Cardiac cycle

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Atrial systol → atrial contraction.

ventride ← systol

Diastol

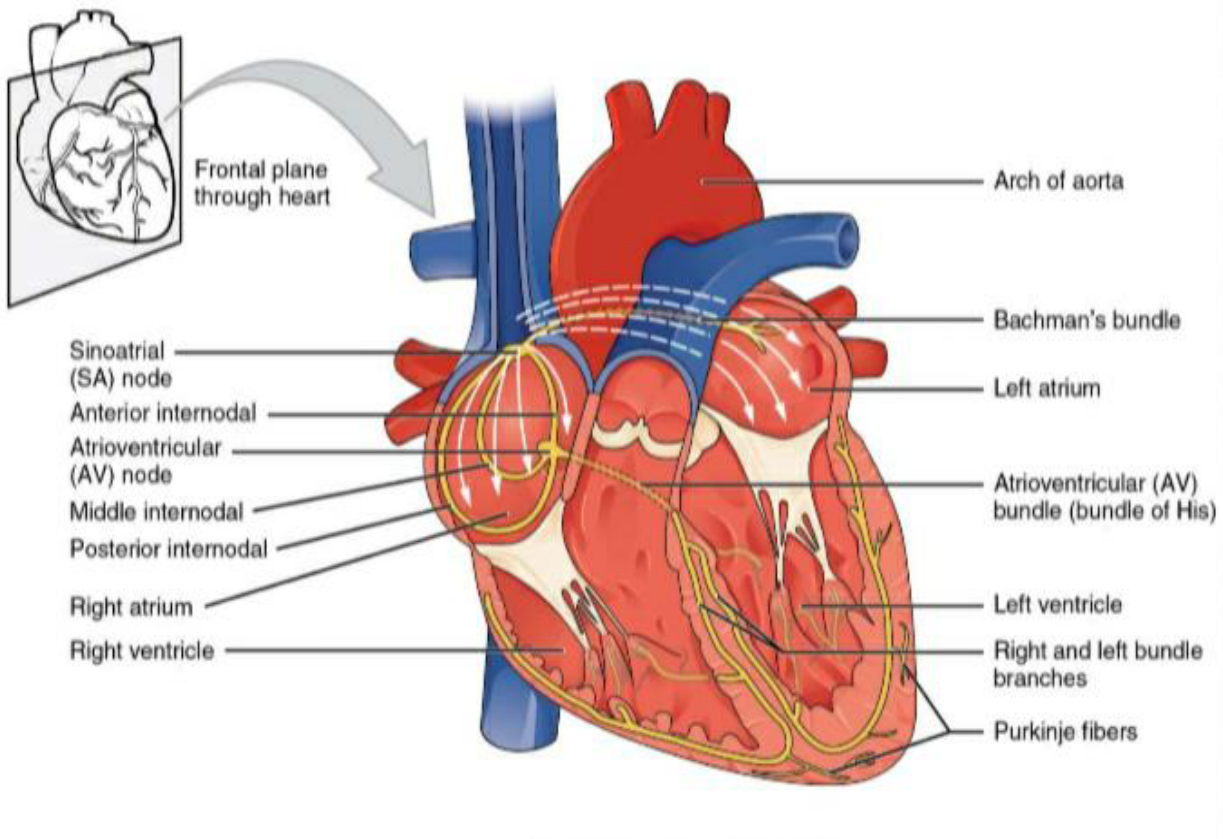
Blood move from High prusser → low prusser.

وين بيتكون ال Action potential ؟! في SA node اللي لحالها بتشحن، بعدين بتنزل ال AP ل 2 Atrium وبعد 2 Atrium ل Av node، ومنها ل bundle of His, Purkinji fiber وبتعمل Contraction في 2 ventricles.

Cardiac cycle معناها انو في عندي coordination / لازم القلب يكون في حالة relaxation عشان يعبي ب Blood بعدين contraction عشان يعمل ejection حتى contraction لازم بالأول يصير ل 2 atrium لحال بعدين ventricles لحال

شو اللي بيخليني AB تنتقل مع atrium ل ventricle ؟! Av node

Av node تعمل delay ل AB بالتالي بيصير Atrial contraction بالأول بعدين ل ventricles


Frontal plane through heart
Arch of aorta
Bachman's bundle
Sinoatrial (SA) node
Left atrium
Anterior internodal
Atrioventricular (AV) node
Atrioventricular (AV) bundle (bundle of His)
Middle internodal
Posterior internodal
Left ventricle
Right atrium
Right and left bundle branches
Right ventricle
Purkinje fibers

- Cardiac cycle is defined as the succession of coordinated events taking place in the heart during each beat
- Each heart beat consists of two major periods called systole and diastole
- During systole heart contracts and pumps the blood through arteries
- During diastole heart relaxes and blood is filled in the heart
- All these events are repeated in a cyclic manner

في 5 event Cardiac cycle .

- أول اشيء لما تكون SA بتشحن ← Cardiac at rest ← كل heart في relaxation

- هيتجي 2 Atrium لـ Blood ، Rt من Superior & inferior vena cava و lt من pulmonary veins . طبيعي

و Blood هيصير prusser فيهم أعلى من 2 ventricles بالتالي هينتقل الدم تلقائيا من 2 Atrium لـ 2 ventricles

والنسبة هتكون 80% ، 80% من الدم الموجود في 2 Atrium هينتقل لـ ventricles "Passivley" بسبب prusser gradient "

المرحلة الثانية بتكون SA خلصت شحن وانتقل AP لـ AV node بيصير عندي Atrial contraction ومسئول عن

20% من الدم الي هما ضلوا بتتغير النسبة في حالة excirse لتوصل 30

Kick systol

atrial systol بيقابلها ventricual diastol " نهاية atrial systol = نهاية ventricual Diastol "

لو بدي أوصف كمية الدم الي دخل كل لـ ventricle " 100% في ventricle " ← في نهاية atrial systol بحكي عنه

End atrial systolic Or End diastolic Volume.

في المرحلة الثالثة :- closed chambers.

في contraction لـ 2 ventricles وبيكون بسيط في هاي المرحلة " semilnar valve لسا مسكرين لأنه الضغط في aorta و

pulmonary عالي ، الـ contraction البسيط الي صار مسئول عن تسكير الـ mitral و tricuspid

في المرحلة الرابعة :- " ejection systol "

contraction هيفتح aortic و pulmonary ، اسمها ejection systol لانه صار بعدها ← ejection systol بسبب انه

contraction الي صار كان قوي كفاية عشان يخلي الضغط أعلى من الـ aorta و pulmonary وفتحوا 2 semilunaris valve

الخامسة

بيصير relaxation في ventricles ← هيقل الضغط ← هيكون في aorta و pulmonary أعلى وبيسكروا semilunaris

EVENTS OF CARDIAC CYCLE

- Atrial Events
- Ventricular Events

Ventricular Events

- Ventricular Systole = 0.27 (0.3) sec
- Ventricular diastole = 0.53 (0.5) sec

Clinical terms

- Systole =Ventricular Systole
- Diastole = Ventricular Diastole

- Atrial systole is also known as last rapid filling phase or presystole.It is usually considered as the last phase of ventricular diastole.
- The duration is 0.11 sec.During this period, only a small amount of blood is forced from atria into ventricles.

Cardiac Cycle

1) The heart at rest: atrial and ventricular diastole

The atria are filling with blood from the veins, and the ventricles have just completed a contraction

The AV valves between the atria and ventricles open, Blood flows from the atria into the ventricles.

→ at the end of atrial systol

2) Atrial systole

80% of blood enters the ventricles passively, but the last 20% of filling is accomplished by atrial contraction which push blood into the ventricles

At the end of atrial systole the ventricles contain the largest volume they will holdduring the cycle (End-diastolic volume (EDV)

3) Isovolumic ventricular contraction

When ventricular contraction begins, AV valves closes but does not create enough pressure to open semilunar valves

With both the AV valve and the semilunar valve closed, blood in the ventricle has nowhere to go

كل valves مسكرة لانو هلا atrio contraction خفيف سحّ valves
عمية الدم الموجودة ثابتة .

4) Ventricular ejection: The heart pumps

As the ventricles contract, they generate enough pressure to open the semilunar valves and push blood into the arteries.

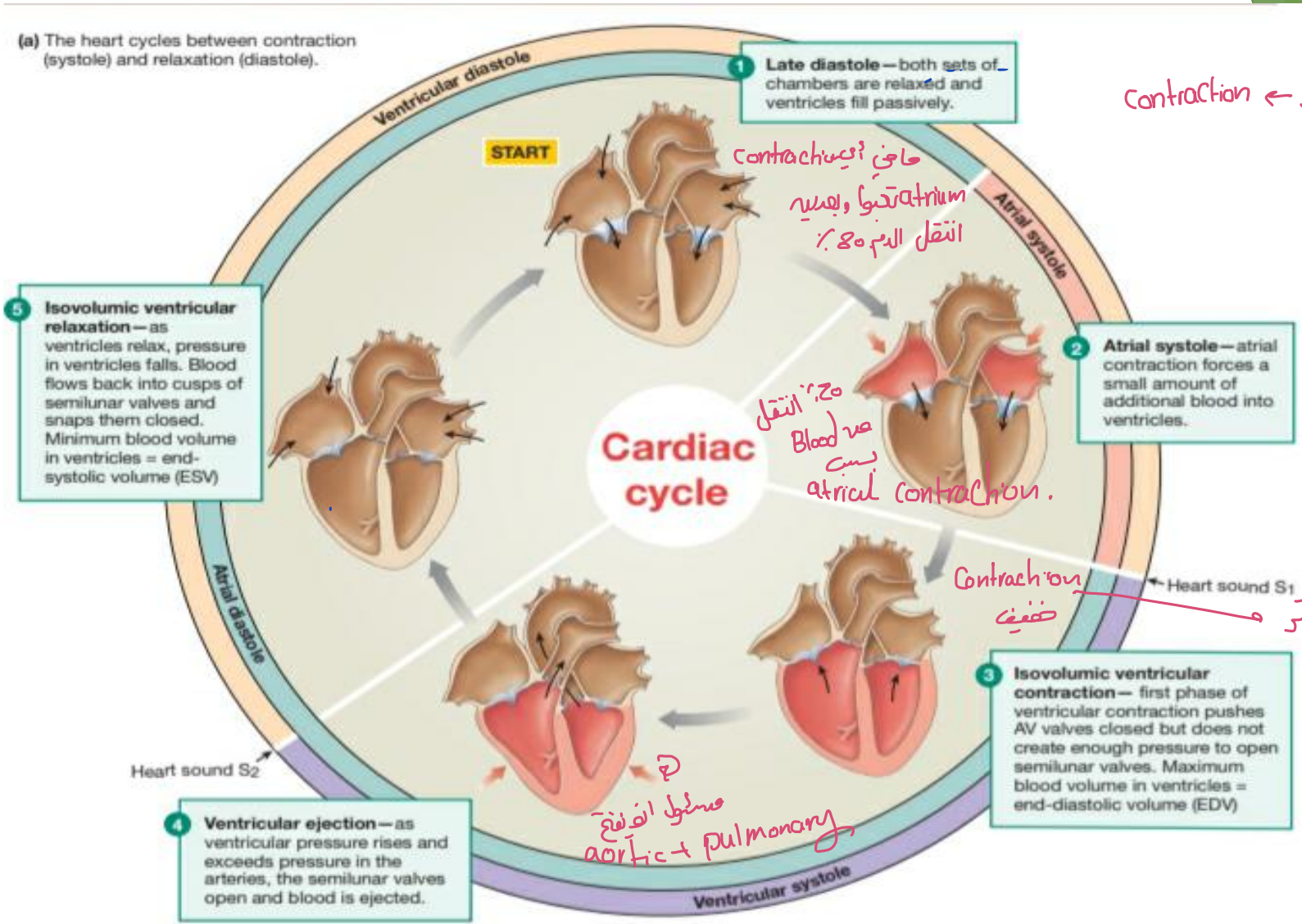
The pressure created by ventricular contraction becomes the driving force for blood flow. High-pressure blood is forced into the arteries

The heart does not empty itself completely of blood each time the ventricle contracts. The volume of blood left in the ventricle at the end of contraction is known as the end systolic volume (ESV).

5) Isovolumic ventricular relaxation

Ventricles relax, pressure in ventricles falls. Blood flows back into cusps of semilunar valves and snaps them closed.

Once the semilunar valves close, the ventricles again become sealed chambers. The AV valves remain closed because ventricular pressure, although falling, is still higher than atrial pressure This period is called isovolumic ventricular relaxation because the volume of blood in the ventricles is not changing.


(a) The heart cycles between contraction (systole) and relaxation (diastole).
Ventricular diastole
START
1 Late diastole—both sets of chambers are relaxed and ventricles fill passively.
Atrial systole
2 Atrial systole—atrial contraction forces a small amount of additional blood into ventricles.
Cardiac cycle
3 Isovolumic ventricular contraction— first phase of ventricular contraction pushes AV valves closed but does not create enough pressure to open semilunar valves. Maximum blood volume in ventricles = end-diastolic volume (EDV)
Heart sound S1
4 Ventricular ejection—as ventricular pressure rises and exceeds pressure in the arteries, the semilunar valves open and blood is ejected.
Heart sound S2
Ventricular systole
5 Isovolumic ventricular relaxation—as ventricles relax, pressure in ventricles falls. Blood flows back into cusps of semilunar valves and snaps them closed. Minimum blood volume in ventricles = end-systolic volume (ESV)
Atrial diastole

- First Heart Sound - closure of atrioventricular valves at the beginning of this phases produces first heart sound

- Second Heart Sound: Closure of Semilunar valves during this phase produces second heart sound.

Stroke Volume

- The amount of blood pumped by one ventricle during a contraction.
- It is measured in milliliters per beat
- Stroke Volume = EDV-ESV (Volume of blood before contraction-volume of blood after contraction)
- For the average contraction in a 70-kg person at rest:135 mL-65 mL = 70 mL, the normal stroke volume

لأنه left side هو كمية Blood اللي بتطلع في beat وحدة واحنا بنكلم عن SV
pulmonary circulation تقريباً بس 0.5 لتر أو أقل ، و systemic ← 35 L
ف احنا هنقيس SV لـ left side وهو كمية الدم اللي موجود في ventricle بعد contraction لمرة وحدة .

End diastolic volume − End systolic

SV = 200 − 150 = 50

لما نقول EDV = 200 يعني اكتر كمية دم وصلت ventricle = 200 .

ESV = 50 كمية الدم اللي ضلت في ventricle

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normall ejection fraction 55 → 75

less than 55 → Hf →

arrythmy بيقل في

- Ejection fraction: the percentage of EDV ejected with one contraction (stroke volume/EDV)
- For the 70-kg man at rest: if the EDV is 135 mL and the stroke volume is 70 mL,the ejection fraction is 70 ml/135 mL, or 52%. This means the ventricle is ejecting 52% of the blood that was in it at the end of relaxation and filling.

EDV = 100 ejection fraction 60% Heart rate = 60.

CO? SV?

60

- Stroke volume is not constant and will increase as contraction force of the ventricles increases.
- For example, if stroke volume increases to 100 ml during exercise, theejection fraction increases to 100 mL/135 mL, or 74%.
- Stroke Volume depends on 3 factors: Contractility, Preload, Afterload

- The preload refers to the amount of blood already in your ventricles when you’re ready to pump it out, and the afterload refers to the pressure against which your heart has to pump that blood.

كمية الدم التي يضخها القلب في الدقيقة

Cardiac Output (CO)

(60 → 100) beat.

- A measure of cardiac performance; A way to assess the effectiveness of the heart as a pump
- CO (Minute Volume): The volume of blood pumped by one ventricle in a given period of time.Because all blood that leaves the heart flows through the tissues
- cardiac output is an indicator of total blood flow through the body.

EDS = 100
ejection fraction = 60%.
heart rate = 60

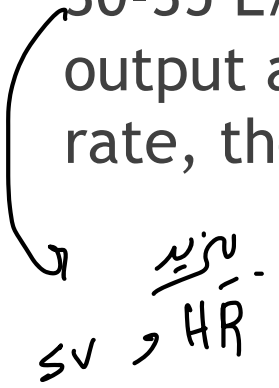
CO ?! 60x60 = 360
SV ?! 60

70 = HR ✓ SV = 70
CO = 70 x 70

- Cardiac output = heart rate × stroke volume
- For an average resting heart rate of 72 beats per minute and a stroke volume of70 mL per beat, we have
- CO = 72 beats/min× 70 mL/beat=5040 mL/min

(or approx. 5 L/min)

- Average total blood volume is about 5 liters. This means that, at rest, one side of the heart pumps all the blood in the body through it in only 1 minute
- During exercise, cardiac output may increase to 30-35 L/min. Homeostatic changes in cardiac output are accomplished by varying the heart rate, thestroke volume, or both.



Conditions that affect CO

- Increased

1. Excitement & Anxiety (Up to 100%)
2. Exercise (Up to 700%)
3. Eating (30%)
4. Fever → metabolic rate ↑ → O_2 demand ↑ → HR ↑ → tachycardia / tachypnea
5. Pregnancy → amount of blood increase — Blood ↑ 50% (from 5 L → 7.5 L).

- Decreased

1. Sitting or standing from lying(30%) → physiological
2. Tachyarrhythmia → contraction مش منتظم فمش هيكون فعال زي normal
3. Heart diseases

لو في Cardiac myopathy ضعف في contraction منتج

mitral regurge → عكس الدم

اللي هتطلع مش كلها

Autonomic Control Of Heart Rate

Parasympathetic Control → brady cardia

The parasympathetic neurotransmitter acetylcholine (ACh) slows heart rate.

Potassium permeability increases, hyperpolarizing the cell so that the pacemaker potential begins at a more negative value. At the same time, • Ca2+ permeability of the pacemaker decreases. Decreased Ca2+ permeability slows the rate at which the pacemaker potential depolarizes.

The combination of the two effects causes the cell to take longer to reach threshold, delaying the onset of the action potential in the pacemaker and slowing the heart rate.

SA node هي اللي بتتحكم في 2 sympathatic و parasympathatic
هتضلها تشحنه لعند ماتوصل لـ threshold هتنتقل الاشارة لعضلة القلب وتقبض.
في para هيزود دخول البوتاسيوم كتير وبيقلل Ca

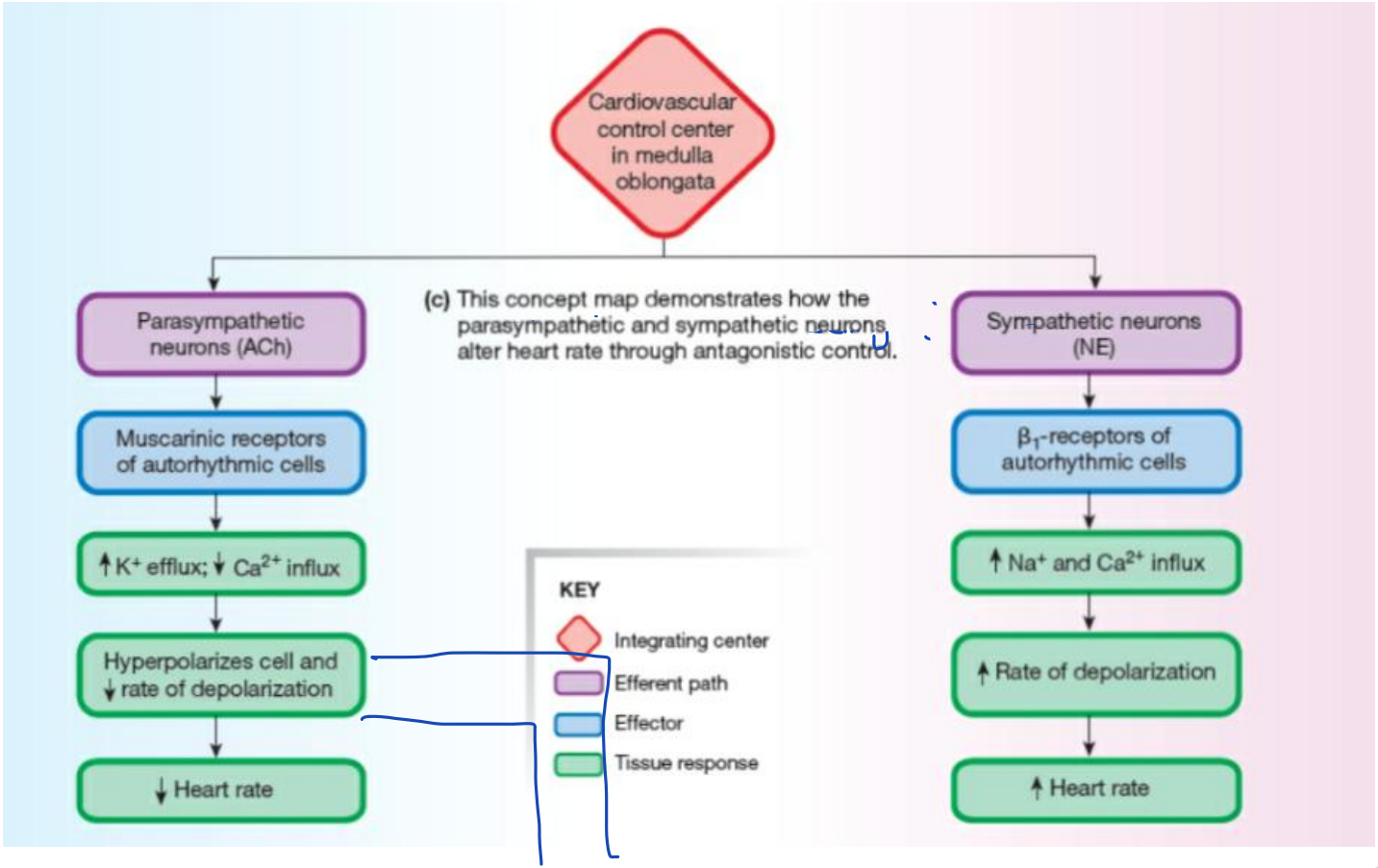
Sympathetic Control

Sympathetic stimulation of pacemaker cells speeds up heart rate. The catecholamines norepinephrine and epinephrine act by binding to and activating b1-adrenergic receptors on the autorhythmic cells increase ion flow through Ca2+ channels.

More rapid cation entry speeds up the rate of the pacemaker depolarization, causing the cell to reach threshold faster and increasing the rate of action potential firing.• When the pacemaker fires action potentials more rapidly, heart rate increases.

Sympathatic receptors → B_1

Parasympathatic Receptors → muscrinic.



(c) This concept map demonstrates how the parasympathetic and sympathetic neurons alter heart rate through antagonistic control.

Contractility

(ability to contract)

- The intrinsic ability of a cardiac muscle fiber to contract at any given fiber length and is a function of Ca2+ interaction with the contractile filaments.
- Normally, as contraction force increases, stroke volume increases

actin يتأثر على

مغطي أماكن ارتباطهم tropomyosin يسمى التي البروتين actin و myosin بين interaction بيصير نتيجة Contraction

Contraction تنقص tropomyosin بيغطي Ca

Contraction اذا زاد متزيد

Inotropy → we describe something related to contractility.

positive
adrenalin → intropic agent → ↑ contractility. +(dejoxin)
β blocker → negative intropic agent → ↓ contractility.

- Any chemical that affects contractility is called an inotropic agent
- Positive inotropic effect: If a chemical increases the force of contraction. For example, the catecholamines epinephrine and norepinephrine and drugs such as digitalis enhance contractility and are therefore considered to have a positive inotropic effect.
- Chemicals with negative inotropic effects decrease contractility

Preload

EDV
How much blood the ventricle can take.
or the amount of blood reach the ventricle.
بيتحكم فيه قدبش ال ventricle هاد stretchable

after loud
بيتحكم فيه
resistance of the aorta.
stenosis لمعنى
resistance
after loud
لازم تضخ بقوة اكبر ventricle

- The degree of myocardial stretch before contraction begins is called the preload on the heart because this stretch represents the load placed on cardiac muscles before they contract.
- The myocardial sarcomere length just prior to contraction. As this is not measurable without removing the heart and cutting it into tiny pieces, clinically it is usually approximated by EDV or, less appropriately, by EDP

- In striated muscles, the force created by a muscle fiber is directly related to the length of the sarcomere, as indicated by the initial length of the muscle fiber. The longer the muscle fiber and sarcomere when a contraction begins, the greater the tension developed, up to a maximum
- As stretch of the ventricular wall increases, so does the stroke volume. If additional blood flows into the ventricles, the muscle fibers stretch, then contract more forcefully, ejecting more blood.

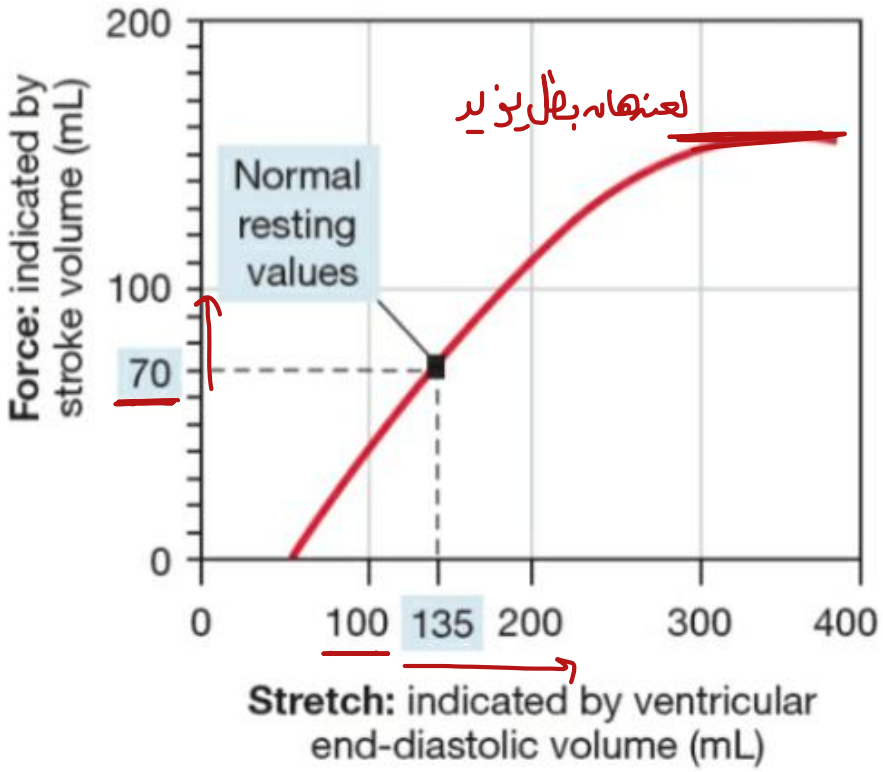
كل ما زاد stretching ← تميزيد contraction تكبر إلى حد معين .

The Frank-Starling Law of the Heart

- This law describes the relationship between stretch and force in the intactheart
- It states that the strength of cardiac contraction is dependent on initial fiber length
- At a cellular level, additional stretch increases:

1. The number of myofilament crossbridges that can interact
2. 2. Myofilament Ca2+ sensitivity

• As additional blood enters the heart, the heart contracts more forcefully and ejects more blood. It means that within physiological limits, the heart pumps all the blood that returns to it.


200
100
70
0
Force: indicated by stroke volume (mL)
Normal resting values
0
100
135
200
300
400
Stretch: indicated by ventricular end-diastolic volume (mL)

- **Venous Return** Preload depends primarily on venous return; the amount of blood that enters the heart from the venous circulation.
- Three factors affect venous return

1. Contraction or compression of veins returning blood to the heart (the skeletalmuscle pump)
2. Pressure changes pump)in the abdomen and thorax during breathing (the respiratory
3. Sympathetic innervation of veins

valve → unidirection of Blood flow
Blood flow it self due to the movment of muscle surrounding the vein

المريض بعد العملية لو ما تحرك ← بيعمل clotting ← thrombus ← DVT ← pulmonary thrombus
↓
Death.

Skeletal Muscle Pump

- Skeletal muscle contractions that squeeze veins (particularly in the legs), compressing them and pushing blood toward the heart.
- During exercise that involves the lower extremities, the skeletal muscle pump helps return blood to the heart.
- During periods of sitting or standing motionless, the skeletal muscle pump does not assist venous return.

Respiratory pump

Created by movement of the thorax during inspiration. As the chest expands and the diaphragm moves toward the abdomen, the thoracic cavity enlarges and develops a subatmospheric pressure.• This low pressure decreases pressure in the inferior vena cava as it passes through the thorax, which helps draw more blood into the vena cava from veins in the abdomen.

بيصير عندي breath لما اخذ
expanshion of the chest
negative prusser.
-ve. ← abdomen و
لهيك الدم من الضغط
العالي للمنخفض

ف IVC بينتقل من
abdomen → chest

ليش ؟! لانه زي ماحكينا في Respiration بيصير Chest وبيقل الضغط
expansion

- The respiratory pump is aided by the higher pressure placed on the outside of abdominal veins when the abdominal contents are compressed during inspiration.The combination of increased pressure in the abdominal veins and decreased pressure in thoracic veins enhances venous return during inspiration.

musculoskeletal بتحل شوي لكن الاساسيه

- Sympathetic innervation of veins
- Constriction of veins by sympathetic activity
- When the veins constrict, their volume decreases, squeezing more blood out ofthem and into the heart.
- With a larger ventricular volume at the beginning of the next contraction, the ventricle contracts more forcefully, sending the blood out into the arterial side of the circulation.In this manner, sympathetic innervation of veins allows the body to redistribute some venous blood to the arterial side of the circulation.

- **Afterload**
- The amount of pressure that the heart needs to exert to eject the blood during ventricular contraction• Ventricular force must overcome the resistance created by blood already filling the arterial system. In other words, to eject blood from the ventricle, The combined load of blood in the ventricle (the EDV) and arterial resistance during ventricular contraction is known as afterload.

- Increased afterload is found in several pathological situations, including elevated arterial blood pressure and loss of stretchability (compliance) in the aorta. •
- To maintain constant stroke volume when afterload increases, the ventricle must increase its force of contraction. This in turn increases the heart muscle's need for oxygen and ATP production.

- If increased afterload becomes a chronic situation, the myocardial cells hypertrophy, resulting in increased thickness of the ventricular wall. Clinically, arterial blood pressure is often used as an indirect indicator ofafterload.
- •Ventricular function can also be assessed noninvasively by echocardiography

ECG في QR بتمثله R

لما نحكي النبض 110/70

systolic=110 ← Diastolic=70

لو واحد في coma وبالعناية بدنا نشوف ضغطه عشان نعمل له systolic ولا Diastolic ؟!

متوسط الاثنين ← MAP هنحتسب ع ←

Mean Arterial Pressure (MAP)

- The average arterial pressure throughout one cardiac cycle (The driving force for blood flow)• Considered a better indicator of perfusion to vital organs than systolic blood pressure(SBP)
- Calculated as: MAP = 1/3 SBP + 2/3 DBP
- Mean arterial pressure is closer to diastolic pressure than to systolic pressure because diastole lasts twice as long as systole

بياخد وقت أطول

لو احد $\frac{90}{60}$

$(\frac{1}{3} \times 90 + \frac{2}{3} 60)$

$30 + 40 = MAP$

$= 70$

Thank you