

# The Cardio-

# VASCULAR

# System

- Anatomy
- Histology
- Pathology
- Pharmacology
- Physiology
- Microbiology

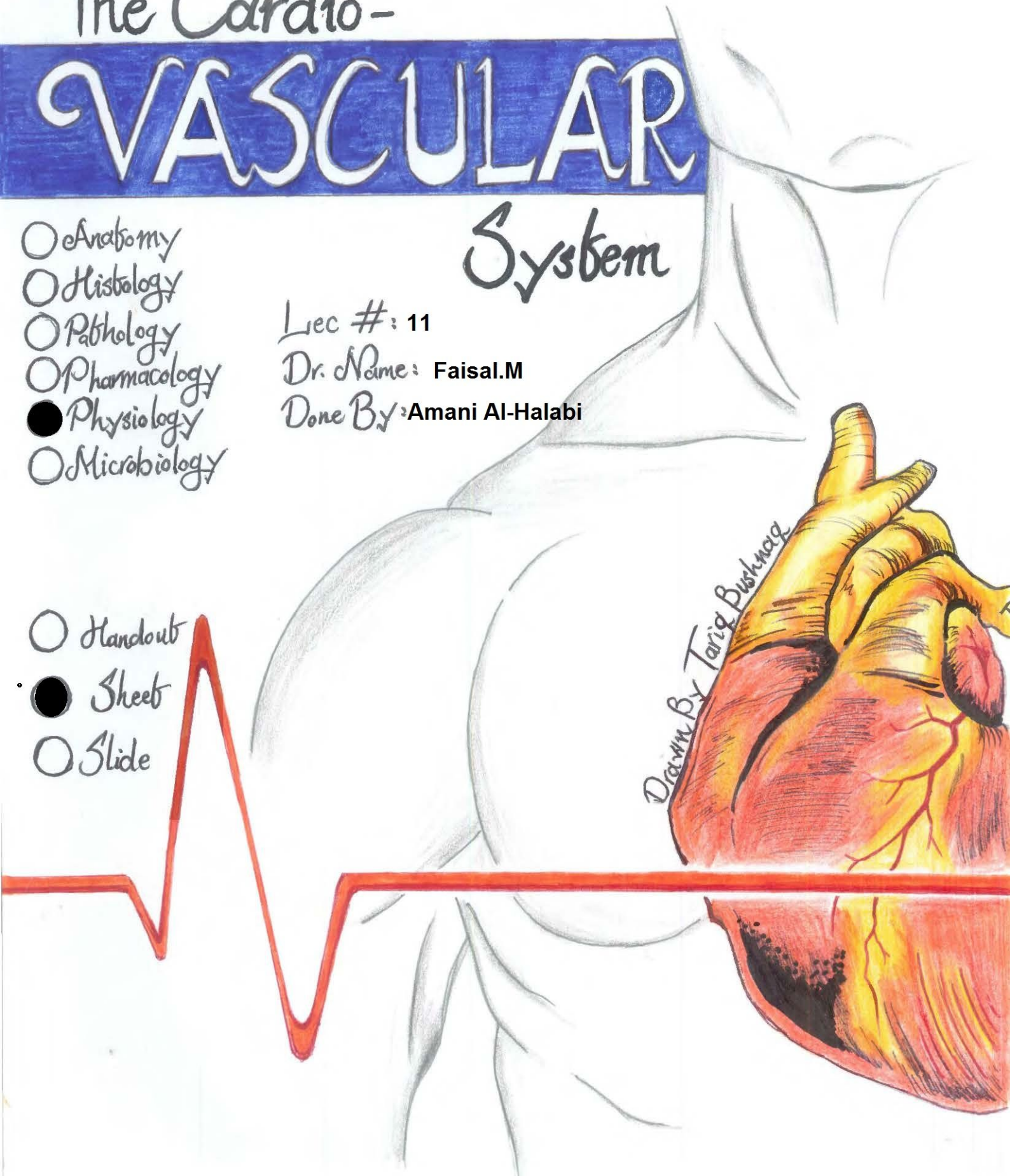
Lec #: 11

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- Handout
- Sheet
- Slide

Drawn by Tariq Bushnaq



## Revision & Cardiac Output & Venous Return

*The doctor in this lecture was repeating many previously discussed topics. Most of them were re-written in this sheet.*

If we're talking about the mathematical representation of a cardiac cycle, we plot the Left Ventricular Volume on the X axis and the, and the Left Ventricular Pressure on the Y axis. (or the Right ones, with changing the values only.)

Again lets define the Afterload: it is the pressure in the Aorta during diastole; thus for the semilunars to open, the intraventricular pressure should be above 80 mmHg. To develop these 80 mmHg, you need certain amount of energy and work. With increasing afterload you need to develop more energy to raise the pressure more, but if the total energy in the system is constant (no increased blood flow in the coronary arteries due to obstruction for example), the available energy will be used more to pump the blood to the Aorta, with LESS SV. [only if you increase the energy + the load on the heart, the SV may be normal or high.]

Remember the 3 diagrams of increasing the Preload, Afterload, and contractility respectively.

- When you increase the preload, the line (1-2) will shift to the right (you increase the EDV, thus increasing the SV; Frank Starling.
- If you increase the afterload, the line (2-3) will become longer. And because the energy in the system will remain the same, the area under the curve (external work) or the SV will decrease.
- If you want to increase the contractility (the SV), you should increase the energy + the amount of blood supplying the cardiac muscle, thus extending the line (1-2) from the left end; decreasing the ESV, with the EDV remains constant.

The SV, in response to increase HR ONLY, is going to decrease. But if the HR increases along with the contractility, there are 2 options; the SV will increase if the contractility is high enough to overcome the decrease in the filling time. Or the SV will decrease if the contractility is not increased to the required limit.

That's why you should not increase the HR for more than a certain limit, for the SV (and the CO accordingly) will decrease.

- Frankstarling: An increase in the length of the muscle within the physiological limits (up to the optimal length), will increase the SV. If you exceed these limits; the SV will start falling = cardiac failure.
- Later on will be talking about "Bainbridge reflex" which indicates that whenever there's an increase in the right atrial volume (+ pressure), the SA node will be compressed more and increase its permeability to Na, thus increasing the HR overall.

- Chemical regulation:
  - High K decreases the contractility.
  - High Ca Increases the contractility.
  - Thyroxine increases the HR.
  - Epinephrine increases both the HR + Contractility.
  
- You need a standard to compare different cardiac outputs among different people (due to the differences in surface areas). That's why we look to calculate the Cardiac Index, which is the CO/surface area, which should be almost the same for all people. [you can get the surface area for a person by plotting his height and age in certain curves.]
  - The Cardiac output is a flow, the amount of blood ejected from either right or left per minute (ml/min or L/min), thus it obeys Ohm law "  $Current = (p_2 - p_1) / r$ ". The current is the flow of blood (CO) which equals the change in pressure (from Aorta (MAP) to Rt. Atrium) / resistance. Pay attention to that the MAP is not the diastolic+systolic/2; because there's a time difference; the diastole is longer so it contributes more to the MAP. The resistance (TPR) or (TSR) is calculated between the Aorta and the Rt. Atrium.
  - Rt. Atrial pressure = 0, so the  $CO = MAP / TPR$ .
  - If you want to increase or decrease the MAP, either you change the CO, or the TPR, or both. [ Remember that  $CO = HR * SV$  ]
  - $EF = SV / EDV$ ,  $SV = EDV - ESV$

# You need to understand and memorize these laws because you'll need to use them in the exam. The problems are not so direct to be solved.
  
- When we say that Aseil has a blood pressure of 110/70, it means that her max ventricular pressure= 110, and the min ventricular pressure=0. Her max systolic pressure in the Aorta=110, while the min diastolic=70.

### Cardiac Output & Venous Return

- CO : the amount of blood ejected from either right or left per minute (ml/min or L/min). Also =  $SV * HR$ .
- Remember that the left CO is slightly higher than the right because of the venous return from bronchials which goes to the left side.
- Venous return: the amount of blood that returns back to the heart either from the superior and inferior vena cava to the right side or from the pulmonary veins to the left side per minute
- The Venous return should equal the CO; what comes goes, and that's the concept of Frank-Starling. If damping occurs → HF.
- Cardiac output that comes out from the aorta will be distributed to the tissues so the CO is the sum of all tissue flows. If there is any increase in blood flow to a tissue, this will lead to an increase in CO to meet the tissue demand. In other words, the need of more or less blood flow to tissue is a factor that regulate CO.
- Tissue's need of more or less blood flow depends on oxygen consumption .
- The Cardiac output is proportional to oxygen consumption (linearly).
- CO is proportional to  $1 / TPR$ , when MAP is constant.

→ A figure in the slides shows the result of an experiment on different levels of exercising.

- You can see that oxygen consumption line and CO line go parallel; the more oxygen consumption (more exercising) the more CO goes.
- Notice the difference in COs between very high level of exercise (athletes) and couch potato (lazy person).

#### Distribution of CO during rest:

- **Kidney:** 1000 ml/min, 20% - 25%, the highest amount. That is due to its function: filtration of blood, not because it needs more blood flow.
- **Heart:** 150 ml/min, 3% or 5% (not that much).
- **Skeletal muscles** at rest: 1000 ml/min, 15%, although it constitutes 40% of body weight. But during exercise, the blood flow to the muscles increases (64%). The CO, and the blood flow to the heart increase too.
- **Digestive system** at rest: 1350 ml/min, 27%. At exercise: very little! That's why you should not eat and then directly go to exercise; the blood will go mainly to your Sk. muscles, and you'll be having an abnormal digestion.

#### Blood flow per 100 gram of organ:

- Adrenal glands have the highest blood flow: 300 ml/min/100g. The reason is that they're too small, so the amount of blood that flows to it in comparison to its weight is very high.
- The Thyroid : 160 ml/min/100g. (Also High due to the low weight.)
- The heart (high blood flow for supply): 70 ml/min/100g.

#### → HR regulation

- The heart rate is regulated **extrinsically** by sympathetic and parasympathetic stimulation; sympathetic increases heart rate (+ chronotropic), while parasympathetic decreases HR (- chronotropic). Stroke volume is regulated by sympathetic stimulation (+ inotropic), BUT not regulated by parasympathetic.

sympathetic stimulation → + inotropic → ↑ stroke volume → ↑ CO.

- **Intrinsically** : according to frank-starling law : ↑ venous return → ↑ end-diastolic volume → ↑ stroke volume → ↑ CO.

[The sympathetic stimulation affects the venous return too, and this will be discussed later on]

→ Again;

- to increase CO, you either increase heart rate or stroke volume or both.
  - what affects the stroke volume are: contractility, preload and afterload.
  - what affects heart rate are: Sympathetic system, chemicals, hormones, etc.
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## Cardiac Function Curve (Cardiac Output Curve)

To draw the curve, plot the work done by the heart vs the atrial pressure.

- The atrial pressure in both atria is almost equal (the Lt. is slightly higher).
- Whenever the Atrial pressure increases (due to increased EDV, so the blood keep moving from Atria to ventricles), the work will increase.
- The work done by the left side of the heart is much higher than that done by the right side (10:1). The scale of the left side is much higher than that of the right. Accordingly the left pressure is much higher.

- Rt. atrial pressure = 0 normally.
- Rt. Atrial pressure is a reflexion of EDV.

Now, let the X axis represents the Rt. Atrial pressure, and the Y axis represents the CO;

- When the Rt. Atrial pressure = 0, the CO = 5.5 L/min.
- Also Normally: If the Rt. Atrial pressure becomes = +2, it means the EDV is increased, SV and CO are increased too up to 15 L/min (Only by Frank-Starling → CO keeps increasing to the plateau (optimal length), then it goes down.
- If you stimulate the heart sympathetically, a +ve inotropic effect will increase the SV with fixed EDV → **HYPEREFFECTIVE HEART** → At any Rt. Atrial pressure value, there's more CO (may reach 35 L/min as in athletes.  
[Athletes have hypertrophied hypereffective heart with high SV, so it's fine to have their HRs lower than normal people. Totally, the CO will be normal.]
- A **HYPOEFFECTIVE HEART** is a one with ischemia/ infarction or sympathetic inhibition → at any value of Rt. Atrial pressure there's less CO.

The heart is surrounded by pericardium, then lungs that are surrounded by pleura. Normally, the intrapleural pressure is negative (-5 to -7 mmHg). This negative pressure is what keeps the lungs inflated.

So, the pressure around the heart is around -5 mmHg, with venous return and CO equal normally 5.5 L/min.

- If the IPP becomes -7 → The venous return which is a flow,  $= (p-p)/r$ , will increase. Here the difference in pressure increases.
- If the IPP increases to become -2 → the gradient decreases, and you need more Rt. Atrial pressure (+3 mmHg) to have the same CO.  
[The whole curve won't be changed, nor the maximum values. The only change is that we are working at higher pressure now (shift to the left.)]

**Cardiac tamponade : pericardial effusion compresses on the heart directly, and increases the intrapericardial pressure. It affects the heart more than IPP. It makes the ventricular filling more hard.**

→ Results in shifting the curve to right. It will slightly affect the maximum but still can reach it. **It mainly changes the shape of the curve.**

- It is a serious condition!

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