



CVS PHYSIOLOGY



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الدكتور: فاطمة ريلات

Cardiovascular Physiology

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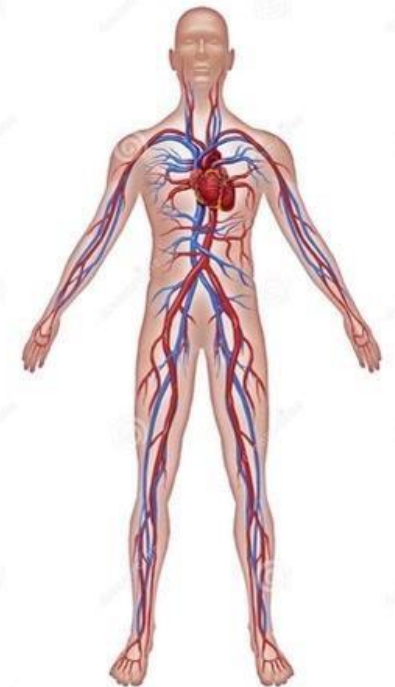
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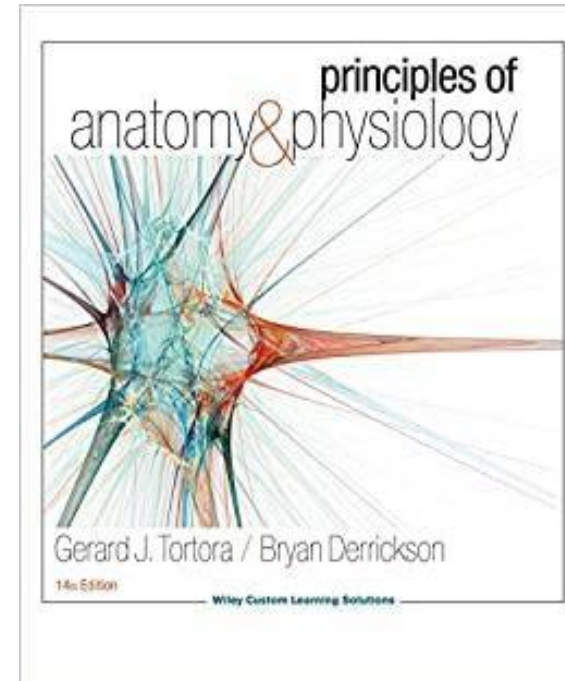
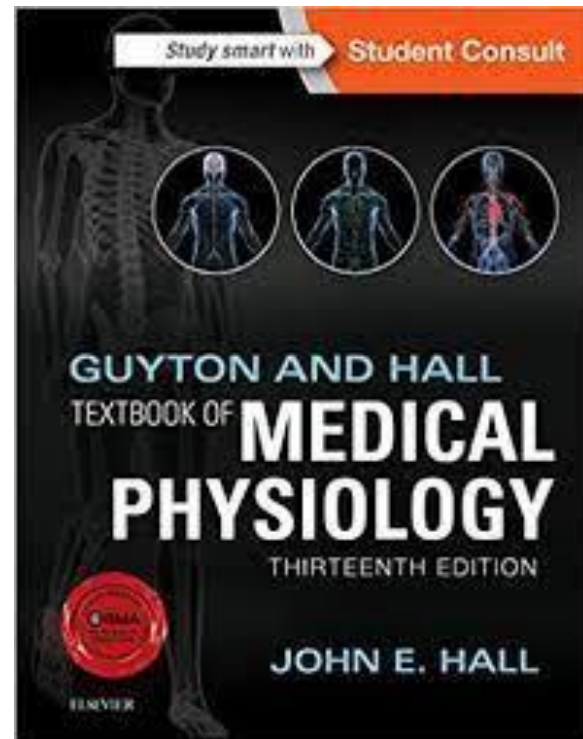
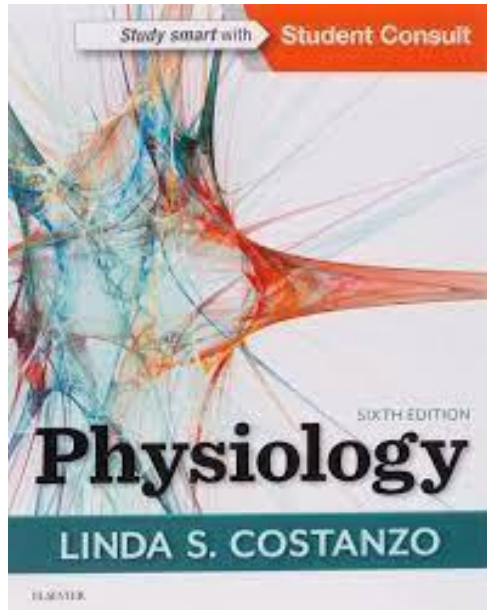
Doctor

Additional info

Important



References



9TH
Edition

Human Physiology

From Cells to Systems

Lauralee Sherwood
Department of Physiology and Pharmacology
School of Medicine
West Virginia University

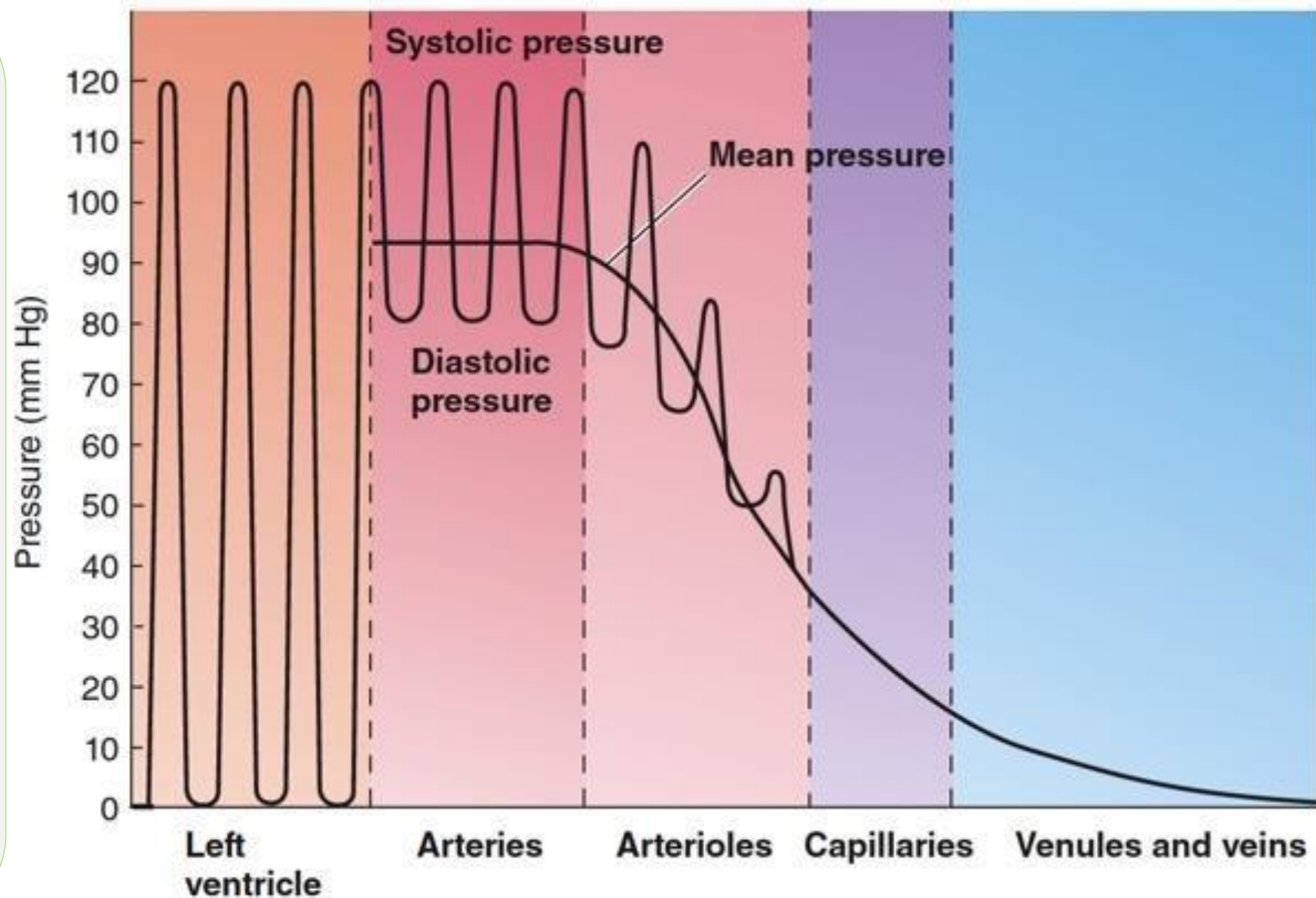
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Vascular hemodynamics-3

- The difference in pressure in major arteries is minimal; because the resistance is very low.
- The systolic, diastolic, pulse and mean blood pressure is almost the same among different large arteries.
- The largest pressure gradient (largest decrease in pressure) is in **arterioles** → have the highest resistance
- The pulsatility of the pressure decreases as we go from arterioles to capillaries, resulting in a non-pulsatile flow (continuous flow)



Arteries

- Because arteries offer little resistance to flow, only a negligible amount of pressure energy is lost in them because of friction.
- Therefore, arterial pressure—systolic, diastolic, pulse, or mean—is almost the same in all arteries.

Pulsation of arterial pressure

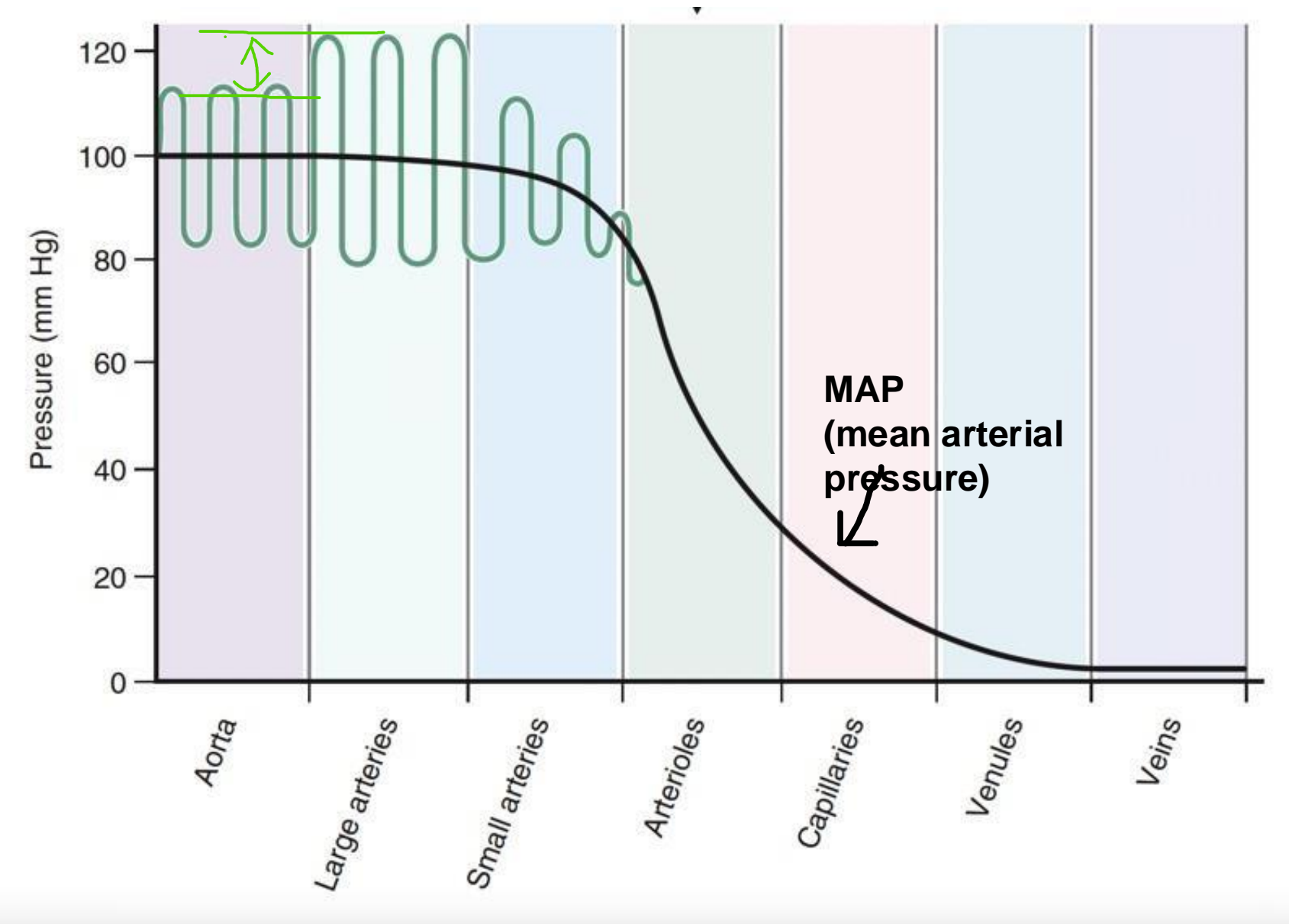
- pulsations of arterial pressure reflect the pulsatile activity of the heart.
- Each cycle of pulsation in the arteries coincides with one cardiac cycle.

Pulse pressure

- Pulse pressure is the difference between systolic pressure and diastolic pressure.
- Two major factors affect the pulse pressure:
 - (1) the stroke volume
 - (2) the compliance of the arterial tree.
 - The compliance is the ability of the vessel to distend and accommodate the volume of blood causing less increase in the pressure.
 - The pressure is the force acting on the vessel wall, so when the vessel distend the force that act on it will be less which will result in lower pressure increase.
 - The compliance term is applied more to the venous side.

Pulse pressure

- In general, the greater the stroke volume output, the greater the amount of blood that must be accommodated in the arterial tree with each heartbeat and, therefore, the greater the pressure rise and fall during systole and diastole, thus causing a greater pulse pressure.
- Conversely, the less the compliance of the arterial system, the greater the rise in pressure for a given stroke volume of blood pumped into the arteries.



The component of the vessel will change from the aorta downstream:

- The aorta : have the highest elastic content but less muscular content.
- The vessels downstream : the elastic part will decrease, and the smooth muscle content will increase.

The pulse pressure in aorta is less than the pressure in the large arteries, why? 🤔

- When the stroke volume is ejected from the left ventricle to the aorta, the high energy of ejection of blood will cause 1. distention of the wall of the proximal aorta, and 2. it will cause pressure waves across the vessel wall caused by the energy of the ejection of the blood that is stored by the walls of the aorta.
- So, there are two sources of pressure in the large arteries here: 1. the pressure waves 2. the blood volume pressing on the wall
- These pressure waves will move with higher velocity than the blood; because of the inertia in blood. (inertia : *القصور الذاتي*)
- pressure augmentation: pressure addition.
- First, the pressure waves will move forward across the walls of the arteries faster than the blood, but the blood will catch the pressure waves at some point and it will be augmented with the blood pressure, that's why we see higher systolic pressure at the arteries compared to the aorta. In addition to that, when the pressure waves reach branching point in the arteries, the change in the diameter (because of branching) will send back another pressure wave called "retrograde waves" and that will also be augmented with the large arteries pressure.
- So basically, aortic pressure = blood pressure only, but the large arteries pressure = blood pressure + pressure waves + retrograde wave pressure.
- Try watch the video in the additional resources (last slide) for a better understanding of pressure waves.

Pulsations in large arteries

- pulsations in large arteries are even greater than the pulsations in the aorta.
- following ejection of blood from the left ventricle, the pressure wave travels at a higher velocity than the blood itself travels (due to the inertia of the blood), augmenting the downstream pressure.
- Furthermore, at branch points of arteries, pressure waves are reflected backward, which also tends to augment pressure at those sites.

- But something doesn't make sense! How does the blood flow from low to high pressure?
- We said before that for the blood to establish a constant flow, we need to maintain a pressure difference where the starting point is higher.
- But here the aorta have a lower pressure than large arteries, is this contradicted?
- Is reality, no. why?
- The mean arterial pressure is the key →
- the mean pressure in the aorta is slightly higher than in large arteries causing pressure gradient.

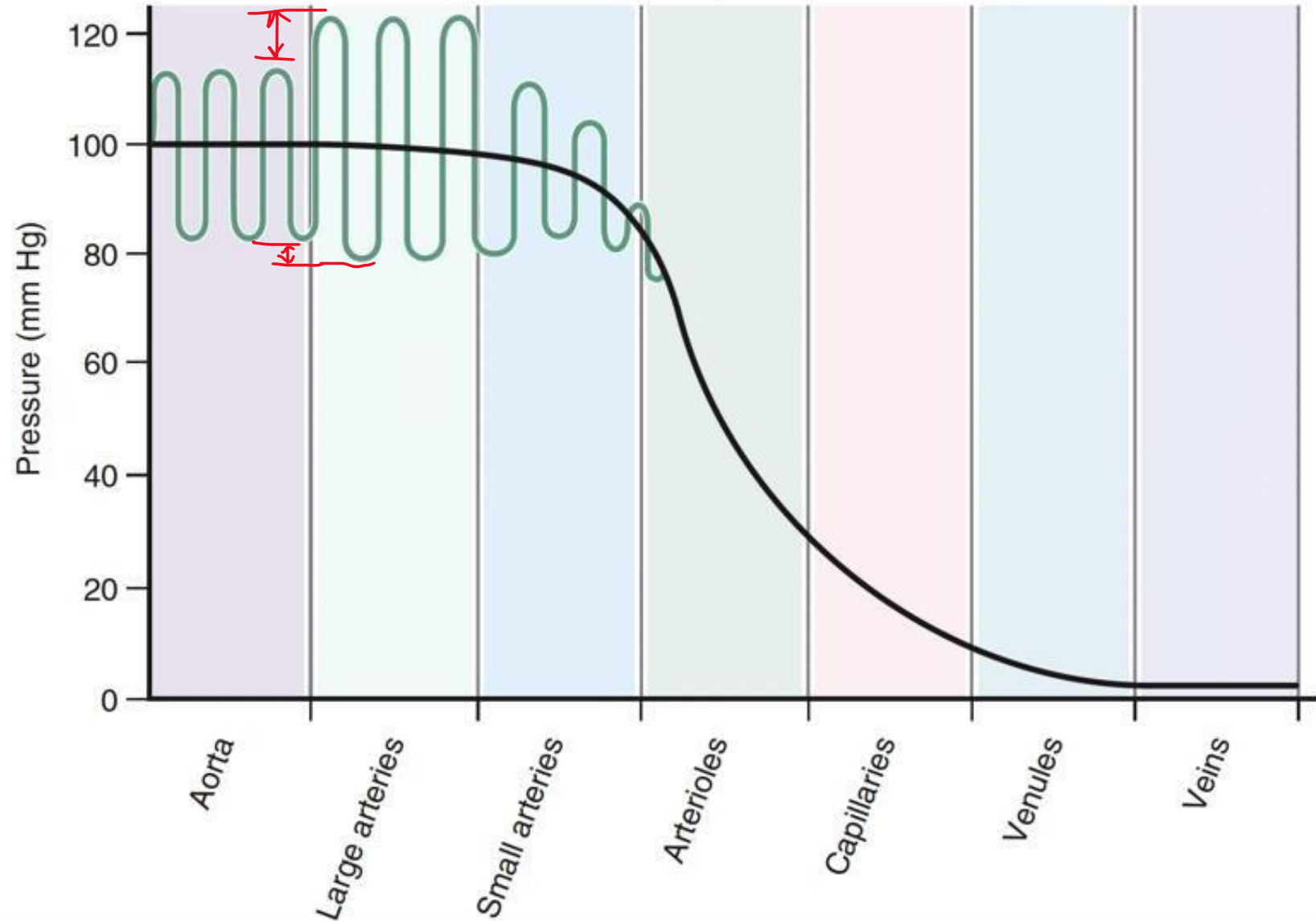
Mean arterial pressure

- the driving force for blood flow in the arteries is the mean arterial pressure, which is influenced more by diastolic pressure than by systolic pressure.
- While systolic pressure is higher in the large arteries than in the aorta, diastolic pressure is lower; thus, mean arterial pressure is lower downstream.

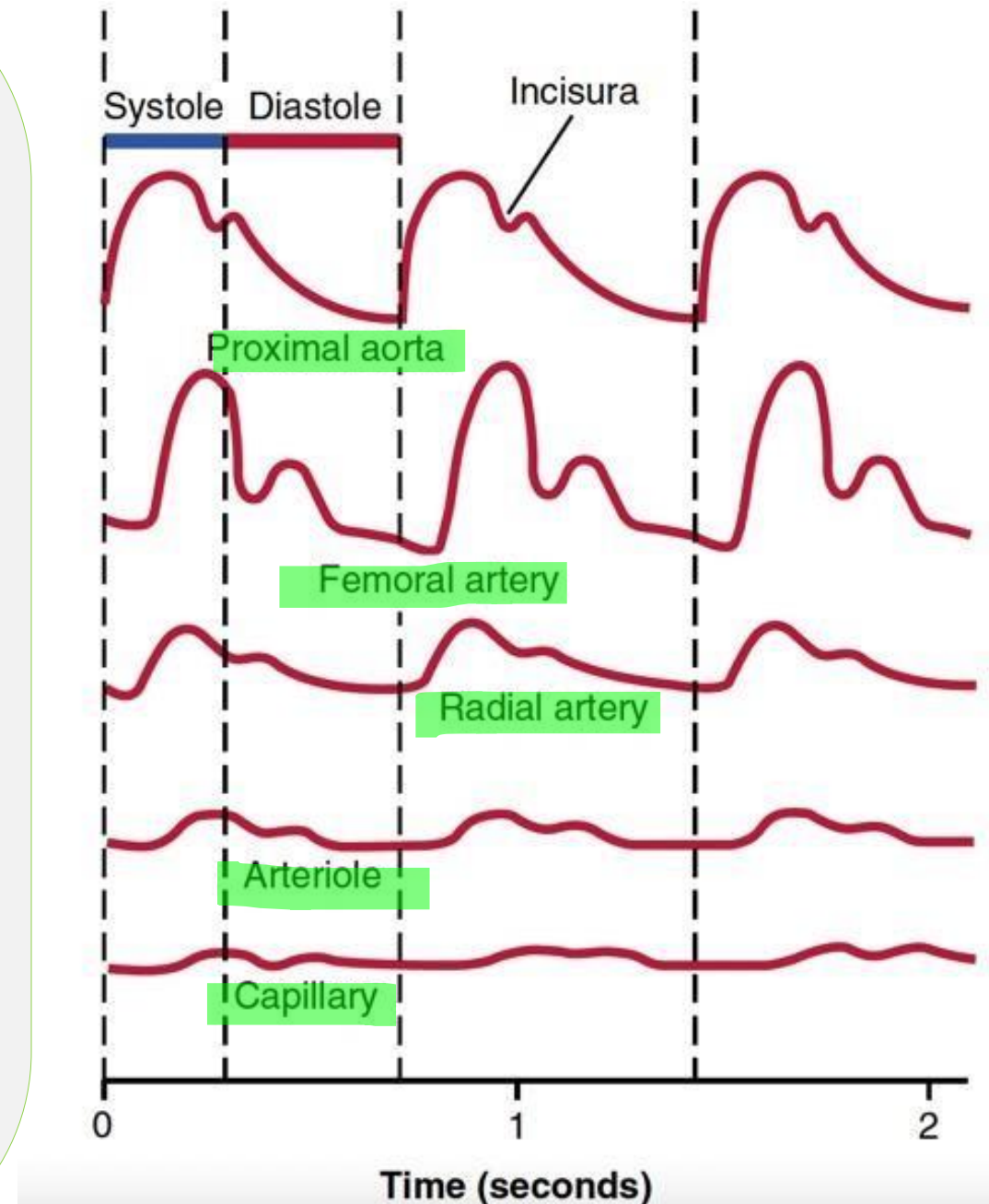
- Higher systolic pressure in large arteries compared to aorta

The mean pressure in aorta is slightly higher than in large vessels.
(the black line represent the mean pressure)

Lower diastolic pressure in large arteries cause the mean pressure more inclined toward the diastolic pressure
→ lead to the blood flow.



- These pressure wave (also called pressure pulse, also pulse wave) move in a certain direction
- don't get confused !
- Pulse pressure: is the difference between the systolic and diastolic pressure.
- The pressure pulse: is the wave that (move across the walls of vessels) represent the changes in pressure.
- The diagram represent the normal pressure pulses in different vessels
- The pressure pulse differ between different vessels as you see (green text in the image).
- Before the diastole, we have dicrotic notch or the incisura; it represents the closure of the aortic valve.
- If you notice, from the aortic down to the capillaries, there is dampening of the pulsation (decrease the pulse pressure) → decrease in the difference between systolic and diastolic pressure
- Until we reach the capillaries, we now have a continuous blood flow with no pulsation (pressure almost the same).
- More clarification → in the aorta there is systolic pressure and diastolic pressure, but in the capillaries the systolic and diastolic pressures are almost the same.



Damping of pulse pressure

- The progressive diminution of the pulsations in the periphery is called damping of the pressure pulses.
- The cause of this damping is twofold:
- (1) resistance to blood movement in the vessels
 - When resistance increase the pressure will decrease, and specifically, the systolic pressure since it's the force that act more on the vessel wall, resulting in decrease in the pressure gradient.
 - As we go down from aorta to capillaries the resistance increase and the pressure decrease
- (2) compliance of the vessels.
 - Compliance is more important in the venous side.

Aortic dicrotic notch

- dicrotic notch (or incisura), is produced when the aortic valve closes.
- Aortic valve closure produces a brief period of retrograde flow from the aorta back toward the valve, briefly decreasing the aortic pressure below the systolic value.

Let's revise some of the cardiac pathology:

➤ **Arteriosclerosis:** it's a hardening in the arteries (**stiffness**) due to thickening and **loss of elasticity** in the arterial wall which will result in a **less compliant** vessel, so it's a pathology in the elastic layers of the arteries, and we see this more with aged patient because the body has a very limited ability to regenerate elastic fibers.

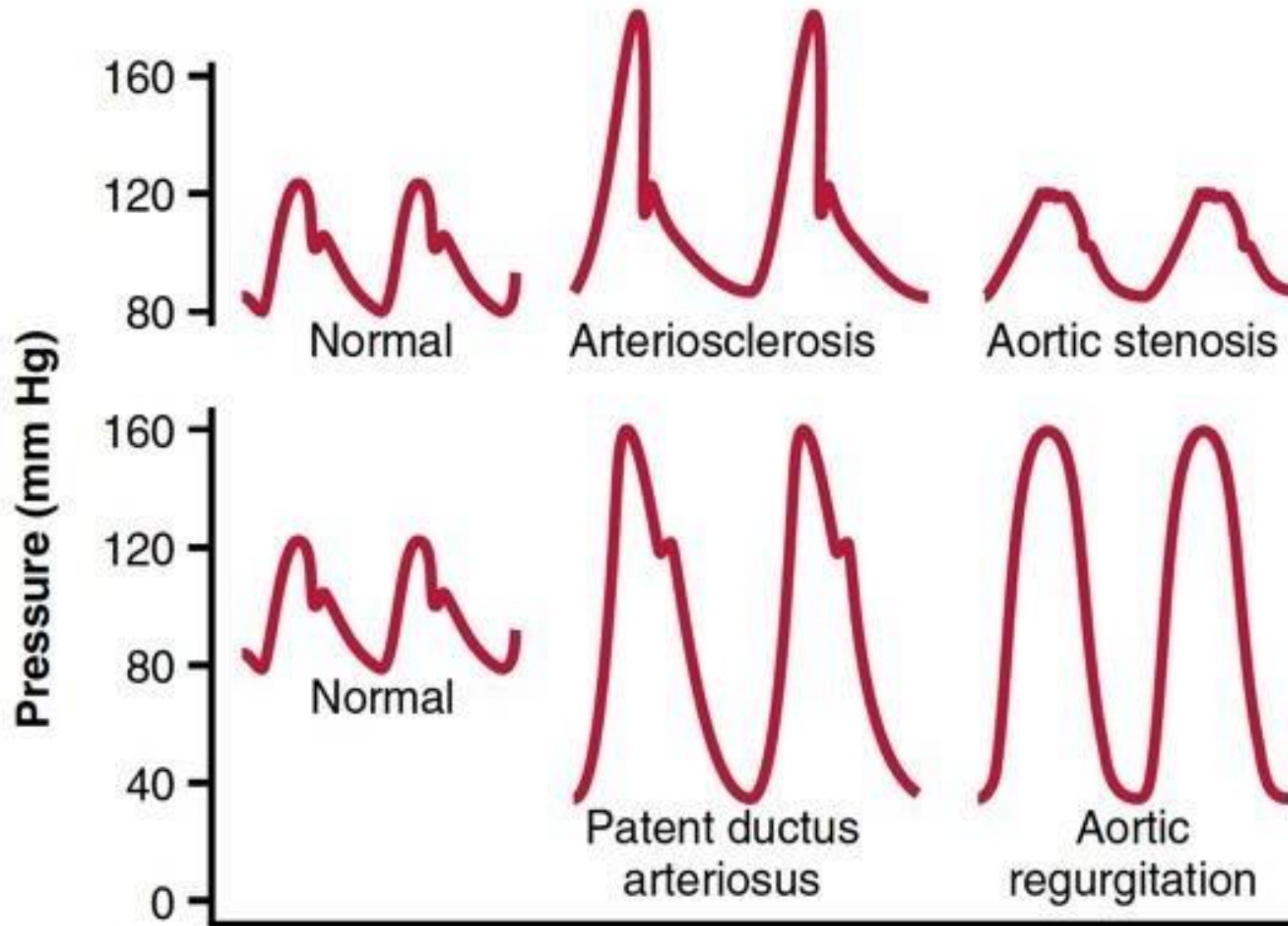
(remember that arteriosclerosis is not the same as atherosclerosis)

➤ **Aortic stenosis:** it's a narrowing in the aortic valve opening which will restricts blood flow from the left ventricle into the aorta during systole, this will lead to a **decrease in the stroke volume.**

➤ **Aortic regurgitation:** here the aortic valve does not close properly, this will allow **the blood to flow back to the left ventricle during diastole**, this will lead to volume overload in the left ventricle and will result in **higher volume pumped in the systole phase (higher stroke volume).**

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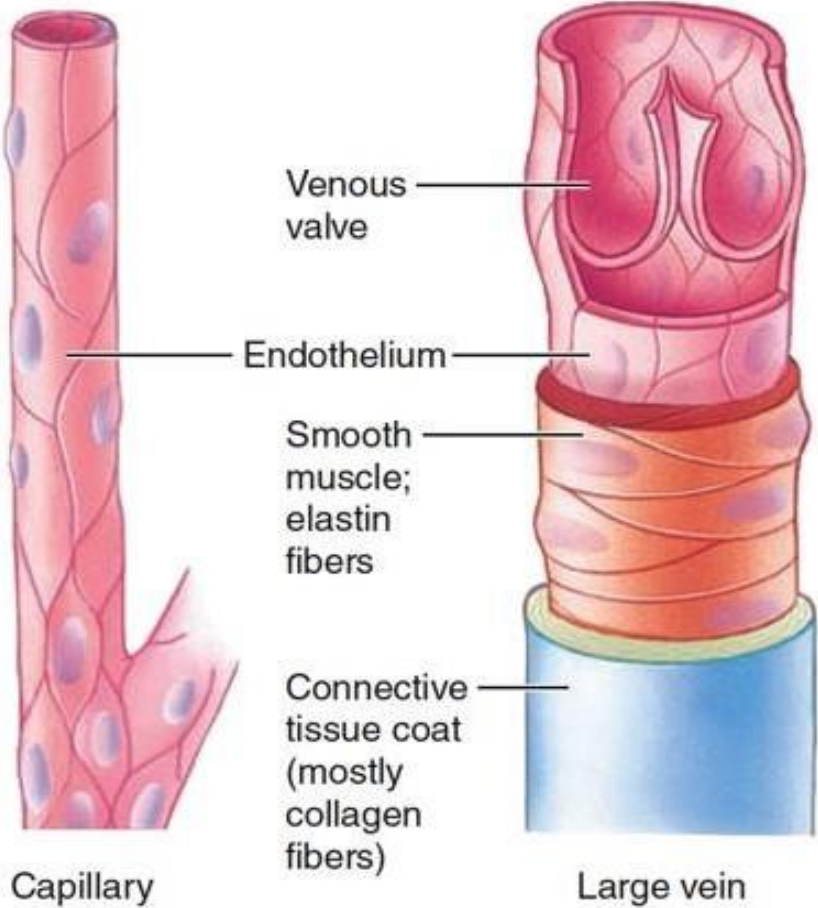
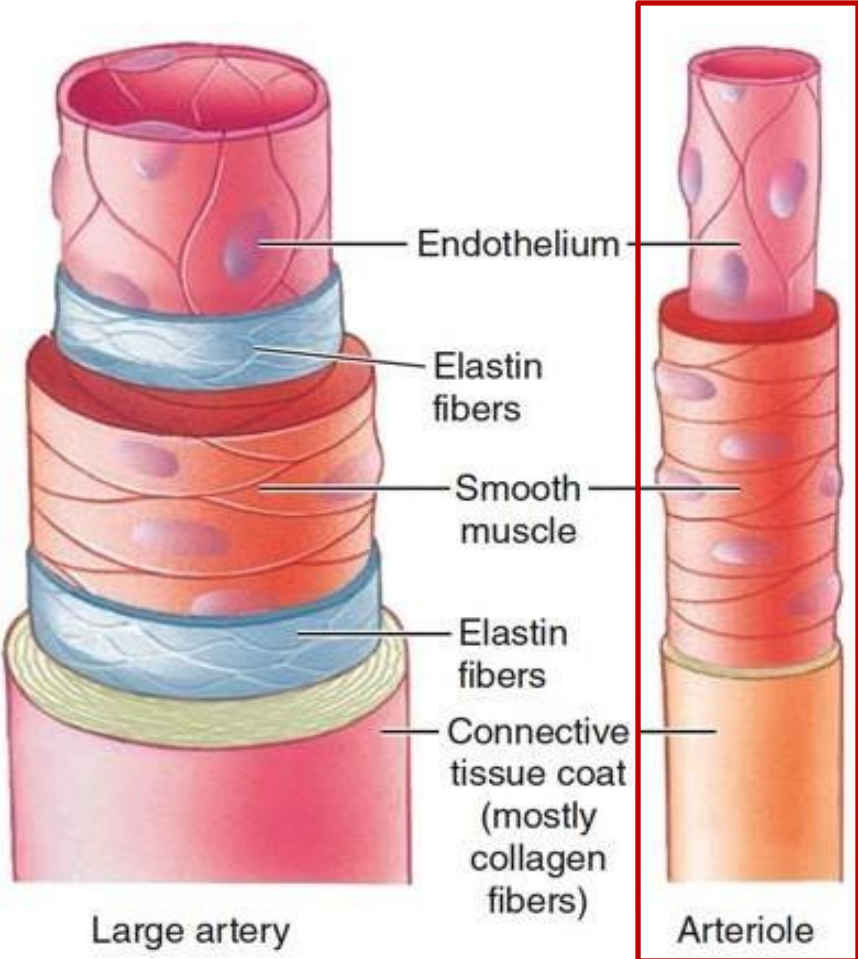
The pressure Pulse Waves in the Aorta



- Remember from the last lecture: (Blood pressure: the force exerted by the blood against a vessel wall.)
- Remember that the dicrotic notch mark the closure of the aortic valve.
- In the normal pulse wave, the systolic pressure reach 120 and the diastolic pressure at 80, and there is dicrotic notch, this is normal.
- In arteriosclerosis pulse wave, due to the stiffness and lack of the elasticity in the aorta, the blood will cause more pressure on the vessel wall (because they can't accommodate this volume), which will result in **increased systolic pressure**. (suspect this with aged patents).
- In aortic stenosis pulse wave, due to decreased stroke volume, the blood will cause less pressure on the vessel wall, which will result in **decreased systolic pressure**. (Notice that the dicrotic notch is less prominent because the aortic valve opening is narrow anyway, so the closure of the valve will not make much difference).
- In aortic regurgitation pulse wave, due to the increased stroke volume, it will **increase systolic pressure**, also because the aortic valve doesn't close, the blood will flow back to the left ventricle which will result in **decreased diastolic pressure**. (Notice that the dicrotic notch is absent because the aortic valve doesn't close properly.)
- the doctor didn't mention anything about patent ductus arteriosus.
- Remember: (pulse pressure = systolic pressure – diastolic pressure), so increased systolic pressure or decreased diastolic pressure will result in increased pulse pressure.

Arterioles

When you hear arterioles -> always remember **Resistance**



Notice that there is no elastin fibers, but there is more smooth muscles

Arterioles

- Arterioles are the main resistance vessels in the vascular tree because their radius is small enough to offer considerable resistance to flow.

- Note that the radius is to the power four!

- Which mean that a small decrease in radius can increase the resistance by a large amount

$$\uparrow R = \frac{8\eta l}{\pi r^4} \downarrow$$

- arterioles are also the site where resistance can be changed by alterations in local, humoral, and neural factors.

Arterioles

the high degree of arteriolar resistance causes a marked drop in mean pressure as blood flows through these small vessels.

- This pressure differential that encourages the flow of blood from the heart to the various organs downstream.
- Arteriolar resistance also converts the pulsatile systolic-to-diastolic pressure swings in the arteries into the nonfluctuating pressure present in the capillaries.

$$R = \frac{8\eta l}{\pi r^4}$$

Why the arterioles are the main vessels responsible for resistance?

Small radius? True but the capillaries also have small radius.

There is another important factor that makes the arterioles more important than capillaries for resistance which is the **ability to change its radius!**, changing its radius mean they can control the resistance by their smooth muscles.

$$\updownarrow F = \frac{\Delta P}{R \updownarrow}$$

Having the ability to change the resistance allow arterioles to regulate the pressure gradient. Not only that, but they can also control the **flow rate!** Essentially, the arterioles control the flow of blood into capillaries by adjusting their diameter.

Arterioles

- The radius (and, accordingly, the resistances) of arterioles supplying individual organs can be adjusted independently to accomplish two functions:
- (1) to variably distribute the cardiac output among the systemic organs, depending on the body's momentary needs,
- (2) to help regulate arterial blood pressure.

Arterioles

- Unlike arteries, arteriolar walls contain little elastic connective tissue.
- However, they do have a thick layer of smooth muscle that is richly innervated by sympathetic nerve fibers.
- The smooth muscle layer runs circularly around the arteriole; so when the smooth muscle layer contracts, the vessel's circumference (and its radius) becomes smaller, increasing resistance and decreasing flow through that vessel.

Arterioles

- The smooth muscle in the arteriole is sensitive to:
- 1. local chemical changes.(things that can change the metabolic activity like $O_2/CO_2/H^+$, this will be discussed later)
- 2. circulating hormones.(e.g. catecholamine)
- 3. mechanical factors such as stretch.
- 4. sympathetic nerve stimulation.

Arterioles

- Vasoconstriction is the term applied to such narrowing of a vessel.
- Vasodilation refers to enlargement in the circumference and radius of a vessel as a result of its smooth muscle layer relaxing.
- Vasodilation leads to decreased resistance and increased flow through that vessel.

Vascular tone

- Arteriolar smooth muscle normally displays a state of partial constriction known as vascular tone, which establishes a baseline of arteriolar resistance.
- Two factors are responsible for vascular tone. First, arteriolar smooth muscle is tonic smooth muscle that has sufficient surface-membrane voltage-gated Ca channels open even at resting potential to trigger partial contraction.
- This myogenic activity is independent of any neural or hormonal influences, leading to self-induced contractile activity.
- Second, the sympathetic fibers supplying most arterioles continually release norepinephrine, which further enhances vascular tone.
- This ongoing tone makes it possible to either increase or decrease contractile activity to accomplish vasoconstriction or vasodilation, respectively.

The arteriolar muscles must always have some sort of contraction even without external stimulus called Vascular Tone. If fully relaxed, vasodilation would be impossible since the radius couldn't increase further.

Pay attention that the vascular tone depends on two factor: intrinsic (Ca^{+2}) and extrinsic (sympathetic), but the intrinsic factor require no external stimulation.

Sympathetic innervation of arterioles

- It is extensively innervated by sympathetic adrenergic nerve fibers. α 1-Adrenergic receptors are found on the arterioles of several vascular beds.
- When activated, these receptors cause contraction, or constriction, of the vascular smooth muscle.
- Constriction produces a decrease in the diameter of the arteriole, which increases its resistance to blood flow.
- Less common, β 2-adrenergic receptors are found in arterioles of skeletal muscle. When activated, these receptors cause dilation, or relaxation, of the vascular smooth muscle, which increases the diameter and decreases the resistance of these arterioles to blood flow.

Why the sympathetic innervation is in the arterioles?

Because it can control the resistance, pressure, flow rate (it's the major resistance vessels)

Vascular tone

- A variety of factors can influence the level of contractile activity in arteriolar smooth muscle, thereby substantially changing resistance to flow in these vessels.
- Unlike skeletal and cardiac muscle in which action potentials trigger muscle contraction, vascular smooth muscle can undergo graded changes in force in response to chemical, physical, and neural factors without undergoing action potentials.
- These agents largely act via second-messenger pathways.
- The extent of contraction of arteriolar smooth muscle depends on the cytosolic Ca concentration.
- The factors that cause arteriolar vasoconstriction or vasodilation fall into two categories: local (intrinsic) controls, which are important in determining distribution of cardiac output, and extrinsic controls, which are important in blood pressure regulation.

Thank you

Additional sources

[great video for pressure pulse!](#)



بِسْمِ اللَّهِ الرَّحْمَنِ الرَّحِيمِ

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V1→V2			
V2→V3			



امسح الرمز و شاركنا بأفكارك لتحسين أدائنا!!