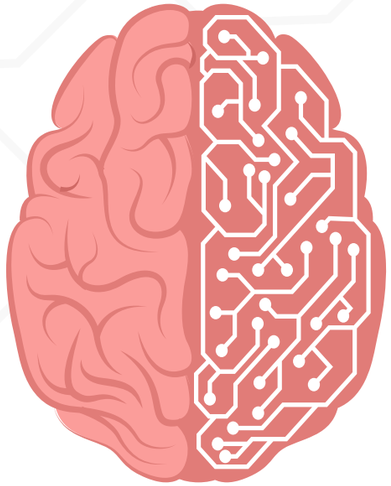
النادي الطلابي
كلية الطب



Physiology - CVS

Done By

Ahmad Ayman & Islam Bilal

Corrected By

Dana Tarawneh

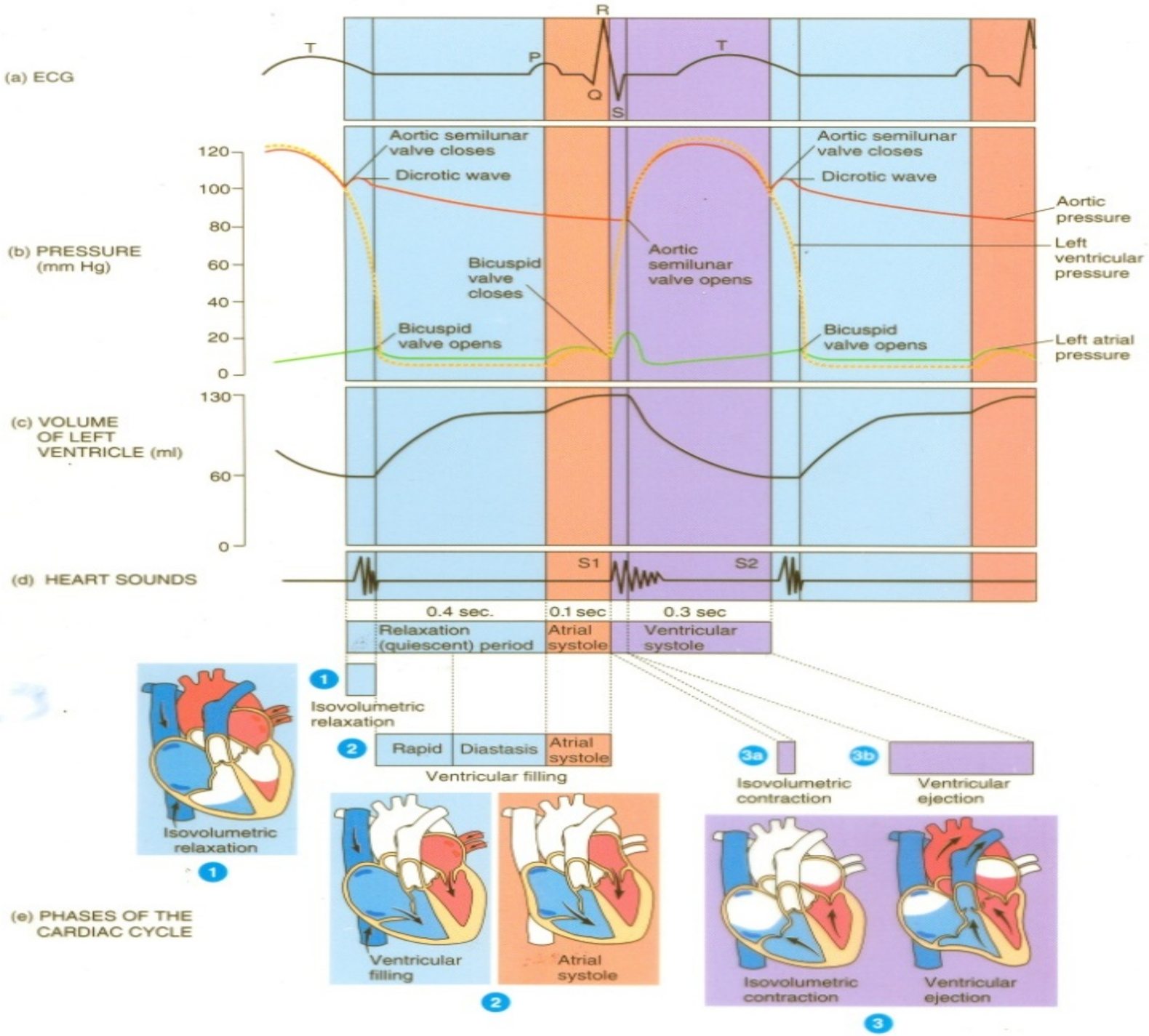
Heart Pump and Cardiac Cycle

Faisal I. Mohammed, MD, PhD

Remember that this subject is very important, you should understand it carefully because this is what you'll be seeing when you go to a patient and see what happens in his heart during one cycle.

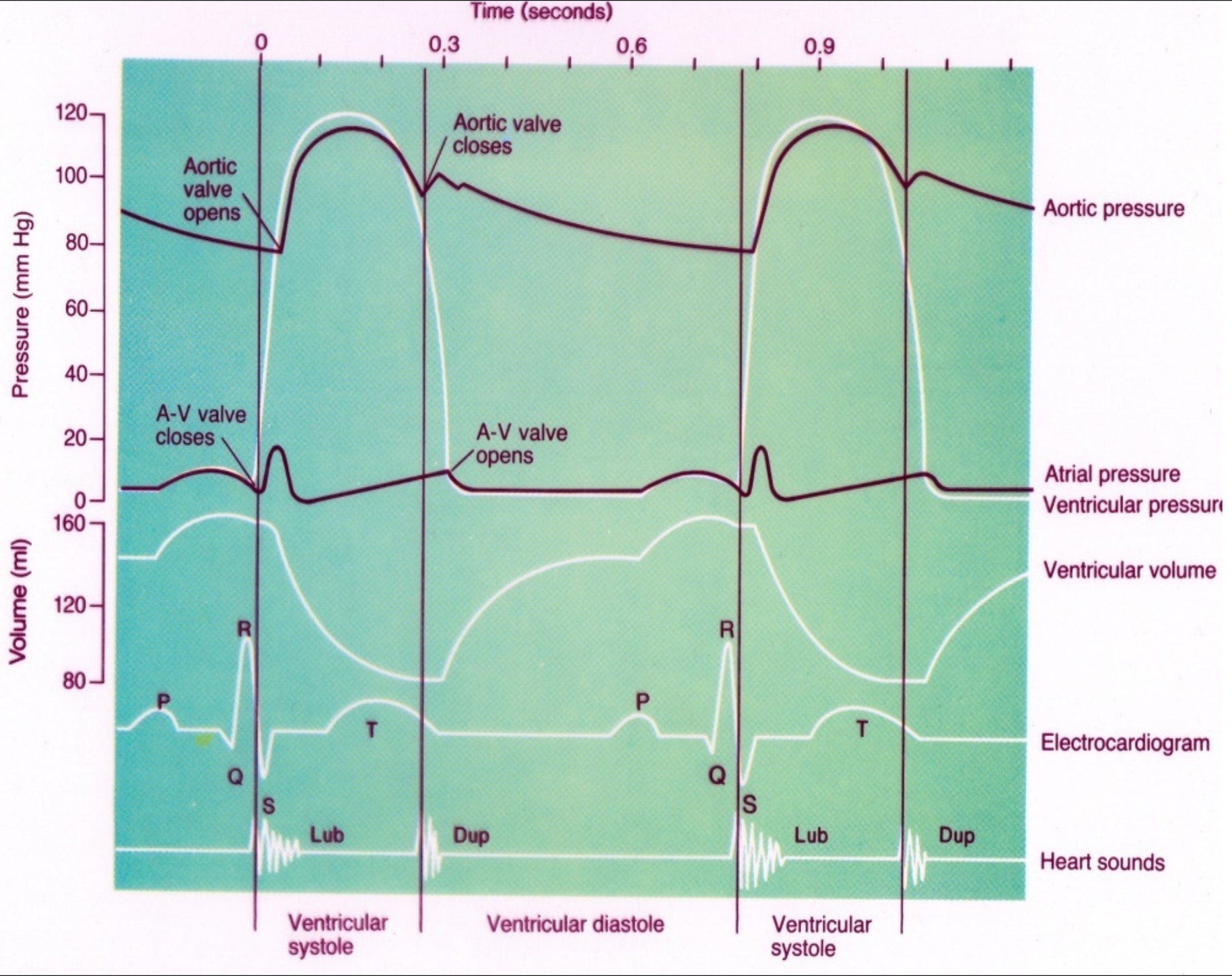
Objectives

- To understand the volume, mechanical, pressure and electrical changes during the cardiac cycle
- To understand the inter-relationship between all these changes
- To describe the factors that regulate Cardiac output and Stroke volume.
- Resources: Textbook of Medical Physiology By Guyton and Hall



Explanation of this figure is in slides 6-11.

Explanation of this figure is in slides 6-11.



- It's important to remember these points (from the ECG lectures) :

1. If the time of one cardiac cycle is 0.8 sec, then the heart rate is 75 beats per minute (BPM).
2. If the cardiac cycle time is 0.6 sec, the heart rate is 100 BMP.
3. If the cardiac cycle time is 1 sec, then the heart rate is 60 BPM.

So, we conclude that heart rate = 60 s / the time of one cardiac cycle .

- Also, remember that :

1. we can get the time of one cardiac cycle from an ECG through calculating the RR interval time; if the RR interval is 0.8 seconds, then the heart rate is 75 (an RR interval of 0.8 seconds means 20 small square ($20 \times 0.04 = 0.8$) & for learning purposes, we consider the heart rate to be 75 and hence the cardiac cycle time is 0.8 sec).
2. The normal ECG has the following components :
   a. The P wave which represents the atrial depolarization
   b. The interval which represents the delay of conduction at the AV node
   c. The QRS complex which represents the ventricular depolarization.
   d. The QT interval which represents, the time of the ventricular depolarization and repolarization combined.
   e. The T wave which represents ventricular repolarization
   f. Another P wave and the cycle repeats.

- The ECG gives us the electrical changes that occur in the cardiac cycle, but how about the mechanical changes ?

  1. Just after the P wave (atrial depolarization) ends, atrial systole (contraction) occurs. Atrial systole takes a time of 0.1 sec, it is represented in orange in slide #4.
  2. Then atrial diastole begins, and it continues through the whole cycle until another P wave comes, so atrial diastole takes 0.7 sec.
  3. The ventricular systole (represented in purple) begins just after the ventricular depolarization ends (just after the QRS complex is recorded), remember that the atrial repolarization (after which the atrial diastole begins) occurs at the same time with ventricular depolarization. hence, ventricular systole occurs after the end of the atrial systole and the beginning of atrial diastole. So, atrial and ventricular systoles should never occur at the same time. The ventricular systole takes 0.3 sec (it continues until the ventricular repolarization, recorded by the T wave).
  4. Then ventricular diastole occurs, and it continues until the next cardiac cycle. So, the ventricular diastole takes 0.5 sec. Note that during the next atrial systole, the ventricular diastole is not finished yet (that's why ventricular diastole takes 0.5 sec instead of 0.4 sec).

- To conclude;
  1. Atrial and ventricular diastoles could occur at the same time, but atrial and ventricular systoles never do so.
  2. Normally, atrial and ventricular diastoles overlap for about 0.4 sec, this is known as the relaxation period.

- Now we're done with the mechanical changes, let's talk about the changes in volume and pressure. Regarding ventricular volume changes, notice that whether you're talking about the left ventricle or the right ventricle, the volume changes are the same in both.

Once the ventricular diastole begins, the AV (tricuspid and mitral) valves open, and the filling phase starts.
Before the atrial systole starts, the volume of blood in the left ventricle reaches up to 100ml. Because of atrial systole (remember that it occurs while the ventricles are still relaxed) there might be an increase in the volume up to 125 ml, so the atrial systole does not contribute to the volume of the ventricles more than 25%. Why is that ?
This is because the filling has started before the atrial systole & as we mentioned earlier, the AV valves open from the beginning of the ventricular diastole. So, during ventricular diastole, the blood that comes to the atria through the great veins goes to the ventricles right away and no force is needed, but when the atria contracts, certain amounts of blood is pushed from the atria to the ventricle a little bit faster.

The volume that we have reached (125 ml) is the volume of blood that is found in the ventricles at the end of the ventricular diastole → it is called the end diastolic volume (EDV).

Now let's discuss the changes in pressure and then we'll return to the changes in the volume.

The changes in volume (the filling and the evacuation) are driven by the changes in pressure.

We reached to a point where the ventricles are filled (at the end of the ventricular diastole), notice that during the ventricular diastole, the pressure in the aorta is around 80 mmHg, however, the pressure in the left ventricle during the diastole is almost zero (it is necessary that the pressure in the ventricle equals zero during diastole in order to facilitate the flow of blood from the atria - which has a little bit higher pressure- to the ventricles).

But when the atria contracts during the ventricular diastole, the pressure in the left ventricle increases a little bit to about 5 mmHg, while the aortic pressure does not change during atrial systole, but once the atrial systole ends and the ventricular systole starts, the pressure in the left ventricle starts to increase.
Once the pressure in the left ventricle exceeds that in the left atrium, the mitral valve closes → at this point, the aortic semilunar valve is still closed because the pressure in the left ventricle hasn't yet exceeded that of the aorta (80 mmHg), so at this point:

1. The four valves (both the atrioventricular and semilunar valves) are closed.
2. The volume of the ventricle is constant.
3. The ventricles are contracting.

This causes a sharp increase in the pressure of the ventricle without any change in the volume & this is known as the isovolumic contraction.

This isovolumic contraction increases until the pressure in the left ventricle exceeds that of the aorta. Once that happens, the aortic semilunar valve opens, and the blood is pumped from the left ventricle to the aorta. Once the blood reaches the aorta, the pressure in the aorta starts to increase, but it remains less than that of the left ventricle by a small amount (around 1 mmHg).
At the end of the ventricular systole (before the diastole), the ventricular pressure becomes less than that of the aorta by a little bit, but blood continues to flow at this point due to the momentum زخم of the blood.

The highest pressure in the left ventricle during systole reaches around 120 mmHg, while the highest pressure that the aorta reaches during systole is around 118 mmHg (but we consider it to be 120 mmHg for measuring purposes).

So, when we measure the aortic pressure from the arm, it is 120 mmHg (the systolic aortic pressure is 120 mmHg, and the diastolic pressure in 80 mmHg).

Once the pressure of the left ventricle exceeds that of the aorta and the aortic semilunar valve opens, the volume of blood in the left ventricle decreases sharply → this is known as fast-rapid ejection.
Note that the ejection that occurs due to the momentum of blood is called slow ejection. After this ejection, about 55 ml of blood remains in the left ventricle & and this volume, that is the volume of blood at the end of the ventricular systole is called the end systolic volume.

As the ventricular diastole starts & the pressure in the left ventricle continues to decrease, the semilunar valve closes & at this point again, the four valves are closed because the AV valves are still closed. This gives us a short period of isovolumic relaxation, after which the pressure in the ventricles becomes less than that of the atria and the AV valves open, the pressure of the aorta also decreases during the ventricular diastole from 120 mmHg to 80 mmHg. BUT just at the moment when the aortic valve closes and because the blood is trying to return to the ventricle, the blood dodges the aortic valve, so the aortic pressure increases a little bit, this sudden increase is known as the dicrotic notch or the incisura.

As we mentioned earlier, once the AV valve opens, the blood can flow freely from the great veins → the atria → the ventricles. This causes a rapid filling of blood which then becomes slow, and it's called slow filling (diastasis).

Then the atrial systole occurs, which pumps a small amount of blood rapidly into the ventricles (remember that filling of the ventricles is not much dependent on atrial contraction) and the cycle resumes.

Considering a complete cardiac cycle :

1. EDV is the largest volume in the cardiac cycle, and it is the volume of blood in the ventricles at the end of the ventricular diastole (almost 125 ml).
2. ESV is the smallest volume in the cardiac cycle, and it is the volume of blood at the end of the ventricular systole (almost 55 ml).

The difference between these two values (the volume of blood pumped from the ventricles) is called the stroke volume. Stroke volume = EDV – ESV . The stroke volume represents the amount of blood that has been ejected from the left or the right ventricles per beat.

Another important concept is the cardiac output, which is the amount of blood ejected from the left or the right ventricles per minute.
Cardiac output (CO) equals the stroke volume multiplied by the heart rate. CO = HR × SV

Example: If the end diastolic volume is 125 ml and the end systolic volume is 55 ml, then the stroke volume is 70 ml, and if the heart rate is 75 BPM, then the cardiac output is almost 5 liters per minute.

Now let's come to the changes in the heart sounds

The 1st heart sound is heard just after the beginning of ventricular systole, that is, when the pressure in the ventricle becomes higher from that in the atria so blood tries to go to the atria but it dodges the closed valve, so this sound appears due to the turbulence of blood around the closed AV valves. This sound is referred to as S1 or the "lup". This sound is a high-pitch sound.
The 2nd sound appears when the semilunar valves close, that is at the end of the ventricular systole in which blood tries to return form the aorta and the pulmonary trunk to the ventricles, but it faces the closed valve. The turbulence of blood around the closed semilunar valves causes the second sound that is S2 or the "dup". it is a low-pitch sound.
The 3rd sound (S3) can be heard due to the rapid flow of blood from the atria to ventricles when the AV valves open, this sound is only heard by people who are talented in music.
The 4th sound (S4) might be heard also at the atrial systole due to the rapid flow of blood.

Notice that the S1 appears just after the QRS while S2 appears just after the T wave

The time between S1 and S2 almost equals the time of the ventricular systole & the time between the S2 and the next S1 is the time of the ventricular diastole.

Remember that if you want to hear these sounds, the time between them is very short :

1. 0.3 sec between S1 and S2.
2. 0.5 seconds between S2 and the next S2.

You must be relaxed to notice that these are two sounds not one.

Regarding the right ventricle and the pulmonary trunk :

1. Same changes in volume with the same number
2. Different changes in pressure (you can see from the figure in earlier slides that the curve is the same, except that the scale is different. The pressure in the pulmonary trunk during ventricular diastole is almost 8 mmHg (compare this to 80 mmHg in the aorta), and the systolic pulmonary pressure is almost 25 mmHg (compare this to 120 in the aorta). The pressure in the right ventricle is zero during diastole and just above 25 mmHg during systole.

The time of ventricular diastole is very important; it is where the ventricles are filled with the blood, so if this time shortens, it means that the amount of blood that is going to fill the ventricle becomes less.

When is the time shortened ?
The time is shortened when there is an increase in the heart rate.
If the heart rate is 100 BPM, the time for the cardiac cycle is 0.6 seconds. In this case, the main shortening here would be in the diastole, the ventricular systole in this case would be 0.29 seconds instead of 0.3, and the diastole would be 0.31 seconds , so the main shortening is at the expense of the diastole.

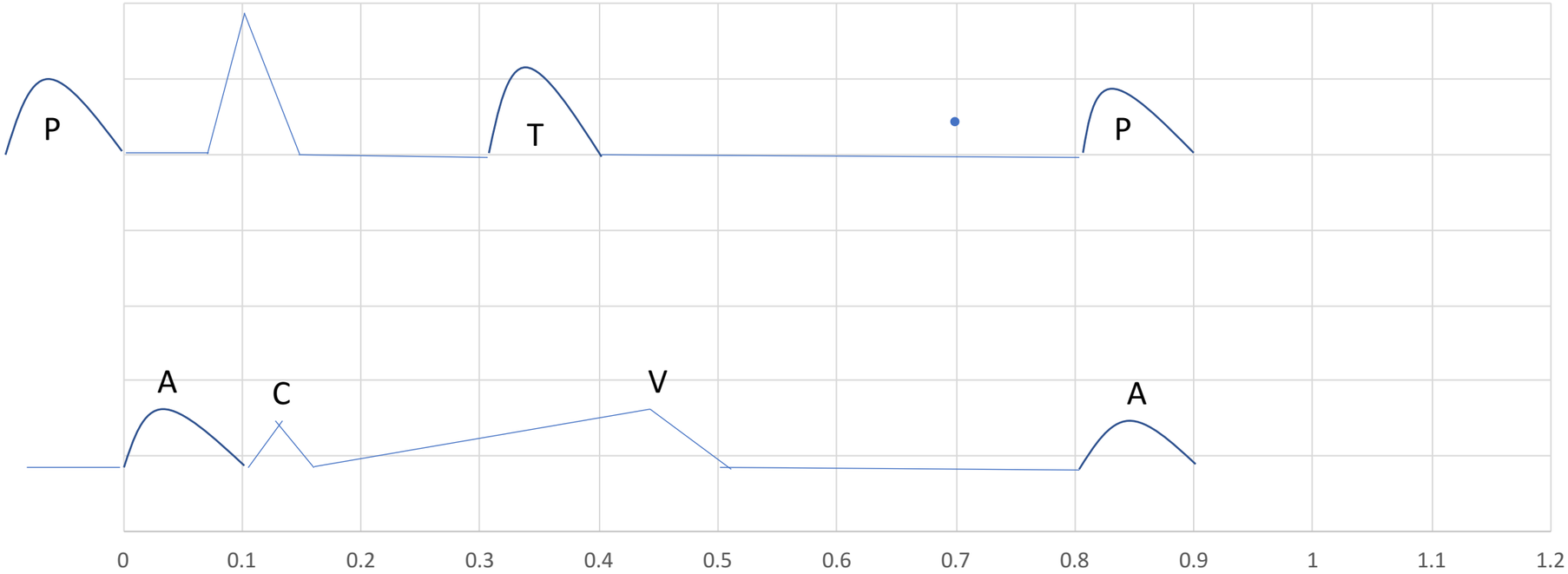
Thus, if the heart rate increases, the time for the diastole is shortened & the filling time is shortened, so the amount of blood that is found at the end of diastole is less which means that the stroke volume is less. But what might compensate this for the cardiac output is the heart rate, because the CO = HR X SV.

if you want to study the changes of pressure in the atria, it would be as the following :

1. During the atrial diastole, the atrial pressure is zero.
2. Once atrial systole occurs, the atrial pressure increases & this causes a wave in the curve known as the A wave.
3. After the atrial systole ends and the ventricular systole starts, the ventricular pressure pushes the atrioventricular valves toward the atria, causing a little increase in the atrial pressure. This increase causes the C wave. Remember that valve prolapse is prevented by the papillary muscles.
4. After that, filling of the atria from the great vein starts, which increases the pressure again due to compliance, then at the end of the isovolumic relaxation, the AV valves open and the atria ejects blood into the ventricles, causing their pressure to drop to zero and the cycle resumes, the wave that appears due to this increase and then decrease is called the V wave.

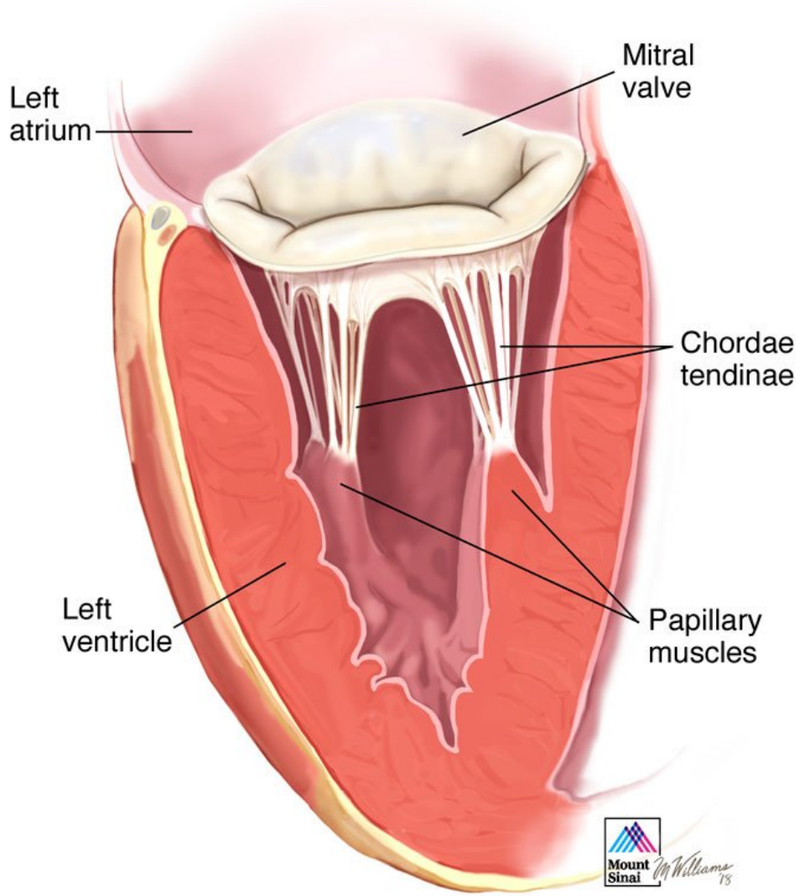
So, A wave is due to atrial systole, C wave is due to ventricular systole, and V is due to the filling and then opening of the AV valves.

This is important because it has a diagnostic value. In AV valve stenosis, you would see that the A wave is peaked (high). In this condition, blood might even leave the right atrium though the valveless superior vena cava causing jugular venous distention. The C wave is peaked in mitral or tricuspid stenosis.



However, if the mitral or tricuspid valves are insufficient, blood will return from the ventricle to the atria during ventricular systole causing the appearance of another wave (not C wave).

What actually prevents the AV valves from prolapsing into the atria during the ventricular systole is the papillary muscles. Those are connected to the valve cusps through tendinous cords known as the chordae tendinea. The papillary muscles are part of the ventricular musculature & these papillary muscles contract during the ventricular systole, so that they pull the valve cusps into the ventricles, preventing their prolapse into the atria so that the valve is stabilized & closed through the push of the ventricular pressure and the pull of the papillary muscles.



Cardiac Cycle

- Cardiac cycle refers to all events associated with blood flow through the heart
  - Systole – contraction of heart muscle
  - Diastole – relaxation of heart muscle

Cardiac Cycle

- Atrial systole 0.1 second
- Atrial diastole 0.7 second
- Ventricular systole 0.3 second
  - Isovolumic contraction 0.01 seconds
  - Rapid ejection period
  - Slow ejection period
- Ventricular diastole 0.5 seconds
  - Isovolumic relaxation 0.02 seconds
  - Rapid filling
  - Slow filling (Diastasis)
  - Atrial contraction

Cardiac cycle …cont

- End diastolic volume (EDV) – End systolic volume (ESV) = Stroke volume (SV)
- SV x Heart rate (HR) = cardiac output (CO)
- Inotropic vs. Chronotropic
- Autonomic control of cardiac cycle (pump)
- Ejection fraction = SV/EDV . The ejection fraction is a measure of the efficiency of the heart & it's calculated as a percentage, the normal EF ranges between 55-75 or 80%, if you remember, in the very first lecture, we talked about a patient with cardiac failure that needed a cardiac transplant because the efficiency of his heart was low, what is the efficiency?

The efficiency is referred to as the ejection fraction which equals SV/EDV. Remember that:

1. SV = EDV – ESV
2. The term chronotropic refers to the heart rate while inotropic refers to the contractility.

Ejection fraction in fact is a measure of contractility, when the contractility increases → the ejection fraction increases and increasing contractility means increasing the stroke volume with a fixed increase in the end diastolic volume.

Phases of the Cardiac Cycle

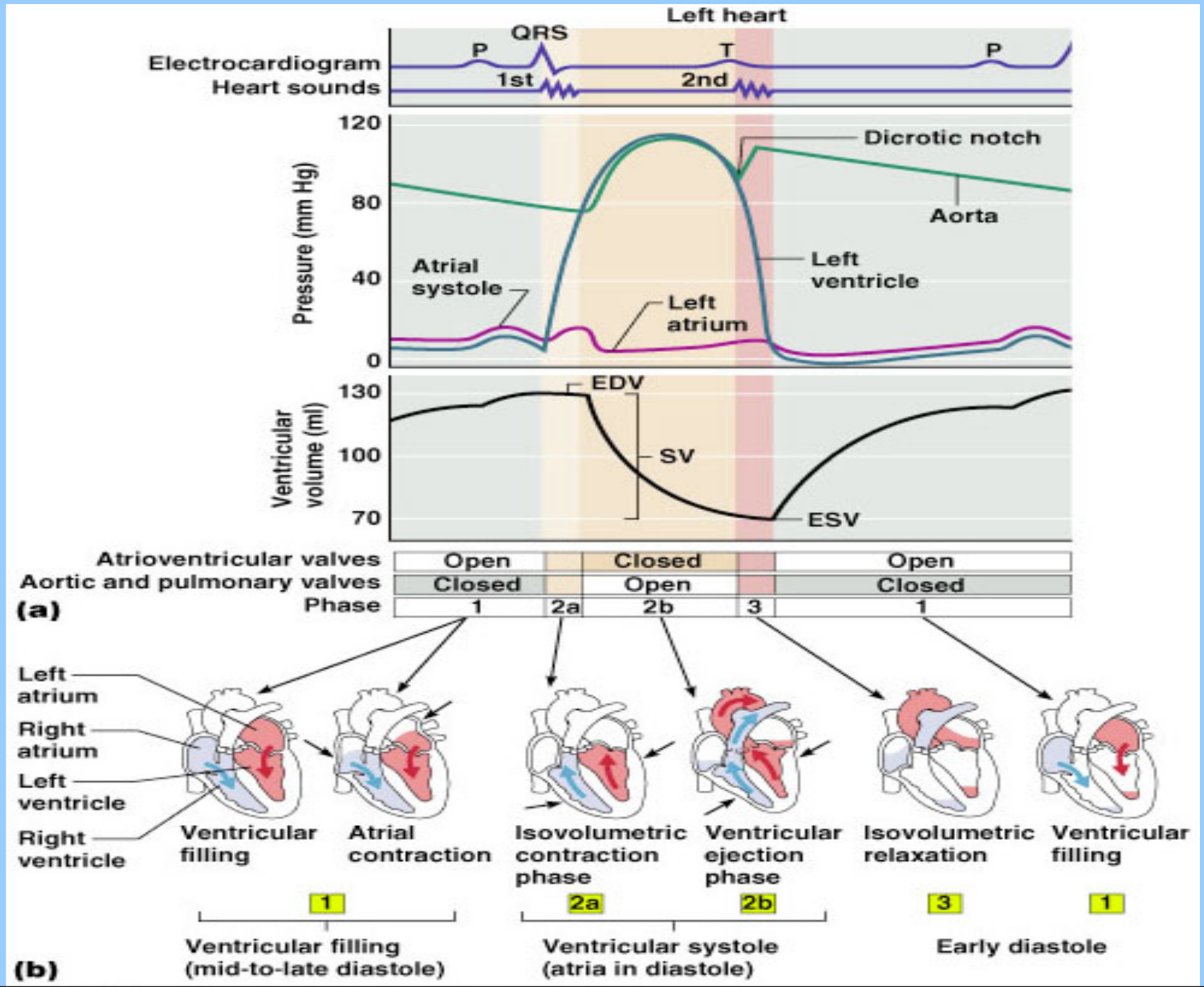
- Ventricular filling – mid-to-late diastole (early diastole is isovolumic relaxation)
  - Heart blood pressure is low as blood enters atria and flows into ventricles
  - AV valves are open, then atrial systole occurs

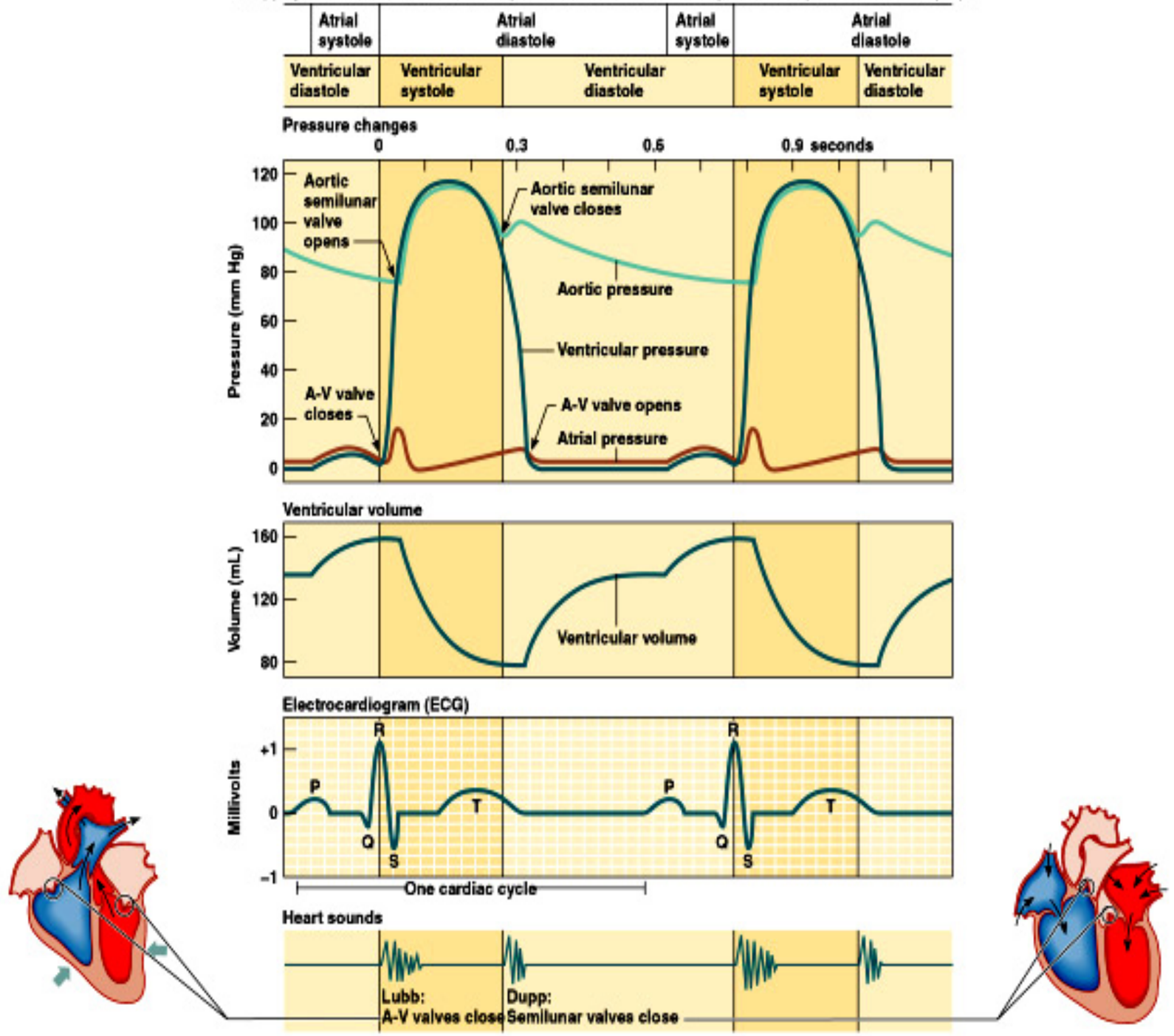
Phases of the Cardiac Cycle

- Ventricular systole
  - Atria relax
  - Rising ventricular pressure results in closing of AV valves
  - Isovolumetric contraction phase
  - Ventricular ejection phase opens semilunar valves

Phases of the Cardiac Cycle

- Isovolumetric relaxation – early diastole
  - Ventricles relax
  - Backflow of blood in aorta and pulmonary trunk closes semilunar valves (remember that this backflow results in a sudden increase in the aortic pressure known as the incisura or the dicrotic notch).
- Dicrotic notch – brief rise in aortic pressure caused by backflow of blood rebounding off semilunar valves

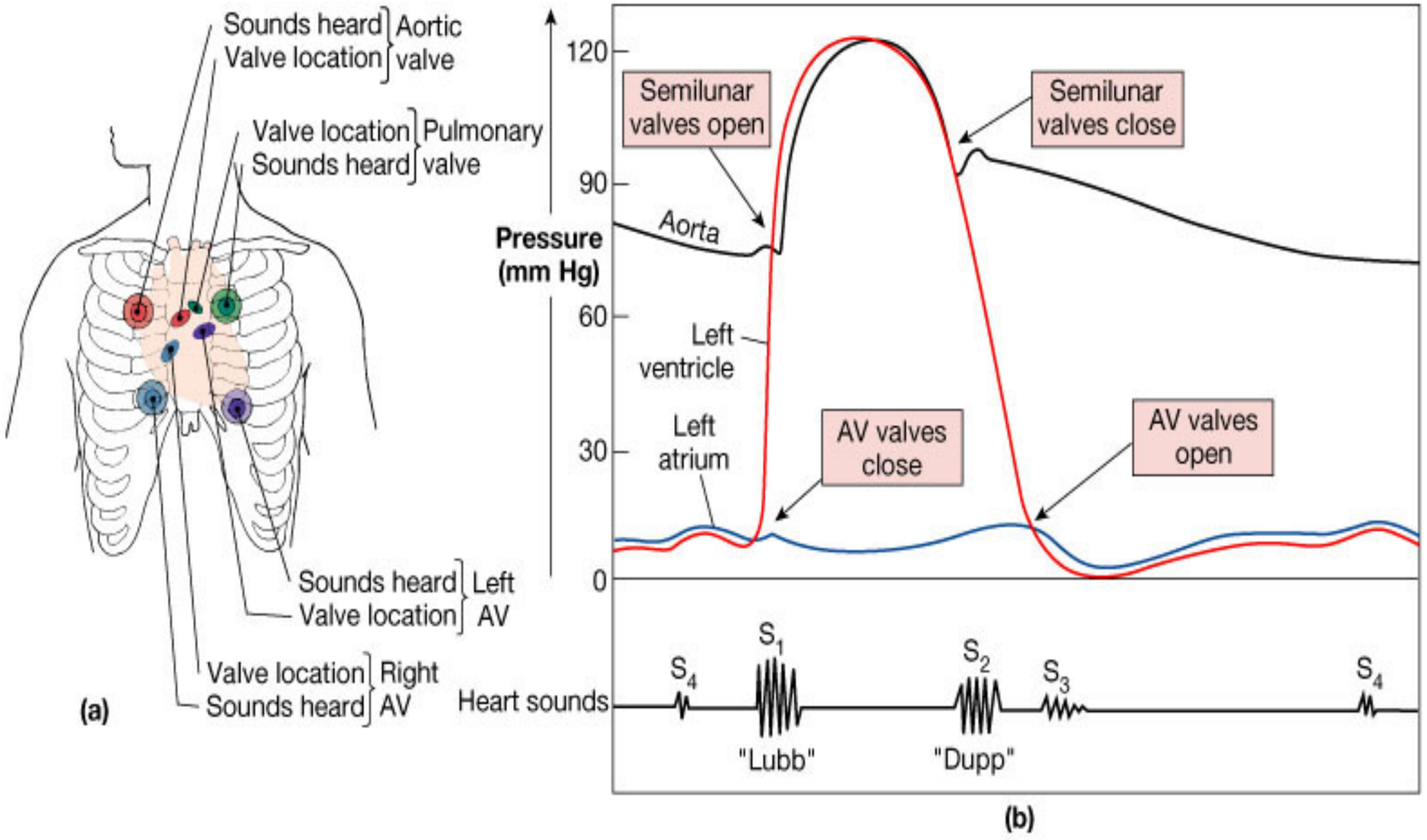

Left heart
Electrocardiogram
Heart sounds
QRS
P
T
P
1st
2nd
Pressure (mm Hg)
120
80
40
0
Dicrotic notch
Aorta
Atrial systole
Left ventricle
Left atrium
Ventricular volume (ml)
130
100
70
EDV
SV
ESV
Atrioventricular valves
Aortic and pulmonary valves
Phase
Open
Closed
Closed
Open
Open
Closed
1
2a
2b
3
1
(a)
Left atrium
Right atrium
Left ventricle
Right ventricle
Ventricular filling
Atrial contraction
Isovolumetric contraction phase
Ventricular ejection phase
Isovolumetric relaxation
Ventricular filling
1
2a
2b
3
1
Ventricular filling (mid-to-late diastole)
Ventricular systole (atria in diastole)
Early diastole
(b)



Changes during Cardiac cycle

- Volume changes: End-diastolic volume, End-systolic volume, Stroke volume and Cardiac output.
- Aortic pressure: Diastolic pressure ~80 mmHg, Systolic pressure ~ 120 mmHg, most of systole ventricular pressure higher than aortic
- Ventricular pressure: Diastolic ~ 0, systolic Lt. ~120 Rt. ~ 25 mmHg.
- Atrial pressure: A wave =atrial systole, C wave= ventricular contraction (AV closure), V wave= ventricular diastole (Av opening)
- Heart sounds: S_1 = turbulence of blood around a closed AV valves, S_2 = turbulence of blood around a closed semilunar valves.

Heart Sounds

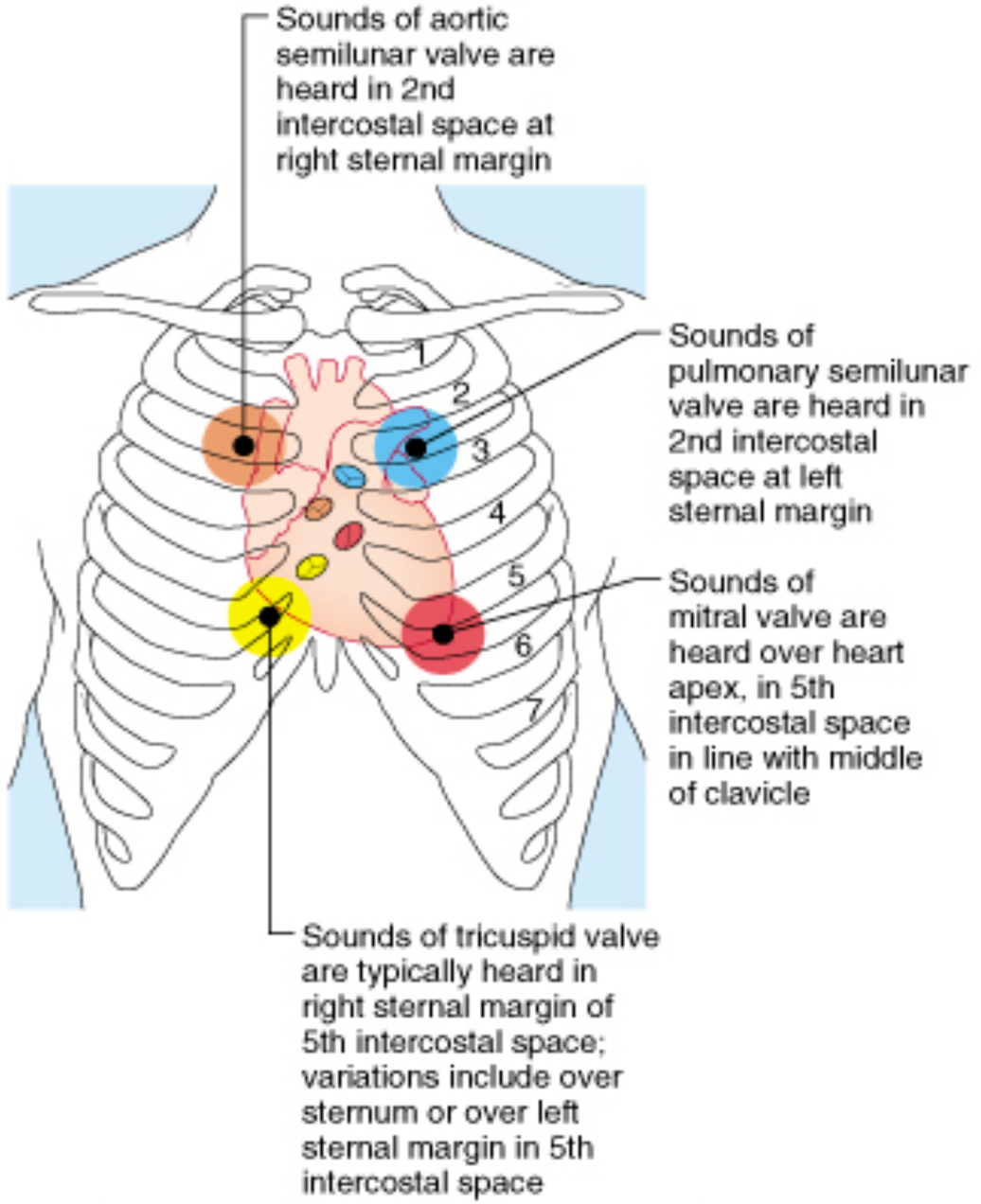


The figure on the left illustrates where you hear the heart sounds. You hear the 2nd heart sound of the aortic valve through putting the stethoscope on the right 2nd intercostal space just parasternal, and the 2nd heart sound of the pulmonary valve is heard in the left 2nd intercostal space parasternal.

The 1st heart sound of the mitral valve can be heard on the left 5th intercourse surface intercostal space around the apex of the heart in the midaxillary line, while the sound of the tricuspid valve (right AV valve) is heard in the right 5th intercostal space on the parasternal line.

Heart Sounds

- Heart sounds (lub-dup) are associated with closing of heart valves



Heart sounds

- 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

Few notes :

- S2 split (normally in children) pathologically in adult

Occurs due to delay in right ventricular emptying causing aortic valve closure occurs before pulmonary valve

- Diastolic murmur —cause: blood move from aortic artery to left ventricle
- Systolic murmur—blood move from right ventricle to right atrium

S1 …(ventricular systole)… S2 …(ventricular diastole … S1

If we hear a sound here we call it systolic murmur

If we hear a sound here we call it diastolic murmur

Cardiac Output (CO) and Reserve

- CO is the amount of blood pumped by each ventricle in one minute
- CO is the product of heart rate (HR) and stroke volume (SV)
- HR is the number of heart beats per minute
- SV is the amount of blood pumped out by a ventricle with each beat
- Cardiac reserve is the difference between resting and maximal CO

- The cardiac output is normally 5 liters, but you might increase your cardiac output to 15 liters at maximal simulation.
- The difference between the maximum cardiac output (let's consider it 15 liters) - the resting cardiac output (which is 5 liters) means that the cardiac reserve is 10 liters.
- The cardiac reserve is the difference between the maximum cardiac output and the resting cardiac output, so if you can increase your cardiac output up to 15 liters maximally, your cardiac reserve is 10 liters. Athletes might increase their cardiac out up to 35 liters, so their cardiac reserve is very high.

Cardiac Output: Example

- CO (ml/min) = HR (75 beats/min) x SV (70 ml/beat)
- CO = 5250 ml/min (5.25 L/min)

Ejection Fraction

- End diastolic volume = 125 ml
- End systolic volume = 55 ml
- Ejection volume (stroke volume) = 70 ml
- Ejection fraction = 70ml/125ml = 56% (normally 60%)
- If heart rate (HR) is 70 beats/minute, what is the cardiac output?
- Cardiac output = HR * stroke volume = 70/min. * 70 ml = 4900ml/min.

Ejection Fraction (cont'd)

- If HR =100, end diastolic volume = 180 ml, end systolic vol. = 20 ml, what is the cardiac output?
- C.O. = 100/min. * 160 ml = 16,000 ml/min.
- Ejection fraction= 160/180%=~ 90%

Regulation of Stroke Volume

- SV = end diastolic volume (EDV) minus end systolic volume (ESV)
- EDV = amount of blood collected in a ventricle during diastole
- ESV = amount of blood remaining in a ventricle after contraction

Factors Affecting Stroke Volume

- Preload – amount ventricles are stretched by contained blood (before it contracts) -it increases SV-

The amount of stretch of the ventricle before it contract is a proportion to the amount of the blood, and this is the EDV.
This is proportional to the resting tension or passive tension. The anesthetist would like to talk about EVP because they can measure the pressure rather than volume.

- Contractility – cardiac cell contractile force due to factors other than EDV – it increases SV-

If there is an increase in the diastole you fix EDV and change the SV.
positive contractility (+ inotropic effect) it means the EDV stays the same and you increase the SV. This means that there is an increase in contractility (+ inotropic effect).
In negative inotropic effect, the EDV stays the same and the SV decreases.

- Afterload – back pressure exerted by blood in the large arteries leaving the heart –it decreases SV-

(Diastolic Blood Pressure) The pressure that the ventricle must develop over the semilunar valve , so it must open the semilunar valve, in case of the left → it's the diastolic pressure in the aorta & in case of the right → it is the diastolic pressure in the pulmonary artery.

Frank-Starling Law of the Heart

- Preload, or degree of stretch, of cardiac muscle cells before they contract is the critical factor controlling stroke volume

Within physiological limits, increasing in the preload increases SV, and increasing the resting tension or the length of muscle increases the force of contraction that increases the ejection of blood thus increases the SV.

Here we don't measure the length of the muscle, it's proportional to the amount of blood that is found in the ventricle before it contracted.

- Slow heartbeat and exercise increase venous return to the heart, increasing SV

When the heartbeat is slow, there is more filling-time, so it increases EDV thus SV increases.

- Blood loss and extremely rapid heartbeat decrease SV

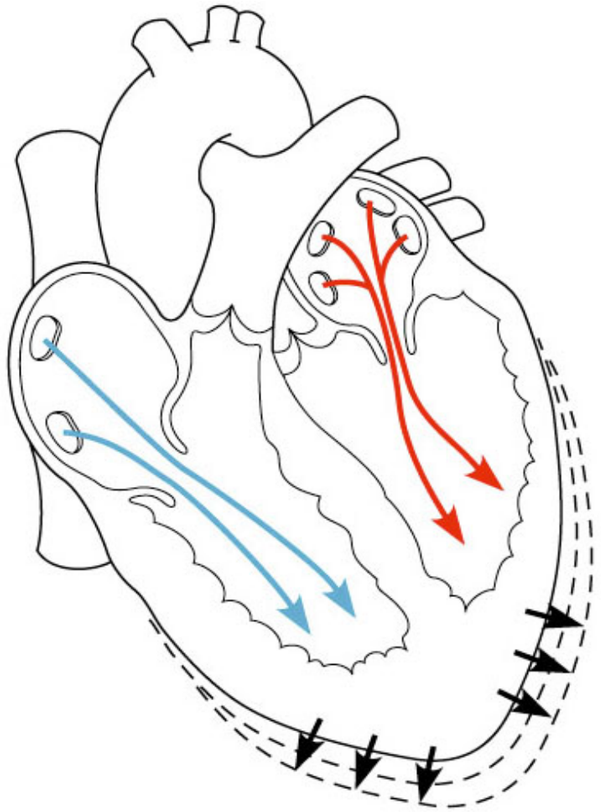
Rapid heartbeat → shortened filling-time → SV decreases.

Frank-Starling Mechanism

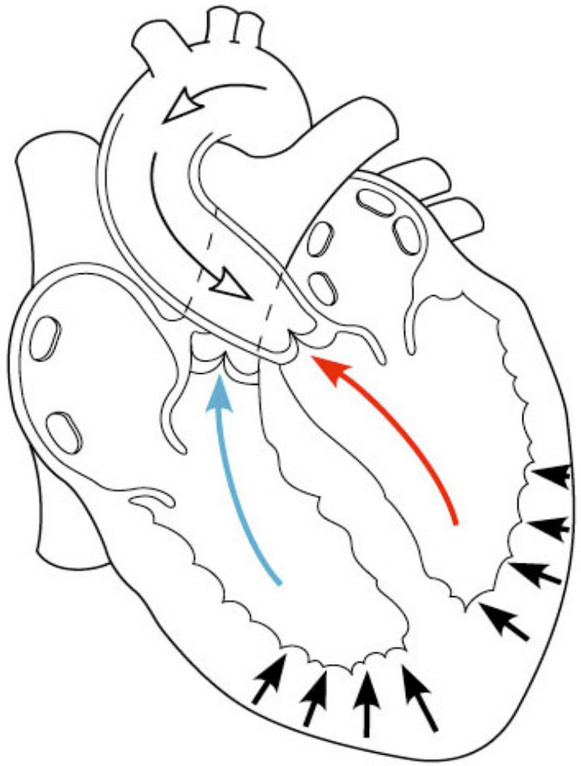
- Within physiological limits the heart pumps all the blood that comes to it without excessive damming in the veins.

- Extra stretch on cardiac myocytes makes actin and myosin filaments interdigitate to a more optimal degree for force generation.

Preload and Afterload



(a) Preload



(b) Afterload

The heart is filled with blood before it contracts, and remember, the higher the preload is, the higher is the SV .

The higher the amount of blood that stays in the ventricle before it contracts (this is proportional to the length / passive tension) the higher is the SV.

How much pressure must we exert in the right or left atrium to open the semilunar valve? if the pressure in the semilunar valve before it contracted was higher, which is the diastolic volume, it means that the SV will be less because most of the force will be imparted just to open the semilunar valve.