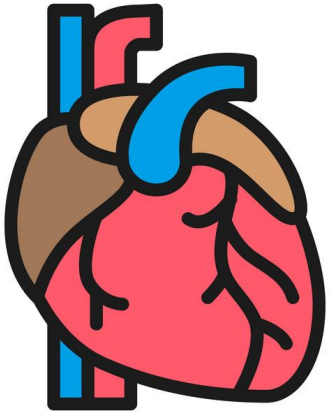
Revised & Reviewed by Abdulaziz Bahammam & Faye Wael Sendi

MED441
KING SAUD UNIVERSITY



Stroke Volume

Color Index:

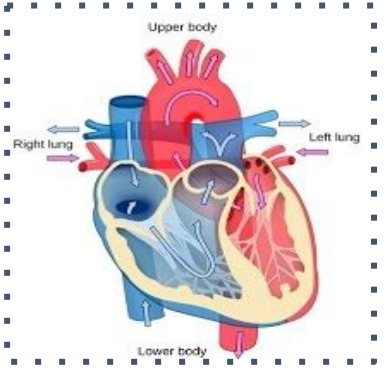
- Main text
- Important
- Girls Slides
- Boys Slides
- Notes
- Extra

Editing File

Objectives

- Define stroke volume, end-systolic volume, and end-diastolic volume ,cardiac output, venous return, cardiac index & cardiac reserve
- Understand the concept of preload and afterload
- Understand the determinants of CO and how CO is regulated
- Understand the factors affecting the EDV (the venous return),affecting the ESV
- Know how cardiac contractility & heart rate changes affect CO
- Identify the factors that affect heart rate
- Know the method for measurement of CO (The direct Fick's method)
- Describe the factors affecting the SV & CO

Remember

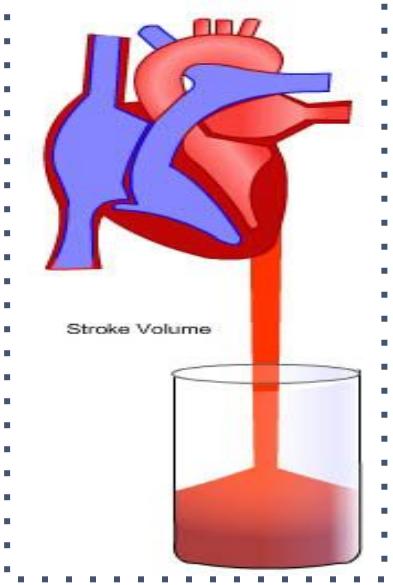


End-diastolic volume (EDV):	**Volume of blood in ventricles at the end of diastole ≈ 110-130mL**
End-systolic volume (ESV):	**Amount of blood left in ventricles at the end of systole ≈ 40-60mL**
Stroke volume (SV):	**Amount of blood ejected from ventricles during systole ≈70-80 mL/beat.**
Ejection fraction (EF):	**Fraction of EDV ejected during a heart beat =SV/EDV ≈ 60-65%.**
VENOUS RETURN	**is the Quantity Of Blood Flowing from the Veins into Right Atrium each Minute CO = VR**

The Stroke Volume

The stroke volume (SV)

Amount of blood ejected from ventricles during systole (per beat).



End-diastolic volume (EDV)

Volume of blood in ventricles at the end of diastole

≈110-130 ml

End-systolic volume (ESV)

Amount of blood ventricles left in ventricles at the end of systole

≈40-60 ml

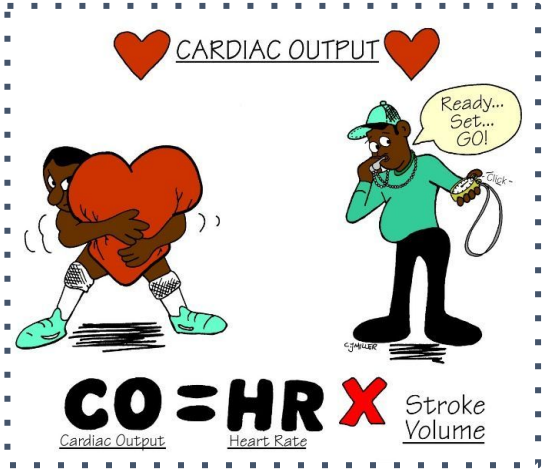
The stroke volume ≈70-80 ml

The Cardiac Output (CO)

- It is the volume of blood pumped by each ventricle per minute = 5L/min.

- Since the normal adult blood volume is about 5 L(blood make 1 cycle), the entire blood supply passes through the body once each minute.

Cardiac output (CO)
= Stroke volume (SV) X heart rate (HR)
= 70*70
≈ 5L/min. in adult at rest



Normal Values of CO at Rest

Resting CO:

- The average CO for the resting adult is 5L/min.
- For men, CO ≈5.6L/min.
- For women, CO ≈ 4.9L/min.

The factors affecting CO:

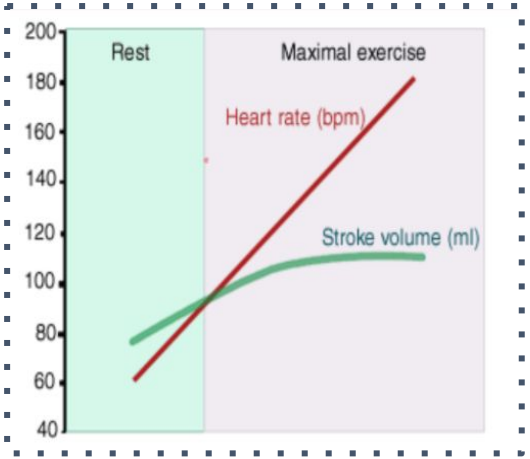
1. Body metabolism
2. Pregnancy
3. Body temperature
4. Sympathetic activity
5. Exercise
6. Hyperthyroidism

Physiological Changes in CO

- During the first 3 hours after meals ,↑ CO by ≈ 30% to enhance blood flow in the intestinal circulation.
- Later months of pregnancy ↑ CO by ≈ 30% due to increased uterine blood flow.
- At environmental temperature above 30°C ,↑ CO due to ↑ skin blood flow . because of the vasodilatation that happen in the skin to maintain the temperature.
- Also at low environmental temperature ↑ CO due to shivering that ↑blood flow to the muscles.
- During anxiety and excitement, sympathetic activity ↑ CO up to 50% - 100%.
- Sitting or standing from the lying position ↓ CO by 20-30%.
- Exercise

Effects of Exercise on Heart Rate, SV and CO

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)
	★ In athletes, maximum CO may be 35L or more - can't increase maximum HR beyond 200 beats/min. Hence, the SV ↑ to 175 ml.(125ml or 175%)



Exercise

⇩

Increase HR

⇩

Increase SV

What is the Cardiac Index (CI)

- Since : CO vary with size of individual, age & gender (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.

Cardiac index: is CO per square meter of body surface area

CI = CO/m2

Normal CI = (3.2 Liters/min/m2 body surface area)

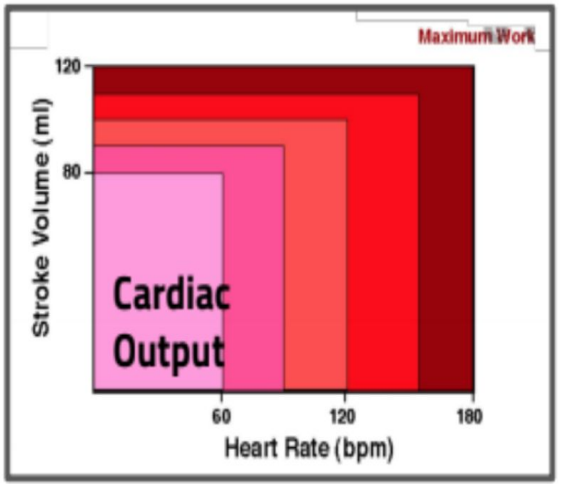
What is the Cardiac Reserve

- During exercise, the CO can ↑ to 20-25 L/min.

- In well trained athletes,

CO can ↑ as high as 35 - 40 L/min.

Cardiac reserve = The maximum volume of blood that the heart is capable of pumping/min — The resting CO at rest



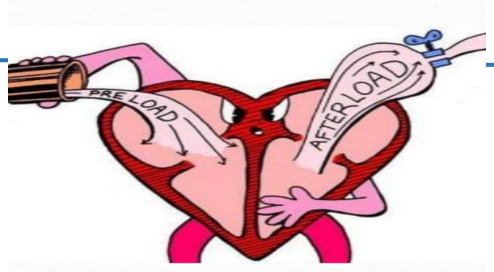
Co in the athletes =35 L\min
Co in the rest = 5L\min
Cardiac reserve = 35-5=30

Factors determine the CO

1. **HR (Heart rate)**
2. **SV (Stroke volume)**

What Are Preload and Afterload

Preload



Afterload

- It is the amount of blood that returns to the heart from veins (VR).
- It is the load on the muscle in the relaxed state.
- ↑VR→ ↑EDV and stretches or lengthens the ventricular muscle fibers.

Preload ↑ in:

- Hypervolemia amount of blood in the body increase
- Heart failure the heart cannot pump the blood

- It is the resistance against which the ventricles contract.
- Afterload on the left ventricle,↑ ,when aortic pressure ↑.
- Afterload on the right ventricle ↑, when pulmonary artery pressure ↑.

Afterload ↑ in:

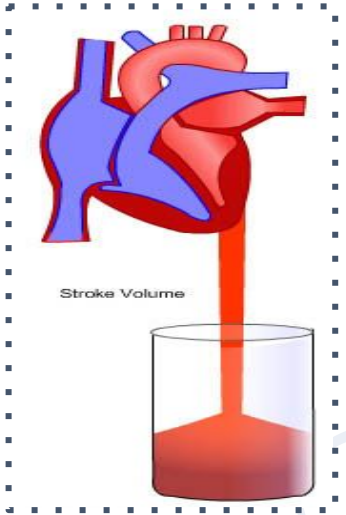
- Aortic/pulmonary stenosis increase in the resistance
- Hypertension
- Vasoconstriction in the pulmonary vessel

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.

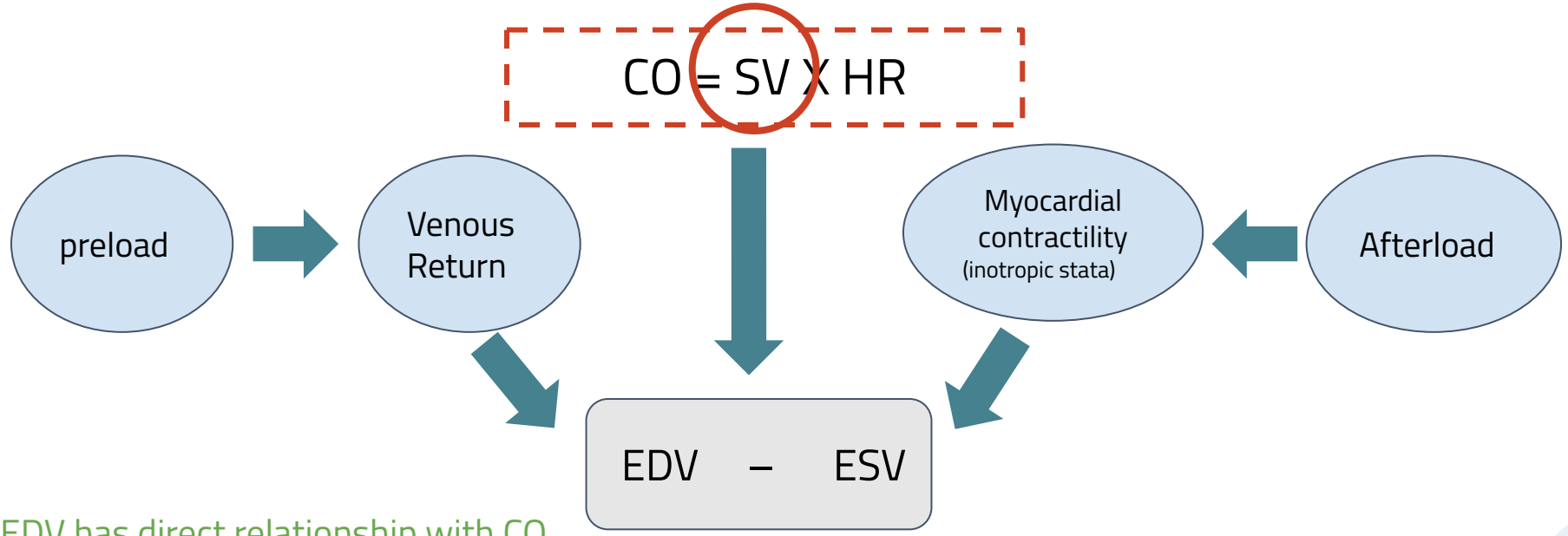
HR — ***There are 2 major factors determining CO*** — ***SV***



To increase the cardiac output

- Increase stroke volume
- Increase the heart rate
- Increase both

Determinants of the CO



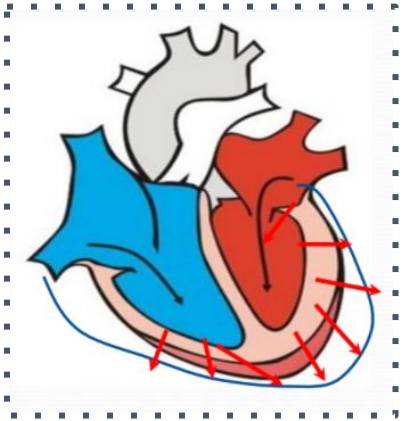
EDV has direct relationship with CO
ESV has inverse relationship with CO

CO = {EDV-ESV} X HR

Any factor that affects these parameters will affect the CO

The End-Diastolic Volume (EDV)

- EDV = is the volume of blood in the ventricles prior to ventricular ejection.



How does the EDV affect the SV and hence the CO?

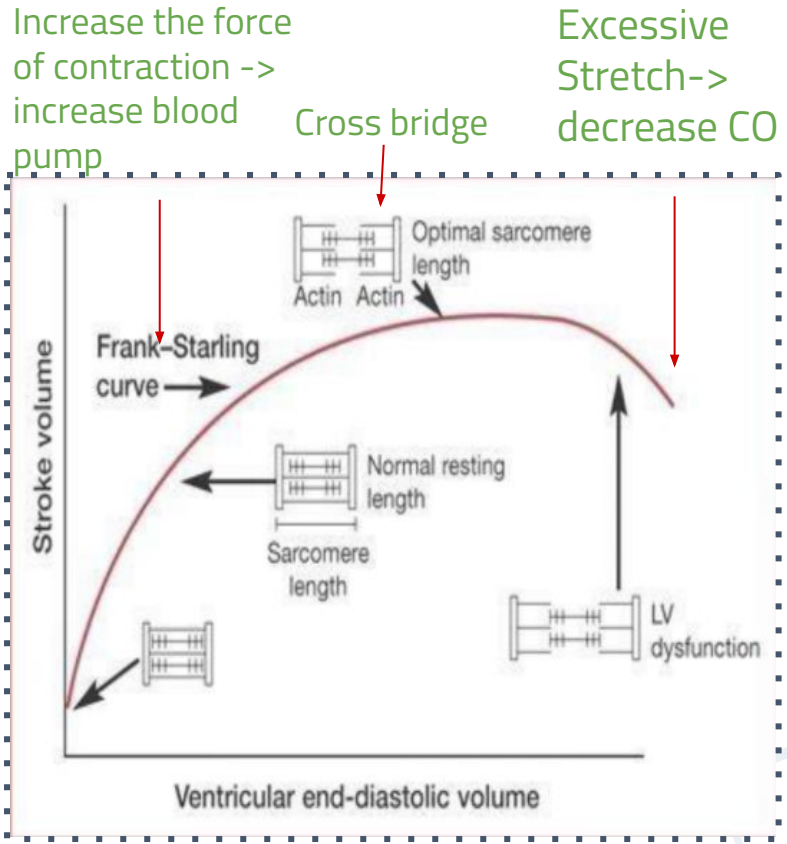
- ↑↑ EDV
- ↑↑ The stretch in the myocardium (↑↑ initial fiber length)
- ↑↑ Myocardial contractility (↑↑ Strength of contraction)

Frank-Starling mechanism (more in next slide)

Frank-Starling's Law

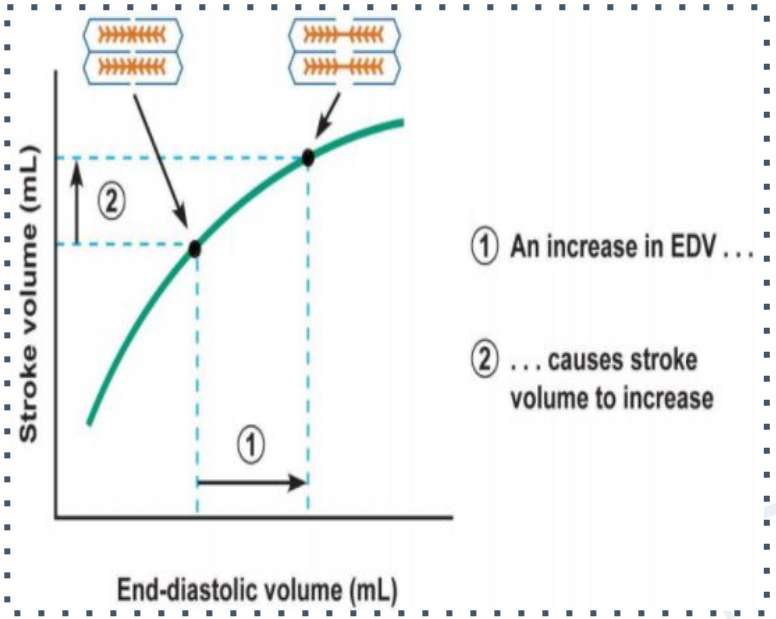
- It is the intrinsic relationship between EDV and SV.
- It reflects the ability of the heart to change its force of contraction and therefore SV in response to changes in VR.
- If EDV (preload) is increased, the ventricular fiber length is also increased, resulting in an increased 'tension' of the muscle

  (i.e SV increases in response to increase of the EDV).



Significance: Frank-Starling's Law

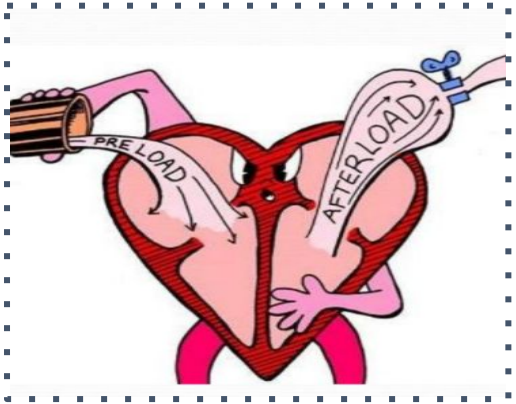
- The greater the stretch of the cardiac muscle the greater would be the force of contraction. i.e.: the energy of contraction is proportional to the initial length of the cardiac muscle fibers (the EDV).
- Why: During stretch, actin & myosin filaments are brought to more optimal degree of sliding, therefore increase force of contraction.
- Within physiologic limits, the heart pumps all the blood that returns to it by the way of the veins.



Factors affecting preload (EDV)

EDV ↑ with:

- ★ Increased total blood volume.
- ★ Increased venous return. Venocontaction
- ★ Increased venous tone. Compression from skeletal muscle
- ★ Increased skeletal muscle pump (exercise).
- ★ Increased negative intrathoracic pressure.
- ★ Stronger atrial contraction.



EDV ↓ with:

- ★ Standing. Gravity
- ★ Decreased venous return.
- ★ Increased intrapericardial pressure. Compression the heart
- ★ Decreased ventricular compliance. Electisty

Indices of left ventricular preload:
Left ventricular end-diastolic volume (LVEDV).
Left ventricular end-diastolic pressure (LVEDP).

End-Systolic Volume (ESV)

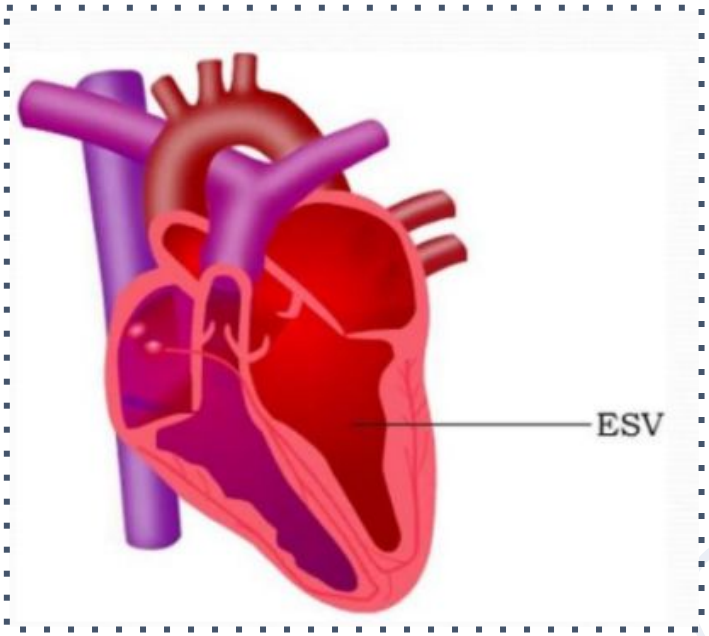
- ESV = The volume of blood remaining in the ventricle at the end of systole.

- ↑ ESV → ↓ stroke volume ↓CO

- ↓ ESV → ↑ stroke volume ↑CO

- **<u>ESV is determined by:</u>**

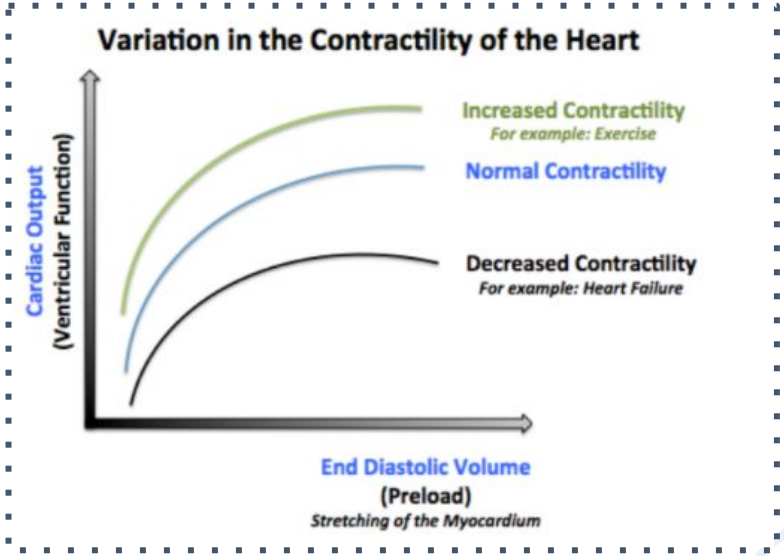
1. Cardiac contractility
2. Afterload



Determinants of ESV

1- Cardiac contractility:

- ↑↑ contractility → ↓↓ ESV → ↑↑ SV & CO
- ↓↓ contractility → ↑↑ ESV → ↓↓ SV & CO



Determinants of ESV cont..

2- Afterload:

- It is expressed as tension which must be developed in the wall of ventricles during systole,

  i.e the load the heart needs to overcome to open the semilunar valves and eject blood to aorta/pulmonary artery.

- Left ventricular afterload represents the force that the muscle must generate to eject the blood into the aorta.
- Right ventricular afterload represents the force that the muscle must generate to eject the blood into pulmonary artery.
- When the aortic pressure (afterload) increases,

  the velocity of shortening of the LV myocardial fibers ↓.

  Hence, the LV eject less blood → ↓ SV →↑ ESV.

- The opposite is true when LV afterload is reduced.

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:

1- Aortic stenosis

2- Arterial hypertension.

3- Vasoconstriction (the peripheral vascular resistance).

Causes of ↑ right ventricular afterload:

1- Pulmonary stenosis

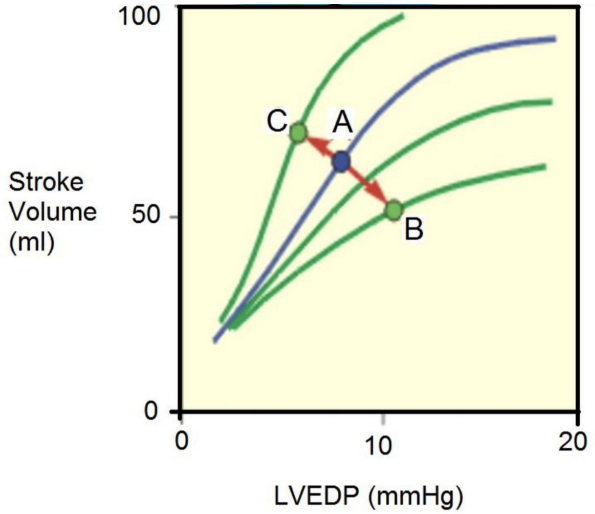
2- Pulmonary hypertension.

3- Pulmonary vasoconstriction.

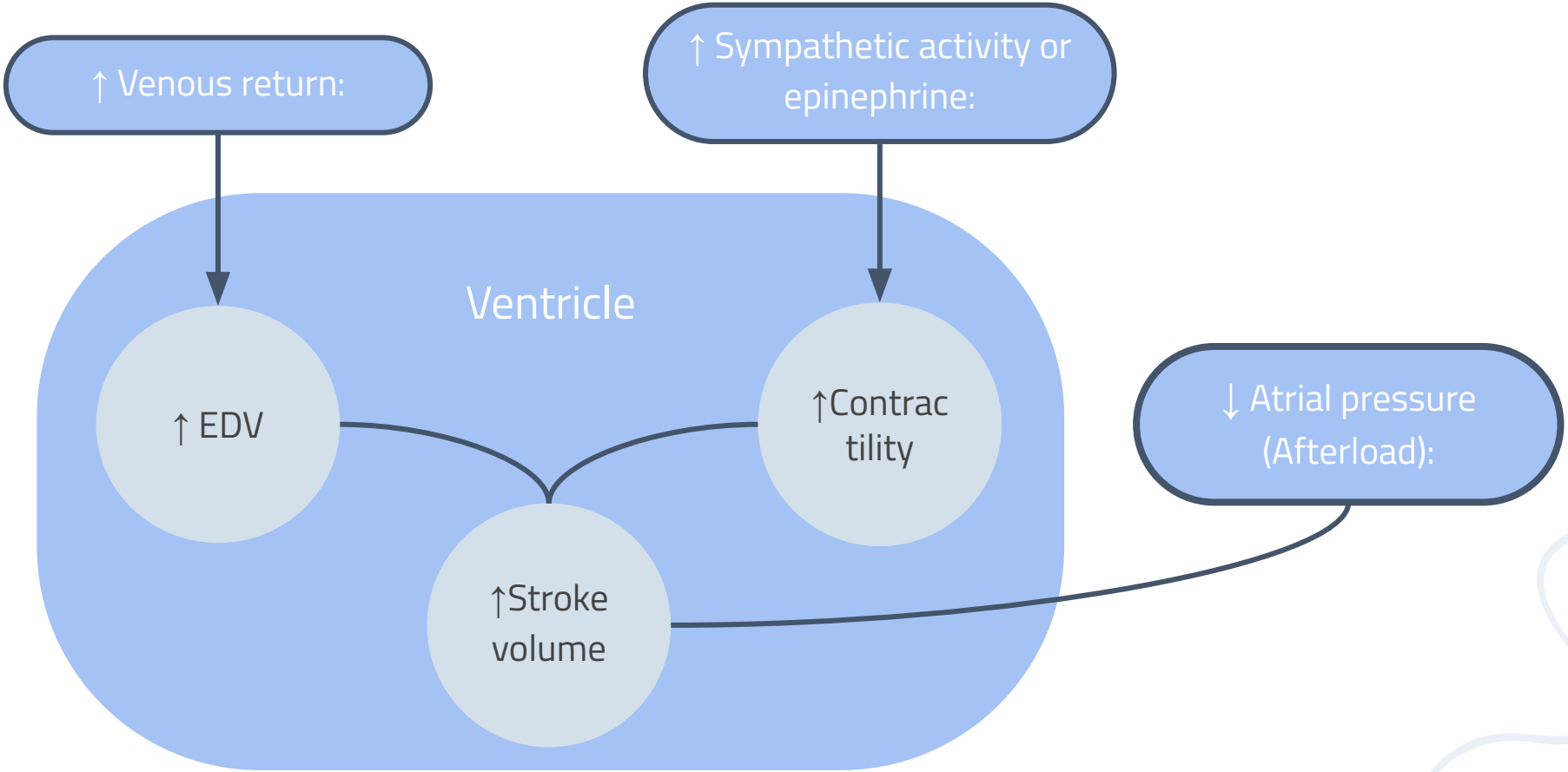
Effect of Afterload on Frank Starling Curve:

- An increase in afterload leads to ↑ ESV and ↓ SV. Thus shifts the Starling's curve down and to the right (from A to B).
- A decrease in 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.



Summary: factors influencing stroke volume



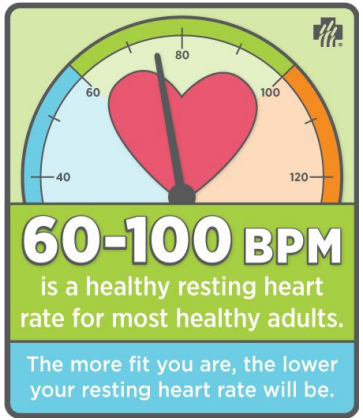
Inotropic, Chronotropic & Dromotropic:

Contractility	❖ **Positive** inotropic agent = ↑ in contractility. ❖ **Negative** inotropic agent = ↓ in contractility.
Heart rate	❖ **Positive** chronotropic agent = ↑ in HR. ❖ **Negative** chronotropic agent = ↓ in HR.
Conduction velocity	❖ **Positive** dromotropic agent = ↑ in conduction velocity. ❖ **Negative** dromotropic agent = ↓ in conduction velocity.

*Female slides only

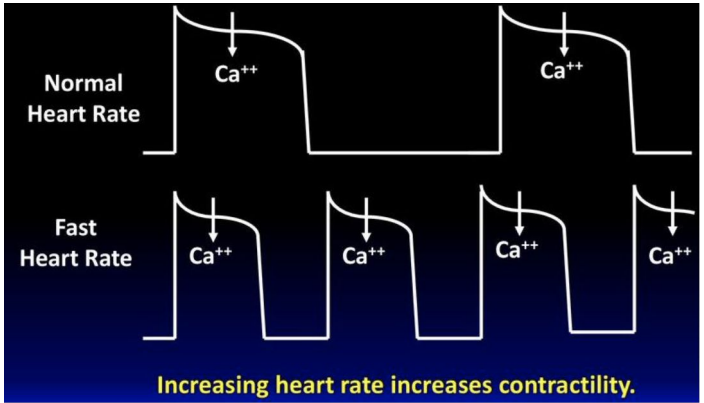
Heart rate:

- 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.
- At heart rates up to about 180, ventricular filling is adequate as long as there is enough venous return, However, at very high heart rates, filling may be compromised to such a degree that CO falls.



Frequency-force relationship:

- The heart rate has an influence on cardiac contractility as well (frequency-force relationship). **Increased heart rate increases contractility**.
- This frequency dependency of force generation is probably due to ↑ Ca2+availability to the contractile proteins within cardiac myocytes as a result of:
  - The ↑No. 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.



Determinants of the CO (HR)

- Since the CO is = SV . HR, as the heart rate increases, CO increases.
- At heart rates up to about 180, ventricular filling is adequate as long as there is enough venous return, and cardiac output per minute is increased by an increase in heart rate.
- However, at very high heart rates, filling may be compromised to such a degree that cardiac output per minute falls.
- The heart rate has an influence on cardiac contractility as well (Frequency-force relationship → due to accumulation of Ca^{2+} ions within the myocytes).

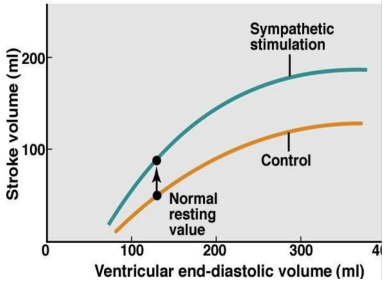
Regulation of heart rate:

1- Autonomic nervous system:

Sympathetic nervous system

Sympathetic nerves increase HR (and the contractility) through noradrenaline release (or adrenaline), which stimulates heart β1- receptors. E.g during stress, crisis or low blood pressure.

Innervate the whole heart.



Parasympathetic nervous system

Parasympathetic nerves (vagus nerve) slow HR (but has little inotropic action). Through Ach release that stimulates muscarinic (M2) receptors.

Innervate the SA and AV nodes, atria and Purkinje system, **but don't innervate most of ventricular myocardium**.

Under normal conditions the SA node is under continuous vagal tone causing its basal firing rate to decrease (Vagal Tone).

*Female slides only

Regulation of heart rate:

2- Physical factors:

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

3- Hormones & drugs:

- Epinephrine and thyroxin increase HR.
- Increased Ca2+ level in blood causes prolonged contraction.
- Reduced Ca2+ level in blood depress the heart.

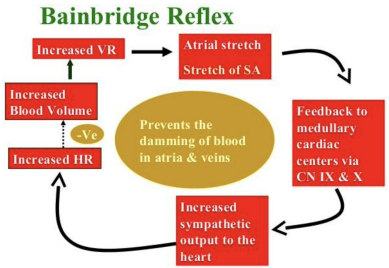
*Female slides only

Regulation of heart rate:

4- Blood volume:

Atrial Reflex (Bainbridge reflex)

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



Factors affecting the HR (Chronotropic)

+ve chronotropic factors	-ve chronotropic factors
- Sympathetic stimulation. -Increased temperature. -Exercise. -Hyperthyroidism.	-Vagal stimulation. -Decreased temperature. -Heart block.

Pathological low or high CO:

Causes of **low** cardiac output:

- Low VR e.g hemorrhage.
- Reduced contractility e.g. heart failure.
- Tachyarrhythmias e.g. atrial fibrillation and ventricular Tachycardia.
- Marked bradycardia e.g., complete heart block.

Causes of **high** cardiac output:

- Hyperthyroidism: the increase in the CO is due to the high metabolic rate → vasodilatation →↑ CO to 50% of control.
- AV fistulas.
- Fever.
- Anaemia.
- Anxiety.

Physiological changes in CO

Males Slides

- ★ During the first 3 hours after meals, the CO is increased by ≈ 30% to enhance blood flow in the intestinal circulation.
- ★ Later months of pregnancy are accompanied by > 30% increase in CO due to increased uterine blood flow.
- ★ At environmental temperature above 30 °C, the CO is increased due to increased skin blood flow. Also at low environmental temperature CO is increased due to shivering that increases blood flow to the muscles.
- ★ Increased sympathetic activity during anxiety and excitement enhances the CO up to 50% - 100%.
- ★ Sitting or standing from the lying position deceases the CO by 20% - 30%.

Effects of exercise on heart rate and SV

Moderate Exercise

HR increases to	200% of resting (140 bts/min)
SV increases to	120% (85ml)
CO increases to	240% (12L)

Severe Exercise

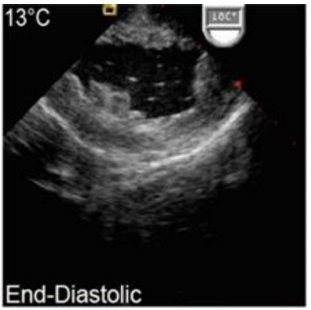
HR increases to	300% of resting (200 bts/min)
SV increases to	175% (125ml)
CO increases to	500% - 700% (25 - 35 L)

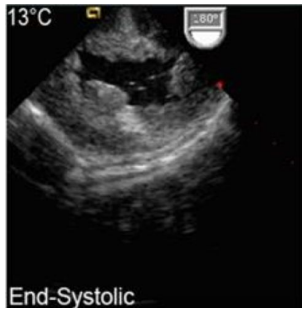
In athletes, maximum CO may be 35L or more - can't increase maximum HR beyond 200 bts - hence - SV increases to 175 ml.

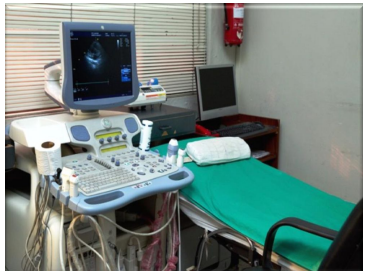
Measurement of cardiac output:

1- Two dimensional Echocardiography:

- Records real-time changes in ventricular dimensions during systole and diastole.
- It thus computes the SV, which when multiplied by HR, the CO will be calculated.

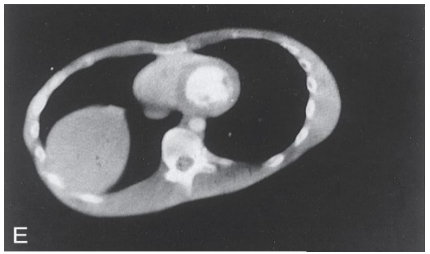


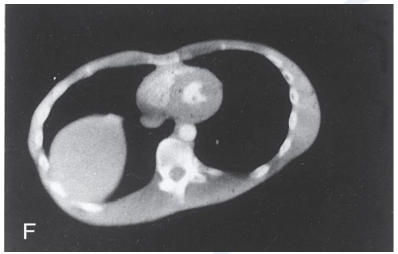




2- Ultra fast computer tomography:

- Can measure changes in ventricular diameter at several depths to estimate changes in ventricular volume.

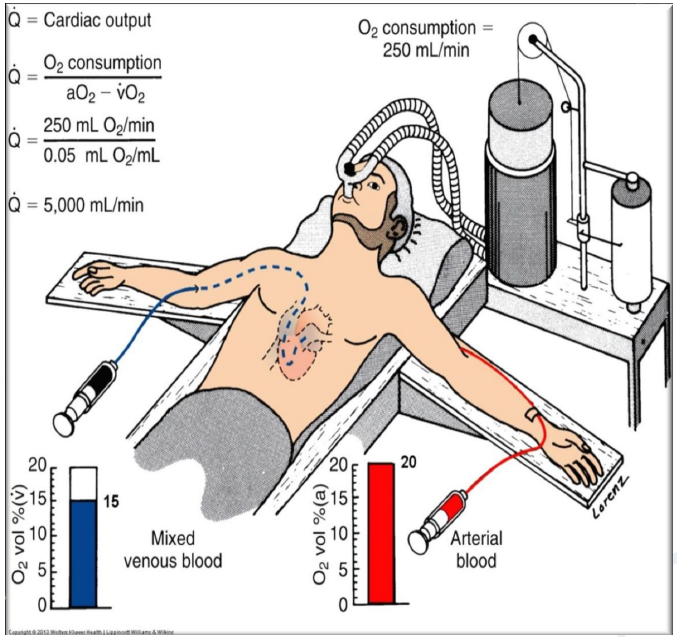




Measurement of cardiac output:

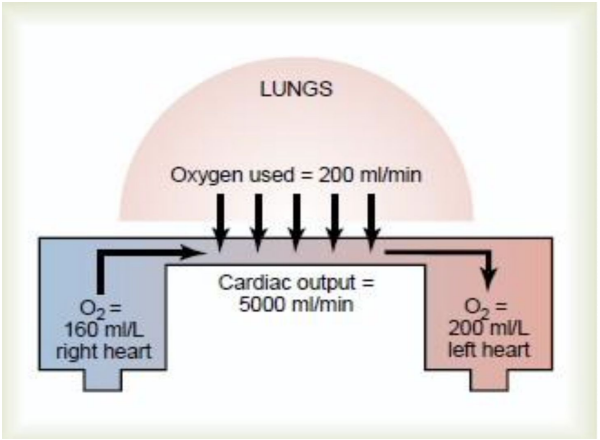
3- Fick's principle:

It assumes that the amount of oxygen consumed = the amount of oxygen delivered by the arterial blood **minus** the amount of oxygen in venous blood of the organ.



$$CO\ (L/Min) = \frac{\text{Total } O_2 \text{ consumption}}{AO_2 - VO_2}$$

AO_2 = Arterial O_2 concentration.
VO_2 = Mixed O_2 venous concentration.



Team Leaders

QBank Team

Rand aldajani

Nawaf Alshehri

Sub Leader

Samiah AlQutub

Team Members

Munira Alsharif

Mansour Aldhalaan

Special thanks to Arwa Almobeirek for designing the theme!