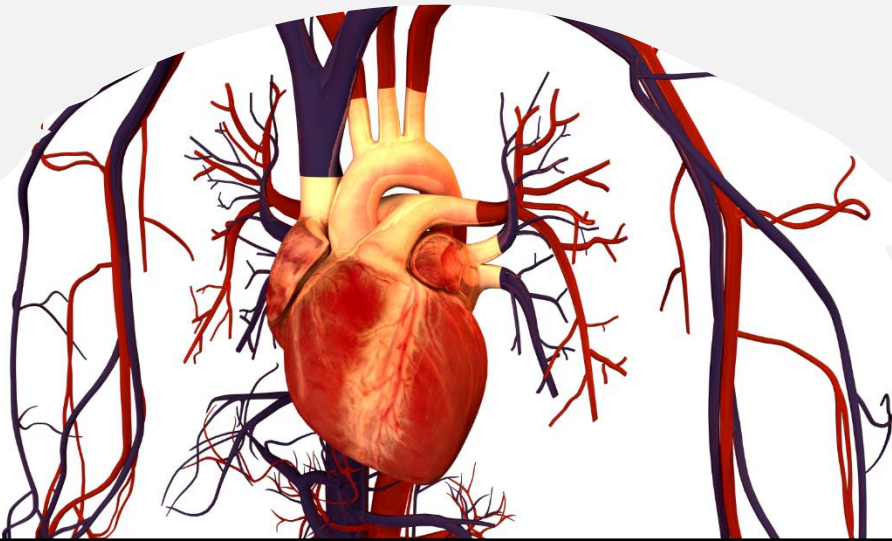




Arterial Blood Pressure

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Arterial Blood Pressure

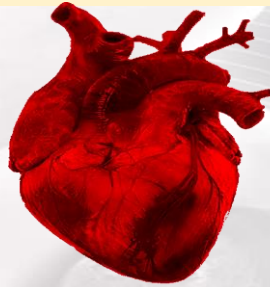
Lecture Outcomes

- ◆ To define & understand the concept of arterial blood pressure.
- ◆ To identify normal values & variations of the arterial blood pressure.
- ◆ To recognize pulse & mean arterial blood pressure.
- ◆ To understand the affecting & determining factors of arterial blood pressure.

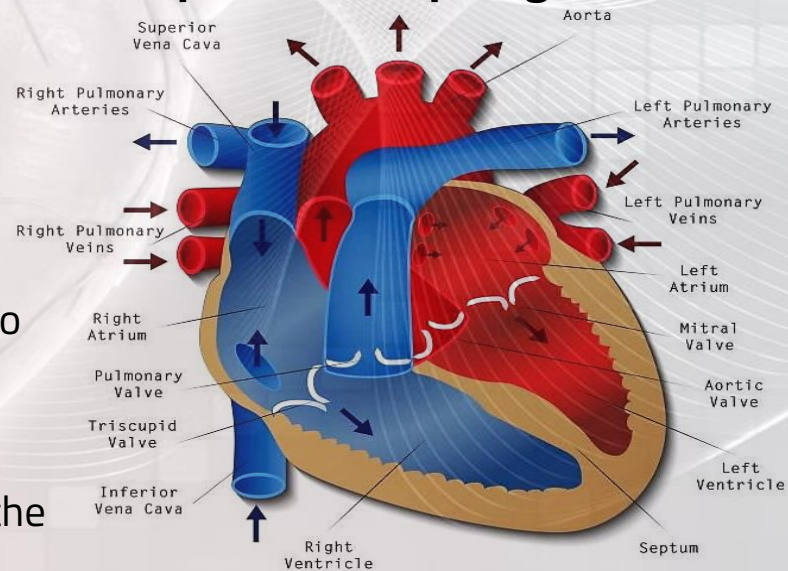
The Cardiovascular System

The cardiovascular system is part of the circulatory system, & consists of:

The heart

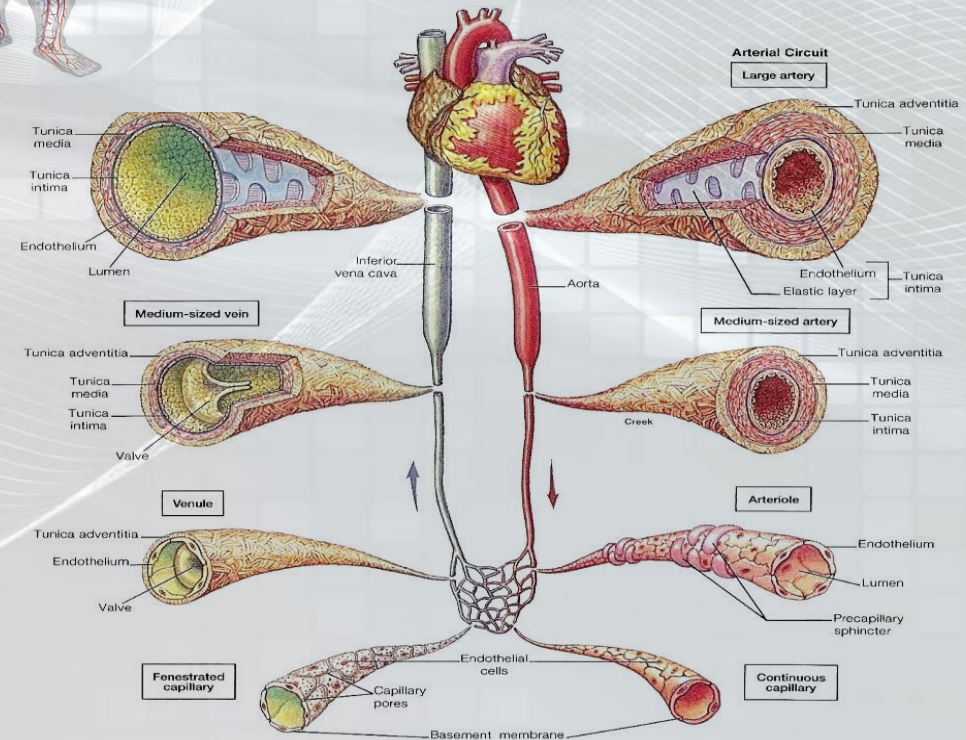
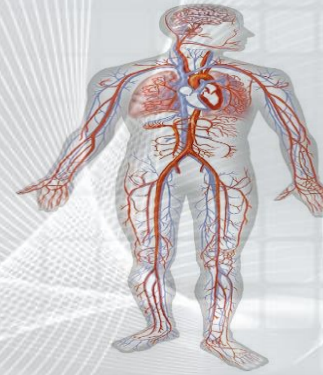


- ❑ A hollow muscular organ, of approximately fist size.
- ❑ Located in the chest - left to midline, behind the sternum, anterior to vertebral column, & superior to diaphragm.
- ❑ Consists of (4) chambers.
- ❑ Having (4) valves.
- ❑ **Right- sided pump:**
Carries deoxygenated blood to the pulmonary circulation.
- ❑ **Left- sided pump:**
Carries oxygenated blood to the systemic circulation.



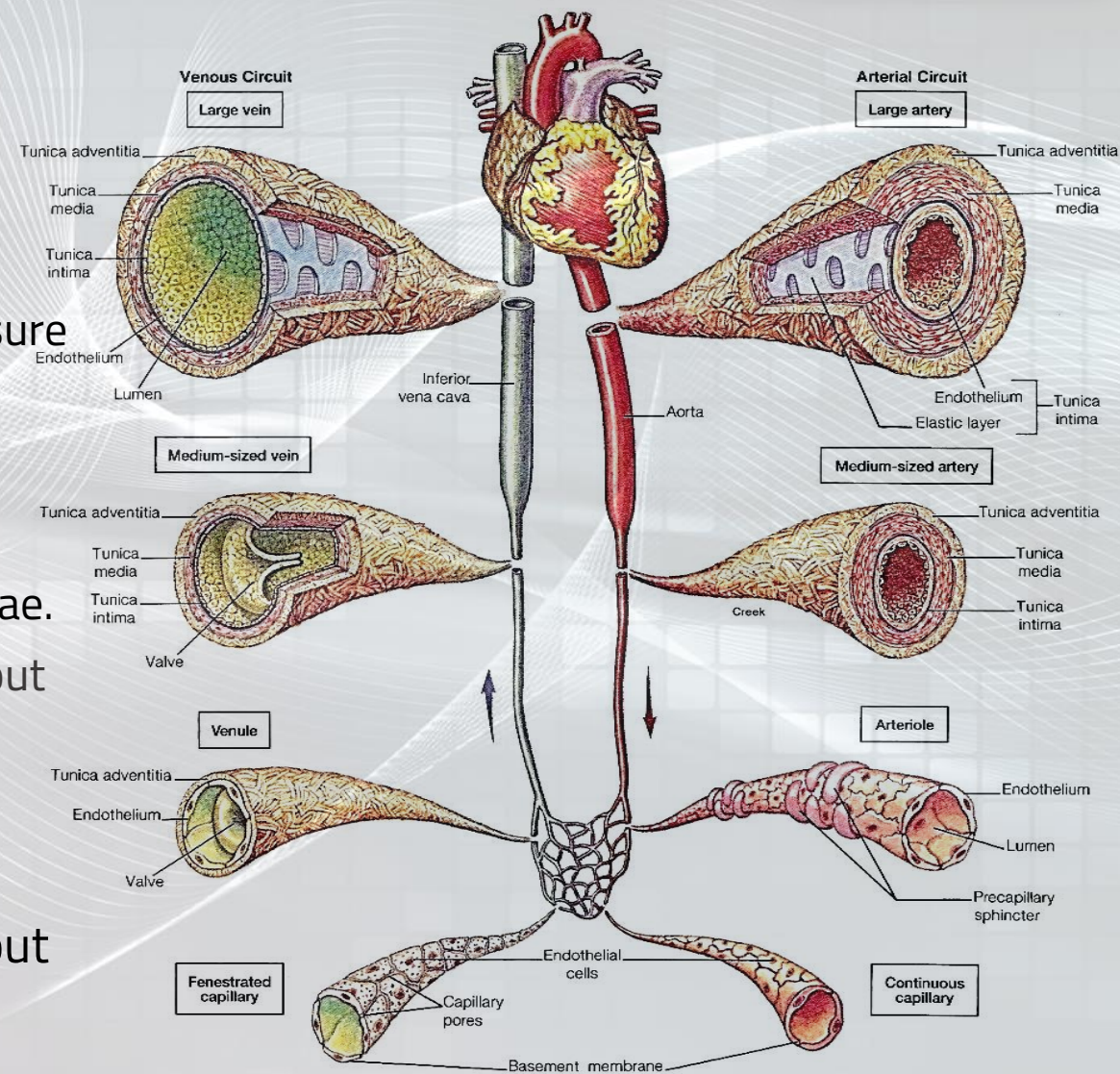
The vascular system

- ❑ Consists of arteries, arterioles, capillaries, venules, & veins.



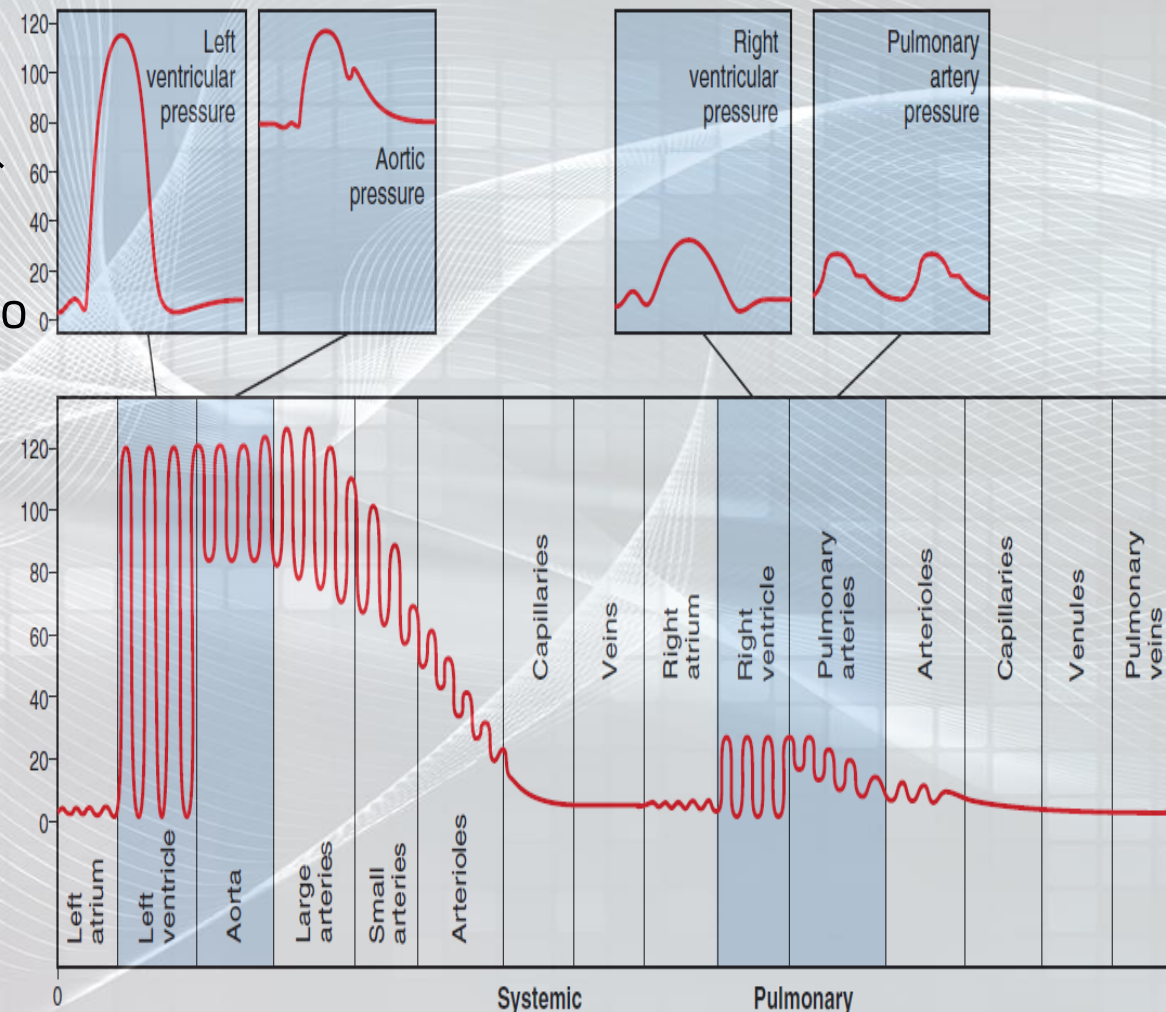
The Vascular System

- ❑ Consists of tube of vessels that blood flows through.
- ❑ These Vessels are arteries & veins.
- ❑ **Arteries** carry blood away from the heart.
 - The largest artery is the aorta.
 - Arteries are thicker to be able to handle the higher pressure of blood moving through them.
 - Arteries branch out into many smaller arteries.
- ❑ **Veins** push blood back to the heart.
 - The largest (2) veins are the superior & inferior vena cavae.
 - Veins have thin walls & accommodate more blood without increased pressure (= higher compliance
 - Veins are called the capacitance vessels.
 - Veins hold most of blood in body ($\approx 70\%$).
- ❑ A complex system connects veins & arteries throughout the body: **arterioles, capillaries, & venules.**
- ❑ Both arteries & veins have three main layers.



Pressure Changes Throughout Systemic Circulation

- ❑ Blood flows down a pressure gradient.
- ❑ Pressure is **highest** at the Heart (driving pressure) & **decreases** over distance.
- ❑ Pressure decreases more than 90% from the Aorta to Vena cava.
- ❑ **Greatest drop** in pressure occurs in **Arterioles** (resistance vessels) which regulate blood flow through tissues.
- ❑ No large fluctuations of pressure in Capillaries & Veins.
- ❑ Blood pressure reaches **120 mmHg** in the Aorta & drops to \approx **2 mmHg** in the Right Atrium.
- ❑ Veins have only 0 -10 mm Hg Pressure.



Blood pressure in different parts of the circulatory system.
~Guyton and Hall.

https://www.youtube.com/watch?v=qWti317qb_w

There are (3) Types of Blood Pressure:

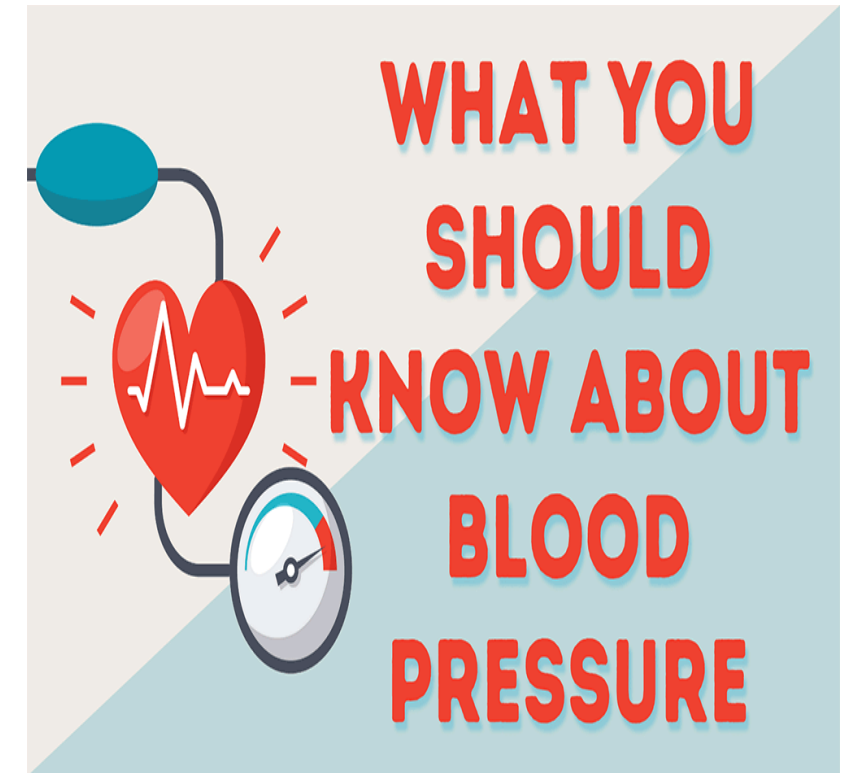
Depending
on nature
of blood
vessels

- **Arterial**
- **Venous**
- **Capillary**



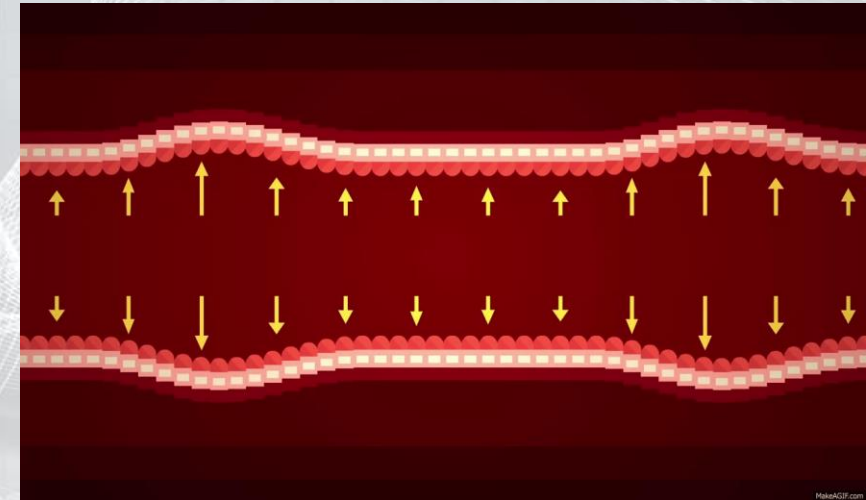
Arterial Blood Pressure

- **Arterial blood pressure (ABP) is one of the most important vital signs.**
- **It is important to keep normal levels of blood pressure for proper blood flow to the body's organs & tissues.**
- **Measured in mmHg, & sometimes in cmH₂O.**
(1mmHg = 1.36 cmH₂O)



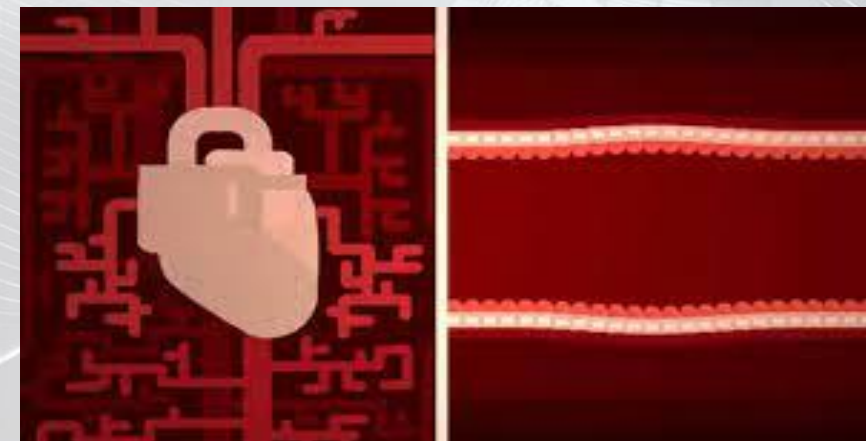
Definition of The Arterial Blood Pressure

Arterial blood pressure (ABP) is the lateral pressure force exerted by the blood flow on the arterial wall against any unit area of the vessel wall.



The pressure force of blood flow is created & determined by the pumping force of the heart.

<https://www.youtube.com/watch?v=J97G6BeYW0I>



Measures of Arterial Blood Pressure

Systolic BP

Maximum force exerted by the blood flow against any unit area of the vessel wall during maximum contraction (systole) of the heart.

Diastolic BP

Minimum force exerted by the blood flow against any unit area of the vessel wall during maximum relaxation (diastole) of the heart.

Normal adult arterial blood pressure measures \approx 120mmHg systolic/80mmHg diastolic.

Both numbers are important to determine the health state of the heart.

~Guyton and Hall

Variations of Arterial Blood Pressure

Arteries are **Pulsatile**, so arterial pressure varies.

- Normal adult Arterial Blood Pressure varies physiologically, with an ideal range of,

90 - 120 mmHg systolic



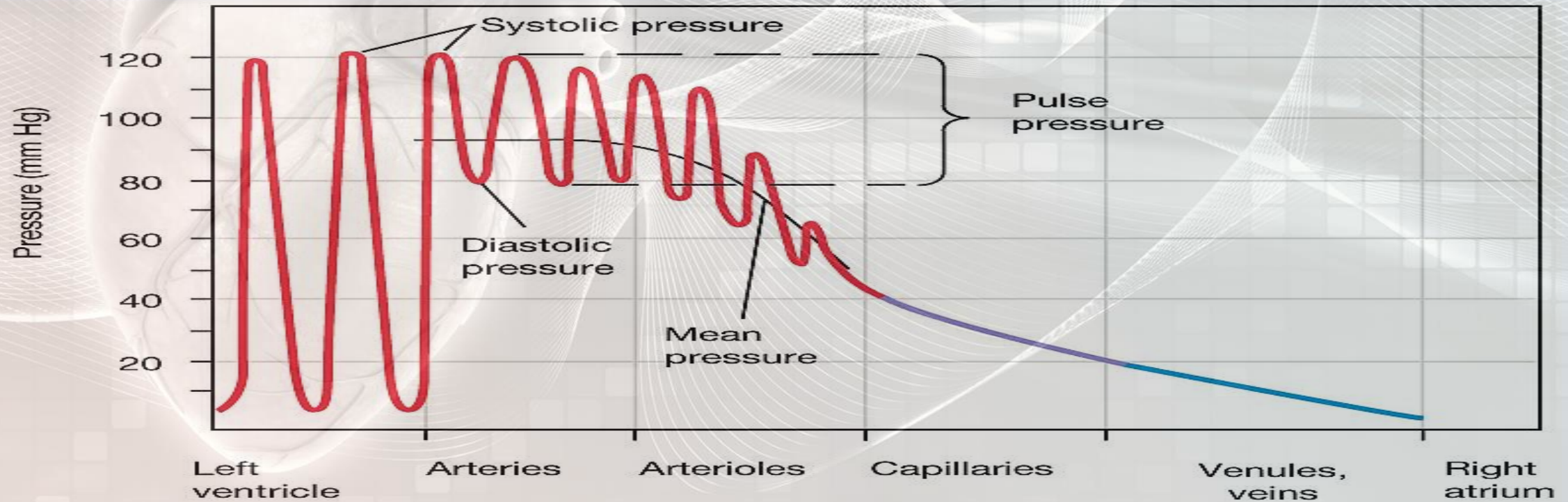
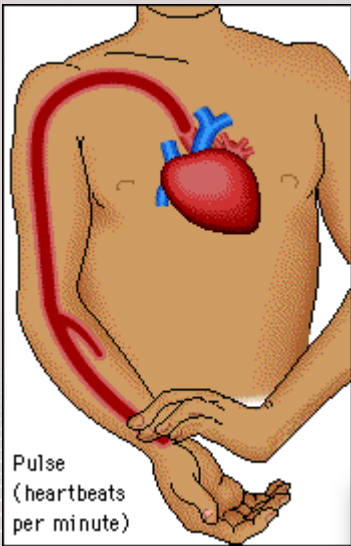
60 - 80 mmHg diastolic

Higher numbers indicate that the heart is working too hard to pump blood to the rest of the body.

Pulse Pressure

- Pulse rate is always same as the heart rate, which is the number of beats per minute (bpm).

$$\text{Pulse Pressure} = \text{Systolic Blood Pressure} - \text{Diastolic Blood Pressure}$$



Mean Arterial Blood Pressure

As arterial pressure varies, a single value is used to represent the overall driving pressure. This value is called the **Mean Arterial Pressure (MAP)**.

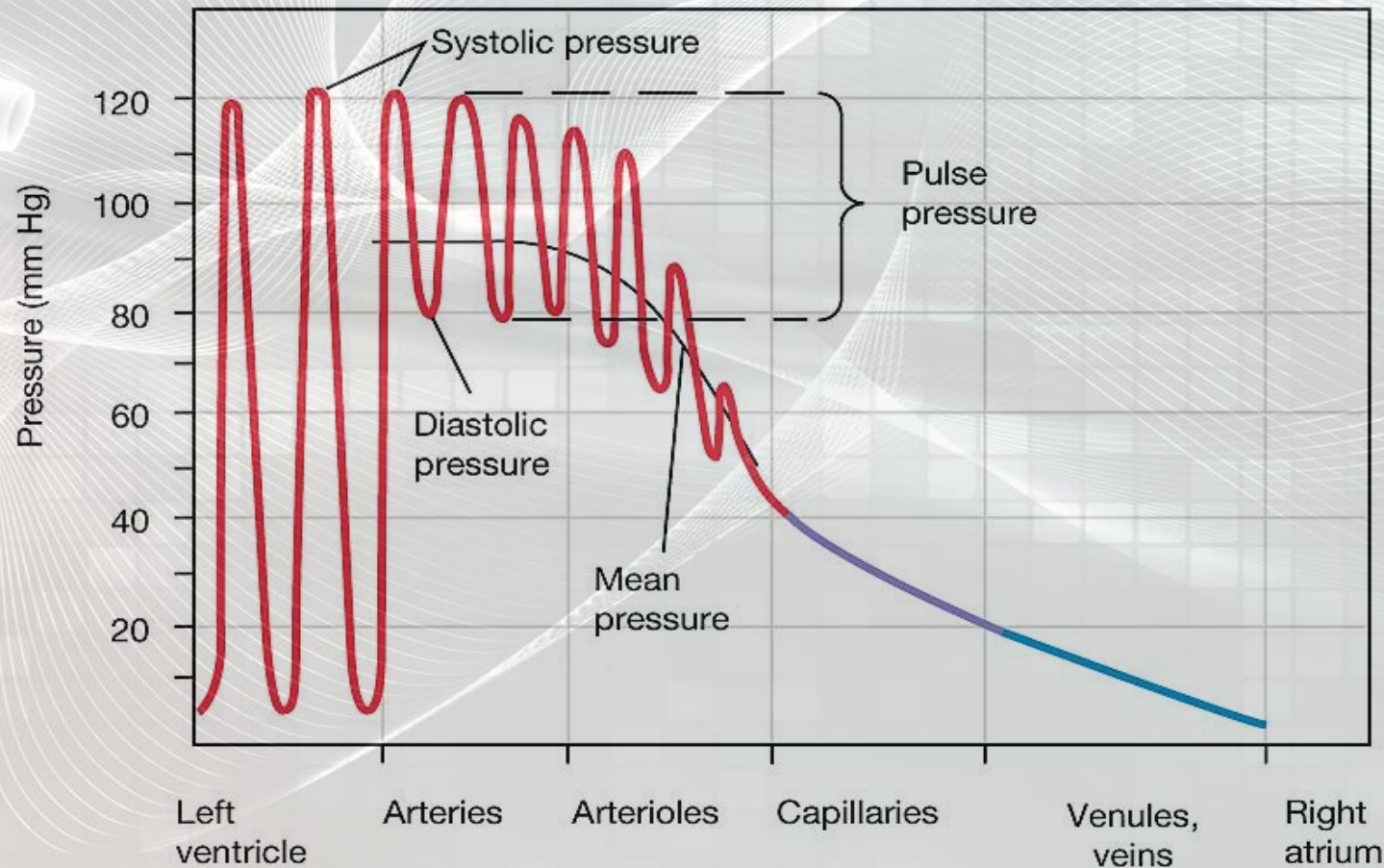
$$\text{MAP} = \text{Diastolic P} + \frac{1}{3} (\text{Systolic P} - \text{Diastolic P})$$

OR,

$$\text{MAP} = \frac{\text{Systolic P} + 2 (\text{Diastolic P})}{3}$$

Mean arterial pressure (**MAP**) is a better indicator of perfusion to the vital organs than systolic blood pressure.

P= Pressure.



Arterial Blood Pressure Guidelines

Old guidelines	New guidelines
Under 120 - NORMAL	Under 120 - NORMAL
120 - 129 HIGH NORMAL	120 - 129 ELEVATED BLOOD PRESSURE
130 - 139 HIGH NORMAL	130 - 139 STAGE 1 HYPERTENSION
OVER - 140 HYPERTENSION	OVER - 140 STAGE 2 HYPERTENSION

Adult BP range:
90 – 120 /
60 – 80 mmHg

Arterial Blood Pressure Chart

American Heart Association (AHA) Blood Pressure Categories Guidelines

Nov
2017



Blood Pressure Categories

BLOOD PRESSURE CATEGORY	SYSTOLIC mm Hg (upper number)		DIASTOLIC mm Hg (lower number)
NORMAL	LESS THAN 120	and	LESS THAN 80
ELEVATED	120 – 129	and	LESS THAN 80
HIGH BLOOD PRESSURE (HYPERTENSION) STAGE 1	130 – 139	or	80 – 89
HIGH BLOOD PRESSURE (HYPERTENSION) STAGE 2	140 OR HIGHER	or	90 OR HIGHER
HYPERTENSIVE CRISIS (consult your doctor immediately)	HIGHER THAN 180	and/or	HIGHER THAN 120

©American Heart Association

heart.org/bplevels

- ❑ The AHA/ACC started synthesizing evidence and publishing guidelines specifically for hypertension (HTN) in **2014**.
- ❑ The last update was in **2017** & the major change at that time was:
 - Lowering the definition of HTN from $\geq 140/\geq 90$ mmHg to $\geq 130/\geq 80$ mmHg.
 - Re-classified "pre-HTN" as elevated blood pressure (120-129/<80 mmHg).

Michael R Goetsch, et al. 2021 "New Guidance on Blood Pressure Management in Low-Risk Adults with Stage 1 Hypertension". <https://www.acc.org/latest-in-cardiology/articles/2021/06/21/13/05/new-guidance-on-bp-management-in-low-risk-adults-with-stage-1-htn>

<https://www.bing.com/videos/search?q=what+is+blood+pressure&&view=detail&mid=A54D3D66D3EF07C8A18AA54D3D66D3EF07C8A18A&&FORM=VRDGAR>

American Heart Association (AHA)

- ❑ The AHA/ACC has released a scientific statement in **2021** offering new guidance for management of stage 1 hypertension among patients with low ASCVD risk.
- ❑ Among low-risk adults (no ASCVD or 10-year CVD risk <10%) with stage 1 hypertension (blood pressure 130-139/80-89 mmHg).
- ❑ Management of stage 1 hypertension starts with nonpharmacologic therapy. If blood pressure remains uncontrolled at 3-6 months, consider starting pharmacologic therapy.

Table: AHA/ACC^a Guideline Recommendations by Blood Pressure Category.

BP ^b Category	Pressure Ranges	Recommendations
Normal BP	<120/<80 mmHg	Promote healthy lifestyle; reassess BP annually.
Elevated BP	120-129/<80 mmHg	Start with nonpharmacologic therapy, reassess BP in 3-6 months.
Stage 1 Hypertension	130-139/80-89 mmHg	ASCVD^c or 10-year CVD^d risk ≥10%: Start with both nonpharmacologic and pharmacologic therapy. Reassess BP in 1 month. If at goal, reassess every 3-6 months. If not at goal, assess for adherence and consider intensification of therapy.
		No ASCVD and 10-year CVD risk <10%: Start with nonpharmacologic therapy, reassess BP in 3-6 months. If not at goal, consider initiation of pharmacologic therapy.
Stage 2 Hypertension	≥140/≥90 mmHg	Start with both nonpharmacologic and pharmacologic therapy. Reassess BP in 1 month. If at goal, reassess every 3-6 months. If not at goal, assess for adherence and consider intensification of therapy.

a: AHA/ACC, American Heart Association, American College of Cardiology.

b: BP, blood pressure.

c: ASCVD, atherosclerotic cardiovascular disease.

d: CVD, cardiovascular disease.

Michael R Goetsch, et al. 2021 "New Guidance on Blood Pressure Management in Low-Risk Adults with Stage 1 Hypertension".
<https://www.acc.org/latest-in-cardiology/articles/2021/06/21/13/05/new-guidance-on-bp-management-in-low-risk-adults-with-stage-1-htn>

Physiological Factors Affecting Arterial Blood Pressure

- **Sex:** Male > Female , equal at menopause.
- **Age:** ABP rises with age, elderly > children, due to atherosclerosis, diabetes, ...
- **Body mass index:** ABP rises with body size.
- **Emotions:** ABP (↑) due to neural & hormonal factors.
- **Exercise:** (↑) ABP due to ↑ venous return.
- **Hormones:** Some hormones like adrenaline, noradrenaline & thyroid H (↑) BP.
- **Gravity:** ABP is higher in lower limbs than upper limbs.
- **Race:** (? dietary factors, or stress)
- **Sleep:** ABP (↓) due to ↓ venous return.
- **Pregnancy:** ABP (↑) due to ↑ in hemodynamics.
- **Temperature:** ABP (↓) with Heat due to vasodilatation, & (↑) with Cold due to vasoconstriction.

Factors Determining Arterial Blood Pressure

- **Cardiac output (Flow.)**
- **Peripheral Resistance.**
- **Blood volume.**

Blood Pressure = Cardiac Output X Peripheral Resistance

MAP

CO

**Stroke
Volume**

**Heart
Rate**

PR

Blood Vessels' Size

Blood Viscosity

Blood Vessels' Length

The Cardiac Output (CO)

Cardiac Output (CO) is the volume of blood pumped by the ventricle per minute.

- CO is expressed in L/min.
- **At rest, CO is 4-6 L/min (\approx 5 L/min),** in healthy adults, when the HR = 70 bpm.
- Since normal total **adult resting blood volume \approx 5 L**, the blood volume circulates through the body once each minute.



The Cardiac Output (CO)

Cardiac Output = Stroke Volume X Heart Rate

- **CO is a function of:**
 - Stroke volume.
 - Heart rate.
- **CO is Determined by:**
 - The Stroke volume.
 - The Heart rate.

Variables Affecting Stroke Volume & Heart Rate

$$\text{Cardiac Output} = \text{Stroke Volume} \times \text{Heart Rate}$$

- Any factor that affects these parameters will affect the CO.

Ventricular Myocardium

Ventricular End-Diastolic Volume (EDV), which is affected by the Preload (Venous Return)

Contractility (Myocardial function)

Afterload (Peripheral Resistance)

SA node

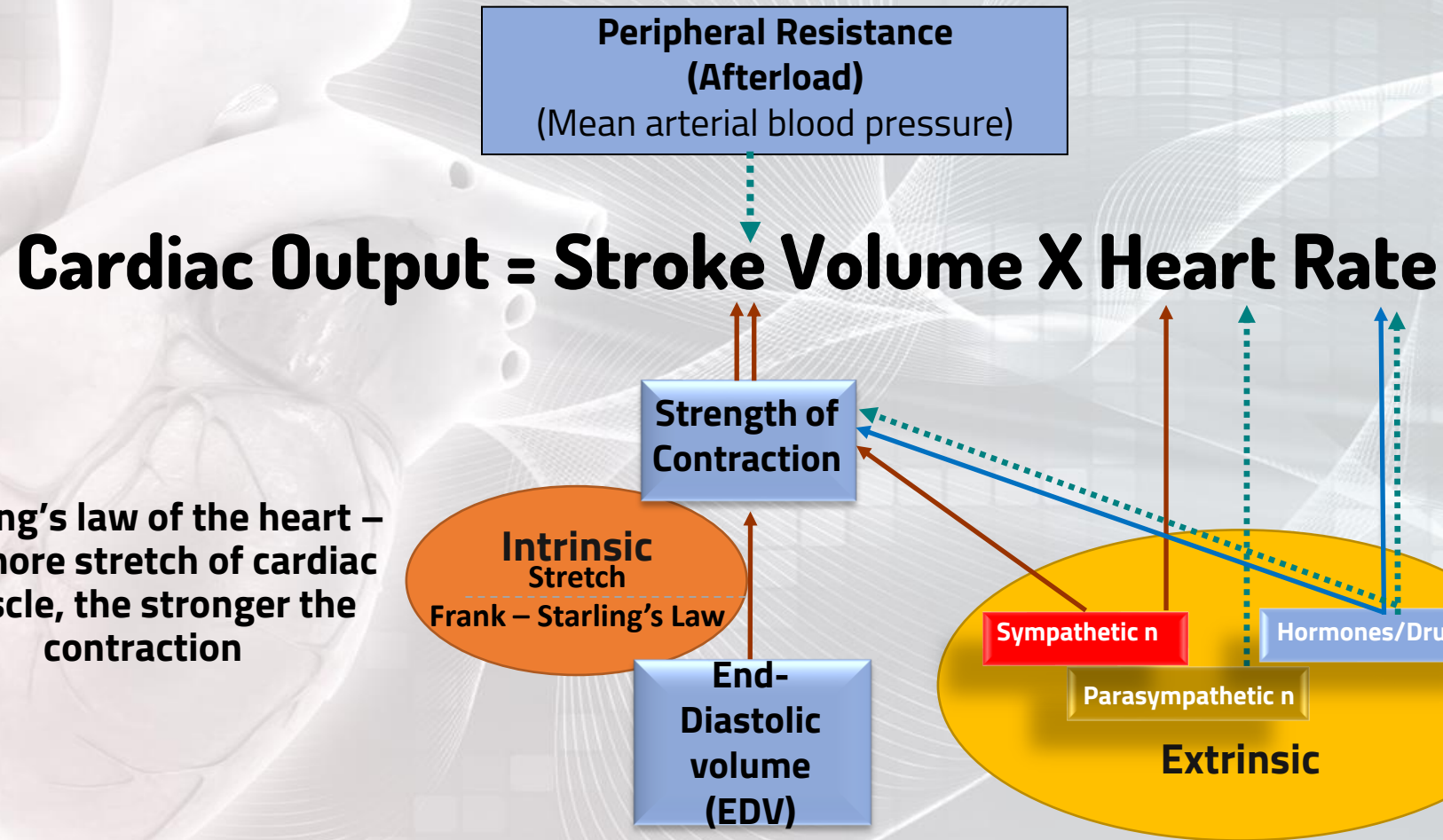
Autonomic Innervation

Hormones/ Drugs

I: Regulation of The Cardiac Output

Cardiac Output = Stroke Volume X Heart Rate
(SV) (HR)

Regulation of The Cardiac Output



The Stroke Volume

- **Stroke volume (SV)** is the amount of blood pumped by ventricle per beat.
- **Stroke volume (SV) \approx 70-80 mL/beat.**
- **Stroke volume (SV) is determined by the:**
 - End- diastolic volume.
 - End- systolic volume.

End-diastolic Volume (EDV) - End-systolic Volume (ESV) = The Stroke Volume

Volume of blood in
ventricles at the end of
diastole
(*End-Diastolic volume*)
 \approx 110-130 mL

Amount of blood left in
ventricles at the end of
systole
(*End-Systolic volume*)
 \approx 40-60 mL

=

*The stroke
volume
(70-80 mL)*

Regulation of Stroke Volume

- Stroke volume = EDV - ESV
- Stroke volume (SV) is regulated by 3 variables:

End- Diastolic Volume (EDV):

Affected by the
Venous return
(Preload).

Contractility (Strength of contraction):

Affected by
Starling's law &
sympathetic
innervation.

Total Peripheral
Resistance (Afterload):

Affected by **size** &
length of blood
vessel. **Thickness**
(**viscosity**) of
blood.

Affects
(EDV)
(ESV)
(ESV)

EDV= End diastolic volume; ESV= End systolic volume.

Preload: (Venous Return)

- **Preload (venous return)**, is the amount of blood returns to the heart from the veins, into the atria.

End- Diastolic Volume: (EDV)

- **End- diastolic volume (EDV)**, is amount of blood presented to the ventricles from the venous return prior to ventricular ejection.
- **When venous return increases, EDV increases & stretches or lengthens the ventricular muscle fibers.**

How Does The EDV Affect The SV & CO? Frank-Starling's Mechanism

- **An increase in the End- diastolic volume (EDV)** amount will increase the myocardial fibers stretch, thus increasing the initial fiber length.
- **The increase in the initial fiber length will increase the strength of myocardial contractility.**

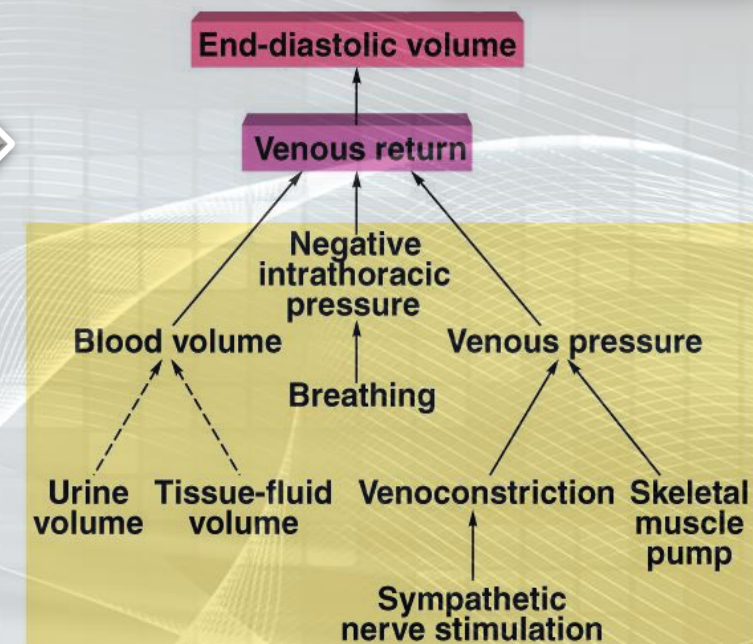
Factors Affecting End- Diastolic Volume

End Diastolic Volume (EDV):

Affected by
the **Venous
return
(Preload)** .

The Venous Return is affected by:

1. Blood volume.
2. Pressure gradient.
3. Gravity.
4. Veno-constriction: caused by sympathetic nervous system.
5. Presence of valves in the large veins.
6. Skeletal muscles pump.
7. Respiratory activity (Deep inspiration \uparrow venous return).



Factors Affecting Venous Return (Preload):

1. Blood volume

- At constant venous capacity, as the blood volume $\uparrow \rightarrow$ the Mean Circulatory Pressure $\uparrow \rightarrow \uparrow$ VR.
- At constant venous capacity, as the blood volume $\downarrow \rightarrow$ the Mean Circulatory Pressure $\downarrow \rightarrow \downarrow$ VR.
- **Venous capacity** is the volume of blood that the veins can accommodate.

Factors Affecting Venous Return (Preload):

2. Pressure Gradient

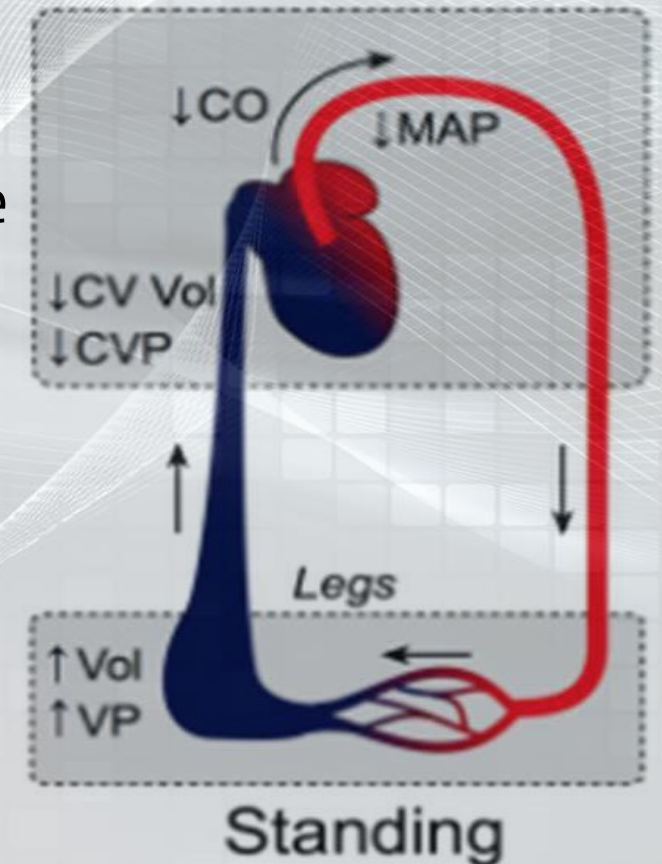
- \uparrow Pressure gradient \rightarrow \uparrow venous return.
- Since the right atrium is the site of venous blood collection from all around the body \rightarrow the pressure inside the right atrium i.e. **Right Atrial Pressure (RAP)** is called **Central Venous Pressure (CVP)**
- The pressure is highest in large arteries & continue to drop throughout the pathway, reaching \approx zero-2 mmHg at **right atrium**.
- The high pressure in the arteries 120 mmHg forces the blood to continually move into areas where the pressure is lower.

Factors Affecting Venous Return (Preload):

3. Gravity

Standing:

- When a person initially stands, right atrial pressure & ventricular EDV falls, which decreases stroke volume by the Frank-Starling mechanism. So, CO & arterial pressure decrease.
- The flow through the entire systemic circulation falls because arterial pressure falls, therefore the pressure gradient driving flow throughout the entire circulatory system is decreased.



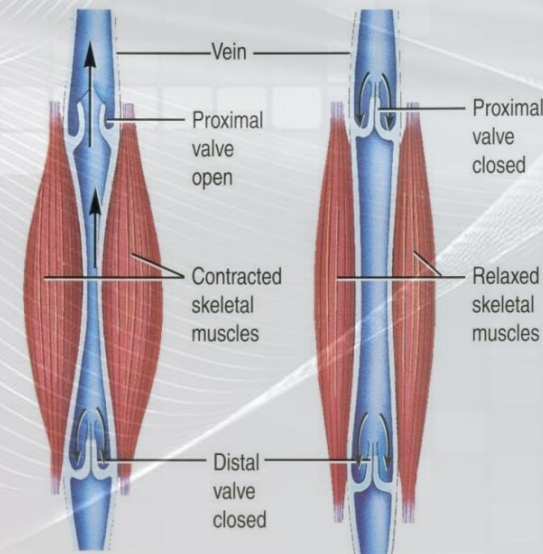
Factors Affecting Venous Return (Preload):

4. Veno-constriction: By sympathetic stimulation \uparrow venous return (VR).

5. Presence of valves: Permit blood to move forward towards the heart & prevent it from moving back toward the tissues.

6. The skeletal muscle pump:

Rhythmical contraction of limb muscles (as occurs during walking, running or swimming) \rightarrow \uparrow VR by the muscle pump mechanism that squeeze the blood vessels between muscle fibers.



(a) Contracted skeletal muscles

(b) Relaxed skeletal muscles

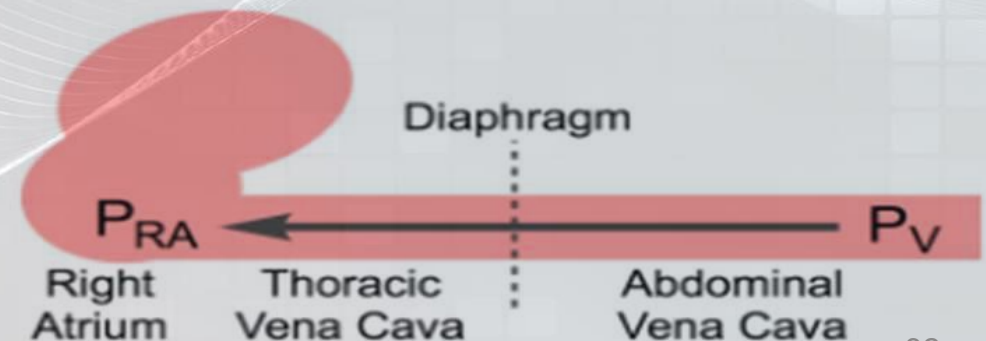
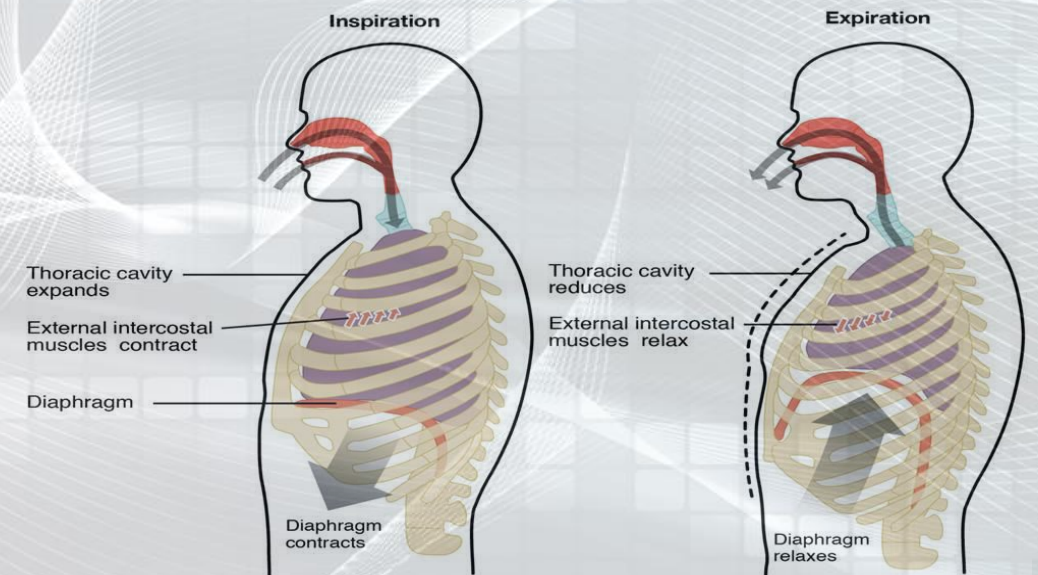


Skeletal muscle pump enhancing venous return

Factors Affecting Venous Return (Preload):

7. Respiratory Activity: Respiratory/Thoracic Pump

- **Inspiration** → ↑ **venous return (VR)** because of a decrease in the right atrial pressure (RAP).
- In **Valsalva maneuver** (forceful expiration against a closed glottis), intrapleural pressure become positive which is transmitted to the large veins in the chest → ↓ VR.



Factors Affecting Venous Return (Preload):

Venous return (VR) is decreased when:

- 1. The Right Atrial Pressure (RAP) is increased.**
- 2. Pumping capability becomes diminished.**
- 3. The Nervous circulatory reflexes are absent.**

Factors Affecting End- Diastolic Volume

EDV is \uparrow with:

- Increased total blood volume.
- Increased venous return.
- Increased venous tone.
- Increased skeletal muscle pump (exercise).
- Increased negative intrathoracic pressure.
- Stronger atrial contraction.

EDV is \downarrow with:

- Standing.
- Decreased venous return.
- Increased intrapericardial pressure.
- Decreased ventricular compliance.

End- Systolic Volume (ESV)

- **End- Systolic volume (ESV) is the volume of blood remaining in the ventricle at the end of systole**

↓ SV → ↑ ESV

↑ SV → ↓ ESV

- **End- Systolic volume (ESV) is determined by the:**

I: **Cardiac contractility.**

II: **Afterload.**

Factors Affecting End- Systolic Volume

Contractility (Strength of contraction):

Affected by **Starling's law** & **sympathetic innervation**.

■ **Intrinsically** affected by: **Frank Starling's law of the heart**, which is affected by the End Diastolic Volume (EDV).

[**Frank Starling's law** states that the force of contraction depends on the initial length of the muscle].

■ **Extrinsically** affected by **sympathetic stimulation, hormones & drugs**.

↑↑ **contractility** → ↑↑ SV → ↓↓ ESV

↓↓ **contractility** → ↓↓ SV → ↑↑ ESV

SV= Stroke volume; ESV= End systolic volume.

Factors Affecting End- Systolic Volume

Total Peripheral Resistance (Afterload):

Affected by
size & length
of blood vessel.
Thickness
(**viscosity**) of
blood.

- Afterload is the resistance to flow against (oppose) ventricular contraction.
- **Afterload increases by** any factor that restricts arterial blood flow: Such as an increase in arterial pressure (vasoconstriction).
- As the **afterload increases**, the **Stroke volume decreases**.
- **In response to all regulatory mechanisms:**
 - Vasoconstriction will $\uparrow\uparrow$ Peripheral Resistance \rightarrow**
 $\downarrow\downarrow$ peripheral Flow \rightarrow $\downarrow\downarrow$ SV \rightarrow $\uparrow\uparrow$ ESV.
 - Vasodilatation will $\downarrow\downarrow$ Peripheral Resistance \rightarrow**
 $\uparrow\uparrow$ peripheral Flow \rightarrow $\uparrow\uparrow$ SV \rightarrow $\downarrow\downarrow$ ESV.

SV= Stroke volume; ESV= End systolic volume.

The Heart Rate (HR)

- ❑ The heart rate is the number of heart beats per minute.
- ❑ Normal heart rate (HR) = 60-100 beats/min
 - ❑ > 100 beats/min → **Tachycardia**.
 - ❑ < 60 beats/min → **Bradycardia**.
- ❑ **As the HR increases**, the CO increases.
 - \uparrow HR up to ≈ 180 bpm, ventricular filling is adequate and CO \uparrow .
 - At very high HR, filling may be compromised to a degree that CO falls.
- ❑ **As the HR decreases**, the CO decreases.
- ❑ The HR has an influence on cardiac contractility as well Frequency-Force Relation.

Regulation of the Heart Rate

The Heart Rate (HR) is regulated by:

- ❑ **Autonomic nervous system** through cardiac control centers in medulla oblongata in the brain stem:
 - Cardiac-accelerator Center (Vasomotor center) → Sympathetic nerve fibers.
 - Cardiac-inhibitory Center → Parasympathetic nerve fibers.
 - **Sympathetic nervous stimulation, increases the HR & the contractility.**
 - **Parasympathetic nervous stimulation (vagus nerve) slows the HR.**
- ❑ **Hormones and Drugs:**
 - Epinephrine, Norepinephrine, or thyroxine hormone, increases the HR.
 - Increased calcium level concentration in the blood, causes prolonged contraction.
 - Reduced calcium level concentration in the blood, decreases the HR.

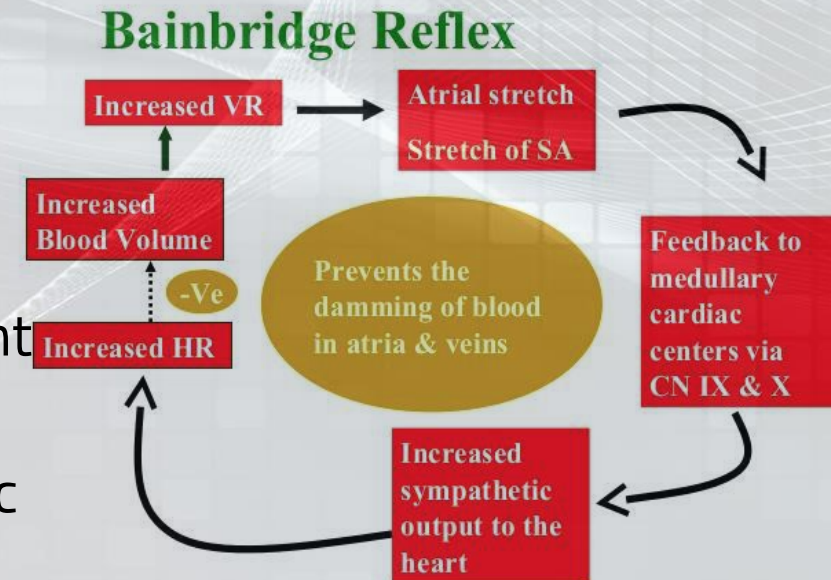
Regulation of The Heart Rate

Physical factors:

- **Age:** Resting HR is faster in fetus and then gradually decreases throughout life.
- **Gender:** HR is faster in females (72-80 beats/min) than in males (64-72 beats/min).
- **Temperature:** Heat increases HR as occurs in high fever. Cold has the opposite effect.
- **Exercise:** Increases HR through sympathetic nervous system.

Blood volume:

- Atrial reflex (Bainbridge reflex).
- This reflex adjusts HR in response to VR.
- Increase blood volume, stimulates stretch receptors in right atrium.
- This triggers increase in HR through increased sympathetic activity.



II: Peripheral Resistance

Peripheral Resistance (R) : Affecting Factors

- ❑ Resistance (R) = tendency of the vascular system to oppose the blood flow (Q).
- ❑ Vascular Resistance (R) is **affected by** the pressure difference (ΔP) & the blood flow (Q).

$$R = \frac{\Delta P}{Q}$$

$$\text{Flow (Q)} = \frac{1}{R}$$

Resistance to flow is influenced by:

Length of the tube (**L**), radius of the tube (**r**), & viscosity of the blood (**h**)

- ❑ In a **human**, the **length (L)** of vascular system is **fixed**. Accordingly, the blood viscosity (**h**) & radius (**r**) of the blood vessels have the largest effects on the resistance.

Poiseuille's Law $R = 8hL / \pi r^4$

Flow (Q) and Poiseuille's Law

- Fluid Flow (Q) through Cylindrical Tubes.
- Flow decreases (↓) when resistance (R) increases.
- Flow resistance decreases (↓) when vessel diameter increases.

$$Q = \frac{\Delta P}{R}$$

$$Q = \frac{(P_i - P_o) \pi r^4}{8\eta L}$$

**Difference
in Pressure**

Viscosity

Length

Radius

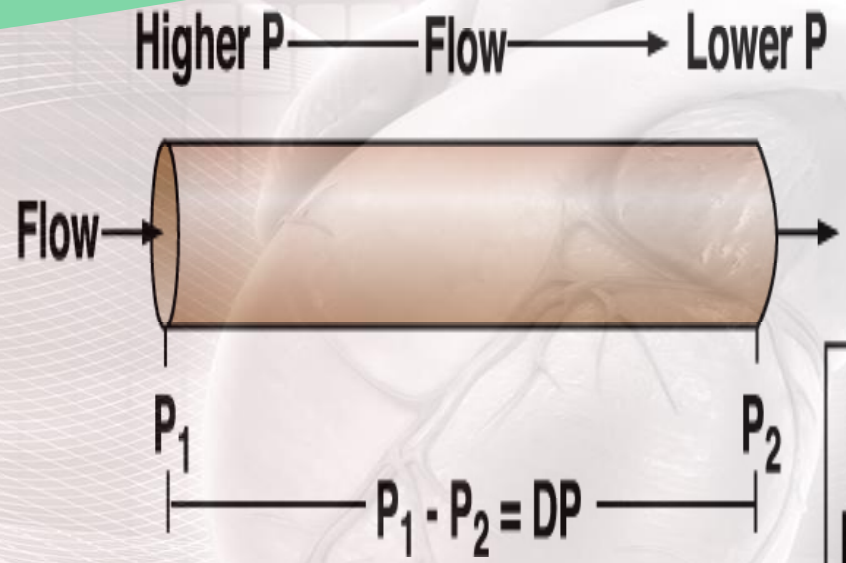
Blood Flow (Q) & Pressure

Blood flows down a pressure gradient.

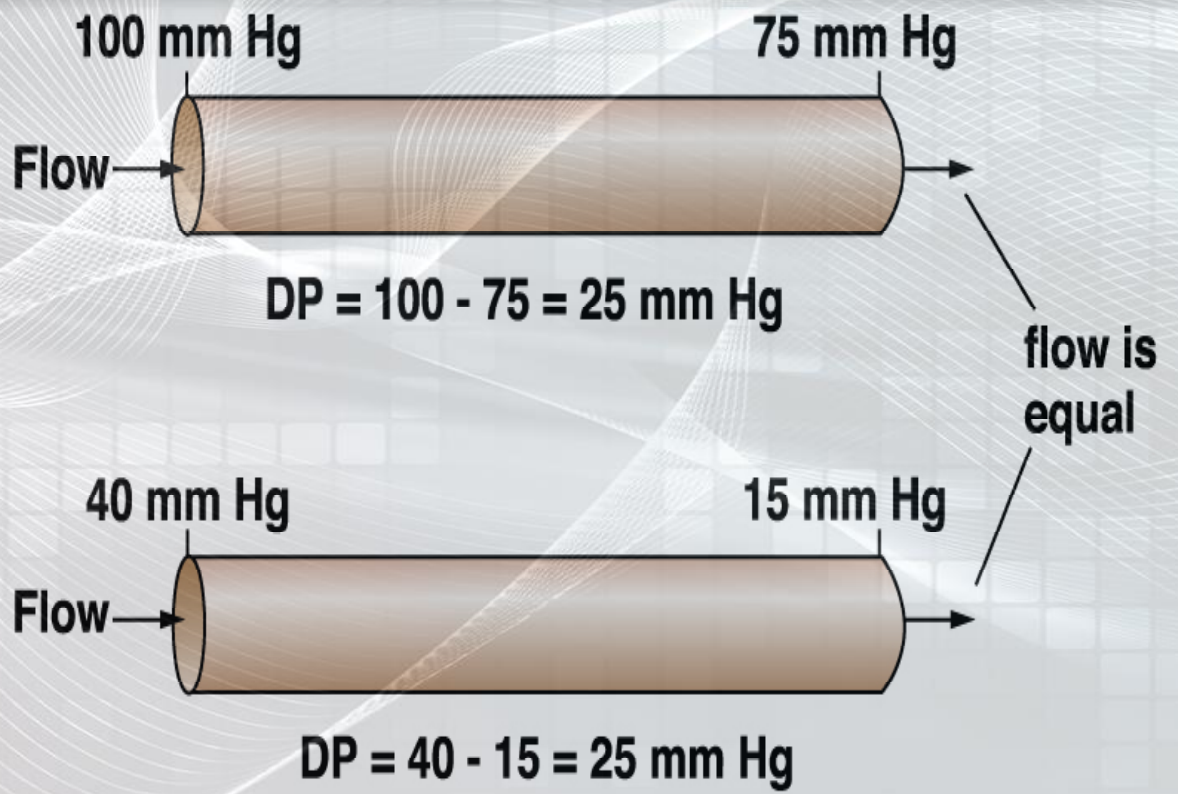
Pressure difference (ΔP) is directly proportional to the Flow (Q).

Resulting pressure is called the **driving pressure** in vascular system

$$Q = \frac{\Delta P}{R}$$



P = Pressure
DP = Pressure gradient



- **Absolute value** of pressure is **not important** to flow (Q), but the **difference in pressure** (ΔP or gradient) is **important** to determine the **flow** (Q).

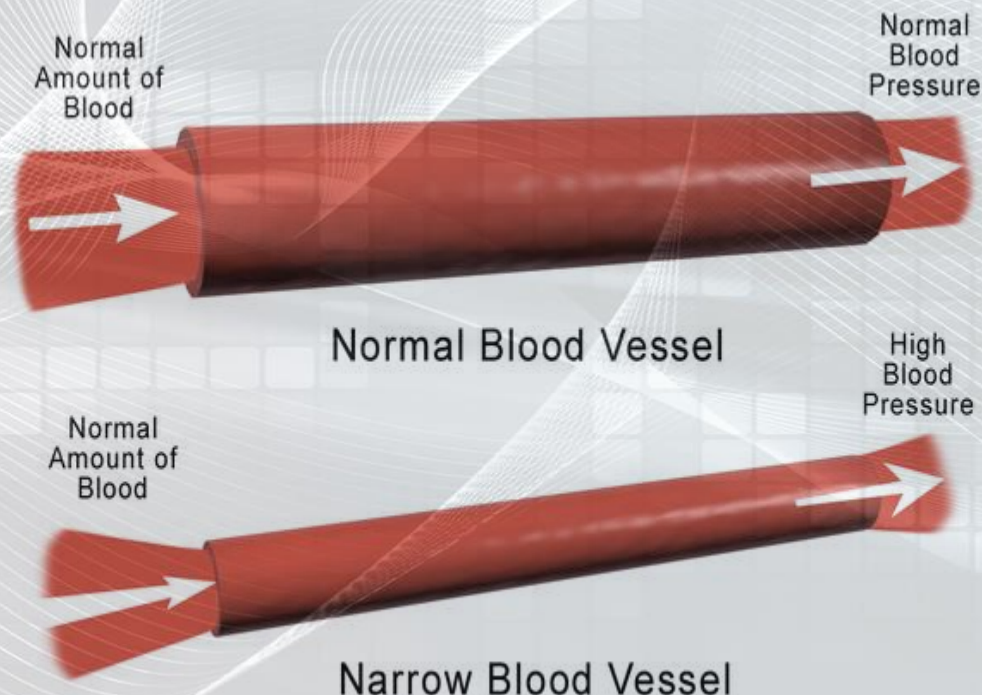
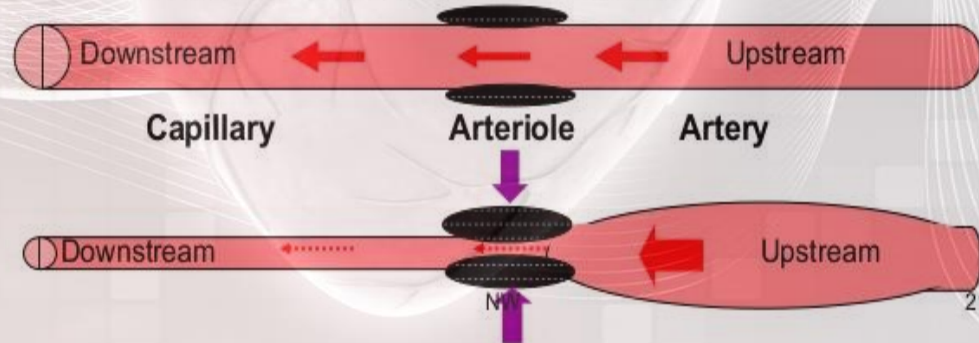
Effect of Radius (r) on flow & Pressure

(r) is **directly** proportional to the **flow** (Q).

(r) is **inversely** proportional to the **pressure** (P).

Vascular tone - effects

- Increased tone in a segment of blood vessel
- e.g. **increased arteriolar tone**
- → decreased radius of arteriole
- → greatly increased resistance to blood flow
- → greatly decreased blood flow across the arteriole
- → the effects on blood volume
 - Increased upstream (in the artery)
 - Decreased downstream (in the capillaries)



Blood Pressure Blood Flow

Factors Affecting Vessel Diameter: Radius (r)

❑ Vasoconstrictors:

- Oxygen (O_2).
- Epinephrine & Nor-epinephrine.
- Angiotensin II.
- Vasopressin (Anti-Diuretic hormone).
- Endothelin-1.
- Thromboxane A_2 .
- Cold.
- α_1 R.

❑ Vasodilators:

- CO_2 & other metabolites.
- Nitric oxide (NO).
- Histamine.
- $[H^+]$
- Adenosine
- Atrial Natriuretic Peptide (ANP).
- Prostacyclin; PGI_2 .
- β_2 R.

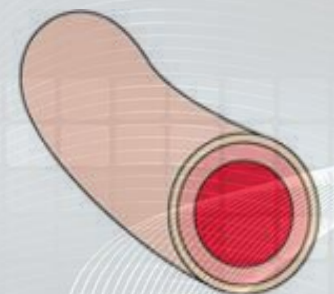
Normal arteriolar tone

Vasoconstriction

(increased contraction of circular smooth muscle in the arteriolar wall, which leads to increased resistance and decreased flow through the vessel)

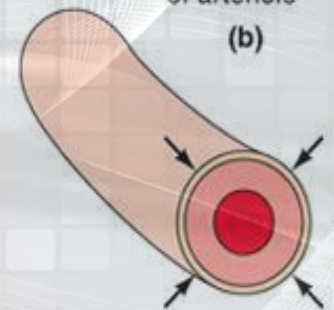
Vasodilation

(decreased contraction of circular smooth muscle in the arteriolar wall, which leads to decreased resistance and increased flow through the vessel)

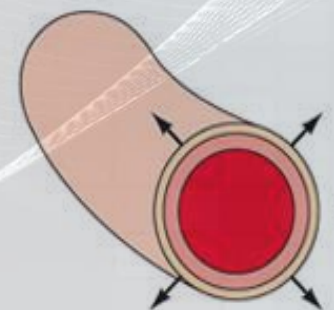


Cross section of arteriole

(b)



(c)



(d)

Caused by:

- ↓ Oxygen (O_2)
- ↓ Carbon dioxide (CO_2) and other metabolites
- ↑ Endothelin
- ↑ Sympathetic stimulation
- ↑ Vasopressin; angiotensin II
- ↓ Cold

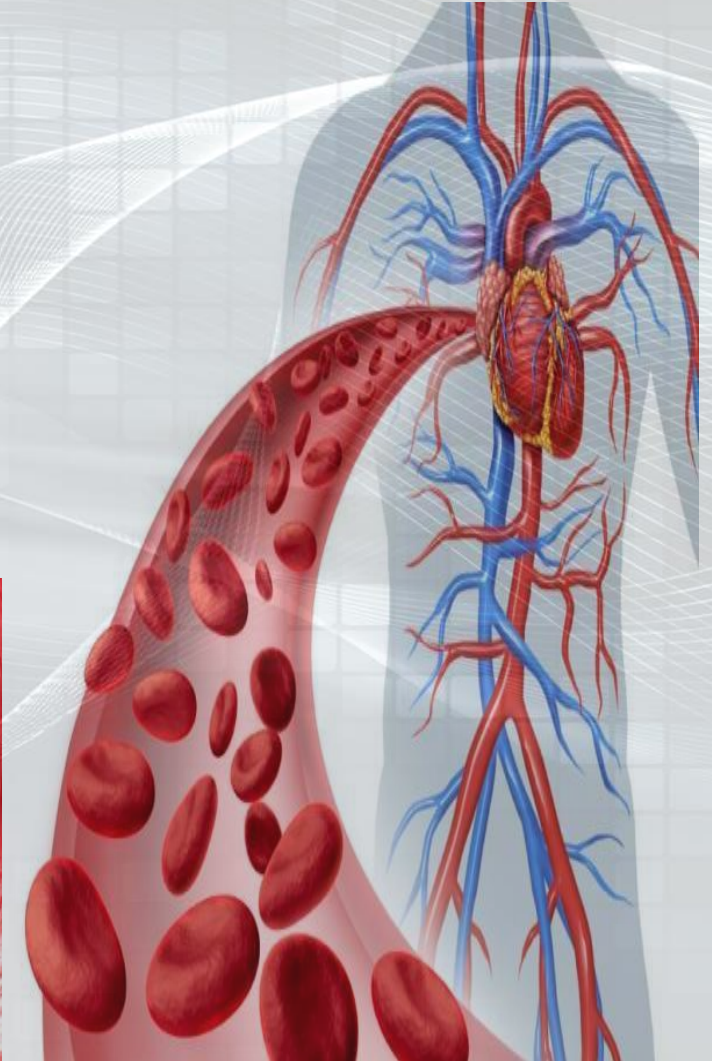
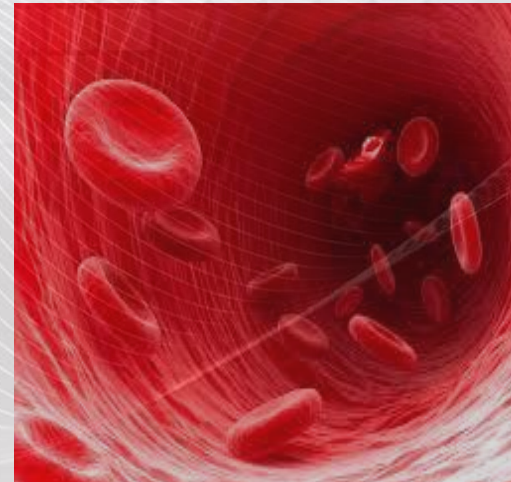
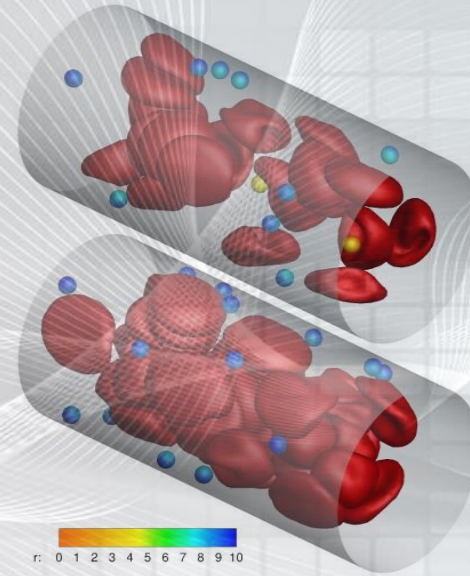
Caused by:

- ↓ O_2
- ↑ CO_2 and other metabolites
- ↑ Nitric oxide
- ↓ Sympathetic stimulation
- ↑ Histamine release
- ↑ Heat

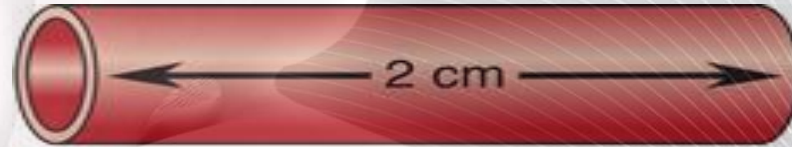
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Effect of Viscosity (η) on flow

- ❑ Blood viscosity (**h**) is the thickness & stickiness of the blood.
- ❑ It is an important factor that determines the resistance of blood to flow.
- ❑ Human blood is five times more viscous than distilled water.
- ❑ Viscosity (**h**) of the whole blood is mainly due to cells, & that of plasma is due to plasma proteins.
- ❑ **Viscosity (**h**) is inversely proportional to the flow (**Q**).**



Effect of Length (L) on Flow



$Q = 10 \text{ ml/sec}$



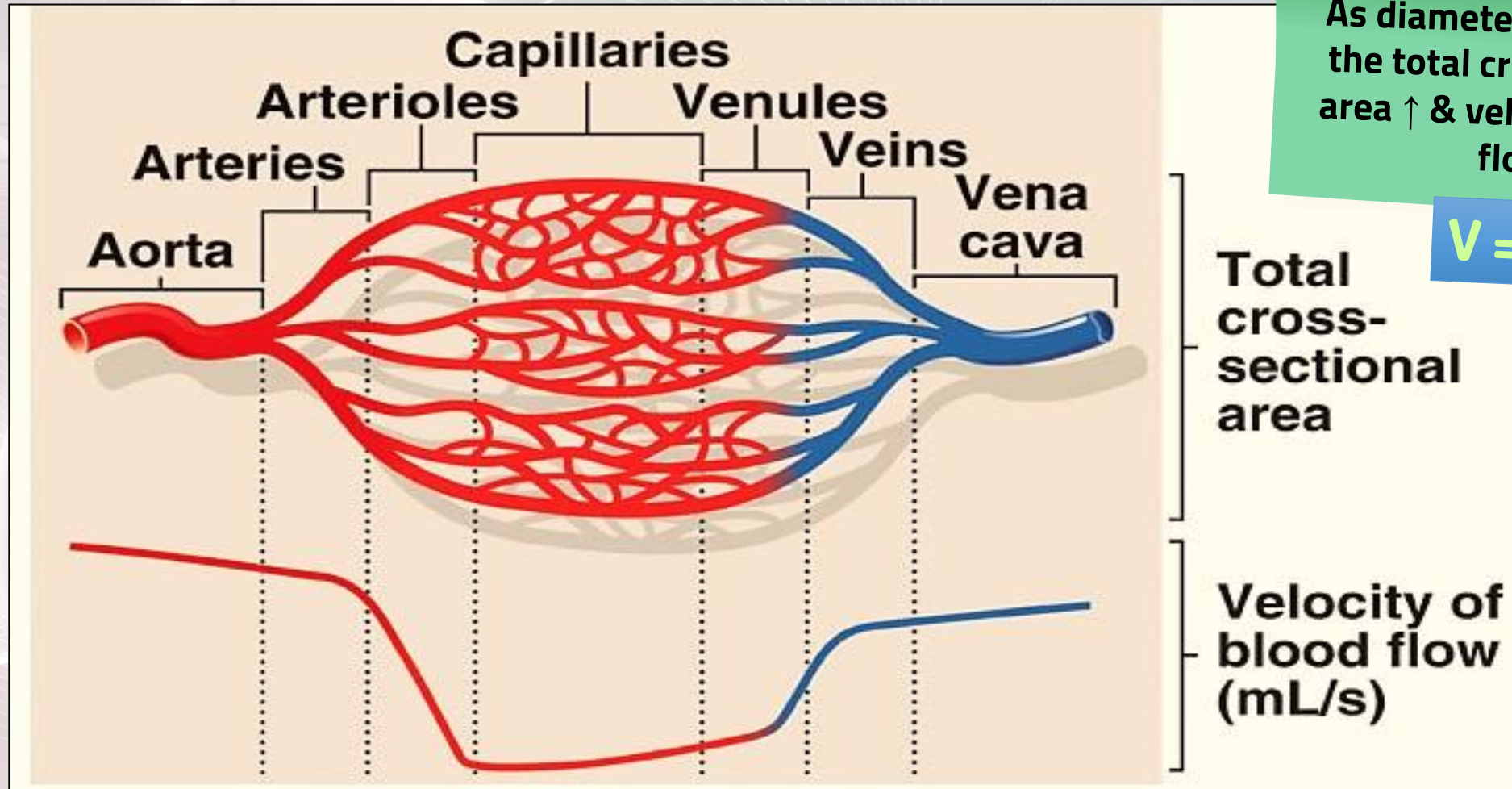
$Q = 5 \text{ ml/sec}$



$Q = 20 \text{ ml/sec}$

Length (L) is **inversely** proportional to the flow (Q).
N.B. In a normal human, length of the vascular system is fixed.

Flow (Q) and Cross-Sectional Area



As diameter of vessels ↓,
the total cross-sectional
area ↑ & velocity of blood
flow ↓

$$V = Q/A$$

V= Velocity; Q= Flow; A= Cross sectional area.

Compliance (C) of Blood Vessels: Affecting Factors

- ❑ **Compliance = Distensibility.**
- ❑ **Compliance** is the volume (**V**) of blood that the vessel can hold at a given pressure (**P**).

$$C = \frac{V}{P}$$

- ❑ **Venous system** has a **large compliance** & acts as a **blood reservoir** (high volume & low pressure).

C= Compliance; V= Volume; P= Pressure.

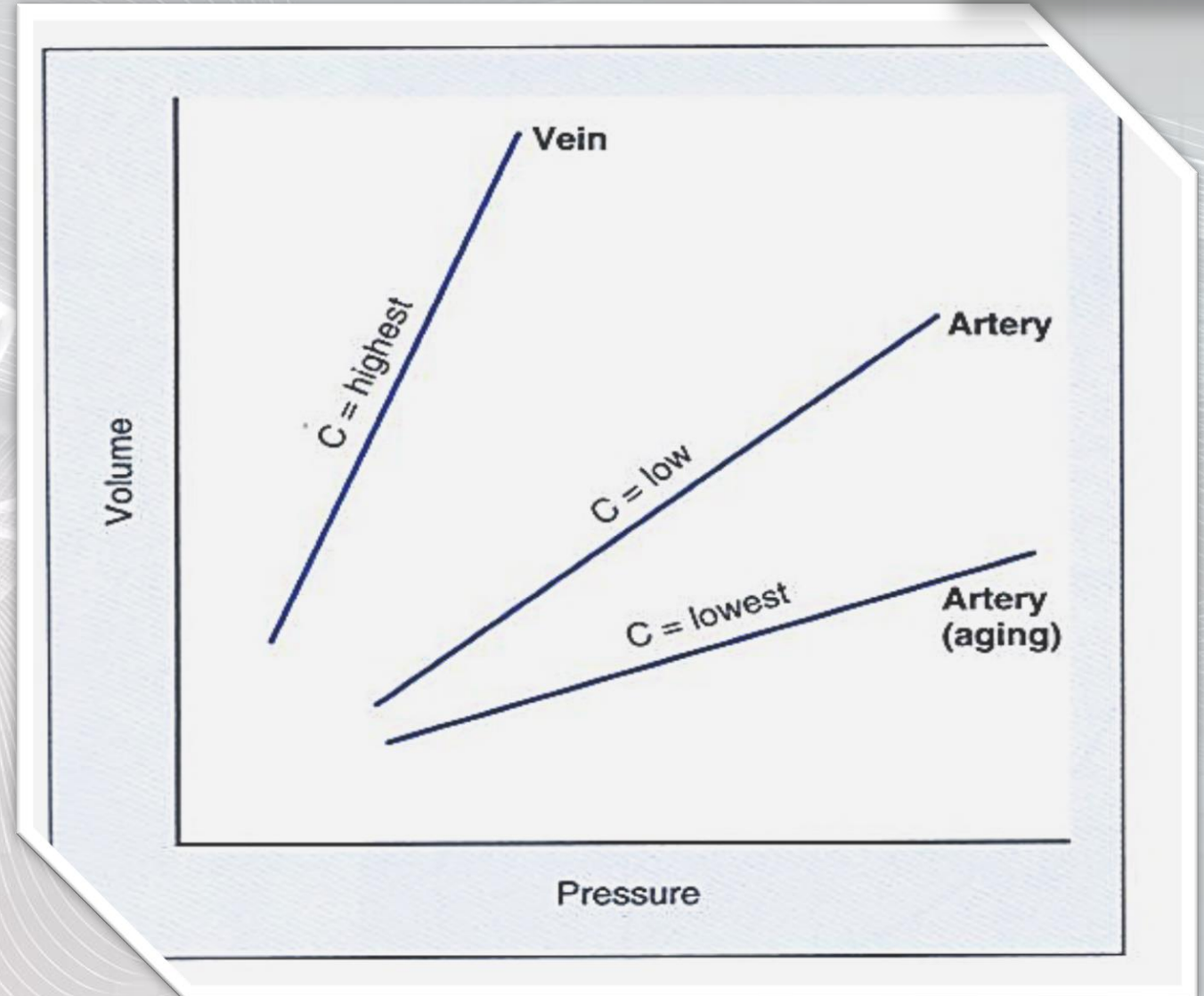
- **Venous system** has the **highest compliance (C)**, while the **arterial system** has a **low compliance (C)**.

$$C = \frac{V}{P}$$

C = Compliance

V = Volume

P = Pressure



Resistance to Flow in the Cardiovascular System

Basic Concepts

Series Resistance

$$R_{\text{Total}} = R_1 + R_2 + R_3$$

More Resistance

Series



Arterioles

Higher R

Parallel Resistance

$$1/R_{\text{Total}} = 1/R_1 + 1/R_2 + 1/R_3$$

Less Resistance

Parallel



Capillaries

Lower R

R= Resistance; R_{Total} = Total Resistance.

Total Peripheral Resistance (TPR):

- Total Peripheral Resistance (TPR) is higher in the **systemic circulation** than the pulmonary circulation.

$$R = \frac{\Delta P}{Q}$$

Systemic Circulation

$$\text{TPR} = \frac{\text{Aortic Pressure} - \text{RAP}}{\text{Flow}}$$

$$\text{TPR} = \frac{120 - 2 \text{ mmHg}}{83.3 \text{ ml/sec (5 L/min)}}$$

$$\text{TPR} = 1.2 \text{ (PRU's)}$$

Pulmonary Circulation

$$\text{Pulmonary R} = \frac{\text{Pulmonary Pressure} - \text{LAP}}{\text{Flow}}$$

$$\text{Pulmonary R} = \frac{15 - 3 \text{ mmHg}}{83.3 \text{ ml/sec (5 L/min)}}$$

$$\text{Pulmonary R} = 0.12 \text{ (PRU's)}$$

TPR= Total Peripheral Resistance; PulR= Pulmonary Resistance; RAP= Right Atrial Pressure; LAP= Left Atrial Pressure; PRU= Peripheral Resistance Units.

(↑) SV

(↑) HR

$CO = \text{Stroke volume} \times \text{Heart rate}$

Peripheral Resistance depends on:

- * Size & length of blood vessel.
- * Thickness (viscosity) of blood.

Peripheral Resistance



↑ CO



↑ PR



(↑)

Blood Pressure

MAP

(↑) **Blood Volume**

Blood Volume depends on:

Balance between fluid intake & fluid loss



Thank You