5- Heart Rate & Its Regulation

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2024-2025

DEFINITION

- It is the number of heart beats per minute.

ITS VALUE

Depends on the intensity of the **vagal tone** on the heart, So HR changes according to:

-Age: in newborn **90-120**/min (no vagal tone)

in adult **65-90** (75/min)

-Sex: in females the HR > in male (weak vagal tone)

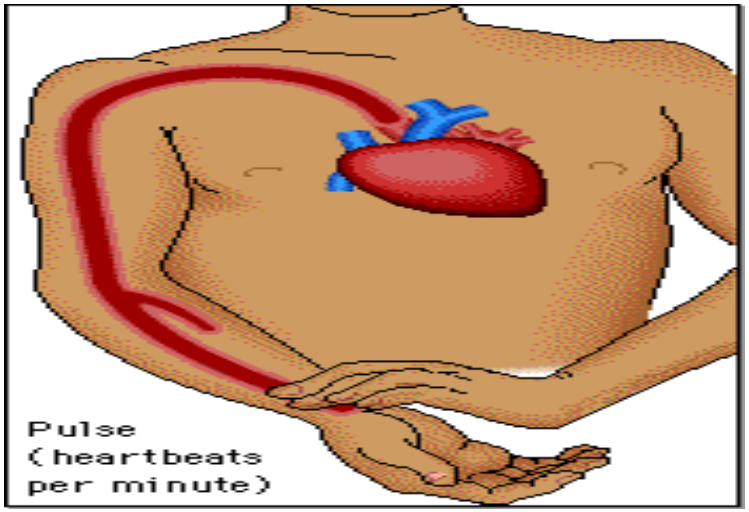
-Muscular exercise: emotion & pregnancy & after meals →↑HR

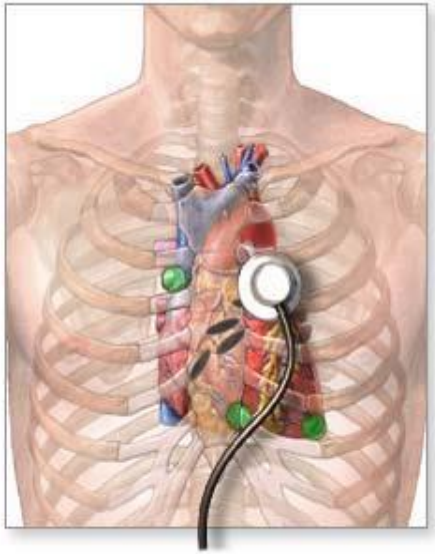
-Athletes: during rest have low HR (↑↑ vagal tone)

-Sleep: strong vagal tone → ↓HR.

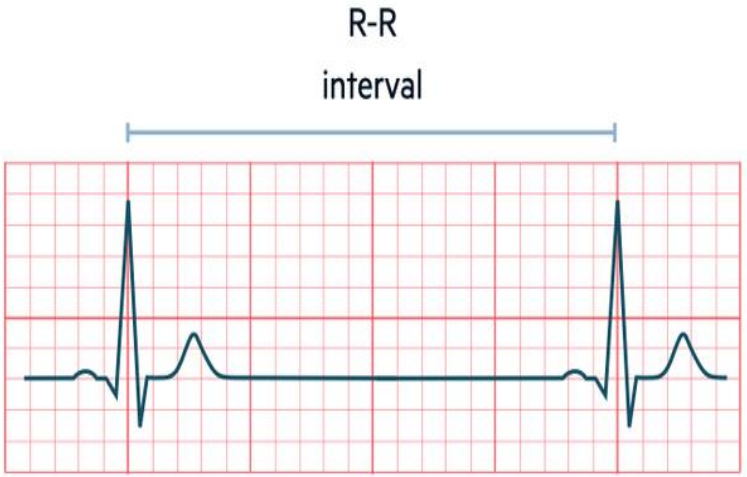
MEASUREMENT:

- From radial artery pulsation counting.
- Heart sounds listening.
- From ECG= 60/time between 2R waves





Auscultation is a method used to listen to sounds of the body during a physical examination



Rate = 300 / (No. of large squares between R waves)

REGULATION OF HEART RATE

- The frequency of discharge of the S-A node (which determine the heart rate is regulated by nervous and chemical factors well as by certain other factors that directly affect the S-A node activity.

1. **Nervous regulation.**

2. **Chemical regulation.**

I- Nervous Regulation Of The Heart Rate

- The heart rate is nervously regulated through the **cardiovascular centers**

- Which control the sympathetic and para sympathetic discharge to the heart.

Medullary Cardiovascular Centers

- **[A] The pressor area (vasomotor center - VMC)**

• Site: located in the ventrolateral part of medulla.

• Its neurons projects downward and synapse with the sympathetic pre ganglionic neurons of the lateral horn cells of spinal cord.

• It is composed of

(l) Vasoconstrictor center (VCC)

During rest, it discharges continuously at moderate rate to the blood vessels via the sympathetic fibers, this is called **(vasoconstrictor tone)**

Its stimulation produces:

(a) Vasoconstriction (VC) of the arterioles which increase the peripheral resistance (PR). So, increases the arterial blood pressure (ABP).
(b) Veno-constriction that leads to increase the venous return (VR), so that the cardiac output and arterial blood pressure are elevated .

(2) Cardiac stimulatory center (CSC)

- It discharges during rest but at low tone via the sympathetic nerve fibers to the heart this is called the sympathetic tone.

- its stimulation produces. ● Increase the heart rate. ● Increase the force of contraction which increase the stroke volume. ● Increased HR and SV lead to increase COP and ABP.

Medullary Cardiovascular Centers

- **[B] The depressor area**
- Site: It is located central and dorsal to the pressor area.
- It is composed of two centers.

(1) Vasodilator center (VDC)

It sends inhibitory impulses to inhibit vasoconstrictor center (VCC).

(2) Cardiac inhibitory center (CIC)

It inhibit the heart along the vagus nerve (vagal tone).

Functions:

1. It decreases the heart rate.

2. Stimulation of the **depressor area** leads to decrease of arterial blood pressure due to:

(a) Decrease the peripheral resistance as a result of vasodilatation.

(b) Decrease in the cardiac output as a result of decreased heart rate.

- **N.B.**

- There is a reciprocal innervation between the **pressor area** and the **depressor area**.
- Impulses which stimulate **the pressor area**, also stimulate **the medullary respiratory area** and vice versa.
- The dominant centers are: Vasoconstrictor center **(VCC)&** Cardio-inhibitory center **(CIC).**

- **The activity of these centers is affected by**

(A) Impulses discharged from certain supraspinal centers

(B) Many reflexes initiated from the CVS itself as well as from several extravascular structures

Supraspinal Centers That Affect the Heart Rate

(1) The cerebral cortex

The cortical influence on the heart rate is evident by alteration of the heart rate as a response in many **Conditioned reflexes**

(2) The hypothalamus and limbic system

These structures are concerned with emotional reactions. Most emotions are associated with tachycardia (e.g. before starting a race). However, severe emotions are frequently associated with bradycardia

(3) The respiratory center

Respiratory sinus arrhythmia

This term refers to the increase of the heart rate during inspiration and its decrease during expiration that commonly occurs normally in young individuals and children. This phenomenon is primarily due to **fluctuations in the strength of the vagal tone to the heart** during the respiratory cycle, which occur as follows:

During inspiration.

a) Inspiratory center sends impulses to stimulate the VMC.

b) Increased intrathoracic negativity —— increased VR — increased RAP — increased HR (**Bainbridge effect or Bainbridge reflex**).

c) Stretch receptors in alveoli — inhibition of the (**CIC**)

During expiration.

These impulses are no longer discharged resulting in decrease of the heart rate.

(B) Reflexes Initiated From The CVS

- **(1) Marey's reflex (Marey's law)**

A rise of the arterial blood pressure (ABP) leads to a decrease of the heart rate and vice versa, provided other factors affecting the heart rate remain constant".

It is initiated by stimulation of the **arterial baroreceptors**

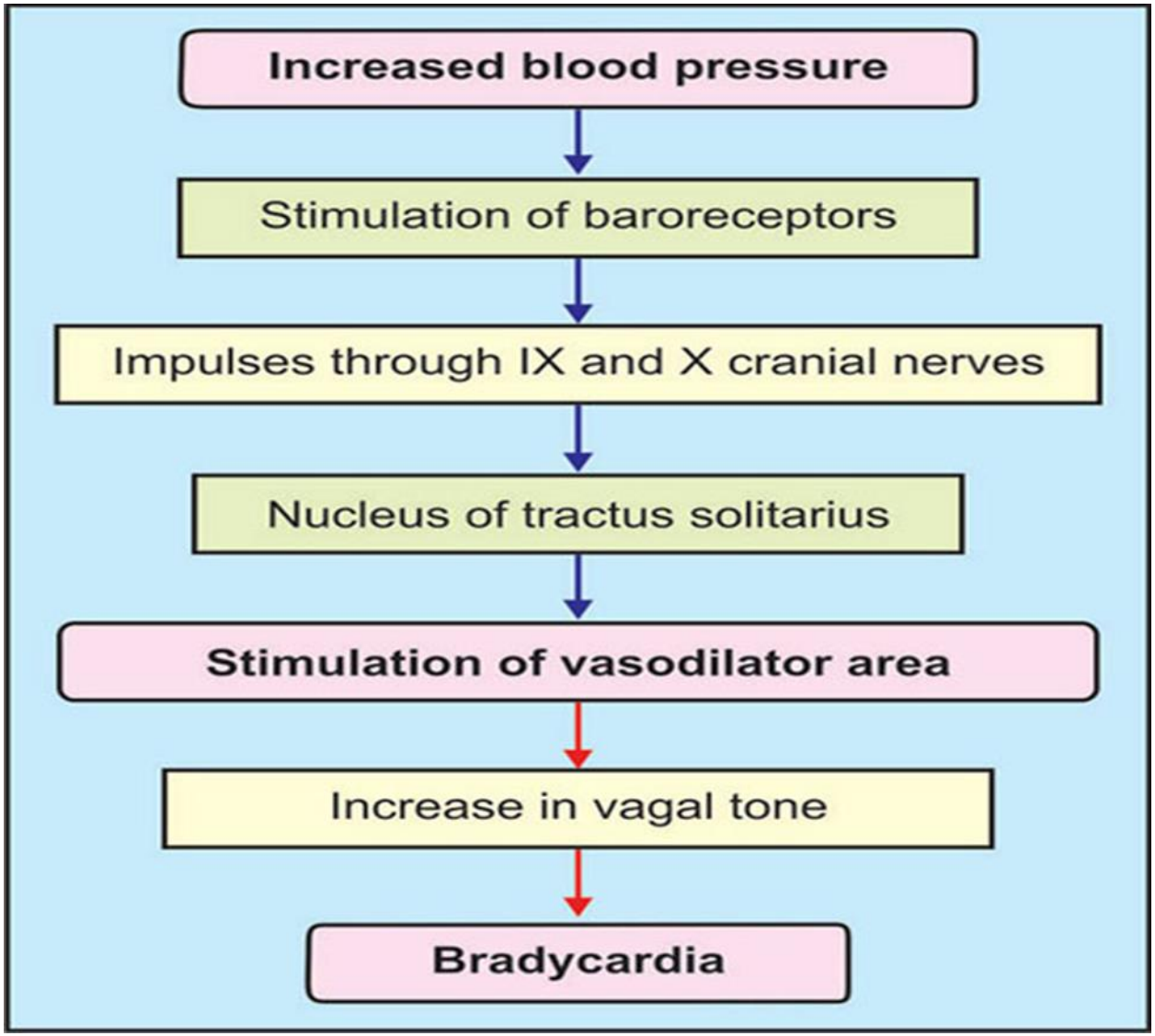
Effects of stimulation of arterial baroreceptors:

(1) Stimulation of both the CIC (resulting in reflex bradycardia) as well as the VDC (resulting in generalized V. D. and hypotension).

(2) Inhibition of' the respiratory center (resulting in temporary apnea).

(3) Inhibition of secretion of the antidiuretic hormone (ADH).

Marey's reflex (Marey's law)



- **(2) Bainbridge reflex**

An increase in the right atrial pressure leads to **heart acceleration**. Impulses are discharged from special tachycardia-producing **type A atrial baroreceptors** via afferent vagal nerve fibers to the medulla where they **stimulate** the **CSC** leading to heart acceleration.

N.B. some authors believe that the resulting increase in the heart rate is due to direct stimulation of the S-A node as a result of stretch and such effect is sometimes called the **"Bainbridge effect"**

(3) Reflexes From The Left Ventricle

(A) **Distention of the left ventricle** leads to bradycardia, temporary apnea. V.D. and hypotension. Such response is initiated by **stimulation of baroreceptors** in the ventricular wall.

- **(B) The Bezold- Jarisch reflex (coronary chemo reflex)**

  Injection of certain substances (e.g. serotonin) into the coronary arteries that supply the left ventricle or Chemicals released from myocardial infarction leads to **bradycardia, V.D. , hypotension & apnea** through stimulation of certain **ventricular chemo-receptors**.

(C) Reflexes Initiated From Extra-vascular Structures

- **(1) From The Lungs**

(A) Stimulation of the **baroreceptors** in the bronchial wall leads to temporary apnea, vasodilatation & hypotension.

But is associated with **tachycardia** due to inhibition of the (**CIC**) i.e. decreased vagal tone

(B) The pulmonary chemo-reflex

- Pulmonary Infarction or injection of certain substances (e.g. serotonin) into the pulmonary vessels leads to bradycardia, V. D. and hypotension through stimulation of certain **pulmonary chemoreceptor**. It is similar to the coronary chemo reflex .

(2) From Skeletal Muscles (Alam-Smirk Reflex)

Voluntary contraction of skeletal muscles leads to an increase of the heart rate". Impulses are generated from receptors in the muscles and joints during contraction and are transmitted to the medulla oblongata where they **stimulate the VCC** and **inhibit the CIC**, resulting in acceleration of the heart.

(3) From The Skin and Viscera

Exposure to cold and moderate painful stimuli from the skin or viscera usually result in reflex increase of the heart rate. However, severe pain (especially from the viscera) is usually associated with bradycardia.

(4) From The Eyes (**Oculo-Cardiac Reflex**)

Applying pressure to the eyeball results in reflex decrease in the heart rate ". Impulses from the eye are transmitted via afferent fibers in the oculomotor nerve to the nervous system where they **stimulate the CIC**.

N.B.: Signals from certain sensitive areas in the body known as the trigger areas (e.g. the larynx, testes and epigastric region) also result in cardiac slowing, and heavy blows to these areas may lead to cardiac arrest.

Cushing's reflex or reaction

- ⇧ Intracranial tension (as in brain tumor) ⇨ **bradycardia**

Mechanism:

- ⇧ Intracranial pressure as in brain tumor or cerebral hemorrhage ⇨ compression on blood vessels in the brain ⇨ ischemia of CNS ⇨ mild ⇩ O2 & ⇧ CO2 ⇨ direct effect on the cardiovascular center ⇨ **generalized VC of arterioles except of brain blood vessels** ⇨ ⇧ ABP ⇨ ⇧ brain blood flow ⇨ correct ischemia.
- This ⇧ ABP ⇨ stimulate the baroreceptors ⇨ reflex bradycardia **(Marey's reflex)**

II- Chemical Regulation Of The Heart Rate

Effect of changes in blood gases

- O2 lack
- CO2 excess

Effects of hormones and drugs

- Adrenaline and Noradrenaline
- Thyroxine, Histamine, Atropine
- Bile salts, Autonomic drugs

(A) Effect Of Changes In Blood Gases

- **(1) O2 lack (hypoxia)**

A moderate 02 lack increases the heart rate by

(a) A direct mechanism (Stimulation or the S-A node activity)

(b) A central mechanism (inhibition of the CIC)

(c) A reflex mechanism (stimulation of the pressor area + respiratory center through exciting the peripheral chemoreceptors in the carotid and aortic bodies).

On the other hand, severe hypoxia decreases the heart rate due to damage of the pressor area as well as inhibition of the S-A node activity.

- **(2) CO2 excess (hypercapnia) & H+ increase**

A moderate hypercapnia and increase **H+** (acidosis) increase the heart rate by the following mechanisms :

(a) Inhibition of the CIC **(b)** Stimulation of the pressor area through exciting the peripheral chemoreceptors (specially by H+ increase)

(c) Stimulation of the pressor area through exciting the central chemoreceptors (only by CO2 excess).

(B) Effects Of Hormones, Drugs And Chemicals

(1) Adrenaline: Small doses increase the heart rate while large doses elevate the ABP which decreases the heart rate through the Marey's reflex.

(2) Noradrenaline: This is a potent V.C. hormone that markedly elevates the ABP, which decreases the heart rate through the Marey's reflex.

(3) Thyroxine: This increases the heart rate by stimulation of the S-A node and increasing the metabolic rate (the formed metabolites cause V.D., which increases the venous return, resulting in the Bainbridge reflex)

(4) Atropine: This accelerates the heart by blocking parasympathetic activity.

(5) Histamine: This is a potent V.D. substance, which leads to marked drop of the ABP, resulting in heart acceleration through the Marey's reflex.

(6) Bile salts: These inhibit the S-A node activity and stimulate the CIC leading to bradycardia .

(7) Autonomic drugs: Sympathomimetic drugs cause tachycardia while parasympathomimetic drugs cause bradycardia

Factors Directly Affect The S-A Node Activity

- **Physical factors**

An increase of the body temperature by **1°C** increases the heart rate by 10-20 beats/minute and vice versa.

- **Mechanical factors**

Right atrial distension directly excite the S-A node leading to tachycardia (**Bainbridge effect**).

- **Chemical factors**

The **S-A node** is **directly excited** by

Mild hypoxia, Catecholamines, Thyroxine, Certain electrolytes and Alkalaemia.

While, it is **directly inhibited** by **Severe** hypoxia and hypercapnia, Bile salts ,Certain electrolytes ,Cholinergic drugs and Acidaemia


Best Wishes!