Physiology

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1) **Intrinsic Factor**: that means in the heart itself or in the muscle of the heart, which is Frank-Starling law.

*The strength of contractions in the cardiac muscle differs from that in the skeletal muscle. In the skeletal muscle, it is dependent on the number of fibers participating in the contraction, while in the heart it’s composed of two unit (syncytium): atrial syncytium and ventricular syncytium, this means the atria act as a single fiber and the ventricle as another single fiber.*

The **Frank-Starling law**: said within physiological limit:
The more blood coming → more stretching → more contraction.

*But keep in mind that when it stretches too much, the elasticity or the distensibility of the myocardium will be destroyed. This is mainly what happens in cardiomyopathy (it’s the dilation of the myocardium beyond the physiological limit for unknown causes mostly). Can cause Heart failure.*

Normally
right atrial pressure = 0
cardiac output = 5.
If the atrial pressure increases, for example in exercise, within physiological limits, the cardiac output increases as well, by:

↑ Stretching (EDV)
2) **Extrinsic factors**: that means outside the heart, which are:

**A. Autonomic Nervous System:**

1. Sympathetic NS: supplies atria and ventricles, so it effects HR and contractility.
2. Parasympathetic NS: it effects only the atria (SA node)
Sympathetic Effect:

The mechanism of HR:
The heart rate is regulated mainly by SA node. Excitation of SA node is by leakage of Na\(^+\) to reach the threshold. The sympathetic effect on SA node is by increasing the leakage, so it will trigger the excitation much faster.

The mechanism of Contractility:
The sympathetic NS facilitates the opening of Ca\(^{+2}\) because during the plateau the contraction happens, and the channels of Ca\(^{+2}\) are responsible for the plateau.

Parasympathetic Effect:
It will do exactly the opposite, it will facilitates the opening the K\(^+\) channels, which will result in “Hyperpolarization” which means less SA node resting potential. (less than -60) \(\rightarrow\) HR

Parasympathetic stimulation: Sleeping)
B. Potassium:

Repolarization is affected by the K⁺.

it affects the heart in 2 ways:

1. The rate:
   A. K⁺ will decrease the heart rate by lowering the resting potential from -60 to -80, so it takes more time to reach the threshold -40.
   Hyperpolarization → Delay in the excitation

   B. K⁺ will make the repolarization longer, because there isn’t enough K⁺.

2. Dilation and weakening of ventricles:
   When we decrease HR the contraction by making the muscle dilated.

In both cases we might lose the patient. That’s why any patient admits to the hospital, with a heart disease, and also if he is older than 40 without any heart diseases, we should test their K⁺ level. The normal level is 3.5-5.5mEq. When level decreases to 2.5 or reaches to 8mEq mostly the patient will die, due to arrhythmia.

Treating patients, particularly with hypertension, we need drugs to reduce blood pressure. These drugs are called Diuretics (مدرات البول); One of them is Lasix (Furosemide).
When we give it to the patient, it reduces the volume, plus increases the secretion of K⁺.
But when we using it, we should give the patient K⁺ supplements,
to prevent the danger of loss of too much \( K^+ \). That’s very important.

Other types of drugs, increase the excretion of water (reduce the fluid volume) and doesn’t effect \( K^+ \) directly. Lasix is stronger diuretic, and is mainly used in patients with severe heart failure or pulmonary edema, in the case of the later we must administer it by IV, because it is an emergency state.

(35:23)

**C. Temperature:**

In the case of mild and moderate increase in the temperature in the early stage of any infection will increase contractility and the heart rate \( \rightarrow \) Tachycardia. But in prolonged and severe temperature (more than 40°) this will effect myocardial contractility (depress it), so we usually we have to reduce the temperature significantly, so we put the patient in cold water (with some ice preferably).
WORK OUTPUT OF THE HEART

It means the energy that is used in the contraction of the heart.

1. Filling phase: the volume and pressure are increasing.
2. Isovolumetric contraction: volume is constant but pressure is increasing because it’s contraction.
3. Ejection phase: pressure is increasing, volume is decreasing.
4. Isovolumetric relaxation: volume is constant again, but the pressure is decreasing.
5. Stroke volume: the distance between Early diastole and Early systole.

\[ SV = EDV - ESV \]
- **EW (External Work):** the work (energy) that is used for the contraction. → normal
  It’s divided into 2 parts:

  **Part I** *(99% of EW)*: to change the pressure of blood (from low pressure in the atrial *venous side* to high pressure in the ventricular side *atrial side*)

  **Part II** *(1-2% of EW)*: to increase velocity of the blood.

  The priority is to generate the pressure because without it the blood won’t flow, the second thing is to give it velocity.

- **PE (Potential Energy):**
  the heart in some stage during exercise, have the ability to increase the SV (output) by contraction at the end of systole, which decreases significantly. (to increase its function when there’s a need to as in the state of severe exercising)

  The blue part is → Diastolic pressure
  The green part is → Systolic pressure
The more there’s increase in the ventricular volume, there’s a little bit increase in the ventricular pressure. Up to about 180-200ml the blood pressure won’t increase significantly, but at the end diastolic volume increase more than 200ml (which happens in the **failing heart** that can’t pump the blood) the EDV will increase significantly, and in the case of **aortic incompetence (aortic stenosis)** the blood will stay in the ventricles, so this will increase the stretch of myocardial cells beyond physiological limits. Preload increases →↑ diastolic pressure The systolic pressure increases up to 280-300 because the contraction increases. After that you will see that the systolic pressure is decreasing, because the heart is failing and contractility is decreasing due to cardiomyopathy. This happens also in **pericarditis** patients.
Normal range is 119-170 the CO isn’t increased with the change of blood pressure beyond 200 the CO will decrease as we explained previously in page 7 and 8.

Do it with passion or not at all 😊
if there’s any mistake please contact us

Good luck