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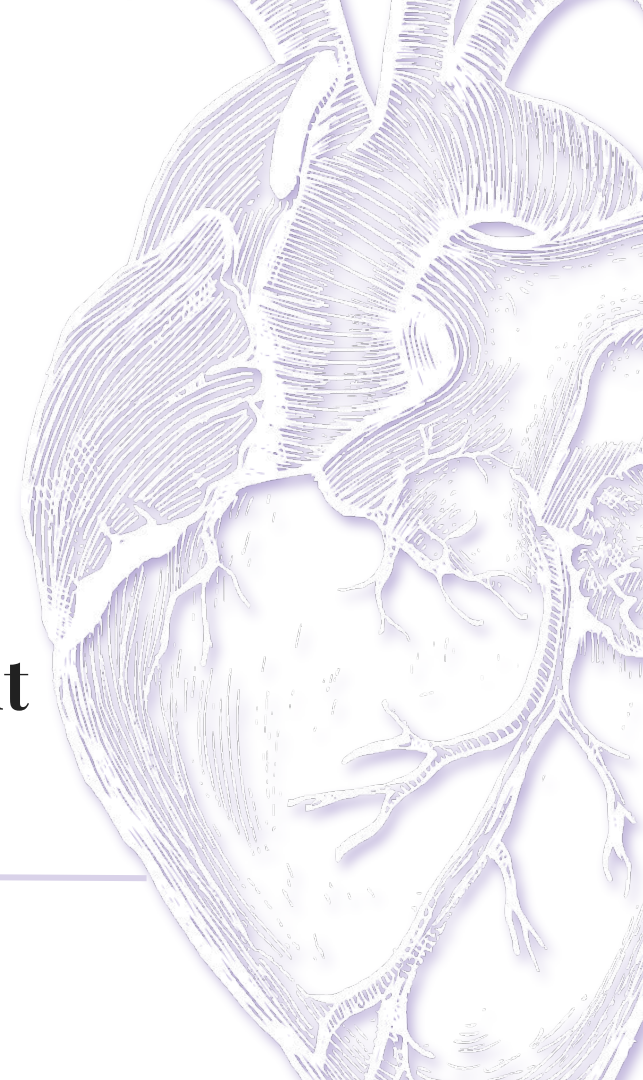


Stroke volume, cardiac output & its regulation (preload, contractility & afterload)

Editing File

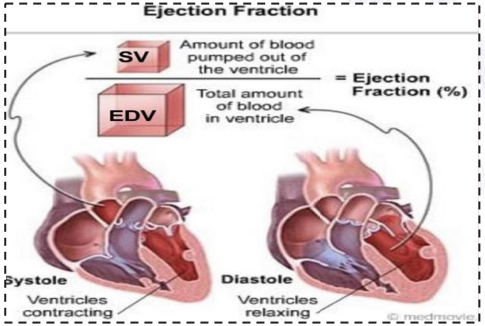
Main text Female's slide Male's slide Important text Doctor's note Extra

Objectives:

- Define stroke volume, cardiac output, venous return, cardiac index , cardiac reserve, end-systolic volume and end-diastolic volume.
- Understand the concept of preload and afterload.
- Know the method for measurement of CO (The direct Fick's method, 2-dimensional echocardiography, ultra-fast computer tomography).
- Understand the determinants of CO and how CO is regulated.
- Understand and describe the factors affecting the EDV (the venous return) & ESV ,SV & CO.
- Know and explain how cardiac contractility & heart rate changes affect CO.
- Identify the factors that affect heart rate.
- Causes of pathological low or high cardiac output .

Remember !

End-diastolic volume (EDV)	كمية الدم الموجودة عند أقصى اتساع للـ Ventricle Volume of blood in ventricles at the end of diastole ≈ 110-120 / 130 mL
End-systolic volume (ESV)	Resistance Volume of blood left in ventricles at the end of systole ≈ 40-60 mL
Stroke volume (SV)	Volume or amount of blood ejected or pumped from/by ventricles during systole (per each **beat**) and it's around 70/75-80 mL/beat
Ejection fraction (EF)	percentage of blood that is pumped out of the heart during each beat / fraction of EDV which is pumped with each heart stroke and it's around ≈ 50-70% , EF is a key indicator for diagnosing heart failure & cardiac health (<40%)

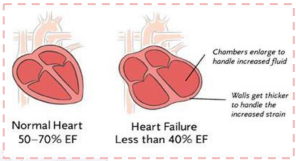


SV= EDV - ESV
These (EDV &ESV) determine the stroke volume

How EDV and ESV determines the stroke volume ?
*EDV ↑ = SV ↑
(EDV) كلما زادت الكمية الداخلة ما قبل الانقباض
(SV) معناه إنه فيه كمية كبيرة بتطلع وقت الانقباض
*ESV↓ = SV ↑
(ESV) لأن كل ما قلت كمية الدم الموجودة في القلب بعد الانقباض
(SV) معناه إن الدم الطالع هو الأكثر
- any factor that affects EDV or ESV will affect SV

نسبة الدم المقذوف

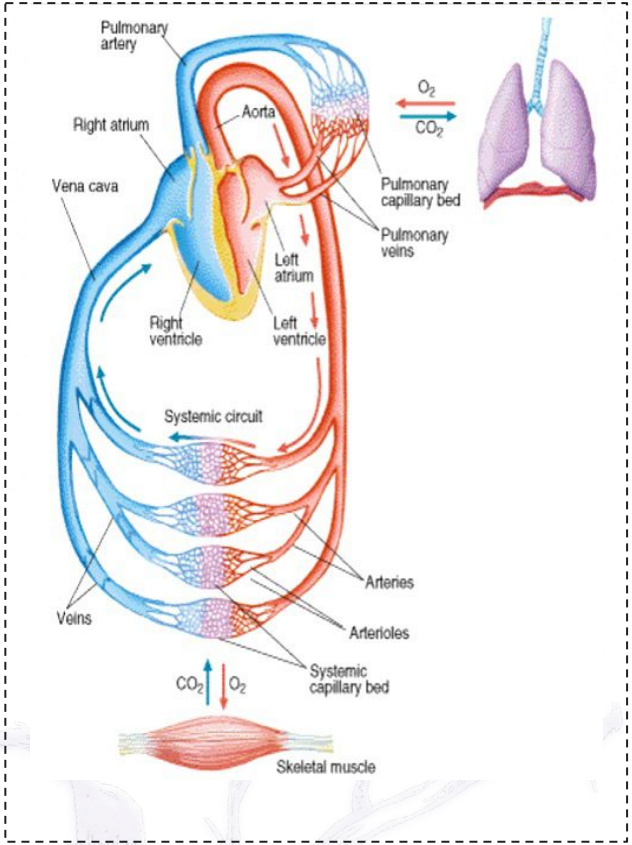
$$\frac{\text{SV or (EDV - ESV)}}{\text{EDV}} \times 100 = \frac{75*100}{120} = 62.5$$



The heart as a pump / coupling of cardiac & vascular functions.

Male's slide

- The ventricles function as the central pumps in the closed circuit of the CVS. Thus, the **cardiac output** should be **equal to** the rate of **venous blood return** to each ventricle.



Cardiac Output

Definition	- The volume or amount of blood pumped /ejected by each ventricle per **minute**. - Around 5L/min in an adult at rest /average adult so entire blood supply passes through the body once each minute .
Equation	**Cardiac output(CO)**= stroke volume(SV) X heart rate (HR) Ex: CO=(70 or 75) X 70= (5 or 5.2)L/min
Normal values	-For resting adult /average =5L/min. - Men ≈ 5.6L/min. - Women ≈ 4.9L/min
Factors affecting it	Extra 1-Body metabolism due to increase in blood flow 2-Pregnancy 3-Body temperature. For every degree the body's internal temperature rises, the heart beats about 10 beats per minute faster. 4-Sympathetic activity 5-Exercise 6-Hyperthyroidism

Physiological Changes in CO

Meals:
During the first 3 hours after meals , ↑ CO by ≈ 30% to enhance blood flow in the intestinal circulation (splanchnic circulation or flow)

Pregnancy:
Later months of pregnancy, ↑ CO by ≈ >30% due to increased uterine blood flow. Also ↑ cardiac index because ↑ surface area

Weather:

- At environmental temperature **above** 30°C, ↑ CO due to ↑ skin blood flow & sweat gland, because of the vasodilatation that happens in the skin to maintain the temperature.
- At **low** environmental temperature ↑ CO due to shivering (muscle contraction) that ↑blood flow to the muscles.

Emotion:
During anxiety and excitement, ↑ sympathetic activity ↑ CO up to 50% - 100%.

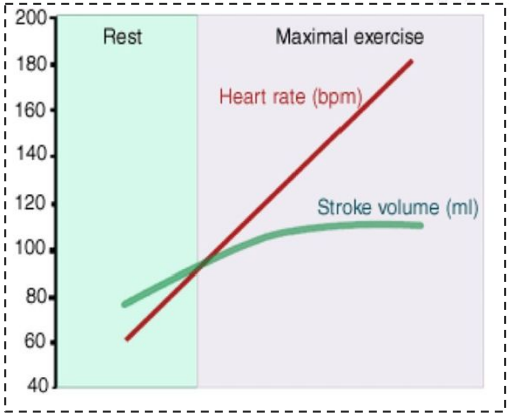
Position: بسبب الجاذبية يقل الدم العائد للقلب
Sitting or standing from the lying position,↓ CO by 20-30%. (if you were in a sitting position and then suddenly stood up , this will cause a decrease in the venous return and blood volume because of **Gravity** —> decrease cardiac output)

Exercise:
more detailed in the next slide

Effects of Exercise on HR, SV & CO

Male's Dr : No need to memorize these numbers.

Moderate Exercise	Severe Exercise
↑ HR ≈ 200% of resting, (140 beats/min)	↑ HR ≈ 300% of resting, (200 beats/min)
↑ SV ≈ 120% (85 ml)	↑ SV ≈ 175% (125 ml)
↑ CO ≈ 240% (12 L)	↑ CO ≈ 500% ~ 700% (25-35 L)



When the heart rate reaches 200 it can't be higher because there will be no time for ventricular filling

Exercise → ⬆️HR → ⬆️SV

In **athletes**, maximum CO may be 35L or more (that's normal for them because they are trained), can't increase maximum HR beyond 200 beats/min. Hence, SV increase to 175 ml.

In **non athletes**, maximum CO may be 30L or more , can't increase maximum SV beyond 125mL. Hence , HR increase to 220 beats/min. Remember normal is 5L.

Why is the HR in athletes not considered high compared to non athletes ?
Because the physiological hypertrophy that is associated with the athlete's heart can lead to a higher stroke volume compared to normal.

Cardiac Index (CI) & Cardiac Reserve

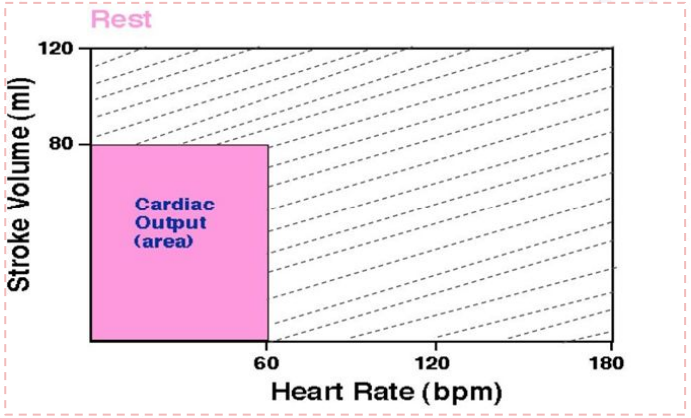
1. **Cardiac Index :**
is CO per square meter of body surface area (cardiac output increases approximately in proportion to the surface area of the body), CI = CO/m²
Normal CI = (3.2 Liters/min/m² body surface area)

For example Pregnant women

2. -Since CO vary with size of individual, age & gender (due to body mass) (e.g; women have smaller CO than men, children have smaller CO than adults).
-Thus it is important to relate heart performance to the size of the individual (and surface area)

3. **Cardiac reserve :** CO at exercise - CO at rest = **Cardiac reserve**
The maximum volume of blood that the heart is capable of pumping/min—The resting CO i.e. at rest
(5L=5-10 حقه بيكون reserve ال إذا , normal = 5L و maximum = 10 L مثل شخص عنده ال)

4. During exercise, the CO can ↑ to 20-25 L/min. In well trained athletes, CO can ↑ as high as 35 - 40 L/min.



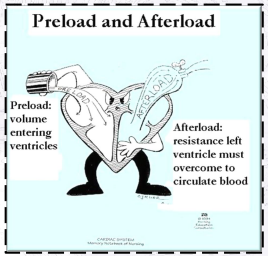
CO in athletes =35 L\min,
CO at rest = 5L\min,
Cardiac reserve = 35-5=30

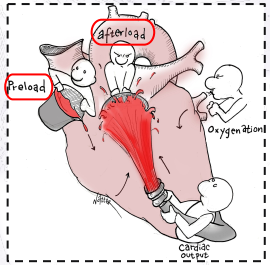
Important slide

Preload & Afterload

Preload Afterload

Preload	Afterload
It is the amount of blood presented to ventricles / that returns to the heart from veins (VR). (It depends on the venous return , if it's right →systemic veins , if it's left →pulmonary veins)	It is the **resistance** against which the ventricles contract. **e.g./Aortic stenosis**
• It is the load on the muscle in the relaxed state. •↑VR→ ↑EDV and stretches or lengthens the ventricular muscle fibers, and that will increase the force of contraction (by frank - starling's law) **Preload ↑ in:** • Hypervolemia (amount of blood in the body increase) • Heart failure (the heart cannot pump the blood) • Exercise • Increased preload:↑venous return → increase in SV based on the Frank–Starling relationship.... loop	• Afterload on the left ventricle↑ when aortic pressure ↑in : • aortic valve stenosis • systemic vasoconstriction • arterial hypertension • Afterload on the right ventricle ↑when pulmonary artery pressure ↑in: • pulmonary valve stenosis • pulmonary vasoconstriction • pulmonary hypertension • Increased afterload: due to an increase in aortic pressure → decrease in stroke volume.





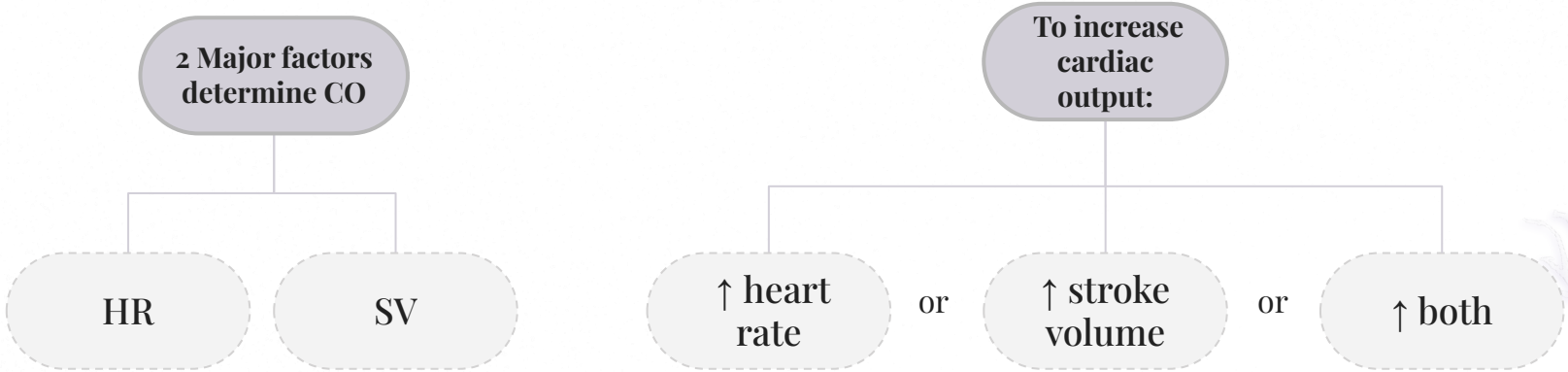
EDV and Preload are related concepts, but they represent different aspects:

-**End-diastolic volume** is the amount of blood in maximum stretch of the ventricle

-**preload** is more physiological , it's the degree of stretch on the myocardial fibers , caused by *End diastolic volume* (: hope you get it

Regulation of CO

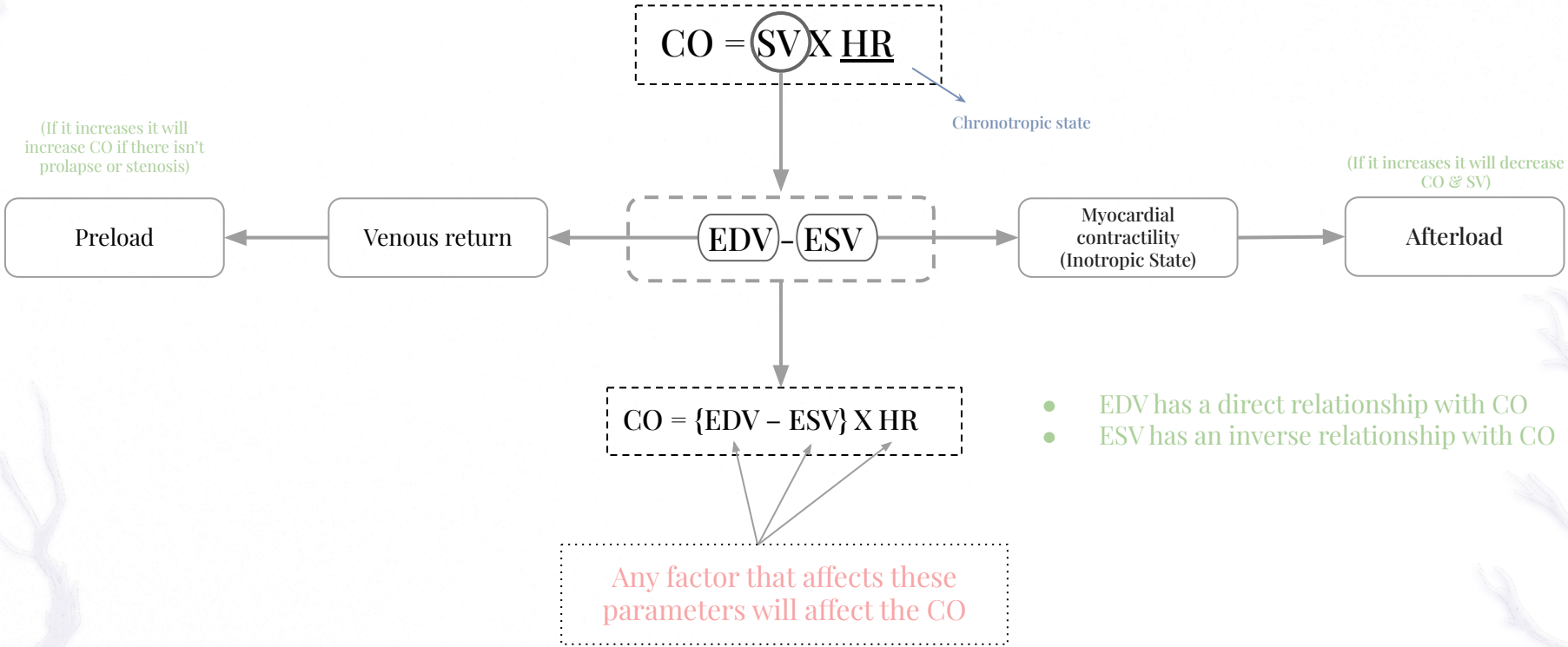
CO is crucial since it is the amount of blood that flows into the circulation and is responsible for transporting substances to and from the tissues. Thus, the body has strict control mechanisms that maintain adequate CO.



What are the factors that regulate the cardiac output?

- Any factor that affects the stroke volume and heart rate whether it increases or decreases them . ↑ stroke volume ↑ both
- We can say any factor that affect EDV and ESV and HR , because the SV=EDV-ESV

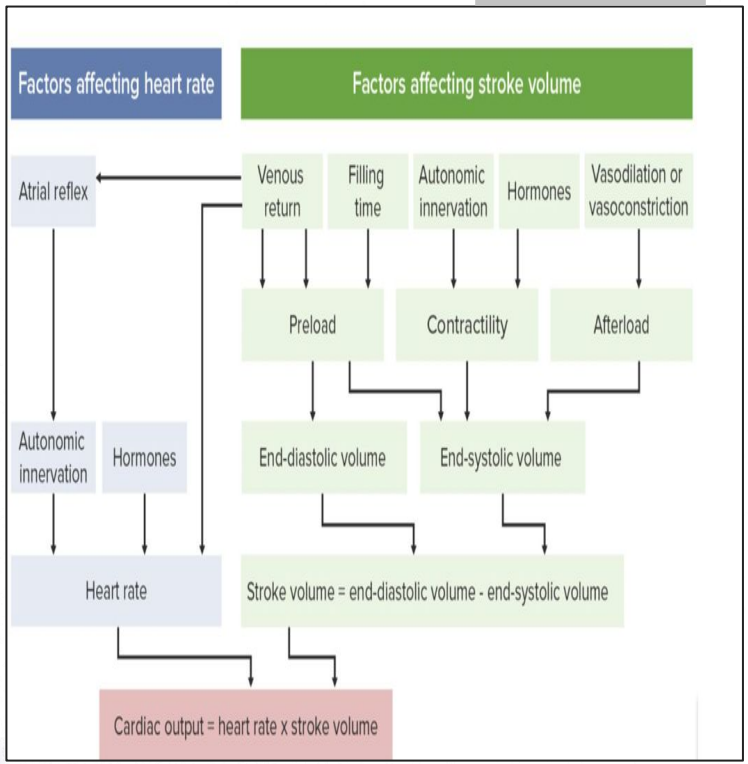
Determinants of the CO



- EDV has a direct relationship with CO
- ESV has an inverse relationship with CO

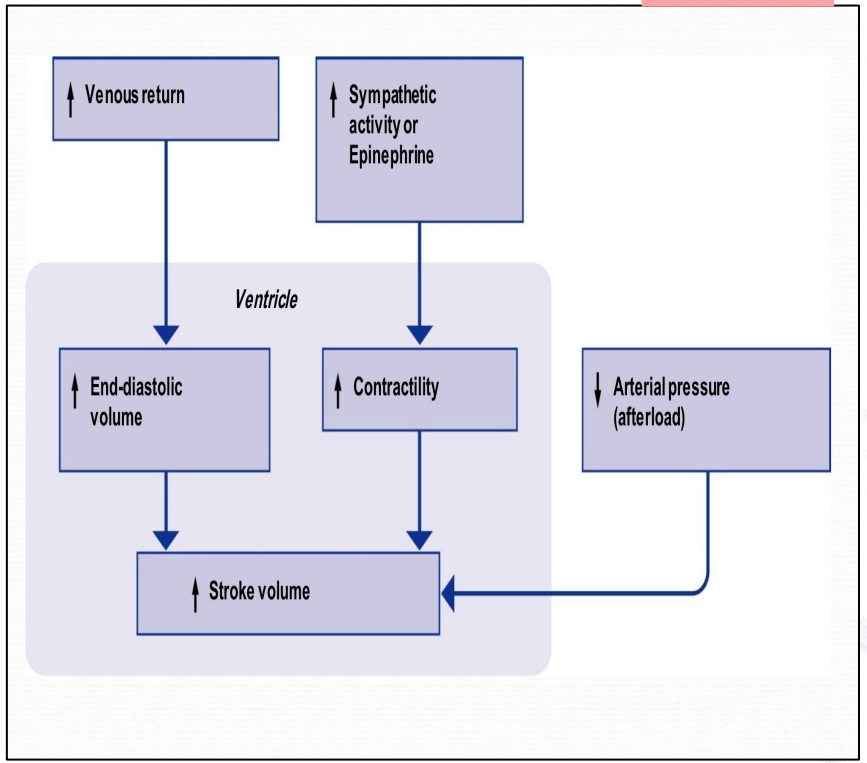
Summaries

Extra slide



Female's slide

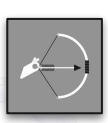
Factors Influencing Stroke Volume



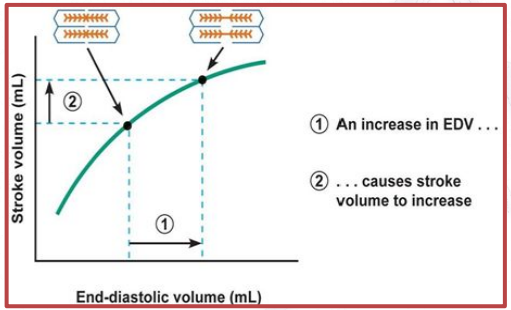
Frank-Starling's Law

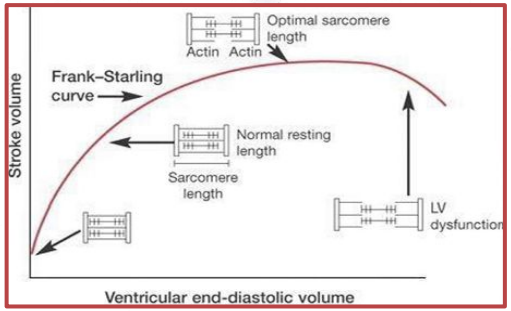
Important slide

- It is the intrinsic relationship between EDV and SV known as starling's law of heart.
- It reflects the ability of the heart /ventricle to change its force of contraction and therefore SV in response to changes in venous return.
- If EDV (preload) is increased, the ventricular fiber length is also increased, resulting in an increased 'tension' of the muscle (i.e SV increases in response to increase of the EDV).
- Within physiologic limits, the heart pumps all the blood that returns to it by the way of the veins (physiological importance: happens to prevent stasis of the blood).
- The **greater the stretch** of the cardiac muscle the **greater would be the force of contraction**. i.e.: the energy of contraction is proportional to the initial length of the cardiac muscle fibers (the EDV) and for the muscle proportional to EDV.
- Why? During stretch, actin & myosin filaments are brought to more optimal degree of sliding(easy to slide on each other), therefore increase force of contraction. The more the stretch of the ventricular wall, the more the ventricles will contract back and eject blood .
- In other words the more you stretch the cardiac muscle the harder it will recoil.



EDV (volume of blood during **diastole**) , stretching Cardiac muscle ⟶ Force of contraction during **Systole**





-**Y axis (stroke volume)** = we can also call it cardiac output, it's the same.

-**X axis (ventricular EDV or atrial pressure)**, if it ↑, it will stretch the muscle.

Within the physiological limit : increase in the initial length of muscle fiber will increase the force of contraction , BUT overstretch will damage the muscle

1- End diastolic volume

- EDV= is the volume of blood in the ventricles prior to ventricular ejection.
- How does the EDV affect the SV and hence the CO?

↑↑EDV→ ↑The stretch in the myocardium (↑ initial fiber length) → ↑ Myocardial contractility (↑Strength of contraction) by Frank-Starling mechanism

Factors affecting preload EDV:

EDV ↑ with:	EDV ↓ with:
- Increased total blood volume. (transfusion) - Increased venous return. - Increased venous tone. (constriction in veins) - Increased skeletal muscle pump (exercise). - Increased negative intrathoracic pressure. - Stronger atrial contraction.	- Standing. Gravity - Decreased venous return. - Increased intrapericardial pressure. Compression of the heart (it will decrease elasticity of the heart) - Decreased ventricular compliance.

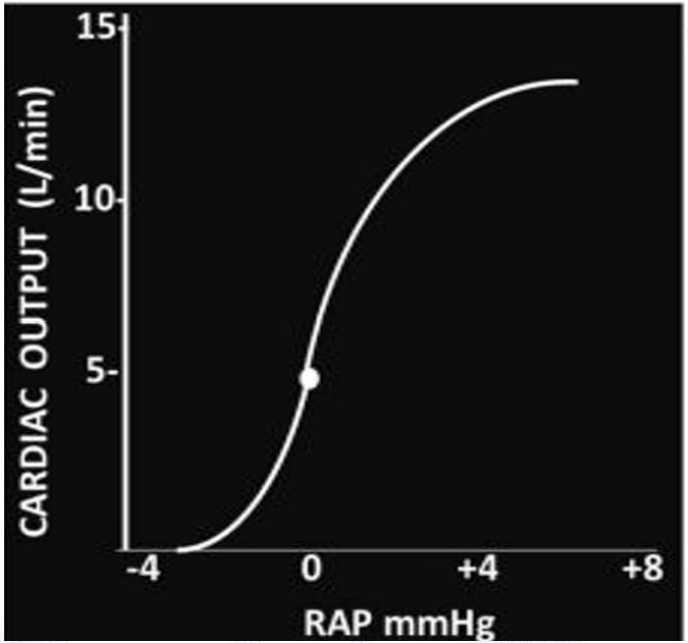
Indices of left ventricular preload:

- Left ventricular end-diastolic volume (LVEDV).
- Left ventricular end-diastolic pressure (LVEDP).

Preload= EDV
Afterload= left ventricular systolic pressure
(لأنه زي ماقلنا الافترلود هو المقاومة اللي بواجهها الدم وهو طالع من الفنتريكل فطبيعي تكون مساوية للضغط اللي على جدار الفنتريكل)

Cardiac Function curve, Starling's Law of the heart

- As the venous return increases, RAP increases, and EDV and End diastolic fiber length increase.
- Increase in end-diastolic volume (i.e., right atrial pressure) produce increases in cardiac output by the Starling mechanism.
- Increase in end-diastolic fiber length produce increases in cardiac output.
- When the mean RAP is about 0 mmHg (note that RAP normally fluctuates with atrial contraction and respiration), the cardiac output in an adult human is about 5 L/min.



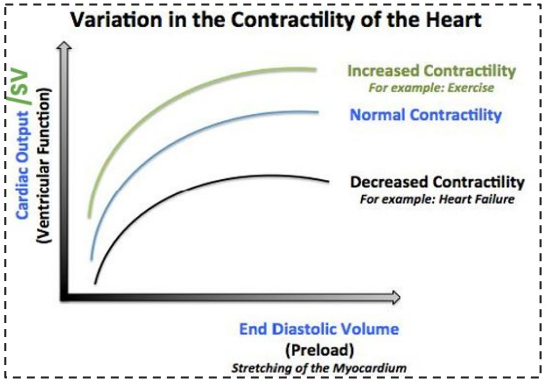
2- The End-systolic Volume (ESV)

ESV = The volume of blood remaining in the ventricle at the end of systole

↑ ESV → ↓ stroke volume
↓ ESV → ↑ stroke volume

ESV is determined by

- Cardiac contractility
  - ↑ contractility → ↓ ESV → ↑ SV & CO
  - ↓ contractility → ↑ ESV → ↓ SV & CO
- Afterload (in next slide)



- **Green line (shifted upward) :** increased contractility will increase the SV —> increase CO because the ESV decreased المتبقي قليل فأكيد الطالع أكثر لأنها نسبة
- **Blue line :** normal
- **Black line :** decreased contractility will decrease the SV —> decrease CO because the ESV increased المتبقي أكثر فأكيد الطالع هو الأقل لأنها نسبة

Factors Affecting Afterload

1. It is expressed as tension (load) developed in the wall of ventricles during systole to open the semilunar valves and eject blood to aorta/pulmonary artery.

2. Left ventricular afterload represents the force that the muscle must generate to eject the blood into the aorta.
   - When the aortic pressure (afterload) increases, The Lt ventricle ejects less blood →↓ SV →↑ESV
   - When the aortic pressure (or Arterial Pressure) is reduced, the velocity of shortening of the LV, myocardial fibers increases. Hence, with ↓ afterload, the LV can eject blood more rapidly and Easily So ... {SV ↑ , ESV ↓}.The opposite is true with increased LV afterload.

3. Right ventricular afterload represents the force that the muscle must generate to eject the blood into pulmonary artery.
   - When pulmonary pressure (afterload) increases, The Rt ventricle ejects less blood → ↓ SV →↑ ESV.

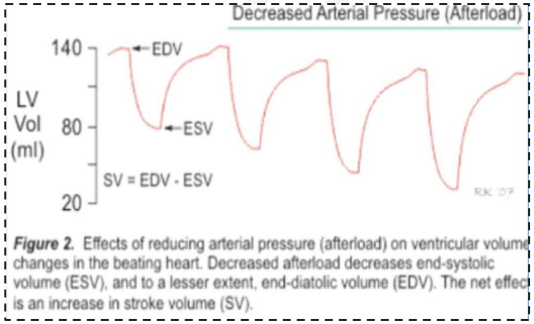


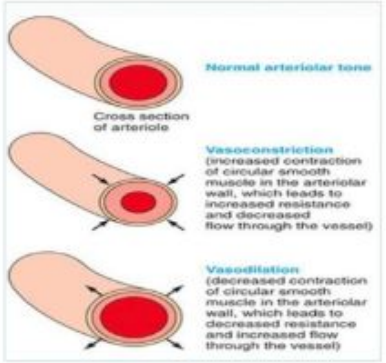
Figure 2. Effects of reducing arterial pressure (afterload) on ventricular volume changes in the beating heart. Decreased afterload decreases end-systolic volume (ESV), and to a lesser extent, end-diatolic volume (EDV). The net effect is an increase in stroke volume (SV).

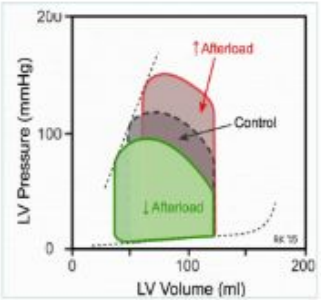
Female's slide

Factors Affecting Afterload

- Afterload ↑ by any factor that restricts blood flow.
- Vasoconstriction ↑↑ the peripheral vascular resistance→ ↑↑ ESV → ↓↓ SV & CO.
- Vasodilatation ↓↓ the peripheral vascular resistance→ ↓↓ ESV → ↑↑ SV & CO.

Causes of ↑ left ventricular afterload:	Causes of ↑ right ventricular afterload:
• Aortic Valve stenosis • Arterial hypertension • Vasoconstriction (the peripheral vascular resistance)	• Pulmonary valve stenosis • Pulmonary hypertension • Pulmonary vasoconstriction



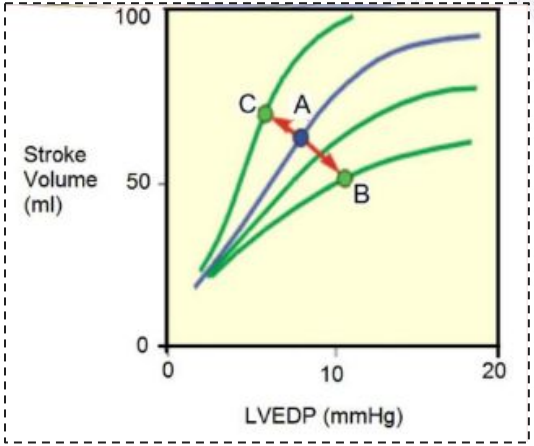


Afterload and CO are inversely proportional , why ? Because that means increase in ESV —> decrease SV —> decrease CO

Effect of afterload on Frank Starling Curve

- Increased afterload leads to ↑ ESV And ↓ SV .Thus shifts the Starling's curve down and to the right (from A to B).
- Reduced afterload ↓ ESV and ↑ SV, Thus shifts Starling's curve up and to the left (A to C)

Explanation: an increase in afterload decreases the velocity of fiber shortening. This reduces the rate of volume ejection so that more blood is left within the ventricle at the end of systole →↑ESV.

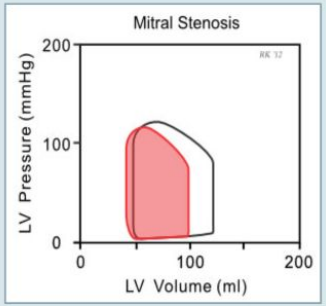


✧ **Diagram called : Frank-Starling's Law**
✧ Here we talk about afterload and cardiac output
✧ **A**= normal
✧ **B** (shift downward)= increase in afterload —> increase in ESV —> decrease in SV and CO
✧ **C** (shift upward) = decrease in afterload —> decrease in ESV —> increase in SV and CO
✧ We can ask you : what point c indicate ? mention two causes for this shift ? and so on .

X axis : we can also call it : LVEDV (mmHg). Because LVEDV and LVEDP are both indicators for left ventricular preload as we said earlier

Diagrams

Stenosis = narrowing of a valve

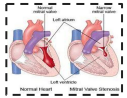


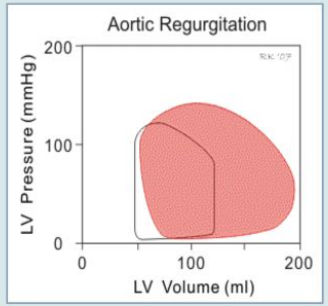
Mitral stenosis

Impaired LV filling

قل الدم المضخوخ لأن الأتريم أصلا متضيق، فما تعبى الفنتريكل كفاية

↓EDV & ↓preload

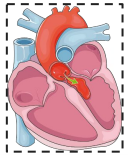


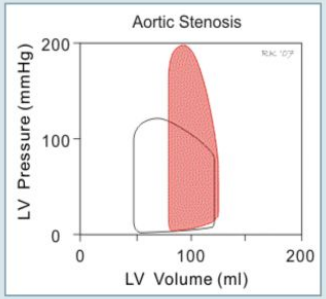


Aortic regurgitation

No true isovolumetric relaxation

هنا بحكم ان الدم يرجع من الاورطي صارت الفنتريكل مليانه بزيادة، لان الاتريم يعبي و الاورطي يرجع فالفنتريكل↑EDV



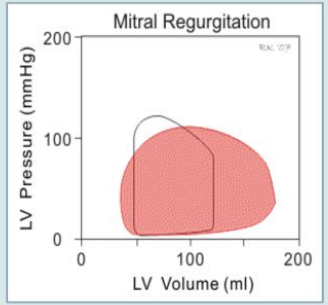


Aortic stenosis

High outflow resistance;
LV
emptying impaired

بسبب التضيق،الدم مايطلع الا بضغط عالي وفوقها يقل الستروك فوليوم و يكثر الدم المتبقي في الفنتريكل

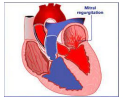
Here the murmur is during the systole



Mitral regurgitation

blood is not only ejected into the aorta but also back up into the left atrium.

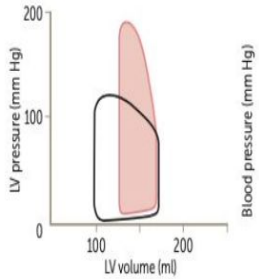
هنا فالانقباض الدم يتسرب للاتريم فأول مايجي الاتريم يعبي بعدها يصير يعبي كمية ضخمه↑ EDV



Thanks to team 443

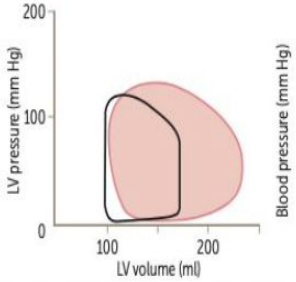
Extra explanation for the Diagrams

Aortic stenosis



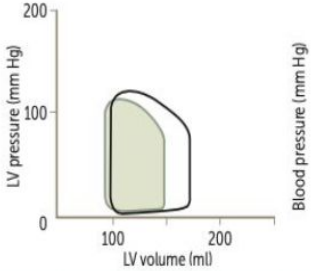
- High outflow resistance; LV emptying impaired..
↑ LV pressure (stenotic valve)
↑ ESV (ejection fraction ↓)
No change in EDV (if mild)
↓ SV

Aortic regurgitation



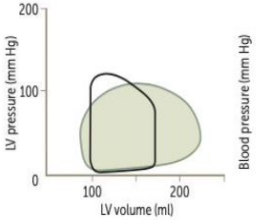
- No true isovolumetric relaxation
↑ EDV
↑ SV
↑ ESV

Mitral stenosis



- Impaired LV filling
↑ LA pressure (stenotic valve)
↓ EDV
↓ ESV
↓ SV
Ejection fraction is initially normal

Mitral regurgitation



↓ESV due to ↓resistance and ↑regurgitation into LA during systole (blood is not only moving to aorta, it's pushed to the atria as well)

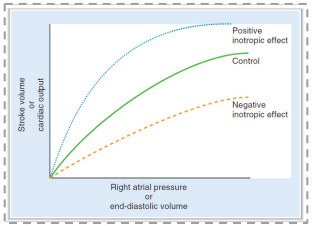
↑EDV due to ↑LA volume/pressure from regurgitation→ ↑ventricular filling

↑SV (forward flow into systemic circulation plus backflow into LA)

Inotropic, Chronotropic & Dromotropic

Contractility

- Positive inotropic agent = ↑ in contractility
- Negative inotropic agent = ↓ in contractility



Heart rate

- Positive chronotropic effect = ↑ in HR
- Negative chronotropic effect = ↓ in HR

Conduction velocity

- Positive dromotropic effect = ↑ in conduction velocity
- Negative dromotropic effect = ↓ in conduction velocity

Heart Rate

Normal heart rate is regular sinus rhythm =60-100 beats/min
(> 100) beats/min → Tachycardia
(< 60) beats/min → Bradycardia

Since the CO = SV X HR, ↑ heart rate →↑ CO. HR is proportional to CO because it increases the the contraction —> increase SV —> increase CO .

At heart rates up to about 180, ventricular filling is adequate as long as there is enough venous return, and CO per minute is increased by an increase in HR.

However, at very high heart rates, filling may be compromised to such a degree that CO per minute falls.

Mechanism of autonomic control of Heart rate

Sympathetic stimulation

Mechanisms:
The ß1-adrenoreceptors on SA node cells are coupled to excitatory G- proteins.

This results in:

1. Opening of Na+ and Ca2+ channels(influx) → speed up the rate of depolarization and hence the heart rate.
2. The net effect is: → swiffer drift to threshold → greater frequency of action potential → increase the heart rate.

Parasympathetic stimulation

Mechanisms:
M2 (muscarinic-2) ACh receptors respond to ACh from the vagus nerve by activating a different G-protein.

This results in:

1. opening of potassium channels in the cell membrane (efflux) →hyperpolarizes the cell and makes it more difficult to initiate an action potential.
2. The net effect is: → slower drift to threshold → lesser frequency of action potential → decrease the heart rate.

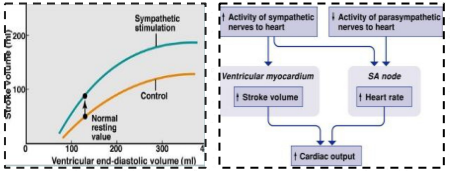
Regulation of Heart Rate

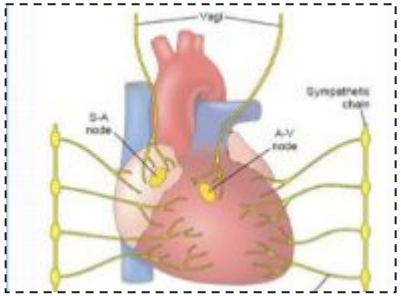
1. Autonomic nervous system

Sympathetic nerves/stimulation

Innervate the whole heart which leads to increase firing of SA node, increase HR and the contractility through noradrenaline or adrenaline release which stimulates heart β1-receptors.

E.g during stress, crisis or low blood pressure.





Notice how the SA node & AV node have vagal innervation, while ventricles do not

Parasympathetic nerves

Innervate the SA & AV nodes, atria, and Purkinje system, and don't innervate most of the ventricular myocardium by vagus nerve which leads to decrease firing of SA node and slow HR, (but has little inotropic action). Through Ach release that stimulates Muscarinic (M2) receptor.

Under normal conditions the SA node is under continuous vagal tone causing its basal firing rate to decrease (Vagal Tone). Usually the SA node has a high ability of firing , but vagal tone will decrease it .This means normally the parasympathetic has more control than the sympathetic.

Female's slide

Regulation of Heart Rate

2. Physical Factors

- Age: Resting HR is faster in fetus and then gradually decreases throughout life
- Temperature: Heat increases HR as occurs in high fever. Cold has the opposite effect.
- Gender: HR is faster in females (72-80 beats/min) than in males (64-72 beats/min) (because males have a stronger vagal tone)
- Exercise: Increases HR through sympathetic nervous system.

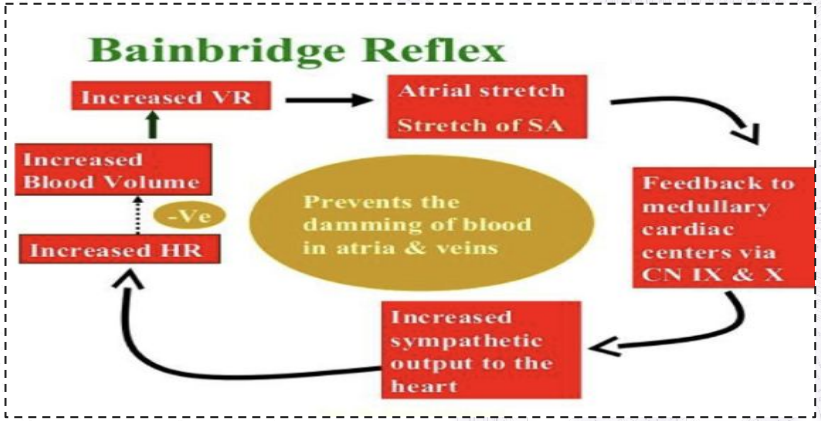
3. Hormones and drugs:

- Epinephrine and thyroxine increase HR. (epinephrine: beascuse it's a sympathetic neurotransmitter , thyroxine : that's why people with hyperthyroidism have increased HR).
- Increased Ca^{2+} level in blood causes prolonged contraction
- Reduced Ca^{2+} level in blood depress the heart

Regulation of Heart Rate

4. Blood Volume

- Atrial Reflex (Bainbridge reflex)
- This reflex adjusts heart rate in response to venous return or blood volume
- Increase blood volume, stimulates stretch receptors in right atrium
- This triggers increase in heart rate through increased sympathetic activity.



442: فيه Stretch receptors في جدار ال Atria إذا إنشد ال Atria تحفز القلب وتزيد ال HR عشان القلب يصرف الدم الي زاحم القلب

When the VR increase due to increase in blood volume –>atrial muscles stretch through **stretch receptors (atrial receptors) NOT through Frank-Starling's Law** .
These stretch receptors send impulses to cardiovascular center —> increases sympathetic activity —>↑HR

Frequency-Force relationship

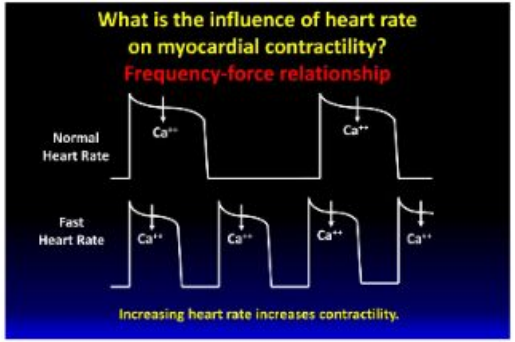
What is the influence of heart rate on myocardial contractility?

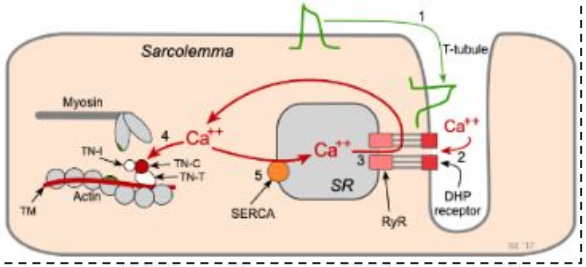
1. The heart rate has an influence on cardiac contractility as well (frequency-force relationship) → due to accumulation of Ca2+ ions within the myocytes

2. Increasing heart rate increases contractility (both relaxation and contraction are active processes , both need energy)

3. This frequency dependency of force generation is probably due to ↑ Ca2+ availability to the contractile proteins within cardiac myocytes as a result of:
   - more of depolarizations/min → more frequent plateau phases → more Ca2+ entry
   - ↑ The magnitude of Ca2+ current→ ↑ the intracellular Ca2+ stores.

Both effects enhance Ca2+ release & uptake by the sarcoplasmic reticulum, thus Ca2+ availability to the contractile proteins with more force generation through cross-bridge cycling





Male's slide

Factors Affecting the Contractility

Positive inotropic effect	Negative inotropic effect
Sympathetic stimulation: ● Adrenaline & Noradrenaline ● Calcium ion ● Caffeine ● Drugs e.g. Digitalis (Digoxin)	Parasympathetic stimulation: ● Acetylcholine ● Potassium ion ● Hypoxia (Decrease oxygen) ● Acidosis ● Bacterial toxin ● Drugs e.g.. Calcium channel blockers, β- Blockers

FORCE- VELOCITY RELATIONSHIP

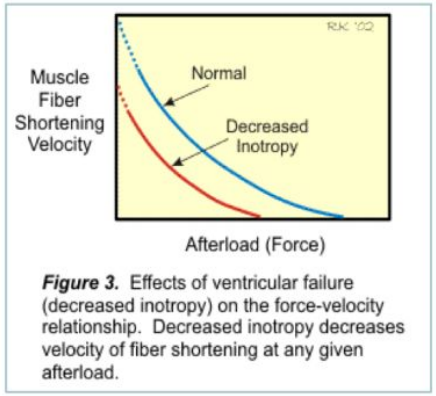
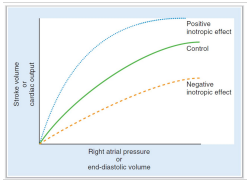


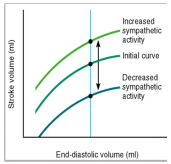
Figure 3. Effects of ventricular failure (decreased inotropy) on the force-velocity relationship. Decreased inotropy decreases velocity of fiber shortening at any given afterload.

- At any given preload and afterload, a loss of inotropy results in decrease in shortening velocity of the cardiac fibers

- Inotropy: force of heart contraction

Frank-Starling's curve





- At rest the heart is under parasympathetic tone.

- Sympathetic nerve stimulation increases cardiac contractility, Noradrenaline enhances calcium entry into cardiac cells

- Parasympathetic stimulation has little effect on contractility due to the innervation pattern of the heart.

Pathological low or high cardiac output

Causes of high cardiac output	Causes of low cardiac output
• Hyperthyroidism: the increase in the CO is due to the high metabolic rate → vasodilatation → ↑CO to 50%+ of control. • AV fistulas (direct connection between Artery & vein) it's an abnormal opening between arteries and veins , so the blood flow from artery to veins , and the pressure in the artery very high , so when it goes to the veins it will increase the venous return —> increase CO الدم يروح على طول من الشريان للوريد • Fever • Anxiety • Exercise • Anaemia	• Low Venous Return (e.g., haemorrhage) • Reduced contractility (e.g., heart failure) • Tachyarrhythmias (e.g., atrial fibrillation and ventricular tachycardia) • Marked bradycardia (e.g., complete heart block)

Measurement of cardiac output

1) Fick's Principle

It assumes that the amount of oxygen consumed = the amount of oxygen delivered by the arterial blood - the amount of oxygen in venous blood of the organ, as shown :

$$CO\ (L/min) = \frac{\text{Total O2 consumption}}{aO2 - vO2}$$

- AO2= Arterial O2 concentration
- VO2= Mixed O2 venous concentration

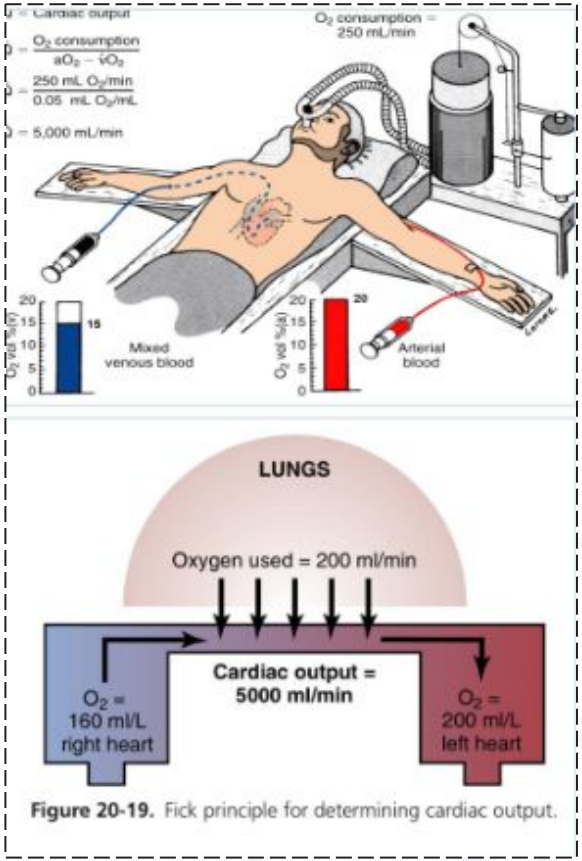


Figure 20-19. Fick principle for determining cardiac output.

Measurement of cardiac output

2) 2-Dimensional Echocardiography

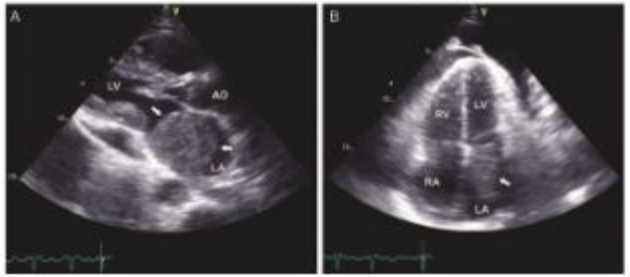
- Records real-time changes in ventricular dimensions during systole and diastole

- It thus computes stroke volume, which when multiplied by heart rate, gives the cardiac output.

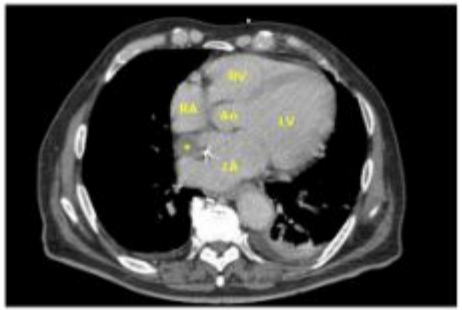
3) Ultra-fast computer tomography

- In pathological low or high CO, can measure changes in ventricular diameter at several depths to estimate changes in ventricular volume.

Dimensional Echocardiography



Ultra-fast CT



Check here for our summary
Highly recommended !!!!!

Sorry but if you will not cheack it راحت عليك المليون

MCQs:

Answers

For more question check our summary file!

1/B
2/B
3/C

1

If the ejection fraction increases, there will be a decrease in?

A	Cardiac output	B	End systolic volume	C	Heart rate	D	Pulse pressure

2

Calculate the CO if ESV= 45ml,EDV=115ml & HR=85 bpm:

A	4.6 L	B	5.95 L	C	13.6 L	D	1.35 L

3

CO on the right side of the heart is equal to what percentage of the CO on the left side of the heart?

A	25%	B	30%	C	100%	D	80%

MCQs:

Answers

For more question check our summary file!

4/C
5/A
6/C

4

A 72-year-old woman who is being treated with propranolol can't maintain her previous exercise routine. Her physician explained that the drug has reduced her cardiac output. Blockade of which receptor is responsible for this effect?

A	α1 receptors	B	β_2 receptors	C	β_1 receptors	D	Muscarinic receptors

5

Which of the following causes an increase in the left ventricular afterload ?

A	Vasoconstriction	B	Pulmonary vasoconstriction	C	Pulmonary hypertension	D	Pulmonary valve stenosis

6

Which of the following causes an increase in the CO?

A	Heart failure	B	Tachyarrhythmia	C	AV fistulas	D	Hemorrhage

SAQ

Define end-diastolic volume, end-systolic volume and stroke volume.	Slide 3
Enumerate the factors that affect CO.	Slide 6
What are the pathological causes of Low & High cardiac output?	Slide 29
Enumerate THREE factors that affects heart contractility, either positively or negatively. (Inotropic effect)	1. Calcium Ion 2. Caffeine 3. Acidosis

Finally you have arrived , we have been waiting for you !!

Meet our team !

Team leaders

Rimaz Alhammad

Noreen Almaraba

Rayan Alshehri

Omar Albaqami

Aljoharah Alyahya

Heroes of the lecture :

Waleed Alanazi

Shoug Albattah

Did you like the lecture ? we mean our work :)

Contact with us! physiology.444ksu@gmail.com