ELECTROCARDIOGRAM

- **As action potentials propagate through the heart, they generate electrical currents** that can be detected at the surface of the body. An electrocardiogram, abbreviated either **ECG or EKG (from the German word Elektrokardiogram), is a recording of these electrical signals.**
- The instrument used to record the changes is an **electrocardiograph.**
- **By comparing these records with one another and with normal records, it is possible to determine:**

(1) if the conducting pathway is abnormal.

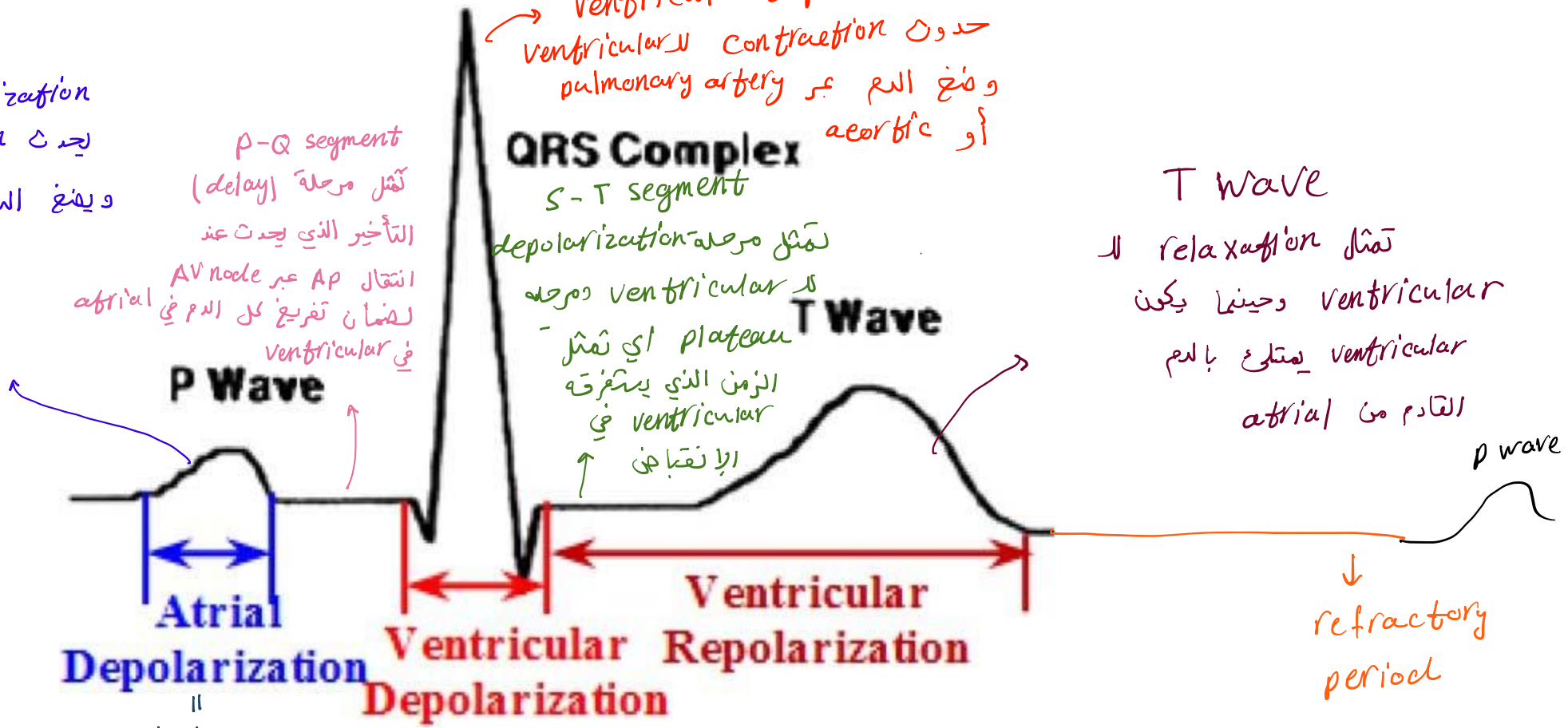
(2) if the heart is enlarged.

(3) if certain regions of the heart are damaged.

(4) the cause of chest pain.

ELECTROCARDIOGRAM

لا يوجد
gradecl



systole
=
ventricular repolarization = contraction

- في حال وجود اختلاف في الرسمة عن الطبيعي يعني وجود مشكلة في القلب سواء في afrial أو Ventricular

ما يعني وجود Arrythmia (عدم انتظام ضربات القلب)

- لماذا QRs أكبر من p wave ؟

1 - لأن حجم ventricles أكبر من afrial و بالتالي عدد الخلايا التي تقوم بالإنقباض أكبر واعوجة أكبر

2 - لأن gap junction في الـ AV node صغيرة وبالتالي عملية depolarization أقل قوة بينما gap junction في bundle of his و purkinji fibers أكبر و بالتالي عملية depolarization (contruction) في ventricula أقوى

ELECTROCARDIOGRAM

- In reading an ECG, the size of the **waves can provide clues to abnormalities.**

1. **Larger P waves** indicate enlargement of an atrium.
2. An **enlarged Q wave** may indicate a myocardial infarction. (نكتة قلبية حادة)
3. An **enlarged R wave** generally indicates enlarged ventricles.
4. The **T wave** is flatter than normal when the heart muscle is receiving insufficient oxygen—as, for example, in coronary artery disease. The T wave may be elevated in hyperkalaemia (high blood K ions level).

موجة T المسطحة : مؤشر على نقص الأكسجين في عضلة القلب (كما في مرض الشريان التاجي) .

- موجة T المرتفعة : مؤشر على اختلال مستويات البوتاسيوم في الدم (فرط بوتاسيوم الدم)

ELECTROCARDIOGRAM

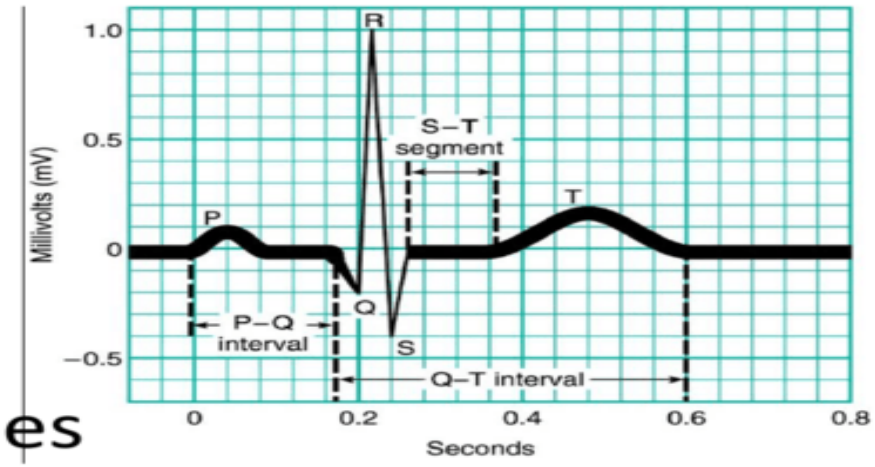
- **Analysis of an ECG also involves measuring the time spans between waves, which are called intervals or segments.**
- **P–Q interval** is the time from the beginning of the P wave to the beginning of the QRS complex. It represents the conduction time from the beginning of atrial excitation to the beginning of ventricular excitation.
- The **S–T segment**, which begins at the end of the S wave and ends at the beginning of the T wave, represents the time when the ventricular contractile fibers are depolarized during the plateau phase of the action potential.

ELECTROCARDIOGRAM

- The **Q–T interval** extends from the start of the QRS complex to the end of the T wave. It is the time from the beginning of ventricular depolarization to the end of ventricular repolarization.

The Electrocardiogram

- The major deflections and intervals in a normal ECG include:
  - P wave - atrial depolarization
  - P-Q interval - time it takes for the atrial kick to fill the ventricles
  - QRS wave - ventricular depolarization and atrial repolarization
  - S-T segment - time it takes to empty the ventricles before they repolarize (the T wave)



CORRELATION OF ECG WAVES WITH ATRIAL AND VENTRICULAR SYSTOLE

- The term **systole** refers to the phase of contraction.
- The phase of relaxation is **diastole**. (relaxation, filling)
- **The ECG waves predict the timing of atrial and ventricular systole and diastole.**

- اي ينقبض atrial بعد ظهور الموجة P
وكذلك ventricular ينقبض بعد ظهور الموجة QRS
وينبسط بعد ظهور T wave

- As the atrial contractile fibers depolarize, the P wave appears in the ECG.
- After the P wave begins, the atria contract (atrial systole).
- The action potential propagates rapidly again after entering the AV bundle. About 0.2 sec after onset of the P wave, it has propagated through the bundle branches, Purkinje fibers, and the entire ventricular myocardium.
- Contraction of ventricular contractile fibers (ventricular systole) begins shortly after the QRS complex appears and continues during the S–T segment.
- Repolarization of ventricular contractile fibers produces the T wave in the ECG about after the onset of the P wave.
- Shortly after the T wave begins, the ventricles start to relax (ventricular diastole). Ventricular repolarization is complete and ventricular contractile fibers are relaxed.

THE CARDIAC CYCLE: PRESSURE AND VOLUME CHANGES DURING THE CARDIAC CYCLE

- **Atrial Systole:**
  - Atrial depolarization causes atrial systole.
  - The ventricles are relaxed (The end of atrial systole is also the end of ventricular diastole (relaxation).

- **Ventricular Systole:**
  - The ventricles are contracting.
  - At the same time, the atria are relaxed.

- **Relaxation Period:**
  - The atria and the ventricles are both relaxed.
  - Ventricular repolarization causes **ventricular diastole**.

Figure 20.16 Phases of the Cardiac Cycle

semilunar valve:
pulmonary valve & aeortic valve

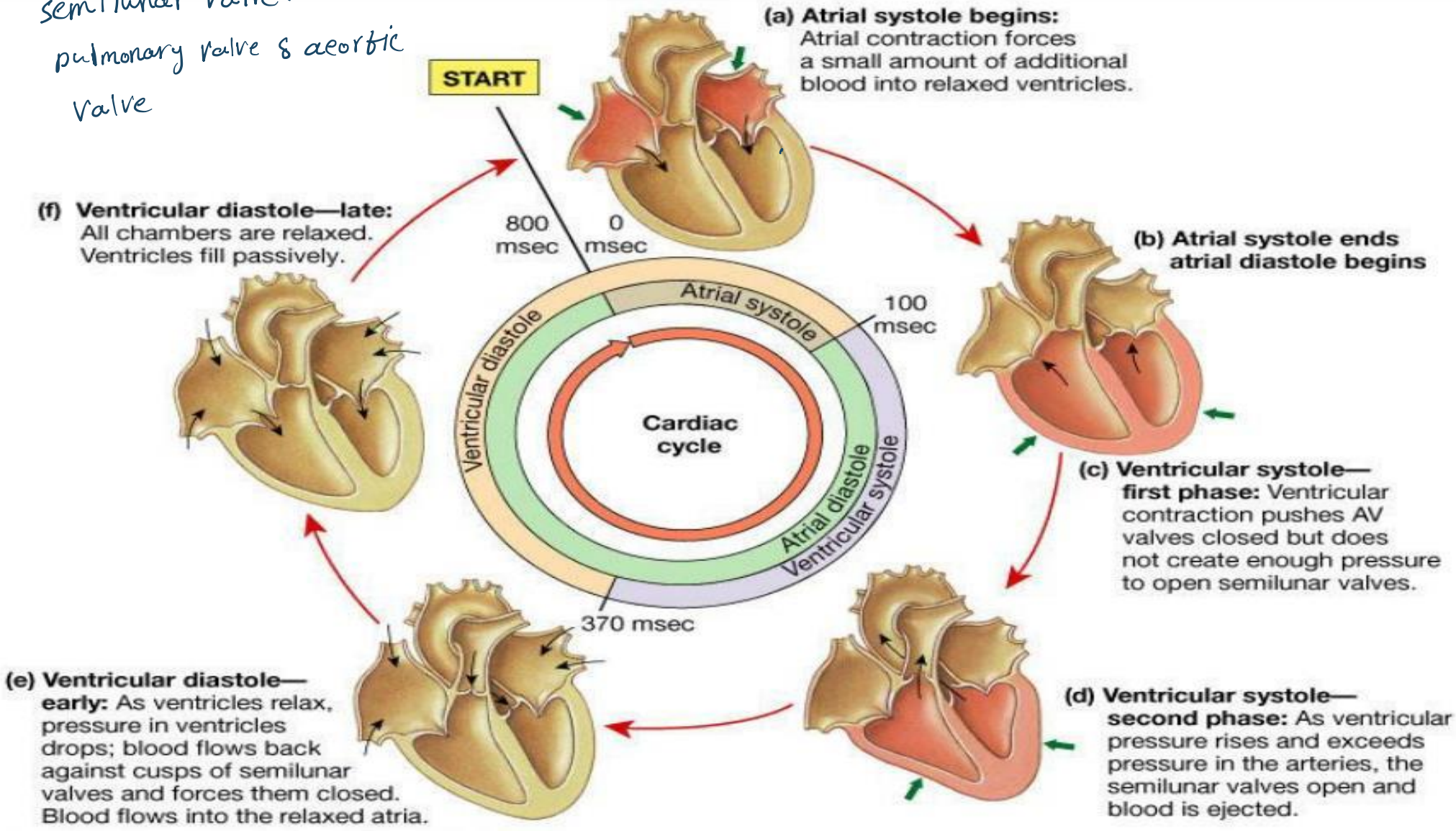


Figure 20.16

semilunar valves : Aeortic valve & palmonary valve

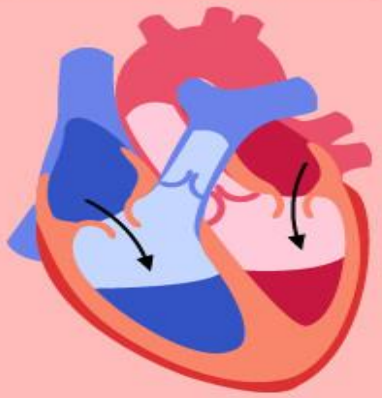
AV valves : Tricuspid valve & Mitral valve

$$\text{blood pressure} = \frac{\text{systole}}{\text{diastole}}$$

PHASES OF THE CARDIAC CYCLE

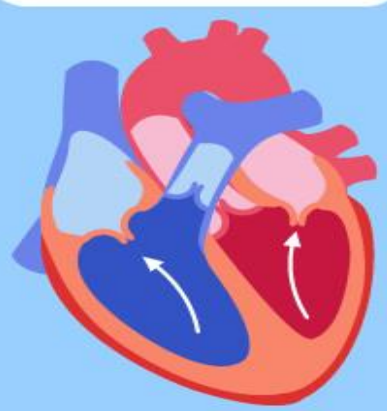
Atriole systole begins

Atrial contraction forces blood into ventricles



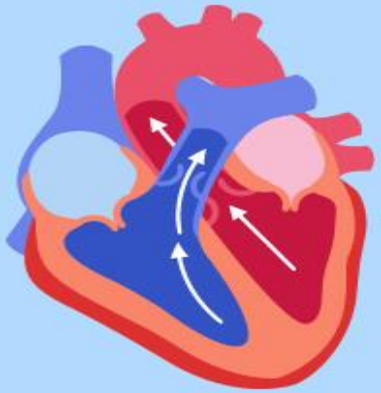
Ventricular systole (first phase)

Ventricular contraction pushes AV valves closed



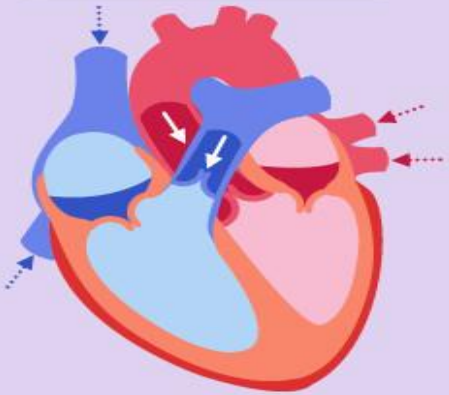
Ventricular systole (second phase)

Semilunar valves open and blood is ejected



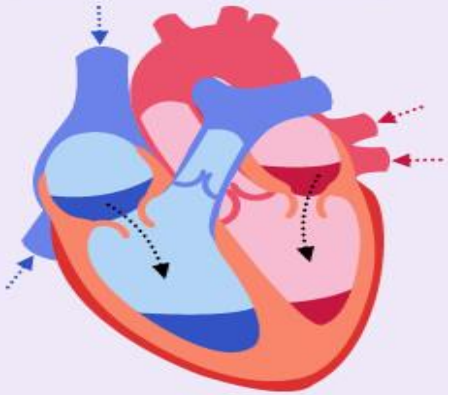
Ventricular diastole (early)

Semilunar valves close and blood flows into atria



Ventricular diastole (late)

Chambers relax and blood fills ventricles passively



P-Wave
Atria depolarization

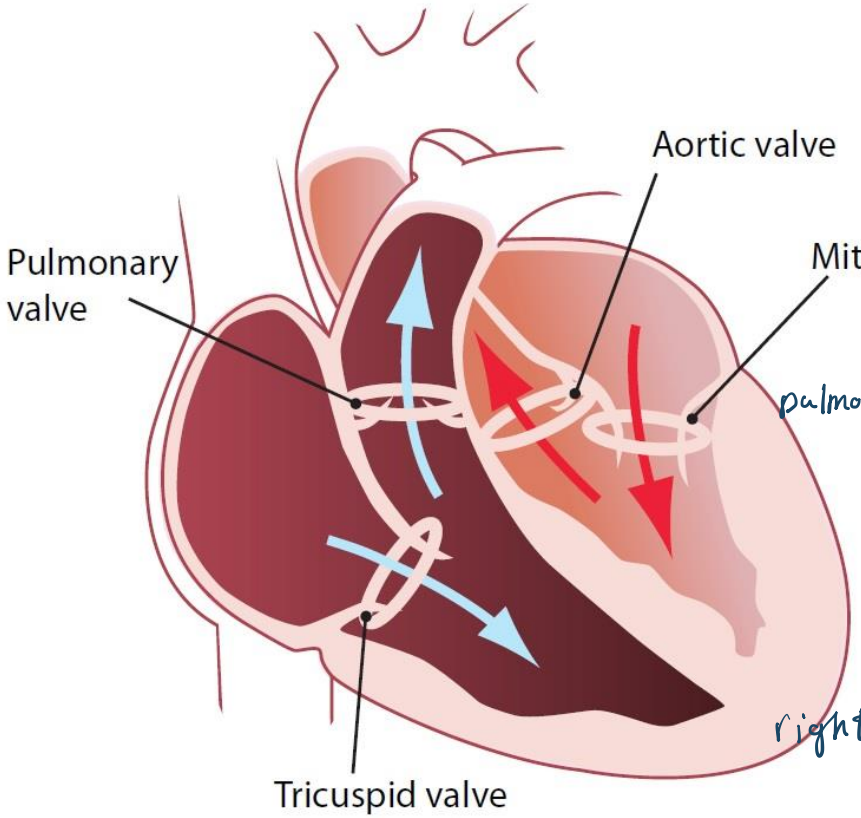
QRS Complex
Ventricle depolarization

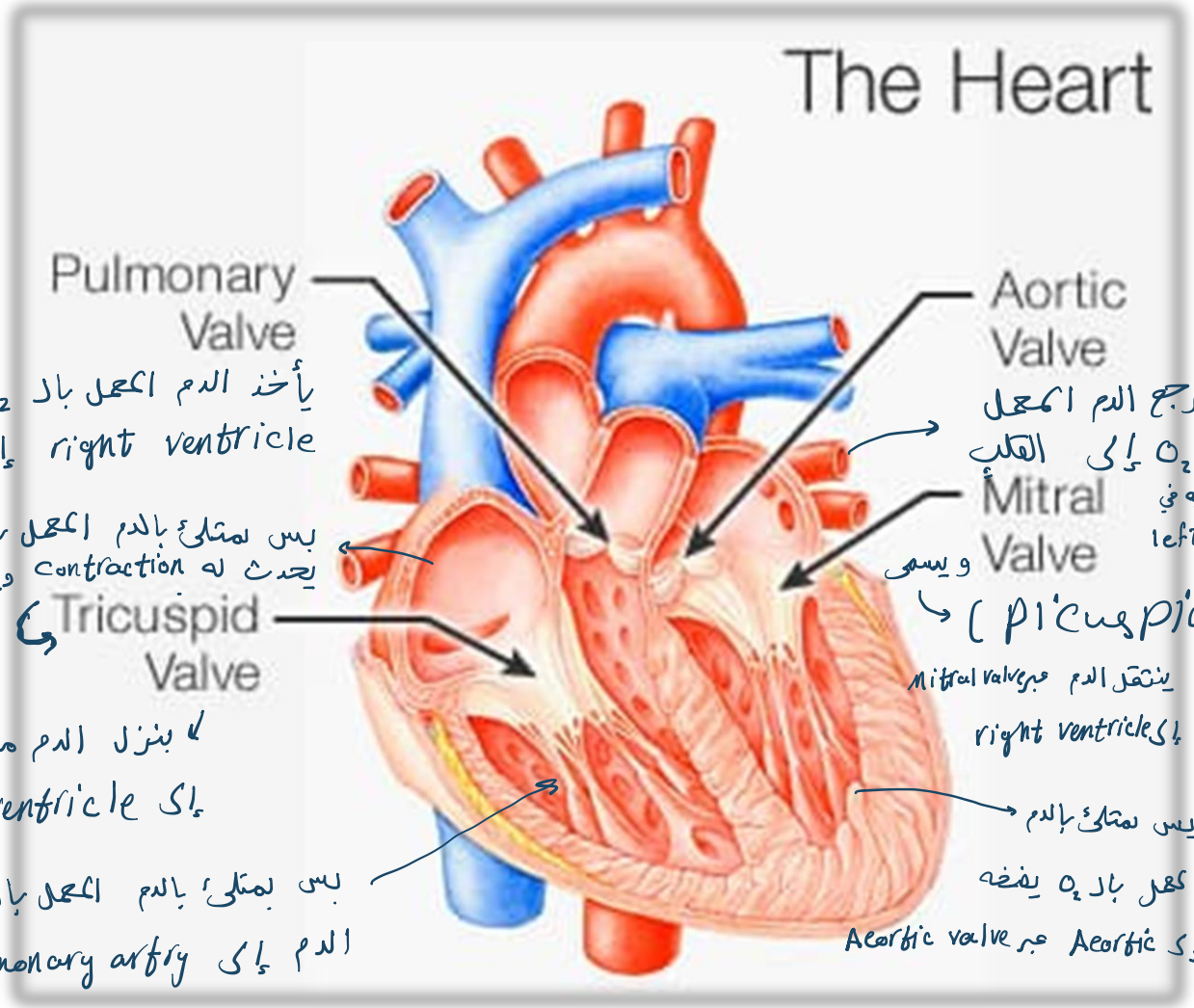
T - Wave
Ventricular repolarization

Atrial Diastole | **Atrial Systole** | **Atrial Diastole**

Ventricular Diastole | **Ventricular Systole** | **Ventricular Diastole**

HEART VALVES





يأخذ الدم المحمل بـ CO_2 من right ventricle إلى pulmonary artery

يبس يمتلئ بالدم المحمل بـ CO_2 يحدث له contraction ويضغ الدم إلى right ventricle

ينزل الدم من right atrial إلى right ventricle

يبس يمتلئ بالدم المحمل بـ CO_2 يضغ الدم إلى pulmonary artry عبر pulmonary valve

يرجع الدم المحمل بـ O_2 إلى القلب يصب في left atrial

ويسمى ← (pi'cuspid)

ينتقل الدم عبر mitral valve إلى right ventricle

يبس يمتلئ بالدم المحمل بـ O_2 يضخه إلى Aeortic عبر Aeortic valve

HEART SOUNDS

- Auscultation, the act of listening to sounds within the body, is usually done with a stethoscope.
- During each cardiac cycle, there are four heart sounds, but in a normal heart only the first and second heart sounds (S1 and S2) are loud enough to be heard through a stethoscope.
- **The first sound (S1),** which can be described as **a lubb sound**, is louder and a bit longer than the second sound. S1 is caused by blood turbulence **associated with closure of the AV valves soon after ventricular systole begins**.
- **The second sound (S2),** which is shorter and not as loud as the first, can be described as a **dupp sound**. S2 is caused by blood turbulence **associated closure of the semilunar (aortic and pulmonary) valves valves at the beginning of ventricular diastole**.
- Normally not loud enough to be heard, **S3** is due to blood turbulence during **rapid ventricular filling**, and **S4** is due to blood turbulence during **atrial systole**

normal ممكن يكون أو abnormal

صوت حركة الدم

abnormal دائما

Heart sounds

ممكن يتم سماع S_3 عند ممارسة جهد بدني ويكون قبل S_1 و S_2 بفترة معينة

- Auscultation – listening to heart sound via stethoscope
- Four heart sounds
  - S_1 – "lubb" caused by the closing of the AV valves
  - S_2 – "dupp" caused by the closing of the semilunar valves
  - S_3 – a faint sound associated with blood flowing into the ventricles
  - S_4 – another faint sound associated with atrial contraction

very loud

very thin + له علاقة بال atrial contraction

ممكن يكون normal أو abnormal

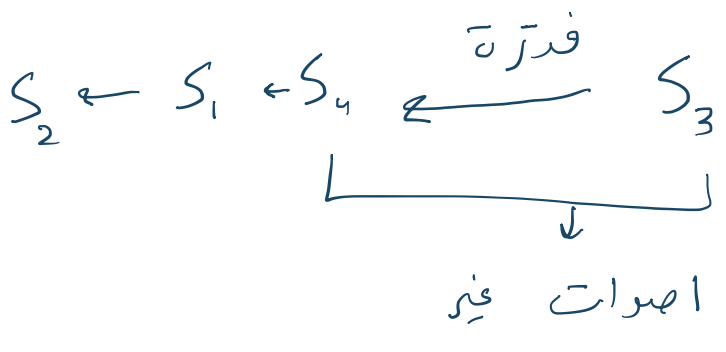
عالية جدًا ventricle لـ atrial قوة اندفاع الدم من

S_3 atrial لـ يظهر هذا الصوت عندما تكون قوة اندفاع الدم من ventricle عالية جدًا في بداية atrial contraction

يظهر هذا الصوت في نهاية atrial contraction قبل S_1 مباشرة

سماع هذا الصوت يعني وجود وضع abnormal +

ترتيب هذه الأصوات :



S_4 يكون قبل S_1 مباشرة

و S_3 يكون قبل S_1 بفترة

CARDIAC OUTPUT

مخرجات القلب

- **Cardiac output (CO) is the volume of blood ejected from the left ventricle (or the right ventricle) into the aorta (or pulmonary trunk) each minute.** Cardiac output equals the stroke volume (SV), the volume of blood ejected by the ventricle during each contraction, multiplied by the heart rate (HR), the number of heartbeats per minute: (كم ml من الدم يطلع من القلب لدقيقة)

- يُظهر مدى قدرة القلب على التكيف مع زيادة الطلب على الدم أثناء النشاط البدني ، مثل التمارين أو الضغوط الفسيولوجية .
- كلما كان الاحتياطي القلبي أكبر ، كان القلب أكثر قدرة على مواجهة الاحتياجات الطارئة

$$CO\ (mL/min) = SV\ (mL/beat) \times HR\ (beats/min)$$

- **Cardiac reserve** is the **difference between a person's maximum cardiac output and cardiac output at rest**. The average person has a cardiac reserve of four or five times the resting value. (الإحتياطي القلبي) (قدرش عضلة القلب بتتحمل زيادة عن normal)

في الشخص السليم ، يمكن أن يكون الاحتياطي القلبي أكبر بـ 4–5 مرات من النتاج القلبي عند الراحة . :

- على سبيل المثال ، إذا كان النتاج القلبي عند الراحة 5 لترات في الدقيقة ، فقد يصل أثناء النشاط إلى 20–25 لترًا في الدقيقة

REGULATION OF STROKE VOLUME

- **A healthy heart will pump out the blood that entered its chambers during the previous diastole.**

SV = end diastolic − end systolic

- **Three factors regulate stroke volume and ensure that the left and right ventricles pump equal volumes of blood**: (1) **preload**, the degree of stretch on the heart before it contracts; (2) **contractility**, the forcefulness of contraction of individual ventricular muscle fibers; and (3) **afterload**, the pressure that must be exceeded before ejection of blood from the ventricles can occur.

in the end of relaxation

PRELOAD: EFFECT OF STRETCHING

- Within limits, the more the heart fills with blood during diastole, the greater the force of contraction during systole. This relationship is known as the Frank–Starling law of the heart.

كم الكمية الثمانية اللي عبت نهاية diastole

نهاية relaxation (diastole الدم اللي بضل بنهاية)

- The preload is proportional to the end-diastolic volume (EDV), (the volume of blood that fills the ventricles at the end of diastole). Normally, the greater the EDV, the more forceful the next contraction.

كمية الدم اللي رجعت من heart لـ systamic circulation

كلما كانت مدة contraction كمية الدم اللي حتنبعي أكبر

- **Two key factors determine EDV: (1) the duration of ventricular diastole and (2) venous return, the volume of blood returning to the right ventricle.**

حجم الدم الراجع على القلب

endsystolicvolume ⟵ chumber في آخر دم بضل بعد systole

CONTRACTILITY

- **Myocardial contractility**, the strength of contraction at any given preload.

sympathetic agonist

- Substances that increase contractility are **positive inotropic agents (promote calcium ions inflow during cardiac action potentials)**, those that decrease contractility are **negative inotropic agents (reducing calcium ions inflow)**.

AFTERLOAD

- Ejection of blood from the heart begins when pressure in the right ventricle exceeds the pressure in the pulmonary trunk, and when the pressure in the left ventricle exceeds the pressure in the aorta.

- **At that point, the higher pressure in the ventricles causes blood to push the semilunar valves open. The pressure that must be overcome before a semilunar valve can open is termed the afterload.**

- **Conditions that can increase afterload** include hypertension (elevated blood pressure) and narrowing of arteries by atherosclerosis.

تصلب الشرايين

REGULATION OF HEART RATE

- **Autonomic Regulation of Heart Rate:**

- **Nervous system regulation of the heart originates in the cardiovascular center in the medulla oblongata**. The cardiovascular center then directs appropriate output by increasing or decreasing the frequency of nerve impulses in both the **sympathetic and parasympathetic branches of the ANS.**
- **Proprioceptors** that are monitoring the position of limbs and muscles send nerve impulses at an increased frequency to the cardiovascular center.
- Proprioceptor input is a major stimulus for the quick rise in heart rate that occurs at the onset of physical activity.
- Other sensory receptors that provide input to the cardiovascular center include **chemoreceptors,** which monitor chemical changes in the blood, and **baroreceptors**, which monitor the stretching of major arteries and veins caused by the pressure of the blood flowing through them. Important baroreceptors located in the arch of the aorta and in the carotid arteries.

REGULATION OF HEART RATE

- **Autonomic Regulation of Heart Rate:**

❖ **Through the sympathetic cardiac accelerator nerves:** In SA (and AV) node fibers, **norepinephrine** **speeds the rate of spontaneous depolarization** so that these pacemakers fire impulses more rapidly and heart rate increases; in contractile fibers throughout the atria and ventricles, norepinephrine **enhances calcium ions entry through the voltage-gated slow calcium ions channels**, thereby **increasing contractility.**

❖ **Through Parasympathetic nerve impulses reach the heart via the right and left vagus (X) nerves:** Vagal axons terminate in the SA node, AV node, and atrial myocardium. They release **acetylcholine**, which **decreases heart rate by slowing the rate of spontaneous depolarization in autorhythmic fibers.** As only a few vagal fibers innervate ventricular muscle, changes in **parasympathetic activity have little effect on contractility of the ventricles.**

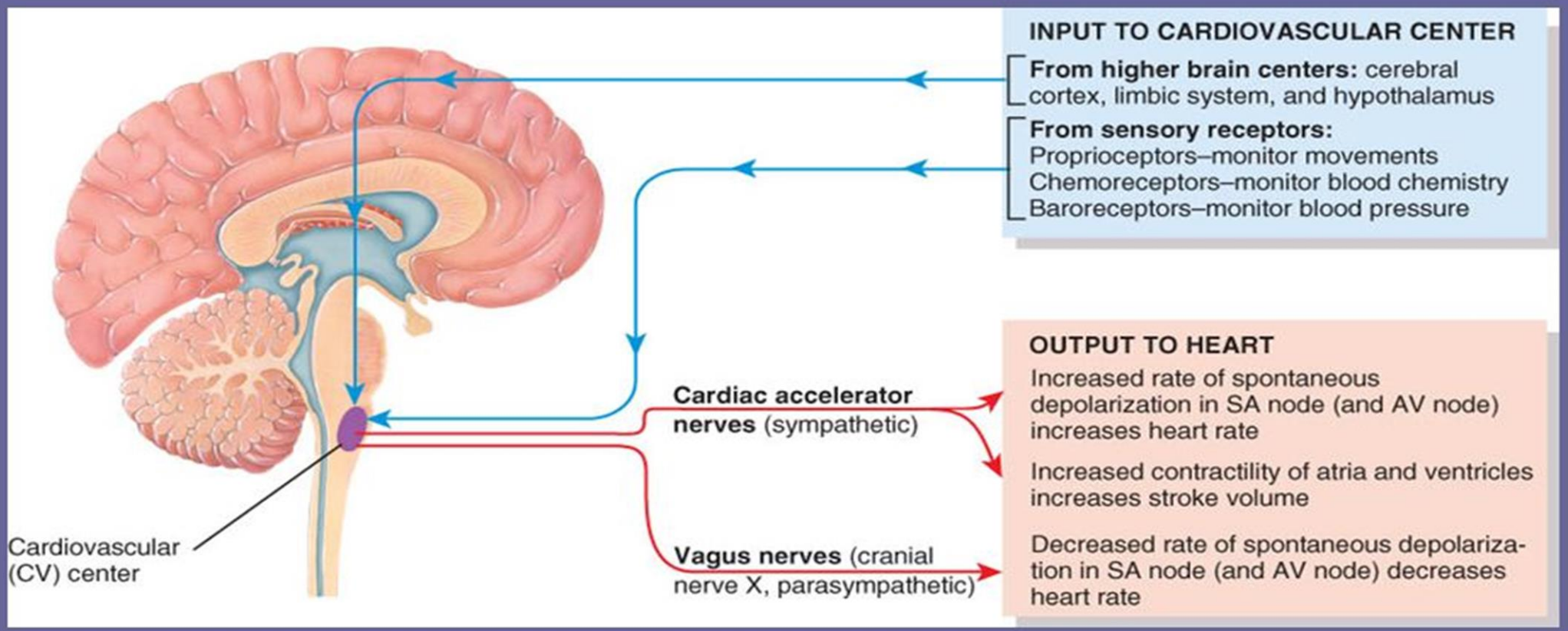
CHEMICAL REGULATION OF HEART RATE

1. Hormones: Epinephrine and norepinephrine (from the adrenal medullae) enhance the heart's pumping effectiveness. These hormones affect cardiac muscle fibers in much the same way as does norepinephrine released by cardiac accelerator nerves—they increase both heart rate and contractility. One sign of hyperthyroidism (excessive thyroid hormone) is tachycardia, an elevated resting heart rate.

2. Cations.: Given that differences between intracellular and extracellular concentrations of several cations (for example, sodium and potassium ions) are crucial for the production of action potentials in all nerve and muscle fibers. Elevated blood levels of potassium ions or sodium ions decrease heart rate and contractility. **Excess sodium ions** blocks calcium inflow during cardiac action potentials, thereby **decreasing the force of contraction**, whereas **excess potassium ions** **blocks generation of action potentials**. A moderate **increase in interstitial (and thus intracellular) calcium ions level** **speeds heart rate and strengthens the heartbeat.**

- كلما زاد Na كلما قل HR لأنو كلما زاد رح يسكر قنوات Ca
لأن Na رح يعوض postiviety وما رح نضطر plateau
tachycardia
فرح تقل ال contraction

Regulation of Heart Rate



OTHER FACTORS IN HEART RATE REGULATION

- Age, gender, physical fitness, and body temperature also influence resting heart rate.

- **A physically fit person may even exhibit bradycardia, a resting heart rate under 50 beats/min.**

انخفاض حرارة الجسم

- During surgical repair of certain heart abnormalities, it is helpful **to slow a patient's heart rate by hypothermia**, in which the person's body is deliberately cooled to a low core temperature.

HELP FOR FAILING HEARTS

- **Cardiac transplantation** is the replacement of a severely damaged heart with a normal heart from a brain-dead or recently deceased donor.

- **Cardiac transplants are performed on patients with end-stage heart failure or severe coronary artery disease**.

لـ مرضى يعاني بتزود القلب بالـ O_2 و التغذية
إذا صار عندها شخرة وكر الشريان إذا كان بنسبة التسكير
رح يفشل القلب ويقوم الشخص بعملية زراعة للقلب 70%

THANK YOU

AMJADZ@HU.EDU.JO

- العلاقة بين Cardiac output و Stroke volume و heart rate علاقة طردية

- كلما كانت كمية الدم اللي حتقبي right atrial أكبر كلما كان venus return أكبر كلما كان preload أكبر كلما كان enddistolic volume أكبر كلما كان stroke volume أكبر كلما كان cardiac output أكبر

Sympathetic ⟸ يعمل Central dijlation و يعمل constriction

general circulation الراجعة للدم عشان يزيد حجم blood vessels لل perpheral

بالقرب من القلب

لأن العلاقة aorfce لل diameter يزيد sympathetic

بين blood flow و علاقة طردية pressur و يعمل constriction لل

organ على الدم الرايح Cappillaries البعيدة عن القلب عشان يقلل

ويزيد الدم الي حيعمل circulation

لما يطلع من ventricle دم بـ after load عالي وضغط عالي على إحدى طبقات blood vessels يصير sensory receptors

baroreceptor: caretoid و aorta بكونو موجودين على sensory receptor اهم activation

- لما ضغط الدم بـ left ventricle يكون عالي بصير baroreceptor لـ activation يكون متفرع من aorta بجمة الرقبة

وهاي receptors بتكون sensetive لقيمة blood pressure فلما تكون كمية الدم كبيرة والضغط عالي بتوسع الشريان ونتيجة لتوسعه بتفتح ion channels ويتكون AP

- baroreceptor بتصل عليها عصبين ← vagus nerve (العصب ١٠) بعمل استجابة parasympathetic
Accsselerator nerve (العصب ١١) بعمل استجابة sympathetic

chemoreceptors ← جسم بيضي على سطح aorta ↓ ionotropic receptor نوعهم (ion channel & receptor) على نفس البروتين

- إذا كان الضغط عالي رح يصير activation لـ bararеceptor ورح يصير فيه dilation للشريان فيتفتح ion channels

ويتكون AP في Vagus nerve بعدين رح ينتقل AP إلى Cardiovascular centre ورح يعمل activation لـ Cardio vascular inhipitory centre

(para sympathetic) إلى حتفرز Ach وتقلل spentenous depolarization في SA node و AV node وبالتالي بتقلل HR

- وإذا كان الضغط قليل رح يصير activation لـ bararеceptor ورح يصير فيه dilation للشريان فيتفتح ion channels

ويتكون AP في العصب 11 ورح ينتقل AP إلى Cardiovascular centre ورح يعمل activation لـ Cardio vascular stimulatory centre (sympathetic)

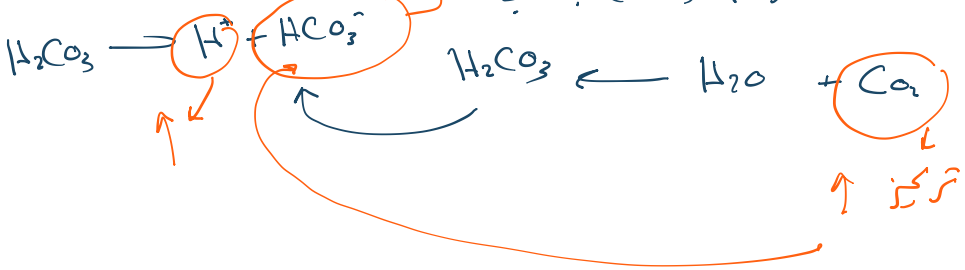
إلى حتفرز النورإبنفرين وتزيد spentenous depolarization في SA node و AV node وبالتالي بتزيد HR

chemoreceptor يتنشغل على Chemichals ← CO_2 أو O_2

بتتحفز بتغير تركيزهم

بتتحفز بال acidity (H^+) من كمية O_2 و CO_2 بالدم

أشكال CO_2 :



كلما زاد تركيز CO_2 زاد تركيز HCO_3 و H^+ (⟸ وكلما زاد H^+ بتتحفز chemoreceptor

نسبة O_2 قليلة = نسبة CO_2 عالية

Hypoxia ↓ hypercapenia

propriceptor ← على skeletal muscle ← كلما صار contraction ← بترجع الدم على القلب

thyroind hermon ⟸ هرمون الغدة الدرقية بزيد HR

الناس اللي نشاط الغدة الدرقية عندهم عالي لأن عمليات الأيض عالية بكون HR عالي