The Cardio-VASCULAR System

- ○ Anatomy
- ○ Histology
- ○ Pathology
- ○ Pharmacology
- ● Physiology
- ○ Microbiology

Lec #: 9

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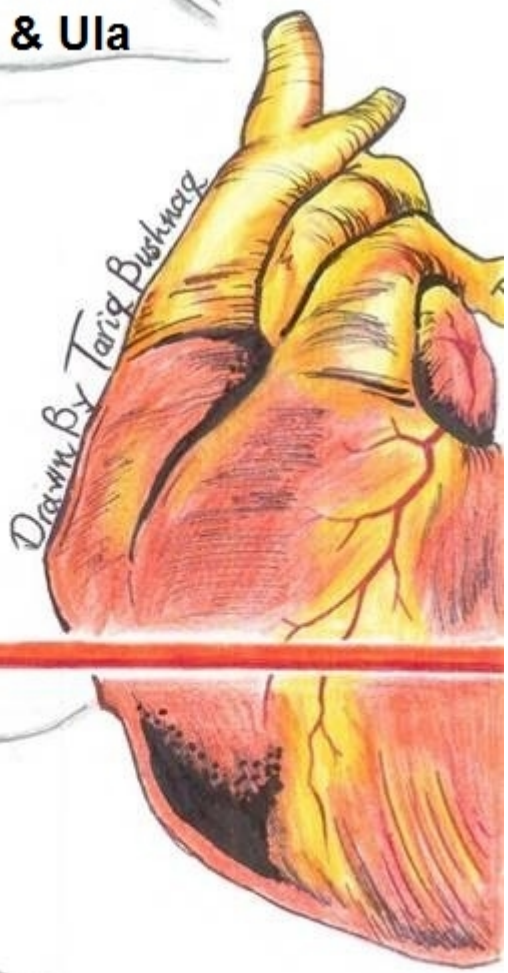
- ○ Handout
- ● Sheet
- ○ Slide



The Cardiac Cycle

Last time, we started talking about the cardiac cycle and the changes which occur in the cardiac cycle. We talked about the volume changes. We talked about systole, diastole and now, we will discuss pressure. We also discussed the electrical changes which are shown in the ECG. Later we will discuss the sounds of the heart to understand the interrelationship

There are two kinds of factors; either the intrinsic factors; via the Frank-Starling law of the heart or the extrinsic factors; which are the autonomic nervous system.

We began by discussing systole and diastole. We said that the atrial systole takes 0.1 seconds and that atrial diastole lasts 0.7 seconds. After atrial systole, ventricular systole begins and lasts 0.3 seconds. Finally, ventricular diastole lasts 0.5 seconds.

As for electrical changes, we record PQRST waves in the ECG. P precedes the atrial systole because **P represents atrial depolarization**. **QRS** precedes ventricular systole because it **represents ventricular depolarization**. **T wave** precedes ventricular diastole because it represents **ventricular repolarization.**

Now, we will discuss the volume changes. We discussed end diastolic volume (EDV). This is the volume of blood remaining in the ventricle, at the end of atrial systole (which is also at the end of ventricular diastole). This is followed by **Isovolumetric contraction, contraction during a short period in which the four valves of the heart are closed. During this period, there is no change in volume, only a change in pressure.** Pressure increases very quickly, to the extent of the pressure of the ventricle being greater than the pressure in the aorta,in the left side, and greater than the pressure of the pulmonary artery on the right side.

When the pressure in the left ventricle exceeds the pressure in the aorta, the aortic semilunar valves will open and blood is ejected from the left ventricle into the aorta. When the pressure in the right ventricle exceeds the pressure in the pulmonary artery (which is around 8mmHg), the pulmonary semilunar valves will open and blood is ejected from the right ventricle into the pulmonary artery. The first part of ejection is called rapid ejection. Then, because the valve is open and blood goes from the ventricle to the arterial system, the ejection is slow until we reach the end of systole.

At the end of systole, there's still a certain volume of blood in the ventricle. This is what we call **end systolic volume (ESV).**

Ula Isleem & Alia Khamis

So, the EDV is around 125 and the ESV is around 55. We calculate the stroke volume by subtracting the ESV from the EDV. Therefore, we define the stroke volume as the difference between the EDV and the ESV.

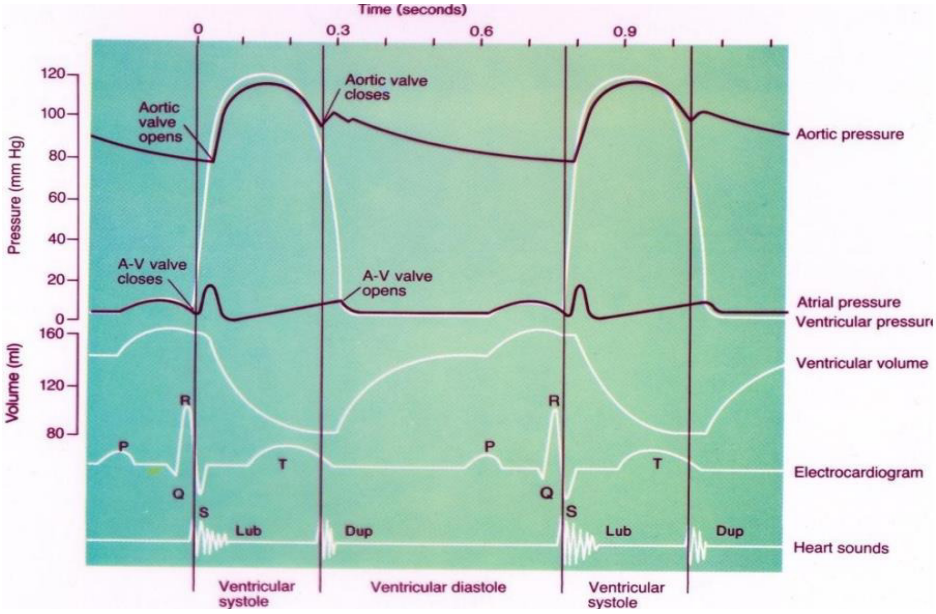
Diastole starts with a short period of **isovolumetric relaxation** (both the AV and semi-lunar valves are closed). The **pressure in the ventricles drops**, and is much LESS than the pressure in their corresponding major vessels (the aorta on the left and the pulmonary artery on the right). Then once the atrial pressure becomes higher than the ventricular pressure, the **AV valves open**, allowing **rapid filling** (due to the collection of blood in the atria during ventricular systole) of the ventricles with blood. This is ventricular diastole. The second stage of blood flow into the ventricles is **slow filling** and is referred to as **diastasis**. **The last stage is due to atrial systole**. We've mentioned previously that **atrial systole is not that important, as it contributes less than 25% of ventricular filling**. That's why atrial fibrillation is not that serious. Atrial systole is not essential for the functioning of the heart.

Pressure Changes

The contour of the waves in different pressures is the same. However, the scale (value) of the waves is not the same. On the right side of the heart, the pressure in the pulmonary artery varies between 8-25mmHg. The pressure in the aorta varies from 80-120mmHg. We use these numbers as textbook values, but in reality, every individual is different from the next.

This is the **aortic pressure**, which is represented by the **black lines**. The **white lines** represent the **ventricular pressure**. This is atrial systole.

Before, the atria begin systole; the **ventricular pressure has to be less than the atrial pressure**. *So, if the atrial pressure is 0, the ventricular pressure should be less than 0.*



When the atria push blood into the ventricles rapidly, the ventricles increase in their pressure, but the atrial pressure is still higher than ventricular pressure during that period. When ventricular systole begins, the pressure starts to build up in the ventricle.

The first increase in pressure in the ventricle raises its pressure above 0. When the pressure in the ventricle is higher than the pressure in the atrium, the AV valve closes. The closure of the AV valve prevents the blood in the ventricle from returning to the atrium. Since, the AV valve is closed, what does the blood do? It starts to move around the closed AV valve. **This movement of blood gives us the 'Lub' sound (low-pitched) or S1, which we hear with a stethoscope. So, S1 coincides with the closure of the AV valve.**

During **isovolumetric contraction**, when the **4 valves are closed** and the ventricle is contracting, **the pressure** in the ventricle gets very high, **very rapidly**. When the pressure of the ventricle rises above the pressure of the aorta during diastole (which is 80mmHg), the aortic semi-lunar valve opens and the blood flows into the aorta.

The **aortic pressure increases** since we are pushing blood through this tube, which is placing pressure on the walls of the aorta. The **ventricular pressure**, however, is still **higher** than the **aortic** pressure. *Why should the pressure in the ventricle remain higher than the pressure in the aorta?*

For blood to flow, there must be a **pressure gradient**, which is from an area of higher pressure to an area of lower pressure.

-If the aortic pressure is higher than the ventricular pressure, the blood will regurgitate into the ventricle.

-Notice in the graph, the white line which represents ventricular pressure is higher than the black line which represents aortic pressure

In the **last stage** of ventricular systole the **aortic pressure** is **slightly higher** than the **ventricular pressure** although blood is still moving from the ventricle to the aorta. *How does this work?*

Didn't we just say that a pressure gradient was necessary? **Movement of blood has inertia, or momentum (زخم)**. Momentum is a force and pressure is a force per unit of area. When the pressure overcomes this momentum, the aortic semi-lunar valve closes. So even at the moment that the aortic pressure is higher than the ventricular pressure, the blood still moves from the ventricle to the aorta. After that initial increase in aortic pressure, the pressure in the aorta gets even higher and the pressure

gradient is restored, causing blood to flow back into the ventricles and the semi-lunar valves to close. The second heart sound, or the **'Dub' sound (high pitched) is the sound of the blood trying to flow back to the ventricles after the semi-lunar valves have closed**.

The blood trying to return to the ventricles from the aorta also has momentum. This blood exerts pressure on the semi-lunar valve creating a notch. This forms what we call a **Dicrotic Notch or Incisura,** and is due to the closure of the semi-lunar valve.

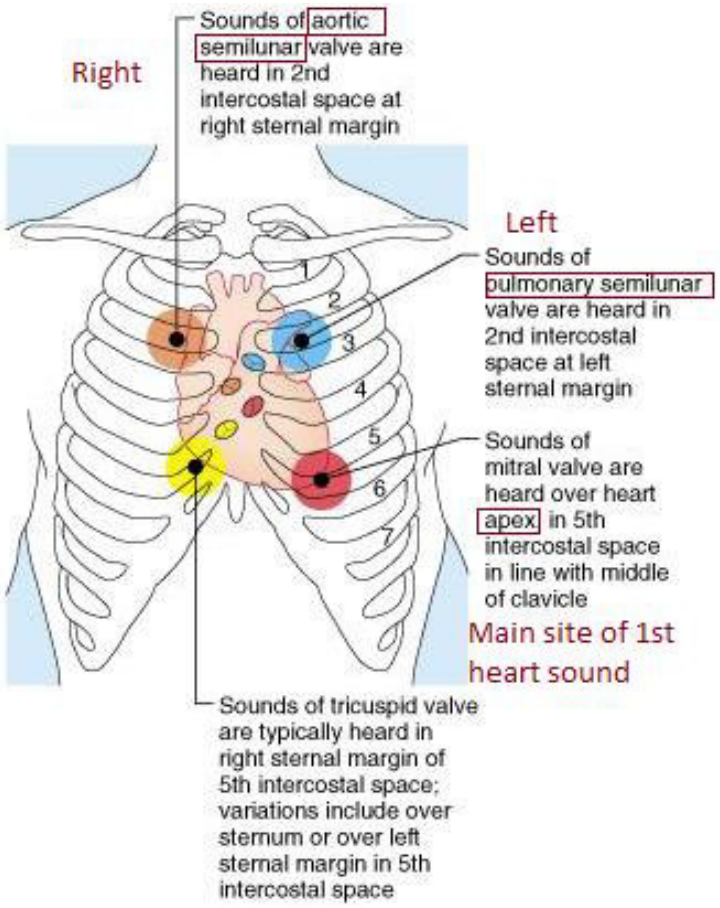
The **highest pressure** in the **ventricle** during **systole** is around **120 mmHg**. During **diastole**, the **aortic pressure** is **80mmHg**. This is why when we measure blood pressure we get a value of **120/80**; 120 representing systole and 80 representing diastole.

In the **left ventricle**, the pressure varies from **0-80mmHg**.

In the **right ventricle**, the contour of the wave is the same, but the values are different. The **pulmonary artery's diastolic** pressure is around **8 mmHg**.

The **systolic pressure** in **pulmonary artery** is around **25 mm Hg.** (Pulmonary artery pressure= 25/8)

The right ventricle pressure varies between 0 and 25 mmHg.



The Heart Sounds

The **first heart sound** coincides with the **QRS** wave of the ECG. The **second heart sound** coincides with the **end of the T wave**. There are two more heart sounds, S3 and S4. The **slow filling** of the ventricles from the blood in the atria makes the heart sound, **S3**. The **rapid filling** of the ventricles from the blood in the atria makes the heart sound, **S4**, due to atrial contraction. **S3 and S4 are more pronounced in valve stenosis** (narrowing of the valve) or during the regurgitation of blood. We don't usually hear these two heart sounds. You can only hear these if you have what's called a good musical ear.

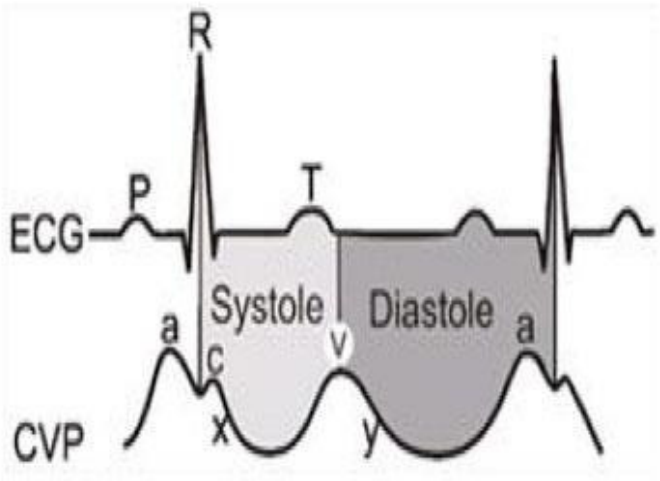
You may hear an abnormal "split" second sound (S2) when the aortic and pulmonary valves don't close at the same time.

The important thing to know here is the time between S1 and S2. The time between these two sounds represents ventricular systole, which is around 0.3 seconds. The time between s2 and the next s1 is 0.5 seconds, which represents ventricular diastole. So these are all the electrical, volume, and sound changes during a single cardiac cycle.

Atrial Pressure

All that's left now is the atrial pressure.

This pressure is most significant in cases of **abnormal pressure**, because it can **exert pressure on the jugular vein**. The atrial pressure is usually zero.



Ventricular Diastole-0.5s (AV valve is open)

The AV valve opens and blood flows from the atrium to the ventricle according to the pressure gradient, causing the pressure in the ventricle starts to increase. Then, the ventricular systole starts and the AV valve closes.

Atrial Systole-0.1s

(Contribution of last 25ml of blood)

Atrial systole occurs during late ventricular diastole. During atrial systole, the atrial pressure increases slightly, and gives us what we call the **A wave**. So, the **A wave is due to atrial systole**.

Ventricular Systole-0.3s (AV valve is closed)

The high pressure in the ventricle pushes the cusps of the closed AV valve upwards, which causes **an increase in the atrial pressure**. The pressure in the atria also

increases because blood is still flowing into the atria, even when the AV valves are closed. The atria then give us a wave which coincides with **isovolumetric contraction (of the ventricles)**. This gives us the **C wave**.

Why doesn't high ventricular pressure open the AV valve?

What prevents the AV valve from opening is Chordae Tendinae which are attached to the ventricular surfaces of the AV Valve. These Chordae Tendinae are anchored by papillary muscles.

So, when the ventricular muscles contract, the papillary muscles contract as well, pulling the Chordate Tendinae down towards the ventricle, thus preventing the valve from everting/prolapsing (turning outwards).

If the Chordate Tendinae are cut or in the case of Myocardial Infarction (the papillary muscle doesn't contract), eversion of valves occur resulting in incompetent valves. Blood then flows back from the ventricle to the atrium (regurgitation), causing acute heart failure.

E.g.: Aortic valve/AV valve/Mitral valve regurgitation means that the valves have everted or prolapsed

Remember:

During isovolumetric contraction of the ventricles (~0.01s), the AV valves are closed and the semi-lunar valves are still closed, no emptying occurs. *Recall that there is no change in volume, only the pressure changes due to contraction.*

When the inter-ventricular pressure exceeds the pressure within the aorta/pulmonary trunk. At the end of isovolumetric contraction the semi-lunar valves open, rapid then slow ejection occur, and ventricular volume and pressure decrease.

Ventricular systole ends, the semi-lunar valves are still closing (by the backflow of blood*) while the AV valves are closed. This is the isovolumetric relaxation (~0.03s)

*Dicrotic Notch/Incisura: a brief rise in aortic pressure caused by the backflow of blood rebounding off semi-lunar valves

The ventricular pressure continues to decrease until it becomes lower than the atrial pressure. Then, the AV valves open.

At the end of isovolumetric relaxation, the AV valve opens. **When the AV valves open, the blood flows from the atria into the ventricles once again.** This gives us the **V wave**.

The pressure increases in the atria until the end of isovolumetric relaxation (until the AV valve opens), which means while ventricular systole was taking place, the atria were filling up with blood **(atrial diastole-0.7s)** causing an increase in atrial pressure. Once the AV valves open, the pressure in the atria drop suddenly. Blood passes to the ventricles rapidly (rapid filling), followed by slow filling (diastasis), then atrial systole once again.

This is the <u>atrial pressure wave</u> and an explanation for the three stages; A, C, and V.

A Wave	C Wave	V Wave
Atrial Systole	Ventricular Systole, Isovolumetric Ventricular Contraction, The AV valve is pushed towards the atria	Isovolumetric Relaxation

Cardiac Output and Reserve

Cardiac Output: the amount of blood pumped by each ventricle

CO=HR*SV

Reminder: The stroke volume is always equal in both ventricles

The cardiac output on the left side is a bit higher than on the right side. *Why?*

This is a result of the drainage of the blood supply of the bronchi into the pulmonary veins.

Cardiac Reserve (Intrinsic Regulation): the difference between maximum and resting cardiac output

-Within physiological limits, an increase in the length of the muscle increases tension.

-We measure the length of the muscle in proportion to the End Diastolic Volume (volume of blood found in the heart before it contracts).

-The force of contraction is directly proportionate to the volume.

-This results in a higher stroke volume as well as an increase in cardiac output.

+Volume (End Diastolic Volume), +Force of Contraction, +SV, +CO

Conclusion: We can keep increasing the stroke volume until the optimum length of the muscle (optimum length of sarcomere) is reached.

-The maximum cardiac output than can be reached is around 15 liters/minute.

-The maximum cardiac output can reach up to 35 liters/minute in athletes.

-The normal (resting) cardiac output our body functions with is 5 liters/minute.

The cardiac output can be increased from 5 to 15 liters/minute intrinsically without any sympathetic stimulation.

The difference between 15 and 5 (10) is the Cardiac Reserve

The Cardiac Reserve in athletes is much higher than that of an average person.

Factors Affecting Stroke Volume

Preload: the load (end diastolic volume) or the amount of tension found in the heart before contraction

Afterload: the amount of tension the heart has to develop to overcome the diastolic pressure in aorta (to open the semi-lunar valves)

Contractility: inotropic effect

(edited version)

This sheet is dedicated to Aseil & Rashid… :*3*