MEDICINE
KING SAUD UNIVERSITY

Physiology Team 436

Revised by
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CVS BLOCK

Cardiac Output

Red: very important.
Green: Doctor's notes.
Pink: formulas.
Yellow: numbers.
Gray: notes and explanation.

Physiology Team 436 – Cardiovascular Block Lecture 8

Lecture: If work is intended for initial studying.
Review: If work is intended for revision.

Objectives

Study Smart: focus on mutual topics.

From the students' guide:

- By the end of this lecture the students are expected to:
- Define cardiac output, stroke volume, end-diastolic and end-systolic volumes.
- Define physiological conditions affecting CO
- List causes of high and low output pathological states.
- Define venous return and describe factors controlling venous return.

The Cardiac Output (C.O)

Video of (Cardiac Physiology of CO) Duration: (8:48 mins)

Video of (Cardiac Output) Duration: (9;39mins)

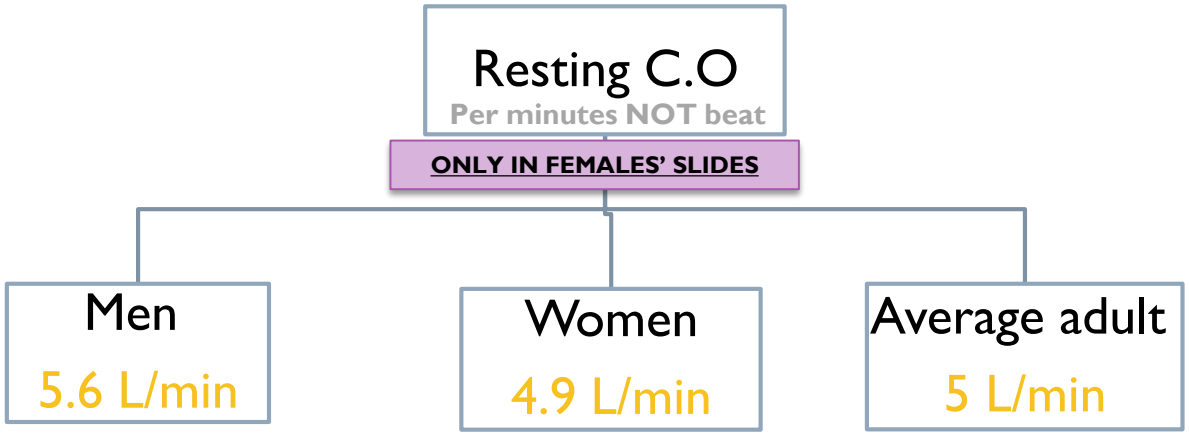
- **C.O.:** is the volume of blood flow ejected from the right or left ventricle per minute. = 5 L\min at rest.
- **Stroke volume:** is the volume of blood ejected from each ventricle per beat = 70 ml\beat at rest
- **Heart Rate =** 72 beats\ min.

Thus, the cardiac output = the volume of blood pumped by one ventricle per beat X the number of beats per minute:

C.O (Q) = Heart rate X stroke volume

- Cardiac output: volume of blood out of heart per minute
- Stroke volume: volume of blood out of heart per beat

- **C.O regulation:** is well regulated according to tissue metabolic demands.
- **Basic determinant of C.O.:** is the O_2 requirements of body tissues, for their metabolic rates.
- **Accordingly,** if the metabolic rate is increased → the CO and VR are increased WHY? to maintain optimal O_2 supply to the active tissues.



Cardiac Reserve: VR and CO

- The cardiac output at rest is approximately 5 L/min.
- The body's blood volume averages 5 to 5.5 liters.
- **Thus,** each ventricle pumps the equivalent of the entire blood volume each minute.
- During exercise, the CO can **increase** to 20 - 25 liters/min and to as high as 35 - 40 liters/min in well trained athletes.
- **The difference** between the resting CO and the maximum volume of blood: the heart is capable of pumping per minute is known as the **cardiac reserve**.

- **Venous return** (VR): refers to the volume of blood entering each atrium per minute.

VR = Heart rate X Diastolic filling volume = 5 L/min

- **Venous return:** flow rate into the heart
- **Cardiac output:** flow rate out of the heart (heart rate)

Coupling of cardiac and vascular functions

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.

ONLY IN MALES' SLIDES

Regulation of the Cardiac Pump

- **The hemodynamic function of the human heart has two components:**
  1. To be filled by the venous return (VR)
  2. To produce an outflow (i.e., cardiac output) with pressure.

- **2 groups of factors that regulate the haemodynamic functions of the heart:**
  1. Factors affecting the pump ability to eject (i.e., CO)
  2. Factors affecting the pump filling (i.e., VR)

- **These factors include:**
  1. Intrinsic properties of the heart and blood vessels → which provide an autoregulation to the heart pump.

(زي كهربائية القلب او تضيق الشرايين والاوردة)

  1. Neuro-hormonal factors outside the CVS → which constitute an extrinsic regulation.

Factors affecting & regulating cardiac output

Video of (Regulation of CO)
Duration: (14:33 mins)

- Heart rate
  - Increase HR
    - Hormones
      - Epinephrin (neurotransmitter)
      - Thyroxine (thyroid hormone which increases HR)
    - pregnancy
    - Sympathetic Nervous system*
      - crisis (e.g. Stress , anxiety , fear .. etc.).
      - High BP
    - Decrease blood volume
      - E.g. Trauma i.e. hemorrhage
    - Exercise**
      - muscle need more O2 → heart need to pump more blood.
    - Bainbridge reflex = Atrial reflex
      - In the right atrium there is Stretch receptor, which response to venous return (increase in pressure) by increasing the heart rate through sympathetic activity.
    - Frequency-force relationship
      - Increase cardiac contractility i.e. hyperthyroidism
    - Increase temperature
  - Decrease HR
- Stroke volume

*Increase contractility (strength of contraction) and BP and SV (volume of blood) and therefore increase CO

**Increases HR then increases CO because muscles need more O2 supply and that is done by increasing HR to pump more blood to the muscles to supply them and remove lactic acid

Physiological/Pathological Changes In Cardiac Output

Physiological changes

- **During the first 3 hours after meal:** 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.
- **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%.
- **Exercise:** (next slide)

Pathological changes

Causes of low CO:

- Low VR (e.g., haemorrhage)
- Reduced contractility (e.g., heart failure)
- Tachyarrhythmias (e.g., atrial fibrillation and ventricular tachycardia)
- Marked bradycardia (e.g., complete heart block)

Causes of high CO:

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

Hyperthyroidsm → more metabolism → more oxygen

Physiological Changes In Cardiac Output

➢ **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.

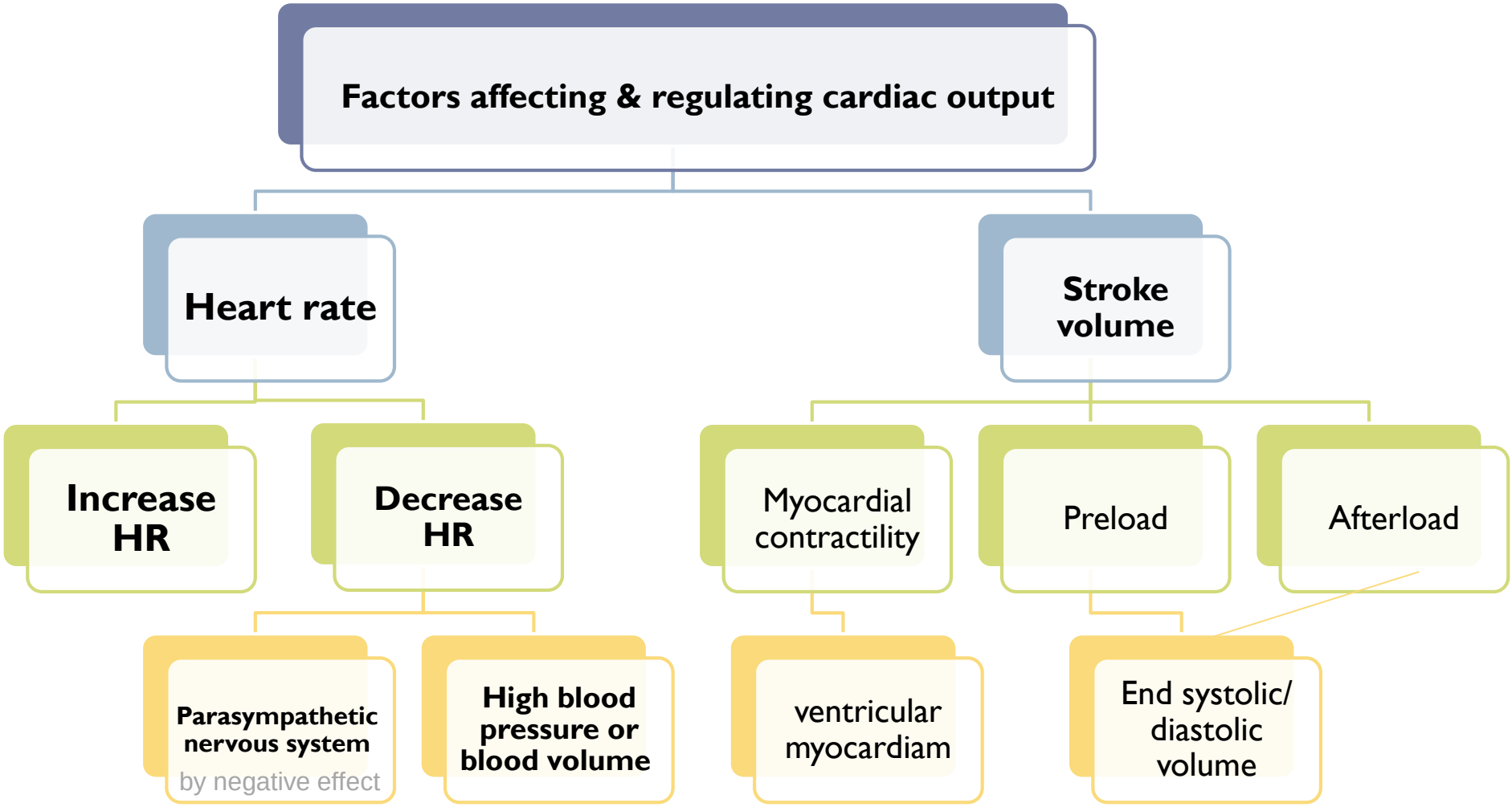
In severe exercise:

- CO is increased by 5-7 times

(due to increase in HR & SV)

Athletes at rest:

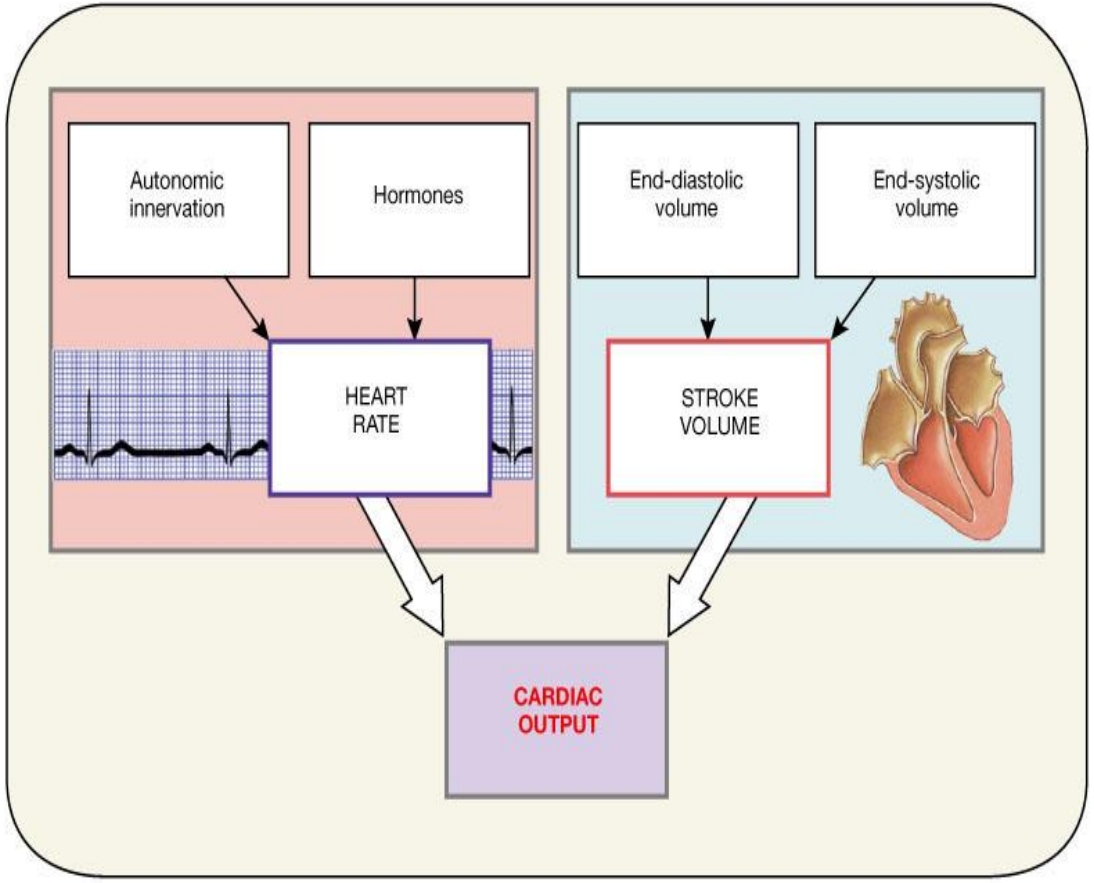
- HR less
- CO is the same
- SV more

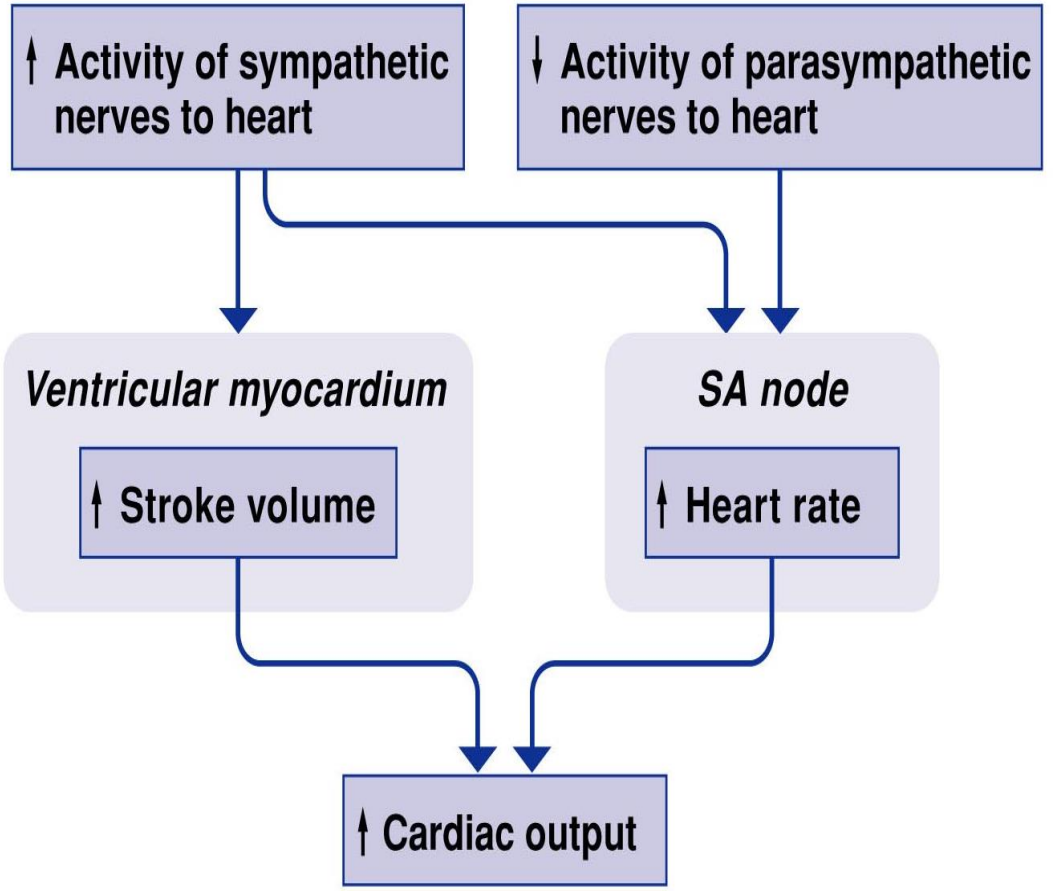


The Bainbridge reflex, is an increase in heart rate due to an increase in central venous pressure. Increased blood volume is detected by stretch receptors (baroreceptors) located in both atria at the venoatrial junctions.

- The stretch receptors are sensitive to venous return.
- volume of blood entering the right atrium will cause stretching in the receptors.
- when the receptors are stretched, they will send signals to the brain which will increase the heart rate.

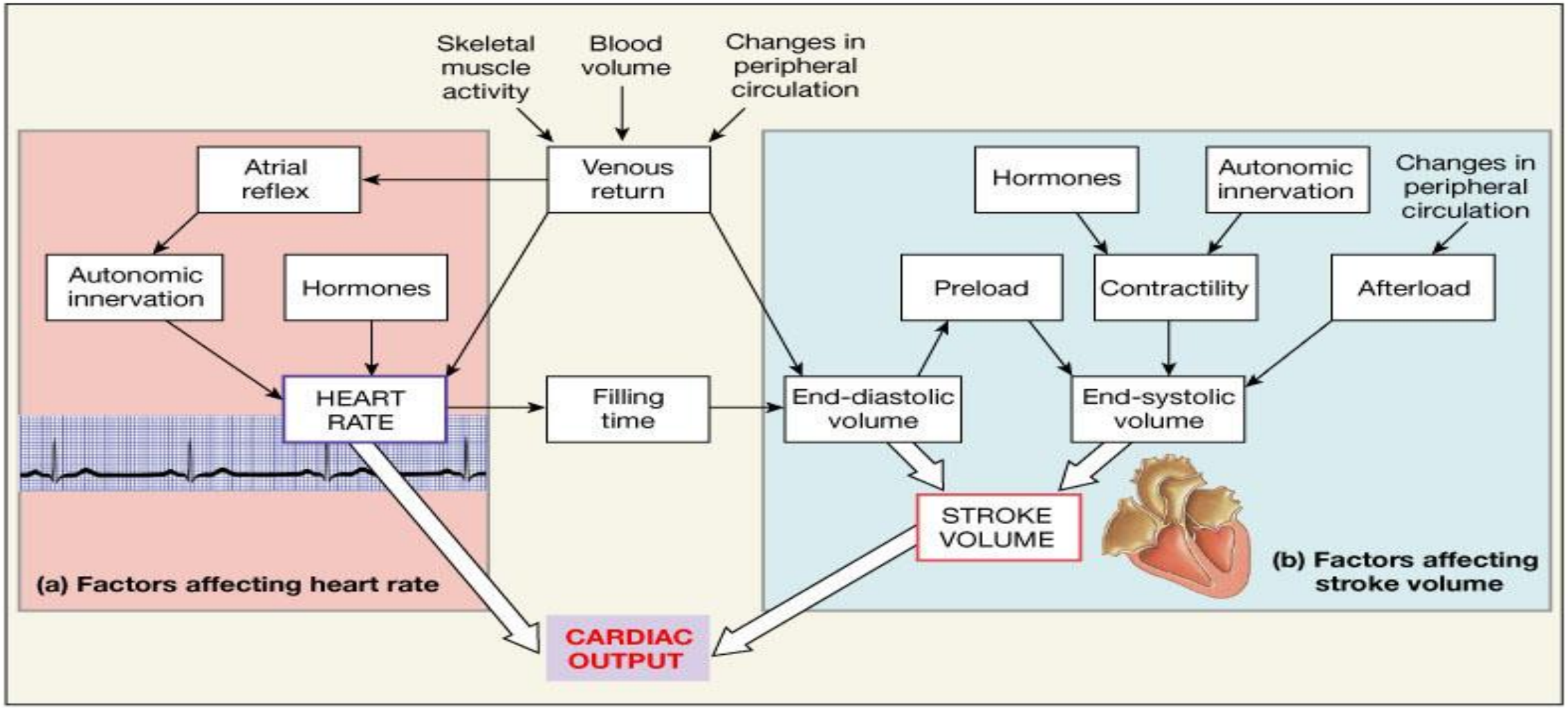
Regulation of CO





ONLY IN FEMALES' SLIDES

Summary



- Read do not skip!

Factors Affecting Cardiac Output

Heart Rate

•Since the **CO is = SV x HR**, as the heart rate increases → CO increases (logically).

• At heart rates up to about 180 Beats/min →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. أللي يبغاك تعرفه انه طالما الوريد يرجع دم كافي لما تكون نبضات القلب عالية من الاذين للبطين ما يكون في مشكلة

• However, at very high heart rates, filling may be compromised to such a degree that cardiac output per minute falls. لما تكون نبضات القلب عالية جدا يكون الشخص معرض انه قلبه ما يطلع كمية كافية

Nerve Supply to the Heart

Sympathetic nerves

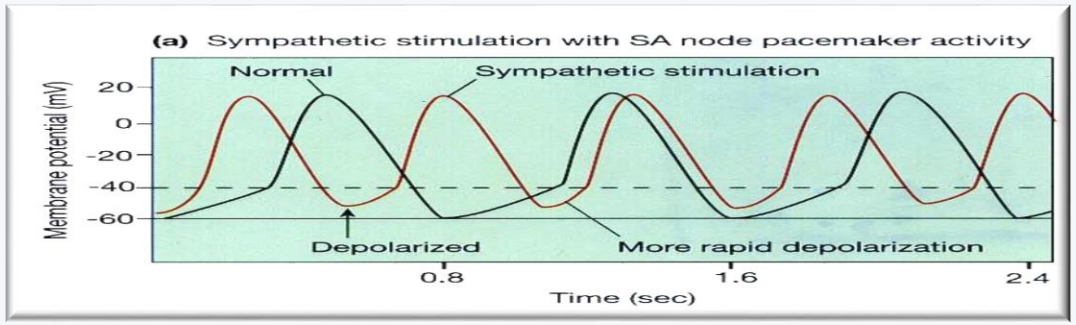
- Innervate the whole heart.
- Release noradrenaline (adrenaline), which stimulates heart β_1-receptors → increase HR

Parasympathetic nerves

- Innervate the SA and AV nodes, and the atria and Purkinje system.
- Do not innervate most of the ventricular myocardium.
- Release ACh that stimulates muscarinic (M_2) receptors → reduce HR
- Slows the heart but has little inotropic action.

CO=Cardiac Output SV=Stroke Volume Atrium = اذين Ventricle = بطين

1- Mechanisms of Neural Control



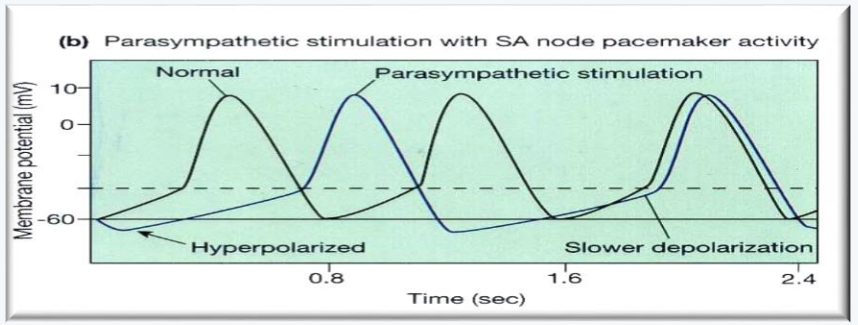
Mechanisms:

The β_1-adrenoreceptors on SA node cells are coupled to excitatory G-proteins →This activates the enzyme adenylate cycles → increases cAMP as a second messenger inside the cell

This results in:

- Opening of Na^+ and Ca^{2+} channels → speed up the rate of depolarization and hence the heart rate.
- Decrease in K^+ permeability by accelerating inactivation of the K^+ channels. Thus, fewer positive potassium ions leave the cell → the inside of the cell becomes less negative → depolarizing effect.

The net effect is: → swifter drift to threshold → greater frequency of action potential → increase the heart rate.



Mechanisms:

M_2 (muscarinic-2) ACh receptors respond to ACh from the vagus nerve by activating a different G-protein.

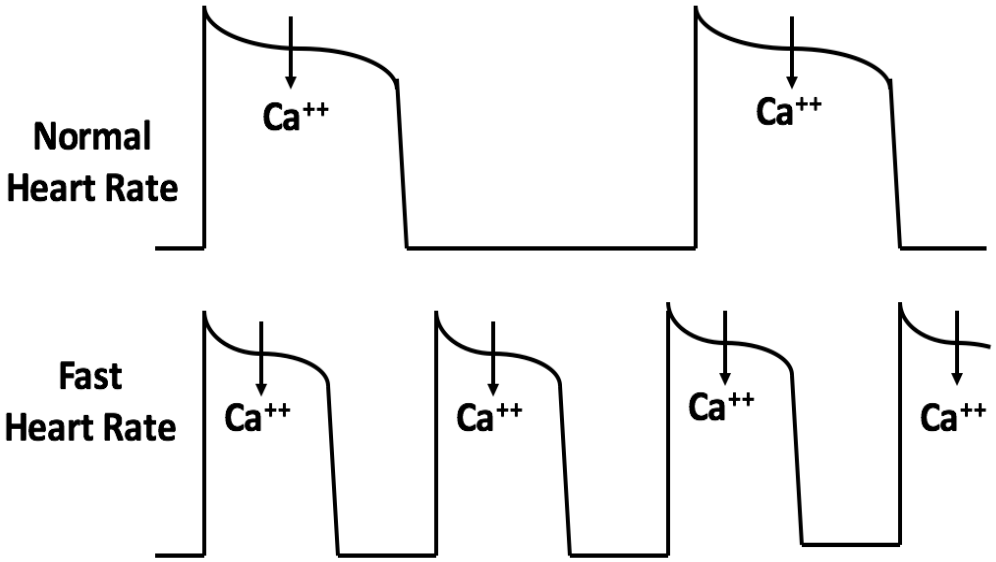
This results in:

- Reduces the levels of cAMP in the cell
- Closure of Na^+ and Ca^{2+} channels
- Opening of potassium channels in the cell membrane
- Hyperpolarizes the cell and makes it more difficult to initiate an action potential.

The net effect is: → slower drift to threshold → lesser frequency of action potential → decrease the heart rate.

2- Frequency Force Relationship

- The force of contraction of the cardiac muscle is increased when it is more frequently stimulated.
- This frequency dependency of force generation in the heart is probably due to accumulation of Ca2+ ions within the myocytes as a result of:
  - The increased in number of depolarizations/min → more frequent plateau phases → more Ca^{2+} entry.
  - The magnitude of Ca2+ current is also increased → increases the intracellular Ca^{2+} stores.
- Both effects enhance the release and uptake of $Ca2^{+}$ by the sarcoplasmic reticulum, thus Ca^{2+} availability to the contractile proteins with more force generation through cross-bridge cycling.
- So, Increase HR due to increase Contractility



كل ما زاد تحفيز عضلة القلب زادت قوة الانقباض
هذا الشيء بسبب احتمالين :1- عندك ديبولارايزيشن بالدقيقة كثير بالتالي يدخل زيادة كالسيوم لان فيز ٢ أكثر
2- الماقنيتيود حق الكالسيوم ازداد مما ادى الى زياة في مخازن الكالسيوم
هذولي الاحتمالين يزيدو كمية ارجاع الكالسيوم وزيادته في الساركوبلازمك ريتكيلم يؤدي الى انقباض اقوى

Cardiac Index and Cardiac Output

Cardiac index: is a ratio or relation

Cardiac Index

It relates the **cardiac output** to body **surface area**.

Thus relating **heart performance** to the **size** of the individual.

The unit of measurement is liter per minute per square meter of body surface area **(L/min/m^2).**

ONLY IN FEMALES' SLIDES

Cardiac Output Measurement

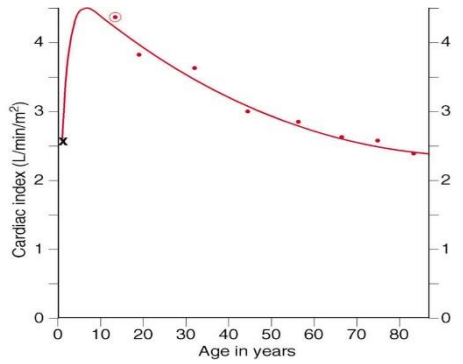
The Direct Fick's Method:

The amount or **volume of any substance** taken up by an organ or by the whole body is equal to:
(The arterial level of the substance — the venous level) X blood flow.

$$\text{Blood flow} = \frac{\text{Amount}}{\text{(Arterial level} - \text{Venous level)}}$$

(Measuring the concentration of any substance in the arteries and veins)

Cardiac index for the human being at different ages:



Methods for Measuring Cardiac Output

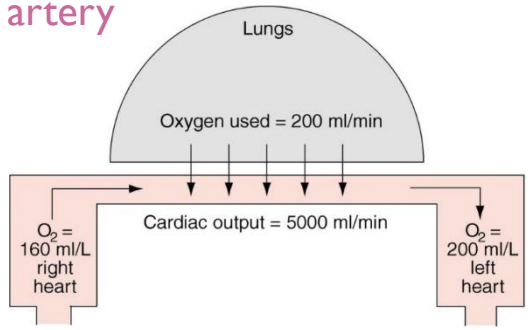
Girls' Doctor said that the scientist and the equation are important but the rest is not so just read it.

Cardiac output can be *measured* using the **Fick principle:**

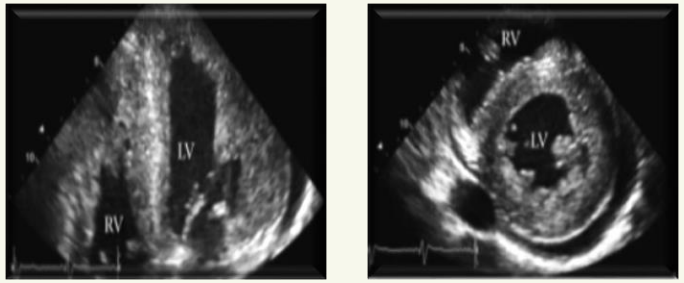
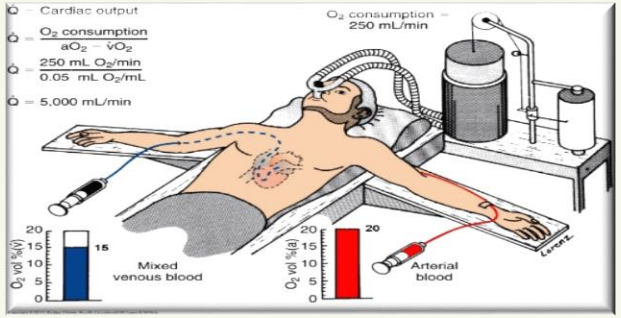
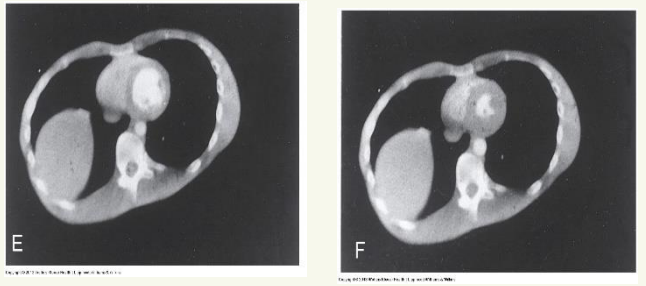
- In the steady state, the cardiac output of the left and right ventricles is equal.

- In the steady state, the rate of O2 consumption by the body must equal the amount of O2 leaving the lungs in the pulmonary vein minus the amount of O2 returning to the lungs in the pulmonary artery.

- Total O2 consumption or the rate of O2 absorption by the lungs can be measured by the rate of disappearance of oxygen from respired air, using any oxygen meter.
- The amount of O2 in the pulmonary veins is pulmonary blood flow multiplied by the O2 content of pulmonary venous blood. Likewise, the amount of O2 returned to the lungs via the pulmonary artery is pulmonary blood flow multiplied by the O2 content of pulmonary arterial blood.
- O2 consumption = cardiac output × [O2] pulmonary vein − cardiac output × [O2] pulmonary artery
- Cardiac output = O2 absorbed by the lungs per minute/arteriovenous O2 difference



Measurement of Cardiac Output

2-Dimensional echocardiography	Fick's principle	Ultra-fast computer tomography
		
• 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	Fick's principle assumes that the amount of oxygen consumed by an organ = the amount of oxygen delivered to that organ by the arterial blood minus the amount of oxygen left in the venous blood of the organ. O2 consumed = O2 delivered-O2 in venous	Can measure changes in ventricular diameter at several depths to estimate changes in ventricular volume.

- Amount of oxygen delivered by the arterial blood = aO_2 . Blood flow through the organ (Q).
- Amount of oxygen left in the venous blood = vO_2 . Q.
- The amount of oxygen consumed by the organ = (aO_2 . Q) - (vO_2 . Q) = Q (aO_2 - vO_2).

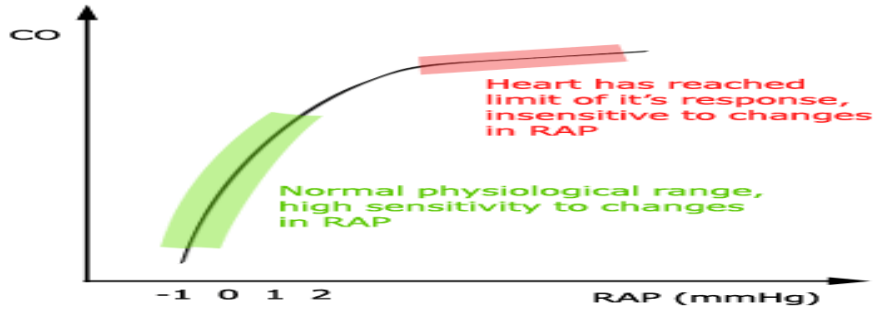
❑ Thus $Q = \frac{\text{The amount of oxygen consumed}}{aO_2 - vO_2}$

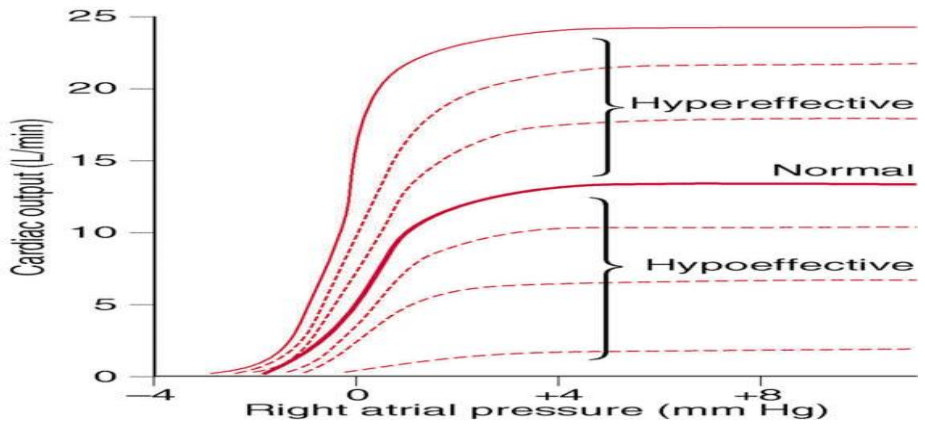
The Heart has Limits for Achievable Cardiac Output (Girl's doctor said just read it)

- **Factors that can cause hypereffective heart:**
  1. Nervous stimulation
  2. Hypertrophy of the heart muscle

- Sympathetic stimulation and parasympathetic inhibition can increase the effectiveness of the heart **via:**
  1. Increasing the heart rate
  2. Increasing the strength of heart contraction

- Increased pumping effectiveness caused by heart hypertrophy 50-75%→ increase in the heart mass of marathon runners→ which increases the plateau of cardiac output 60-100%.





This figure shows the normal cardiac output curve at each level of right atrial pressure.The plateau level of this normal cardiac output is 13 L/min. This means that the heart can pump an amount of venous return up to 2.5 times the normal venous return before the heart becomes a limiting factor in the control of cardiac output.

Quiz

- https://www.onlineexambuilder.com/stroke-volume/exam-140345

Link to Editing File

(Please be sure to check this file frequently for any edits or updates on all of our lectures.)

References:

- Girls' and boys' slides.
- Guyton and Hall Textbook of Medical Physiology (Thirteenth Edition.)

Thank you!

اعمل لترسم بسمة، اعمل لتمسح دمعة، اعمل و أنت تعلم أن الله لا يضيع أجر من أحسن عملا.

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