



PHYSIOLOGY

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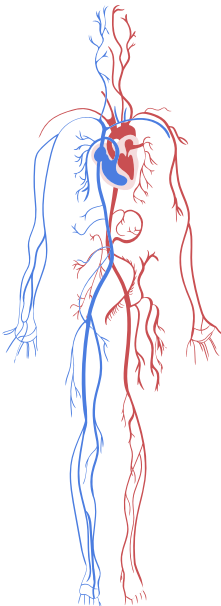
MID | Lecture 3

Arteries

وَلَقَدْ خَلَقْنَا الْإِنْسَانَ وَنَعْلَمُ مَا تُوَسْوِسُ بِهِ نَفْسُهُ وَنَحْنُ أَقْرَبُ إِلَيْهِ مِنْ حَبْلِ الْوَرِيدِ
اللهم إنا نعوذ بك من شرور أنفسنا ومن سيئات أعمالنا

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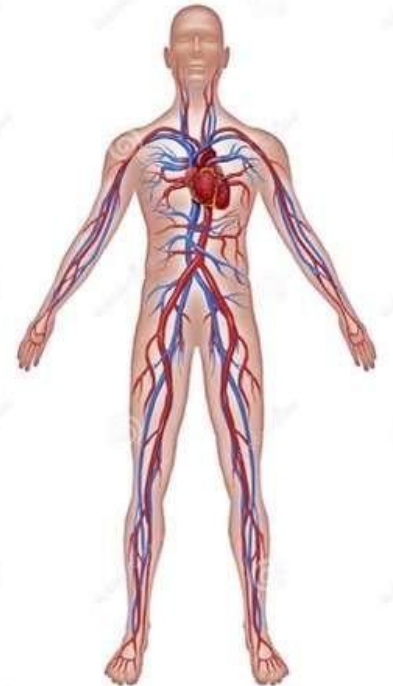


Vascular Physiology

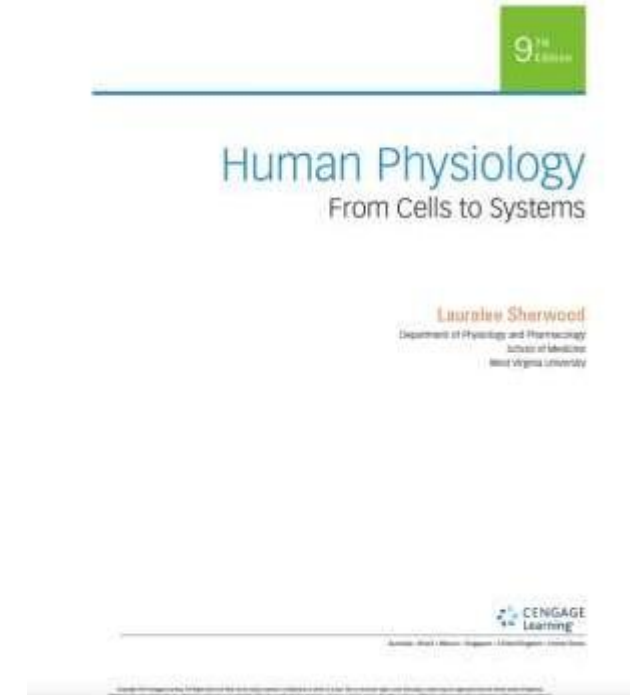
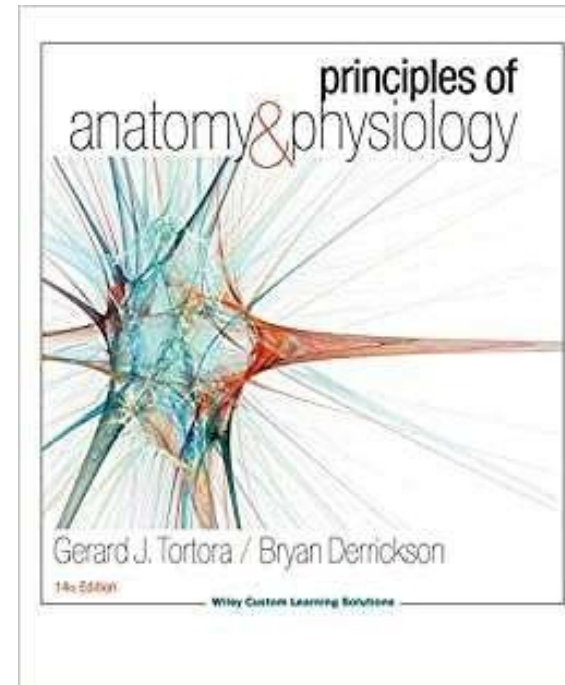
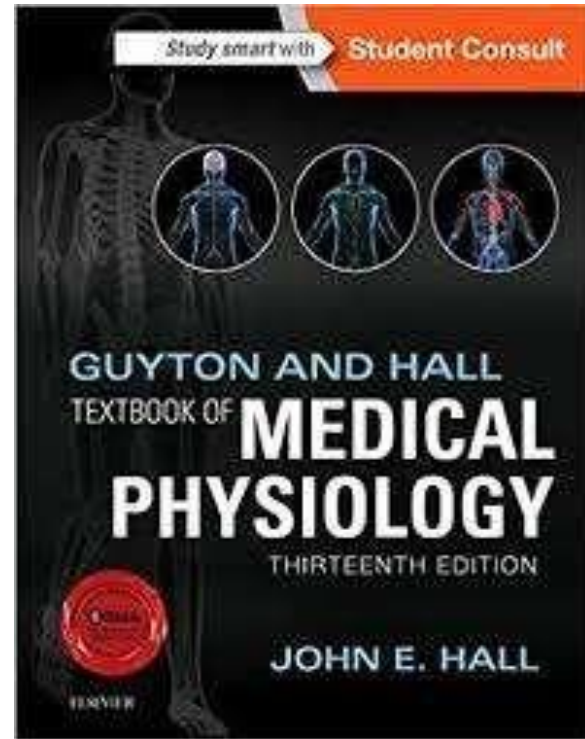
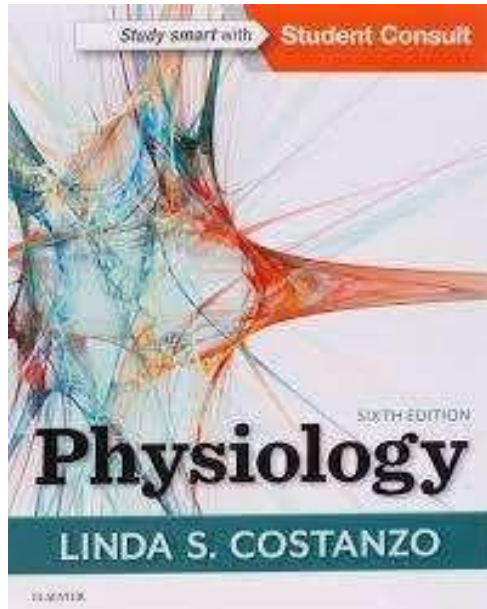
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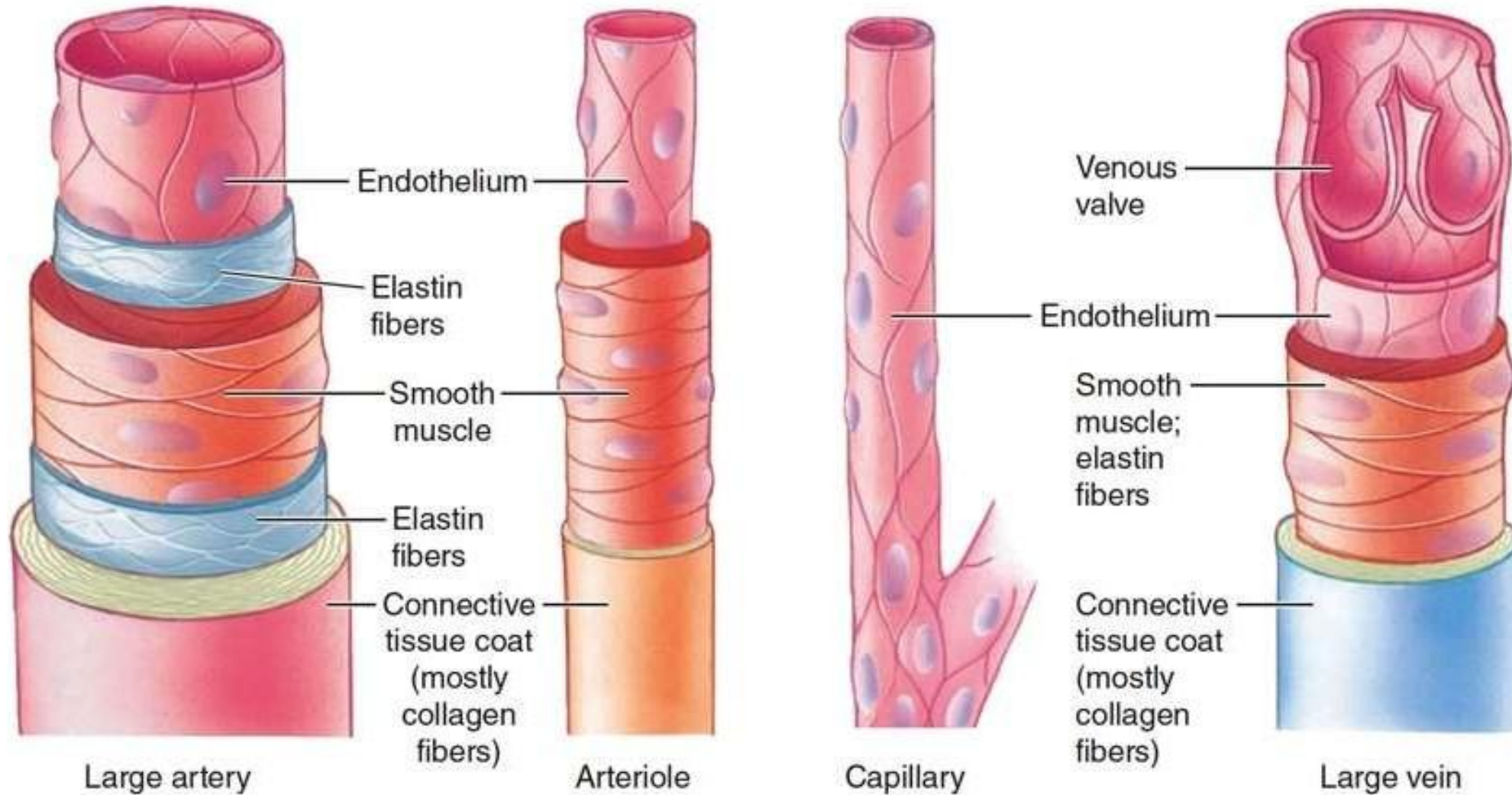
References



Lecture Overview

- Two problems are going to be discussed, as a result of the heart's ejection blood rapidly in relatively high volumes:
 1. High blood pressure can cause injury, so how does the aorta, the direct artery against which blood is ejected, accommodate that blood pressure and doesn't rupture?
 2. The heart beats and ejects blood intermittently, and continuous blood flow is required for the tissues, to whom the pulsatile flow is inconvenient, so, how do blood vessels maintain a continuous blood flow?

Arteries



Arteries

- ❖ Arteries are specialized to:
 - Serve as **rapid-transit passageways** for blood from the heart to the organs.
 - Act as a **pressure reservoir** to provide the driving force for blood when the heart is relaxing.

Conduit Arteries: Roles and Functions

- They're Large arteries act as **conduits** transporting blood from the **high-pressure** left ventricle, the heart, to the downstream vessels and capillaries.
- Blood ejected from the heart has **high pressure and high volume**, creating what is called **stressed volume** in systemic arteries.
- In contrast, blood present in capillaries and veins is under **low pressure**, representing **unstressed volume**.

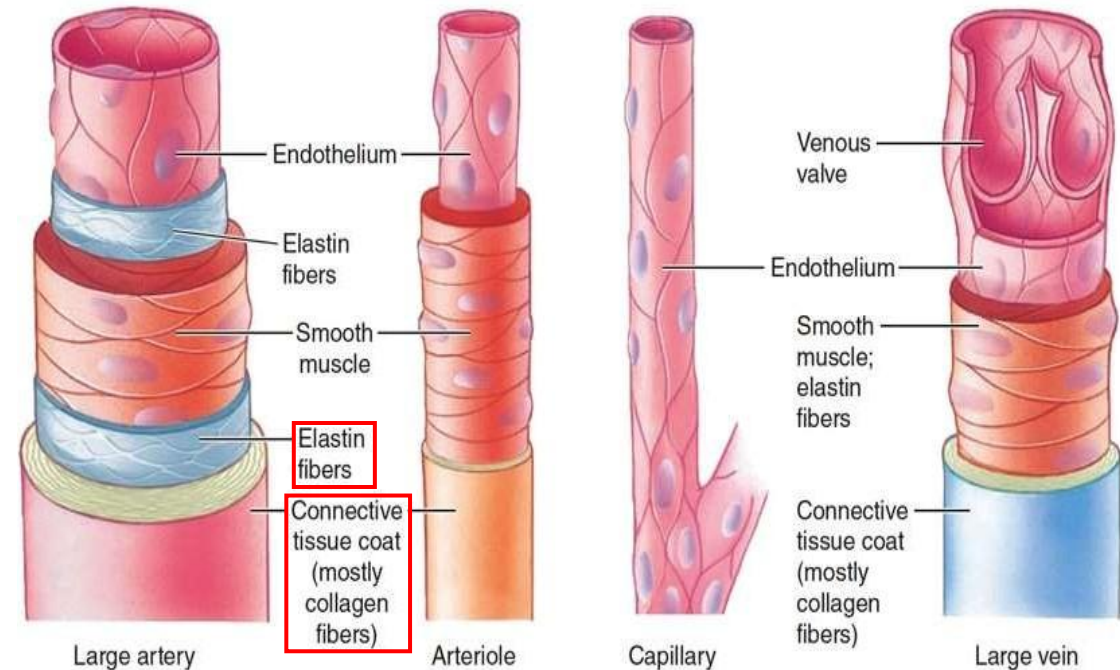
Now, how are arteries **protected** against that **high pressure**?

- The LV ejects blood at ~120 mmHg, systolic pressure, to the aorta then to major arteries.
- The arterial wall structure **must** withstand that pressure **without injury**.

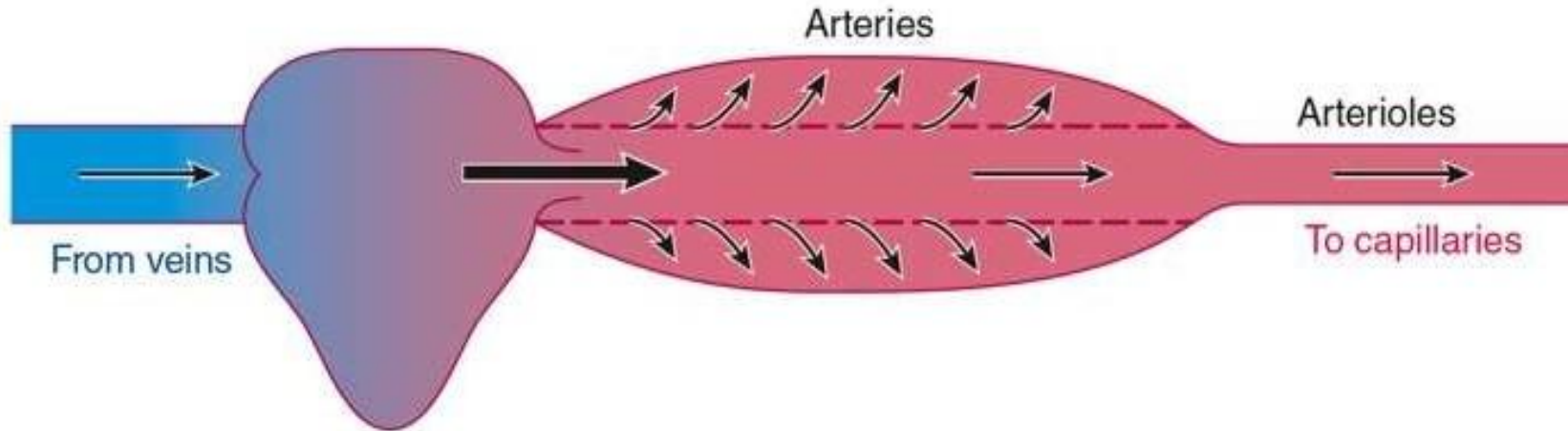
➤ Arterial wall prevents injury via:

- **Collagen:** Provides **tensile strength** to prevent over-stretching and rupture; maintaining structural integrity.
- Elastic Fibers (**Elastin**): Allow for distensibility (**stretch**) and **recoil** during diastole, lower pressure.

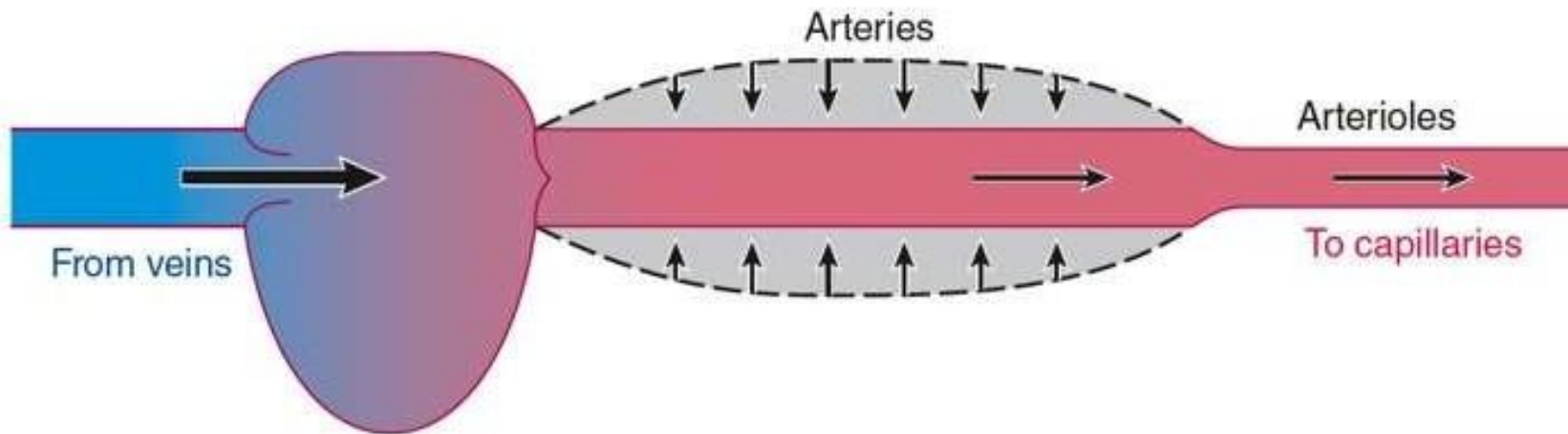
✓ **Collagen** is the one that primarily **prevent** injury. While, elastic fibers are responsible for stretch rather than tensile strength.



The Windkessel Effect



(a) Heart contracting and emptying



Refer to this picture in the following few slides

Summarizing the Windkessel Effect

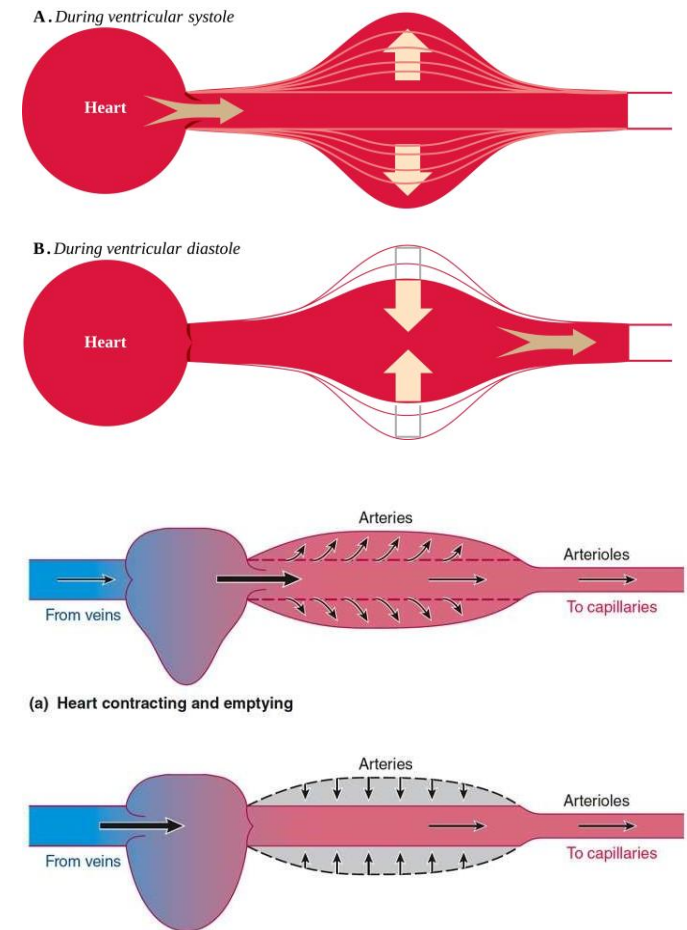
You can read this summary before or after you dive into the next slides, which I somehow felt vague, or simply skip it.

A. During ventricular systole:

- When the left ventricle ejects a high-pressure bolus of blood during systole, the arteries yield and **distend**; this is crucial because it buffers the force, preventing the **pressure** from becoming **dangerously elevated**.
- Once the **ventricle stops** pumping during diastole, the arterial walls **passively recoil**. This **recoil exerts pressure** on the blood, pushing it forward to maintain a continuous flow throughout the entire cardiac cycle. Without this mechanism, blood flow would be intermittent - starting during systole and ceasing completely during diastole, which is inconvenient.

B. During ventricular diastole:

- The second concept focuses on the **active properties** of the vessel wall structure, which contains significant smooth musculature and elastin fibers.
- Beyond passive elasticity, blood vessels are "myogenic," meaning they react to physical forces. If an **artery** is suddenly inflated or **stretched** by increased pressure, **the smooth muscle layer instinctively "fights back" by contracting**. This active contraction works alongside the passive elastic fibers to manage vessel diameter and contributes to the overall recoil phenomenon observed in the vasculature.
- Increasing pressure, with an area having more BP than the next, allows the continuous flow.

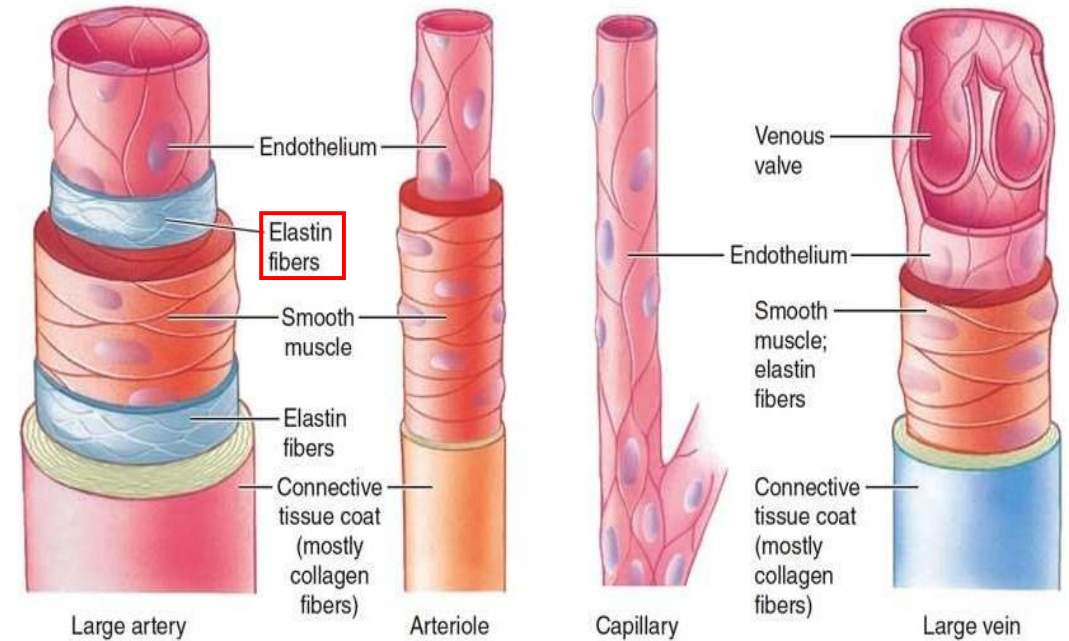


Elastic Arteries

- Elastic large arteries **expand** to temporarily hold the excess volume of ejected blood, **storing some of the pressure energy** imparted by cardiac contraction in **their stretched walls**.
- When the **heart relaxes** and temporarily **stops pumping** blood into the arteries, the stretched arterial walls passively **recoil**.
 - This elastic recoil exerts pressure on the blood in the large arteries during diastole.
- The **pressure** pushes the excess blood contained in the arteries into the vessels downstream, **ensuring continued blood flow** to the organs when the heart is relaxing and not pumping blood into the system.

Elastic Fibers (Elastin)

- They allow for distensibility (**stretch**) and **recoil** during diastole, lower pressure.
- ❖ **Distribution of Elastic Fibers:**
 - It differs between different vessels:
 - The **aorta** has the highest elastin content.
 - Elastin decreases progressively in branches downstream.



Compliance and Distensibility

- **All blood vessels are distensible**, meaning they can increase diameter with increased internal pressure.
- **Compliance = change in volume / change in pressure.**
 - Higher compliance, means that the vessel can accept more blood with minimal increase in pressure.
- **Veins are the most compliant vessels** because they operate under **low, unstressed pressure** and can accept large volumes easily.
- Large arteries are compliant, but significantly less so than veins.

The Windkessel Effect

Dampening Pulsatility

- The problem is that heart pumps **intermittently**, stop-and-start, so how would blood flow remain continuous?
- Large arteries must tolerate high pressure and **convert pulsatile** cardiac output **into continuous** blood flow, that's required for tissues:
 - **During systole:**
 - The left ventricle ejects **stroke volume**.
 - Elastic aorta **stretches**, accommodating part of the volume and storing potential energy in the wall.
 - ✓ This **reduces downstream systolic pressure** and protects capillaries.
 - **During diastole:**
 - Aortic valve closes, so no more ejection, and LV pressure drops to ~0 mmHg during diastole.
 - Stored energy in the elastic wall is released as recoil (passive recoil), generating diastolic blood flow and maintaining continuous perfusion.
- Overall effect: **Dampens pulse pressure and ensures continuous flow, and arterial diastolic pressure of ~80 mmHg (rather than 0 mmHg).**

Stressed vs Unstressed volumes

- Blood ejected from the heart has **high pressure and high volume**, and the volume of blood contained in the **systemic arteries** is called the **stressed volume** (meaning the blood volume under **high** pressure).
- The volume of blood contained in the **veins and capillaries** is called the **unstressed volume** (meaning the blood volume under **low** pressure).

Biophysical Forces on the Vessel Wall

Pressure, tension and shear stress

1. **Pressure**; intra-, extra- and trans-mural.

- Intramural: The perpendicular **force** exerted **by blood** volume **against** the vascular **wall**.
- Extramural: **External pressure** on the vessel.
- Transmural Pressure: The **gradient** between pressure inside the vessel and pressure outside.

$$\text{Transmural} = (\text{Intra-mural}) - (\text{Extra-mural})$$

2. **Tension**:

- Wall Tension (Laplace's Law): (**Tension = Pressure * Radius**)
 - Larger radius → greater tension → greater risk of rupture.
 - Since the **aorta** has a very large radius, wall tension is extremely high.
 - **Thick connective tissue**, containing numerous collagen, is required on the outermost layer to **resist** this tension and **prevent rupture**.

3. **Shear Stress**:

- A longitudinal **frictional** force occurring between flowing blood and endothelial cells.

Blood pressure

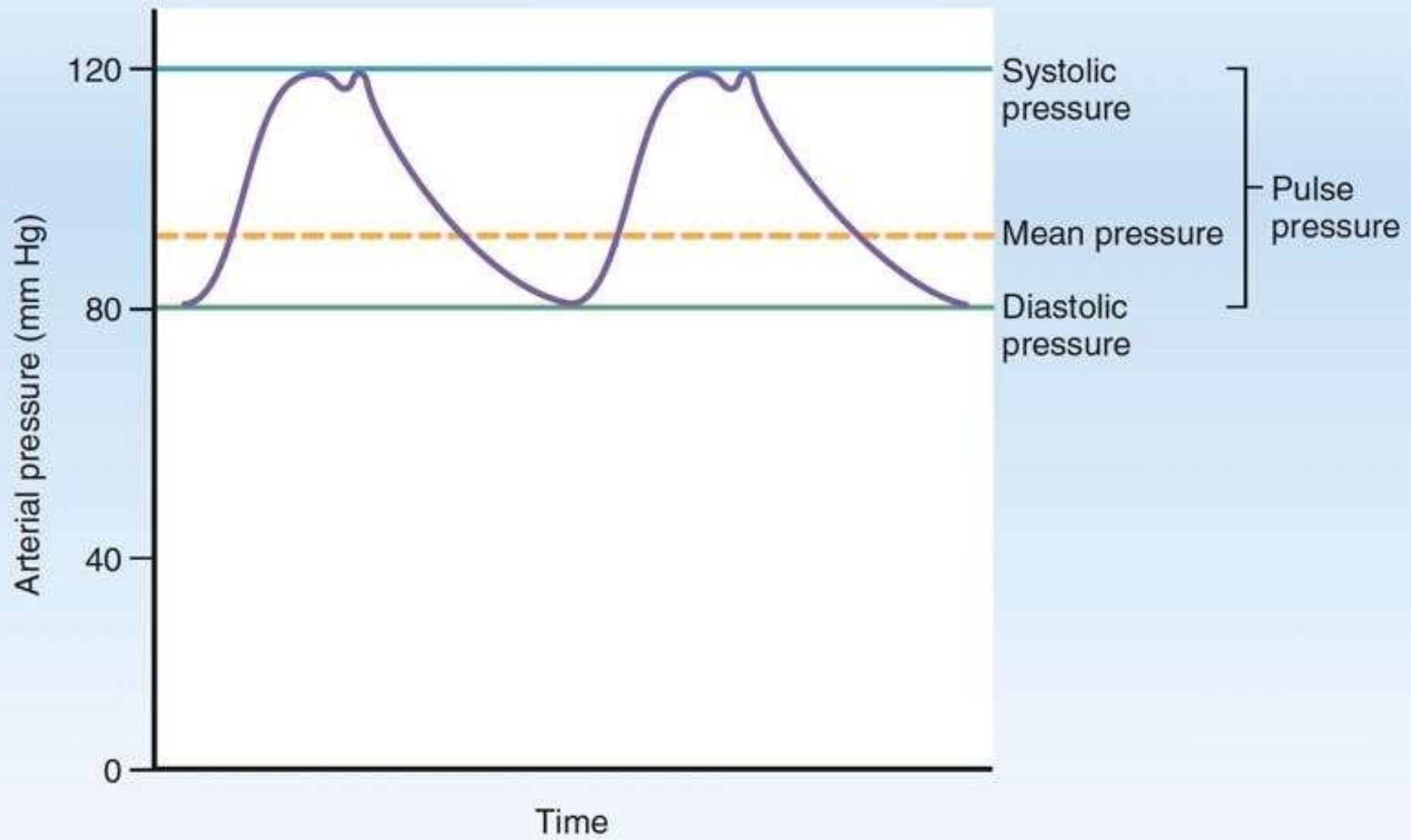
- **Blood pressure:** the force exerted by the blood against a vessel wall.
- It **depends on the** ⁽¹⁾**volume of blood** contained within the vessel and the ⁽²⁾**compliance**, or distensibility, **of the vessel walls** (how easily they can be stretched).

❖ Types of Blood Pressure:

- **Systolic pressure** (SBP): the maximum pressure exerted in the arteries when **blood is ejected** into them during systole.
- **Diastolic pressure** (DBP): the minimum pressure within the arteries when **blood is draining** off into the rest of the vessels during diastole.

Factors Increasing Systolic Blood Pressure

1. Increased **contractility** of the heart.
2. Increased **stroke volume**.
3. Reduced arterial elasticity (less stretch) → higher transmitted systolic pressure.



Blood pressure

- Although **ventricular pressure falls** to 0 mm Hg during **diastole**, arterial pressure **does not** fall to 0 mm Hg.
- **Pulse pressure (PP)**: the difference between systolic and diastolic pressures.

$$\text{Pulse Pressure (PP)} = \text{systolic BP (SBP)} - \text{Diastolic BP (DBP)}$$

- Because the pulse can be felt each time the ventricles pump blood into the arteries, the **pulse rate is a measure of the heart rate**.

Aortic stiffness and its effects on Blood Pressure⁽¹⁾

- **With aging and pathological states** (e.g., diabetes, accumulation of **Advanced Glycation End Products - AGEs**), arterial elastin undergoes:
 1. Fragmentation
 2. Progressive loss
 3. Replacement by stiffer collagen fibers
- Elastin **cannot regenerate**, so these changes cause a **permanent** loss of arterial elasticity, producing **aortic stiffness** (arteriosclerosis).

Aortic stiffness and its effects on Blood Pressure⁽²⁾

- **During Systole:**
 - A stiff aortic wall **cannot expand** to accommodate the Stroke Volume.
 - More blood is transmitted forward immediately → **elevated systolic pressure**.
- **During Diastole:**
 - Reduced elastic recoil → **less stored energy released back into the bloodstream**.
 - Leads to **lower diastolic pressure** and reduced organ perfusion, especially coronary circulation (which depends heavily on diastolic flow).
- **Net effect on Pulse Pressure (PP):**

Pulse Pressure (PP) = Systolic Pressure – Diastolic Pressure

 - Aortic stiffness causes: ↑ Systolic Pressure, and ↓ Diastolic Pressure.
 - This will result in a widened Pulse Pressure, which is a hallmark of aging vasculature.
- **Clinically:**
 - Persistent elevation of systolic pressure with normal/low diastolic pressure forms will lead to **Isolated Systolic Hypertension**, which is common in older adults.

Aortic stiffness and its effects on Blood Pressure⁽³⁾

- **The higher the systolic pressures**, the greater the shear stress and endothelial injury in peripheral arteries.
- **The more reduced diastolic pressures**, the more diminished perfusion to organs requiring constant flow (e.g., brain, kidneys, heart).

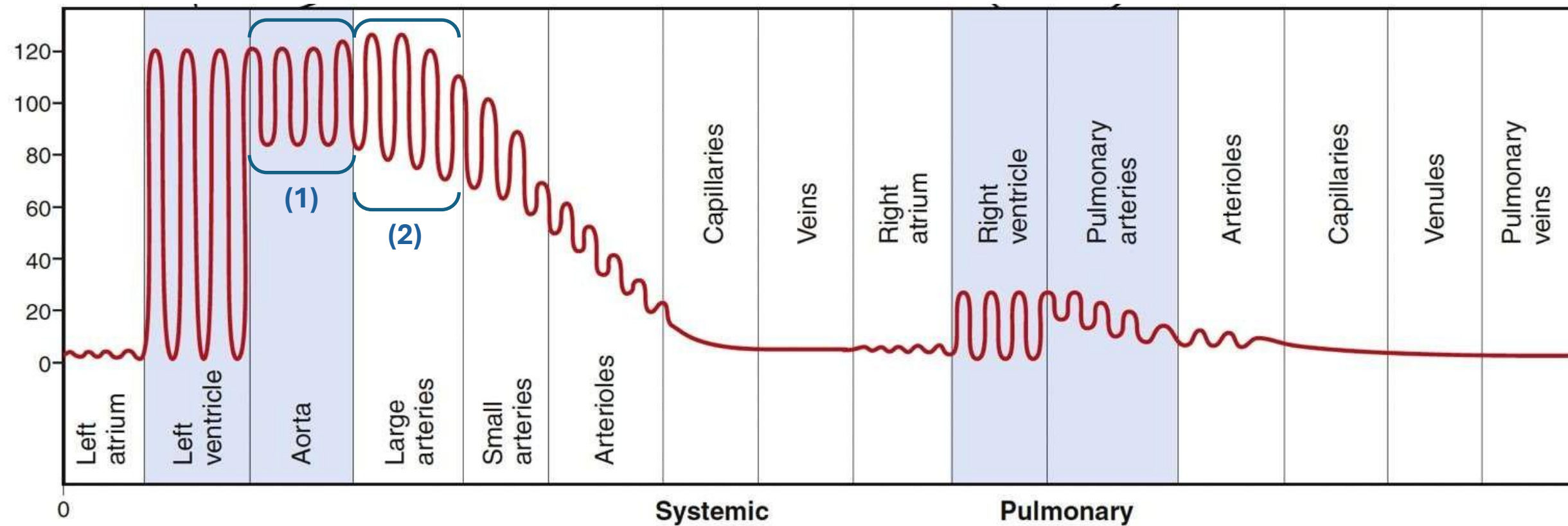


illustration of the figure

*(1) As shown in the figure, the aortic pressure rises to about 120 mmHg during systole and falls only to around 80 mmHg during diastole, unlike the left-ventricular pressure, which returns nearly to zero. This difference is mainly due to **the Windkessel (elastic-recoil) effect**, which makes the aorta act as a pressure reservoir that **maintains the diastolic pressure** and **dampens the change in pressure (the pulse pressure)**.*

(2) When we examine the portion of the pressure graph that represents the large systemic arteries pressure, two important questions immediately arise.

- ✓ **The first question** is: Why does the systolic pressure in the large arteries appear slightly higher than in the aorta? **(A)**
- ✓ **The second question** is: If the systolic pressure increases in these large arteries, does this mean that their overall pressure becomes higher than the aorta, and wouldn't this contradict the basic rule that blood must flow from high pressure to low pressure? **(B)**

illustration of the figure

(A) Wave Reflection:

- ❖ When the heart contracts and ejects blood into the aorta, the blood presses against the arterial walls and creates a **pressure wave** that moves faster than the actual blood flow because the blood has inertia (قصور ذاتي) and needs time to accelerate.
- ❖ As this pressure wave travels along the aorta and reaches branch points (bifurcation) or changes in vessel stiffness, part of the wave reflects backward toward the heart. When this **reflected wave meets the next forward-moving wave**, the two waves add together, producing a slight increase in the systolic peak in the large arteries (**Magnification effect**).

This picture isn't required

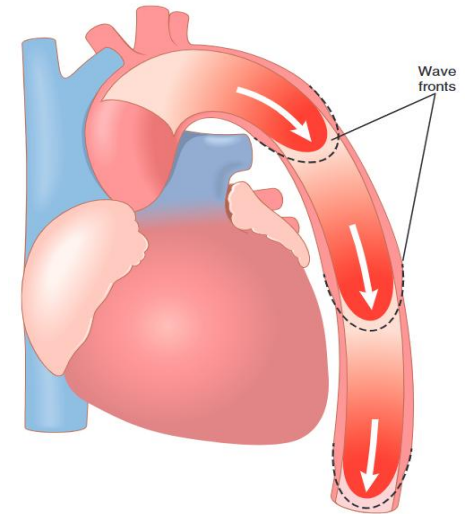
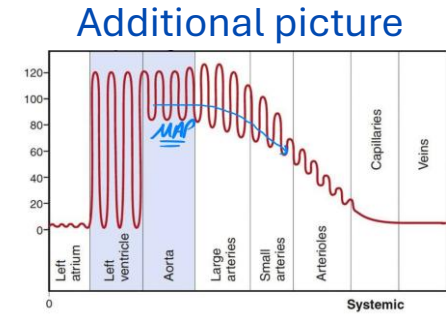


Figure 15-5. Progressive stages in transmission of the pressure pulse along the aorta.

illustration of the figure

(B) Mean Arterial Pressure:



- ❖ Although there is a slight increase in systolic pressure in the large arteries, the overall pressure in these arteries remains lower than that of the aorta. This is because what truly determines the overall driving force of blood flow is the **mean arterial pressure (MAP)**, not the systolic peak.
- ❖ **MAP** is equal ($\frac{1}{3}$ Systolic Pressure + $\frac{2}{3}$ Diastolic Pressure), which means it is more strongly influenced by diastolic pressure.
- ❖ When we look at the figure, we see that even though the systolic pressure in the large arteries rises slightly due to wave reflection, the diastolic pressure actually falls compared to the aorta. This reduction in diastolic pressure **shifts the MAP downward**, keeping the overall pressure in the large arteries lower than the pressure in the aorta.
- ❖ **MAP** could also be measured by (Diastolic Pressure + $\frac{1}{3}$ Pulse Pressure)

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 rate** = number of pulse beats felt in superficial arteries (e.g., radial artery), counted for 60 seconds; a professional may count for 15 seconds and $\times 4$, **to estimate the heart rate** because each pulse represents one systolic–diastolic 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.

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.

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.

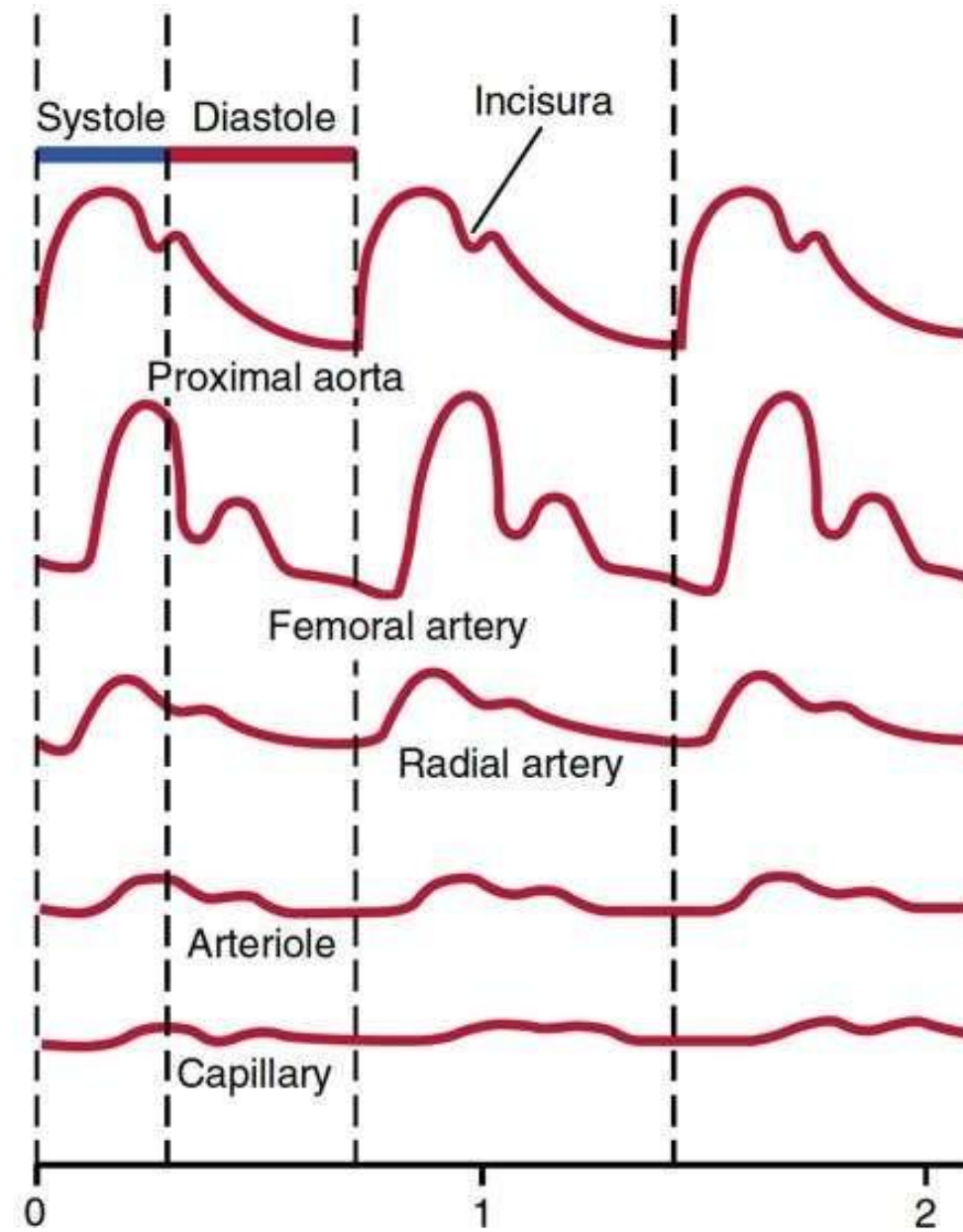


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As we see in the figure:

- The aortic pressure curve shows the normal systolic peak, diastolic fall. When we move forward in the circulation—from the aorta down to the capillaries—two things happen clearly:
 1. **The mean arterial pressure (MAP)**, which is the overall pressure, begins to decrease steadily as we go from the aorta → large arteries → arterioles → capillaries.
 2. **The pulse pressure (PP = systolic – diastolic)** also decreases from the aorta downward, but with one exception:
- In the large arteries, the pulse pressure becomes slightly increased compared to the aorta, and this is due to **wave reflection**.
- After the large arteries, the pulse pressure **drops sharply** as we enter the arterioles and then the capillaries.

illustration of the figure (Con...)

- This overall fall in pressure and the damping of pulse pressure happen because:
 - **Compliance decreases** as vessels become stiffer going forward.
 - **Resistance increases** sharply in the arterioles.
- Therefore, the pressure curve becomes **progressively flatter**, and starting from the arterioles you can see the systolic–diastolic difference narrowing, until in the capillaries the pressure becomes almost a **straight line** with minimal pulse pressure.

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
 - (2) compliance of the vessels.

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.

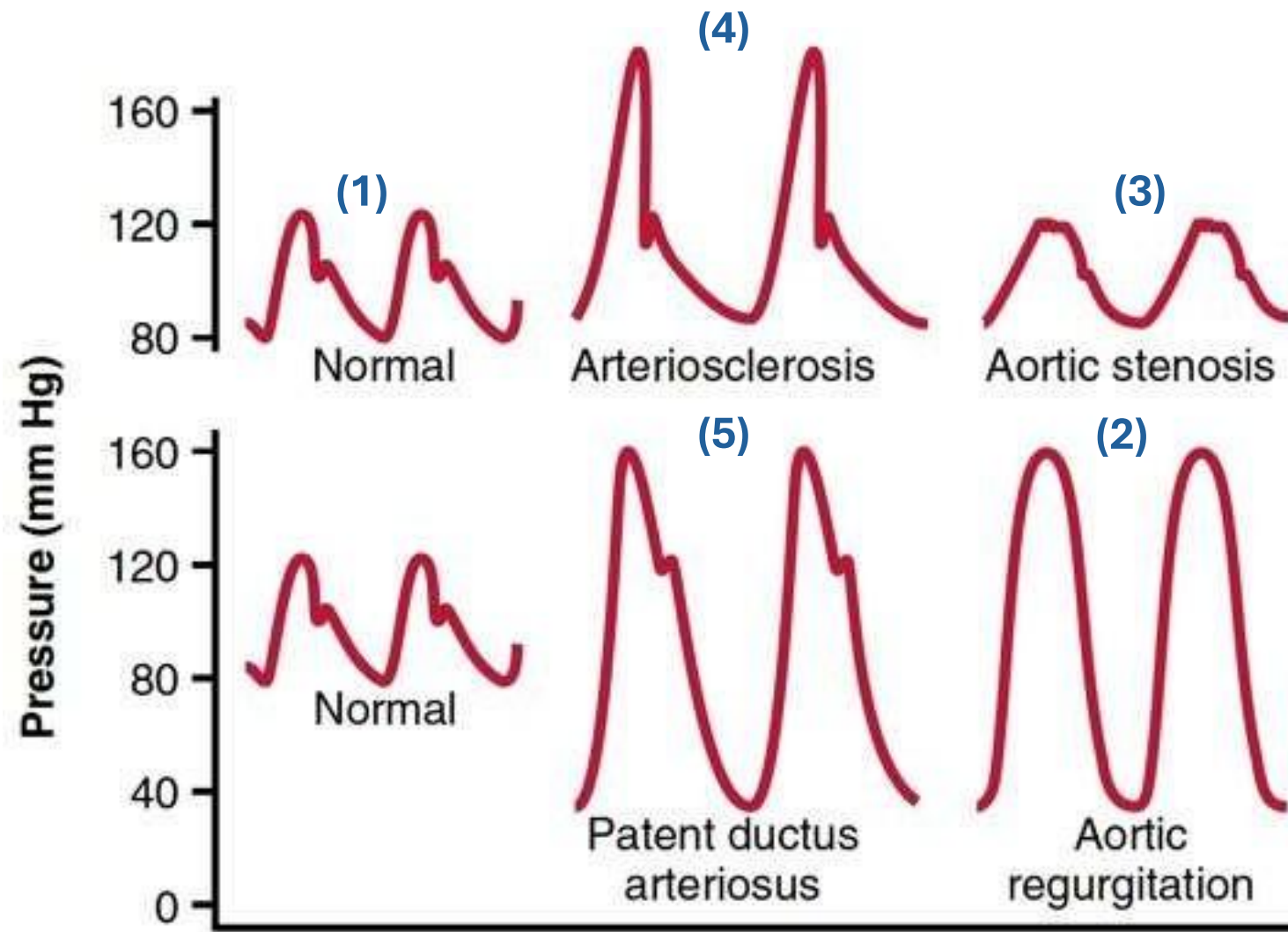


illustration of the figure

(1) The aortic pressure curve begins with diastolic pressure (80 mm Hg), which represents the pressure in the aorta when the heart is at rest. After the left ventricle contracts during systole, the pressure rises to about 120 mm Hg, reflecting the forceful ejection of blood into the aorta. Once the systolic phase ends, **the aortic valve closes**, and the aortic recoil (the natural elasticity of the aorta) causes a temporary backward flow of blood, creating the **dicrotic notch (or incisura)**. This notch represents the brief regurgitation, which will increase the aortic pressure slightly before the pressure begins to drop during the diastolic phase as the heart rests.

illustration of the figure

(2) In cases like **aortic regurgitation**, the **aortic valve** does not close properly, leading to an absence of the dicrotic notch. Since the valve fails to seal completely, blood flows back into the left ventricle, reducing the typical backflow seen in a normal pressure curve. This results in a decrease in diastolic pressure to around 40 mm Hg (compared to the normal 80 mm Hg). The excessive backflow forces the left ventricle to work harder, **increasing stroke volume** to compensate for the lost blood. This leads to a higher workload for the heart, requiring stronger contractions and more blood ejected in the next phase.

(3) In **aortic stenosis**, the aortic valve will be **narrowed**, making aortic valve **closure harder**. As a result, the dicrotic notch becomes very small or may not be seen clearly. Systolic pressure may be slightly elevated, while diastolic pressure may remain normal.

illustration of the figure

(4) In **arteriosclerosis**, which is different from atherosclerosis, the aortic wall **becomes stiff (due to loss of the elastin content)**, increasing resistance to blood ejection from the left ventricle. This causes systolic pressure to rise, diastolic pressure may be normal or decreased, and the diacrotic notch is still visible due to the stiff wall.

(5) In the case of **patent ductus arteriosus (PDA)**, the ductus, which is normally closed after the embryonic stage, remains open or opens intermittently. This allows blood to flow from the aorta back to the pulmonary trunk, **reducing aortic blood volume and lowering diastolic pressure**. Systolic pressure may be normal or slightly elevated, and the diacrotic notch may be absent or less visible depending on the flow through the ductus.

Mean arterial pressure

- The mean arterial pressure (MAP) is the average pressure driving blood forward into the tissues throughout the cardiac cycle.
- MAP, not the systolic or diastolic pressure, is the pressure that is monitored and regulated.
- MAP is not the halfway value between systolic and diastolic pressure.
- The reason is that arterial pressure remains closer to diastolic than to systolic pressure for a longer portion of each cardiac cycle.
- At resting heart rate, about two thirds of the cardiac cycle is spent in diastole and only one third in systole.

Arterial blood pressure

- $MAP = DBP + 1/3 PP$
- 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 essentially the same in all arteries.

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.

MAP Calculating Example

➤ Question:

Given Systolic Pressure (SP) = 100 mmHg and Diastolic Pressure (DP) = 70 mmHg, calculate the Mean Arterial Pressure (MAP).

✓ Method 1:

$$\text{MAP} = (1/3 * \text{SP}) + (2/3 * \text{DP})$$

$$\text{MAP} = (1/3 * 100) + (2/3 * 70)$$

$$\text{MAP} = 33.3 + 46.7 = 80 \text{ mmHg}$$

✓ Method 2:

$$\text{PP} = \text{SP} - \text{DP}$$

$$\text{PP} = 100 - 70 = 30$$

$$\text{MAP} = \text{DP} + (1/3 * \text{PP})$$

$$\text{MAP} = 70 + (1/3 * 30)$$

$$\text{MAP} = 70 + 10 = 80 \text{ mmHg}$$

Clinical Application

- ❖ In **the large arteries**, the systolic pressure, diastolic pressure, and pulse pressure (the difference between them) are **almost preserved** and do not change much along the arterial tree. Therefore, measuring these pressures in the arm provides a good indication of the pressures in other large arteries, such as the femoral artery.

Thank you



**PHYSIOLOGY
QUIZ
LECTURE 3**

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Corrections from previous versions:

Versions	Slide # and Place of Error	Before Correction	After Correction
V0 → V1			
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