CVS - Final -Dr. Fatima material 💜

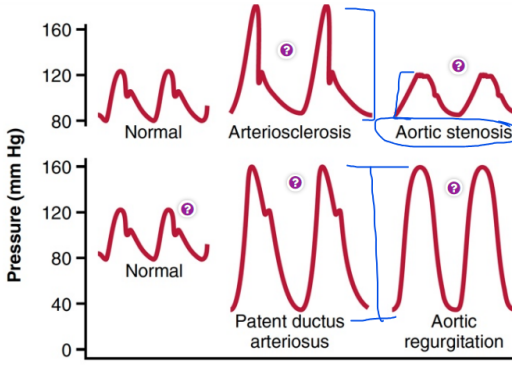
- vascular system function is transport of (o2 , hormones , nutrients).
- distribution of blood to organs is not equal and doesn't match their actual need (size, function) like reconditioning organs: **skin, GI , kidneys "more amount than needs"**.
- distribution is parallel to give organ same content of blood without other organs waste products, with monitored blood supply to each organ based on the case.
- brain needs adequate supply , doesn't tolerate decreased amount for more than 4 min , top priority to keep it constant .
- blood flow needs driving force = pressure difference = pressure gradient = heart pumping "diastole- systole" , not absolute pressure .
- opposing factor is resistance, to slow flow to exchange, resistance depends on diameter (r^4, large diameter less resistance) , length(longer= more resistance), viscosity (polycythemia = high viscosity = high resistance),
- any **neurological** symptoms after APC (history, physical, investigation) , CT(faster) , MRI more specific, vascular obstruction= stroke .
- laminar flow cause parabolic shape , because of faster flow in center away from walls.
- turbulent flow is **not always** pathologically, could be physiologically due to branching.
- laminar flow is silent , turbulent cause sound due to vessel diameter change.
- Reynolds number = turbulent tendency: velocity **x** diameter **x** density **/** viscosity.
- anemia cause low viscosity and more velocity due to more cardiac output= more turbulent=murmurs can be heard .
- thrombosis causes low diameter, and high velocity, velocity is more important than diameter because it is "flow / area "causing more turbulent.
- elastic fibers in arteries give blood in diastole "recoil", in systole = Strech to accommodate pressure. old age cause degeneration of elastic causing arterial stiffness causing less diastolic pressure & more systolic causing injury.
- difference between diastolic and systolic we call it pulse pressure, increased in arterial stiffness (low diastole - high systole).
- pulse rate is counting systolic pressure in superficial artery by fingers.
- **mean arterial pressure (MAP) at rest** is diastolic pressure + **⅓** pulse pressure.
- pressure in left ventricle is high in systole and almost zero in diastole .
- pressure in arteries as high in ventricle in systole but never zero in diastole because of elastic & diameter .
- in arterioles = smaller diameter , high resistance , decrease pressure .

$$F = \frac{\Delta P}{R} \qquad \text{Flow rate} = \frac{\pi \Delta P r^4}{8 \eta L}$$

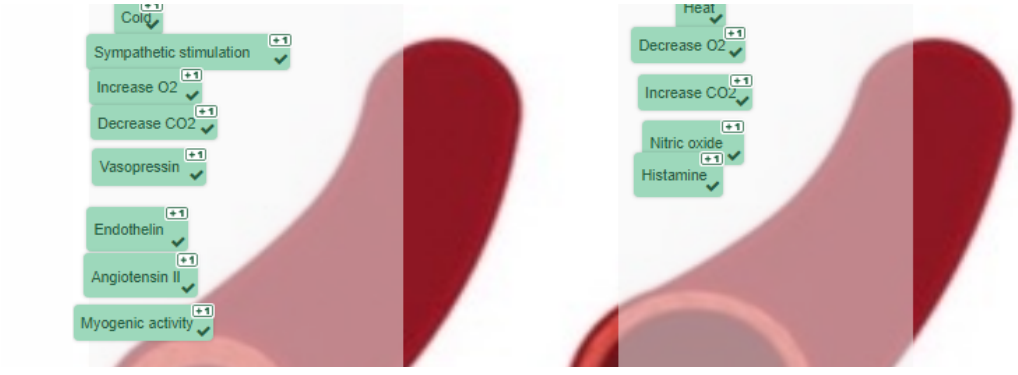
$$Re = \frac{v \cdot d \cdot \rho}{n}$$

- pulse pressure rises in old age atherosclerosis , patent ductus arteriosus (lower diastolic) ,aortic regurgitation (no valve = blood return= increase systolic - decrease diastolic) .

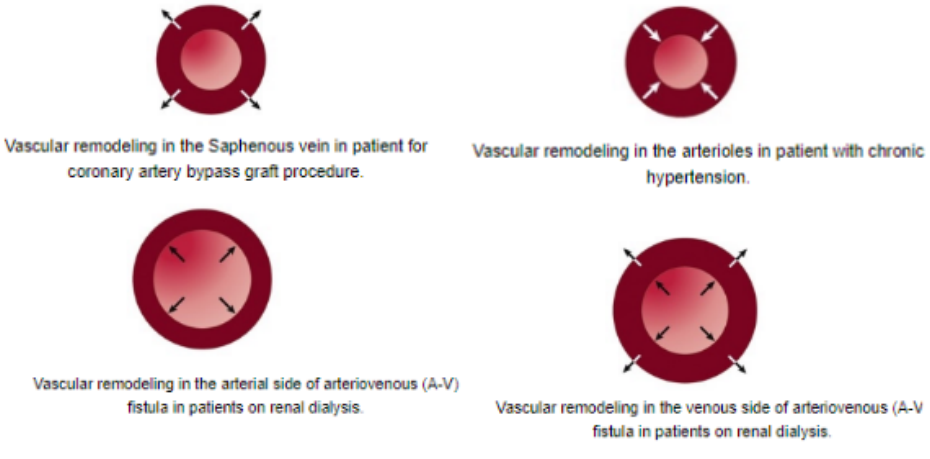
- pulse pressure decreased in aortic stenosis .



- Arterioles = main resistance vessels 2/3 = smaller radius = lesser pressure , nonfluctuating pressure ,continuously monitor tissue needs.
- Arteriolar smooth muscle= (sufficient Ca++ self-induced contractile)+ sympathetic fibers.
- tunica media of arterioles : Smooth muscles with sympathetic + local - hormonal factors, contract = increase resistance = decrease blood flow .
- terminal arterioles “metarterioles” : not continuous , as a sphincter controlling flow to capillary bed .
- total resistance to blood flow is equal to the sum of the resistances of each vessel: R(total) = R1 + R2 + R3
- For blood vessels arranged in parallel, the total resistance to blood flow is expressed as follows: 1/R(total) = 1/R1 + 1/R2 + 1/R3 , so way lesser total resistance .
- The total conductance (the reciprocal of resistance) for blood flow is the sum of the conductance of each parallel pathway.
- amputation of a limb removes a parallel circuit and reduces the total vascular conductance and total blood flow while increasing the total peripheral vascular resistance.
- local factors affect blood flow like physical (stretch, heat , cold, stress) & chemical (metabolic”O2” + histamine).
- locally : NO is an important vasodilator, whereas endothelin is a potent vasoconstrictor . during allergic reactions, histamine is released and acts as a paracrine as major cause of vasodilation in an injured area.
- acute control : rapid change , up to minutes , (vasodilation or constriction)
- long-term control : slow , better for needs , increase on decrease in number or size of vessels .
- adenosine, carbon dioxide, adenosine phosphate compounds, histamine, potassium ions, and hydrogen ions are vasodilators , less oxygen or nutrients cause vasodilation .
- The cyclical opening and closing is called vasomotion.
- reactive hyperemia : opening after vessel block , cause 7 times flow .
- more functional = required more nutrients = vasodilation = active hyperemia = 20 times .
- **Elevated blood pressure will induce vasoconstriction.**
- high arterial pressure stretches the vessel, reactive vascular constriction results.
- increase arterial pressure = increase arterioles resistance as compensatory =constant flow rate = blood flow autoregulation.



- Norepinephrine and epinephrine are sympathetic vasoconstrictors .
- ⬆angiotensin II & Vasopressin & Ca++ = are vasoconstrictors .
- ⬆H+ & CO2 & bradykinin & histamine & K+ & Mg ++ = are vasodilators .
- increase in metabolic demand = increase in capillaries number & size= if not pathological .
- angiogenesis =vascularity adjustment to match needs fast in young , slow in older .
- Vascular endothelial growth factor (VEGF), fibroblast growth factor, platelet-derived growth factor (PDGF), angiogenin, hypoxia inducible factors (HIFs)= promote angiogenesis
- angiogenesis steps : dissolution of basement >>rapid reproduction>> extended cords>> fold over into a tube>> connects with another tube >>capillary loop .
- angiostatin and Endostatin block angiogenesis .
- long term tension (hypertension)= inward eutrophic remodeling in arterioles +hypertrophy+ thickening + Lumin dilation + stiffness in arteries .
- in CABG - implanted vein get thicker like artery .
- in A-V fistula in Renal failure patients = large artery and large vein , thickening in artery + thickening and increase Lumin diameter in vein .



- sympathetic cause major vasoconstriction .
- **MAP = CO x TPR ,** MAP = zero before right atrium , should be sufficient driving pressure& not too extra work on heart .
- change in CO could change TPR , CO depends on (SV , HR , sympathetic) , TPR (viscosity ”red cells” & radius)
- Sympathetic : many vasoconstriction in GI , and less in muscles &heart.
- Arterioles have no sympathetic or parasympathetic (except abundant parasympathetic in penis and clitoris)
- vasodilation in penis by NO , in elsewhere by less constriction -in heart by epinephrine.

- cardiovascular center on medulla regulate blood pressure , also hypothalamus control blood flow to skin to regulate temperature .
- buffer system= baroreceptors, regulate artery pressure, arteriolas pressure is auto - min.
- aortic arch baroreceptors is carried to the brain stem on the vagus nerve.
- chemoreceptors in carotid bodies , are sensitive to O2 pressure in arteries , causing sympathetic vasoconstriction in arterioles , related to respiratory .
- brain is intolerant of decreases in blood flow , central chemoreceptors less sensitive .
- ischemic brain = PCO2 immediately increases and pH decreases = arteriolar vasoconstriction = increase in TPR = blood redirection to brain .
- **Cushing reaction :** Tumer compress brain vessels - less blood - immediate blood redirection to brain .
- stretch receptors in atria and pulmonary = low-pressure receptors detect any increase , then stretch to cause renal sympathetic to decrease reabsorption -dilation “increased Na+ and water excretion” . also go to hypothalamus to decrease ADH .
- increase artery pressure = decrease heart rate , , increase in vein pressure = increase HR .
- From adrenal , norepinephrine binds to A1 = vasoconstriction , epinephrine binds mainly to B2 on heart cause dilation . in GI mostly A1 so sympathetic constriction .
- Vasopressin = ADH = water retention .
- renin–angiotensin II–aldosterone system (RAAS) is slower - hormonal- restore arterial pressure.
- hypertension leads to atherosclerosis , radius narrowing then increases TPR .
- Complications of Hypertension: (1) left ventricular hypertrophy maybe followed by systolic heart failure.
  (2) stroke.
  (3) heart attack.
  (4) renal failure.
  (5) retinal damage.

capillary

- selective perfusion of capillary beds based on needs determined by the degree of dilation or constriction of sphincters
- simple diffusion exchange , O2 and CO2 are highly lipid soluble cross cells , Water-soluble substances need aqueous clefts “less diffusion”
- the greater the number of open capillaries, the greater the surface area for diffusion.
- transcytosis to exchange large proteins .
- slow flow in capillaries = time to exchange
- v=q/a , velocity is flow across distance pre time. velocity of flow is inversely proportional to the total cross-sectional area . low velocity means high resistance means smaller diameter = more cross sectional area (**largest in capillaries).**
- many capillaries = 750 times larger total cross section than aorta .
- In the capillaries, pressure decreases further for two reasons:

1. 1. frictional resistance to flow.
2. 2. filtration of fluid out of the capillaries.

- capillaries are continues , fenestrated or sinusoidal.
- exchange depends on concentration gradient (passive) , bulk flow (hydrostatic and colloid osmotic). not directly with cells ,
- Ultrafiltration at capillary beginning , reabsorption at the end , blood < osmotic

Lymphatic system

- filtrated more than reabsorbed , is picked up by the lymphatic system.
- functions : retain extra fluid & protein , defense , transport fat .
- “lymph pump” & skeletal muscle contraction (affected by sympathetic stimulation) = lymph flow , One-way valve prevent backflow ,
- Edema = fluid accumulation ECF , causes : increase hydrostatic pressure (filtration) , decrease oncotic pressure (absorption- due to protein) , increase hydraulic “kinetic ” .

veins

- 64% blood reservoir , high capacity , low resistance .”large volume under low pressure”
- Compliance of the arteries is much lower than that of the veins.(low volume under high pressure)
- Changes in venous capacity directly influence the magnitude of venous return.
- venous return refers to the volume of blood per minute entering each atrium from the veins. increase venous retain by : Sympathetic stimulation (constriction), skeletal contraction against gravity , one-way valve , sub-atmospheric pressure in chest , cardiac suction (valve downward , ventricle emptying).

Hemorrhage

- low pressure = baroreceptor reflux = sympathetic =⬆ HR , CO , contractility , constriction.
- low pressure = ⬆RAA = ⬆aldosterone = water + Na retention = increase blood volume .
- increase fluid reabsorption in capillaries.