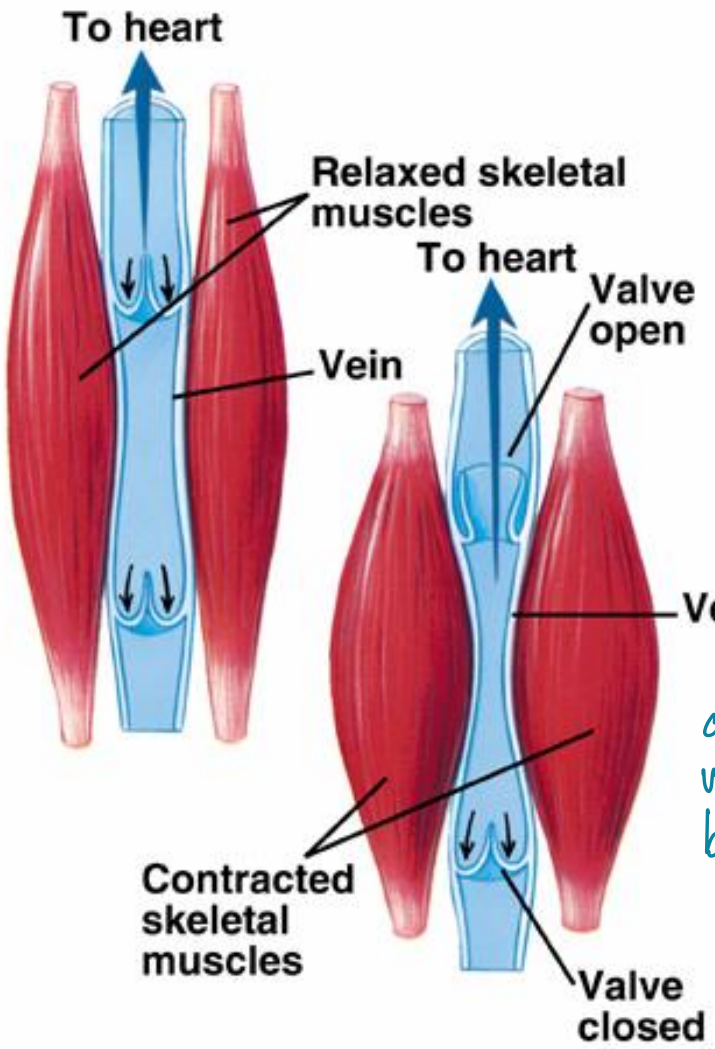


Figure Effect on the cardiac output curve of different degrees of sympathetic or parasympathetic stimulation.

* this slide represent the muscular pump which help with venous return

* it's skeletal muscle



Exercise and muscle pump

during exercise the muscle contract pressing the veins between the muscles so the blood can't go backwards → this is important in elderly who doesn't move causing swelling in the legs due to accumulation of blood

walking decrease the effects of gravity on the blood

The Myocardial Contractility

heart failure : decrease in myocardium contractility so the patient can't produce CO that is appropriate to the physical activity

↳ it increase the cardiac output due to increase in stroke volume & decrease EDV

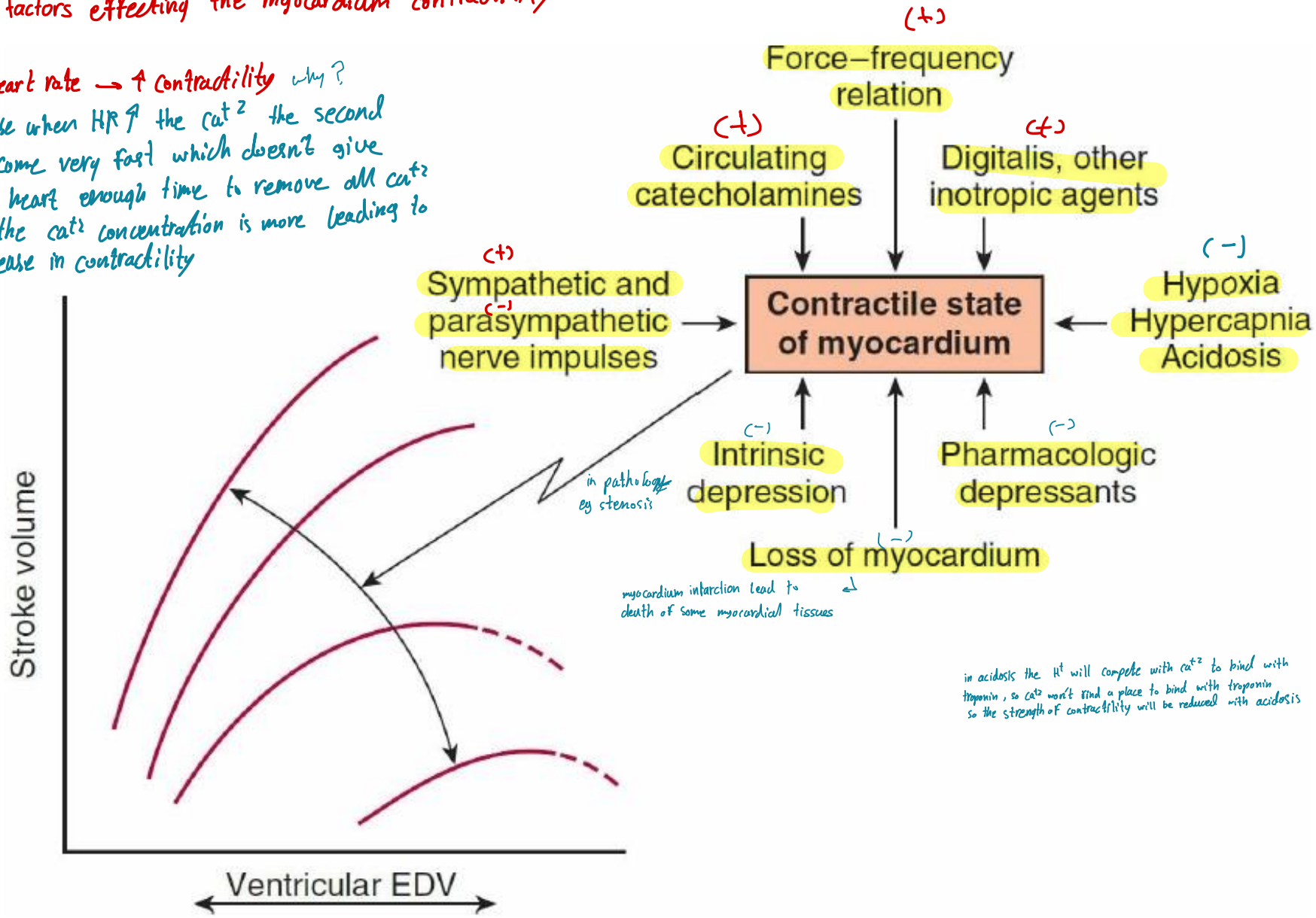
- Sympathetic stimulation and epinephrine enhances the contractility and squeezing out a greater percentage of the blood out of the ventricle leading to more complete ejection (**i.e. less ESV**).
- Heart failure can affect one or both ventricles. It is the inability of the cardiac output to keep pace with body's demands for supplies and removal of wastes. It is due to reduced cardiac contractile power despite the sympathetic compensatory reflex. The most ***two*** common reasons are;
  1. Damaged myocardium due to impaired coronary circulation ischemia / atherosclerosis
  2. Prolonged pumping against chronic elevated resistance (i.e. elevated after load)

     stenosis in valves

factors effecting the myocardium contractility

↑ heart rate → ↑ contractility why?

because when HR ↑ the Ca^{+2} the second AP come very fast which doesn't give the heart enough time to remove all Ca^{+2} so the Ca^{+2} concentration is more leading to increase in contractility



in acidosis the H^+ will compete with Ca^{+2} to bind with troponin, so Ca^{+2} won't find a place to bind with troponin so the strength of contractility will be reduced with acidosis

Effect of changes in myocardial contractility on the Frank–Starling curve

The after load

The total peripheral resistance the heart is facing is mainly due to; physiological resistance not pathological, not arterial

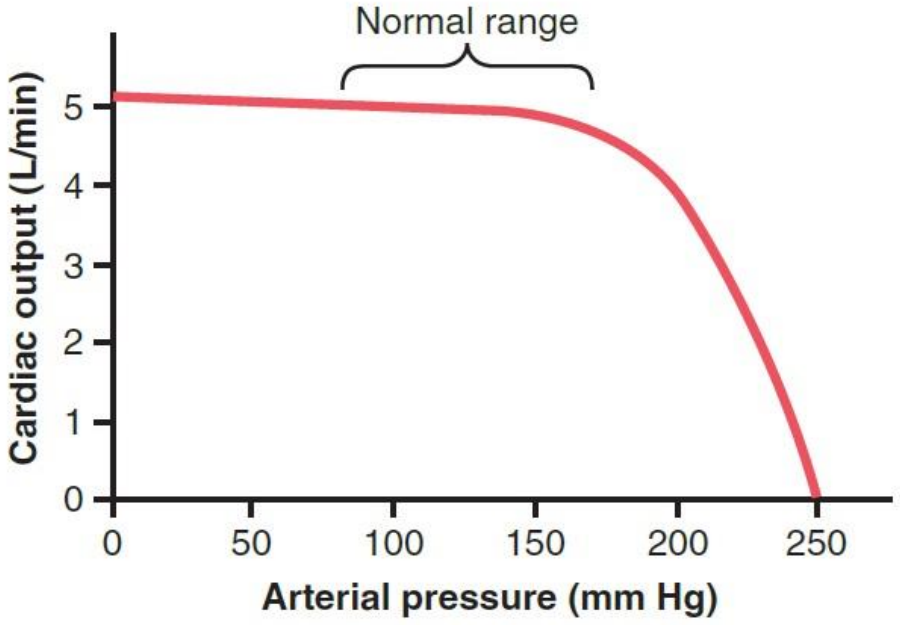
1. Arteriolar resistance. (most important) — because arterioles are smaller in diameter making it hard for the blood to pass through them which increase the resistance
2. Major arterial compliance. → the ability of the heart to stretch → easier to stretch → less resistance → vice versa. e.g. in the aorta contain yellow elastic tissue → when LV contract the aorta become like a ballon make it easier to blood to pass but if the aorta was rigid due to atherosclerosis it would increase the resistance, this is the most cause of hypertension in adults
3. Blood volume within the arterial tree at the moment of ventricular ejection. (least important) — if we push 50ml of blood throug the aorta face an amount of resistance, when we push other 50ml the resistance would be the same or greater? → greater because the first 50ml caused the aorta to stretch talking all the elasticity of the aorta so when the next 50ml come can't stretch the aorta again making it harder to pass so more resistance, so we can conclude the more blood in the aorta before SV the ventricle will face greater resistance so the ↓ in blood volume in our body the easier the heart to pump, eg. eating salty food ↑ blood volume → ↑ resistance vise versa, that is why in fasting it is easier because blood volume is decreased

Note: The higher the after load the higher the ESV, the lower is the SV and the CO. However, the CO will be re-adjusted by the increase in the preload (Frank-Starling law of the heart). Therefore, hypertensive patients have relatively normal CO.

they took the heart of a dog and put a metallic ring (adjustable) around the aorta to increase the resistance / after load they conclude that the ESV increased & SV, CO are decreased, but within minutes the heart is larger & pumping the same CO but with higher pressure this is called Re-adjusment of the heart
what happen is when they constricted the ring around the aorta at this point CO decreased but there is still normal CO in the circulation that is going back to the heart through the IVC & SVC so the blood volume in ventricle is increased (EDV increased) so the preload increased leading to increase contraction strength so the CO become the same as previous with higher pressure → this is what happen in hypertension patient, until what point can the heart re-adjust the CO? → 160 mmHg

Effect of Increasing the Arterial Pressure Load (i.e. Afterload) on Cardiac Pumping

The increase in the arterial pressure in the aorta does not decrease the cardiac output until the mean arterial pressure rises above about 160 mm Hg.



Energy and O_2 utilization by the heart

1. Like skeletal muscle, the energy is derived mainly (70-90%) from oxidative metabolism of fatty acids; other nutrients are lactate and glucose.
2. O_2 consumption is taken as a measure of the chemical energy liberated during cardiac work.

what factors determine the amount of O_2 consumed by the heart
increase these factors increase O_2 consumption

3. O_2 consumption is determined by the intra-myocardial tension (resting tension), the contractile state of the myocardium, and the heart rate. An increase in aortic pressure (i.e. increased afterload) is associated with a greater increase in O_2 consumption than an increase in preload (***LaPlace law***).

① how much are the myocardial fibbers stretched before the heart contract
↑ tension ↑ O_2 consumption

② decrease afterload
decrease O_2 consumption

③ ↳ will be explained later

Energy and O_2 utilization by the heart (cont.)

4. The high energy cost of increasing afterload reflects the large amount of energy expended to do internal work during isovolumic contraction (increases the amount of energy that must be expended to stretch internal elasticities).
5. The product of heart rate and aortic pressure correlates closely with cardiac oxygen consumption.

Energy expenditure α HR × P

CO can be increased in 2 ways 1) increase HR but the SV is fixed 2) increase SV & the HR is fixed the first way consume less energy so it's better our body in exercise use the first way so the heart won't change EDV so tension won't be increased so O_2 consumption at it lowest, but this doesn't happen in heart transplant patient

6. An increase in cardiac output due to an increase in heart rate consumes less O_2 than that due to an increase in preload.

angina treatment is by giving a vasodilator for veins i. decrease venous return so the heart will be less tens

how much ATP is produced by catabolism of fatty acids mostly and how much ATP is consumed to pump blood

7. Cardiac efficiency is not more than 25%. In persons with heart failure, this efficiency can decrease to as low as 5 to 10 percent.

produced energy / consumed energy = eficiency

75% is used to overcome the myocardium resistance

Methods for measuring Cardiac output

- *Direct:* by the use of flow-meter, usually used in experimental animals.
- *Indirect:* Such methods can be applied to humans. These methods are;
  1. Oxygen Fick's method.

$$CO = \frac{\text{Oxygen utilization by body (ml } O_2\text{/min)}}{\text{Arterial } O_2 \text{ content (ml } O_2\text{/L bl.)} - \text{Mixed venous } O_2 \text{ content (ml } O_2\text{/L bl.)}}$$

  2. Indicator dilution method.
  3. Ultra Sound (echo).

Test:

Q. Which of the following will promote an increase in the stroke volume of the heart?

A. A reduction in venous tone
B. A pneumothorax
C. Dehydration
D. General anesthetics
E. Skeletal muscle contraction

Test:

Q. The factor common to most changes in cardiac muscle contractility is the:

A. Amplitude of the action potential
B. Availability of cellular ATP
C. Cytoplasmic calcium concentration
D. Rate of neural stimulation
E. The magnitude of the EDV