HEMODYNAMICS :-

- CVS
  - Systemic circulation – Left heart is the pump
  - Pulmonary circulation – Rt hrt is the pump

① Systemic – Lft hrt → Aorta → Systemic arteries → Sys capillaries → Systemic veins → Rt atrium

② Pulmonary – Rt hrt → Pulmonary arteries → Pulmonary capillaries → Pulmonary veins → Lft atrium

- In steady state conditions cardiac output of both lft hrt & Rt hrt is equal – since hrt is a closed circuit.
- B/W Lft hrt & Rt hrt the local systemic capillary beds are arranged parallel to each other and recieve a fraction of cardiac output

Whereas B/W Rt hrt & left only one Pulmonary capillary bed is present.

Cardiac output of lft ventricle
- 5% – Cutaneous
- 5% – Coronary
- 15% – Cerebral
- 25% – GIT
- 25% – Renal
- 25% – Musculoskeletal

- Pressures in Systemic Circulation :-

① P Lft atrium – 5-8 mm/Hg

② P Lft ventricle
- diastole ≈ 0 mm Hg
- Systole – 120 mm Hg

Systolic pressure is the pressure in major arterial tree when the lft ventricle is at peak of its contraction during systole.

Diastolic pressure is the min pressure present in major arteries during ventricular diastole.

(3) Aorta – Sys 120 mmHg
dias 80

Elastic tissue more
Maintain systolic pressure by stretching
Elastic recoil – diastolic pressure

Mean Arterial pressure = diastolic pressure + 1/3 Pulse pressure
$= 80 + \frac{1}{3} \times 40$
$= 80 + 13 = 93$ mmHg

Pulse pressure = Systolic – diastolic
$= 120 - 80 = 40$ mmHg

(4) Arterioles – Tonic activity – semi constricted
↳ Major resistance to flow of blood

More smooth muscle
Resistance vessels

High sym activity
Splanchnic arterioles
Cutaneous arterioles
Renal arterioles
→ α_1 Adrenergic – Vasoconstriction

Musculoskeletal – β_2 adrenergic – Vasodilatation

As blood passes thru arterioles – pressure dissipates due to resistance.

(5) Capillaries – P = 30 mmHg
except Renal glomerulus P = 50 mmHg

Exchange Vessels
Tot surface area of all capillaries is far greater than the diameter of Aorta
Hence P (↓).
Exchange nutrients & metabolic prod.

– During (↑) activity in a tissue → local vasodilators are prod → Arteriole smooth muscle relaxes → diameter (↑) Resistance (↓) Blood flow (↑).

⑥ Venules :- Capillaries unite to form Venules.
→ Veins
P Venules = 15 mm Hg

- Vein's diameter >> Arterial diameter
↳ Capacity to store blood v high
∴ Act as reservoir of blood - 70% of Tot blood
Hence called Capacitance Vessels

Artery	Vein
① Thick wall narrow lumen	① Wide lumen narrow wall
② High pressure system	② Low pressure system
③ Provide blood to exchange vessels	③ Drains blood from exchange vessels.
④ Blood vol is under high Pressure. - stressed volume ← Small vol	④ Blood vol is under less pressure - Unstressed vol ↳ 70% of blood

- When there is strong Veno constriction unstressed vol is ↓ and blood flows to the hrt & then arteries and gets converted to stressed vol

- When there is Veno dilation ⇒ Venous return reduced cardiac output is ↓ → stressed vol ↓

- MAP - main driving pressure in the systemic arteries

- Pressures in Pulmonary circulation :-
① Rt Ventricle - Systole - 0 to 25 mm Hg
↳ systolic pressure less than that of Lft
Lft ventricle has thicker wall, circulation is systemic which is large.

(2) Pulmonary artery – 25 mm Hg Systolic
8 mm Hg Diastolic

(3) Pulmonary capillaries – 8 mm Hg
↳ Blood gets oxygenated.

(4) PV – 5 mm Hg

(5) Mean Arterial P – 15 mm Hg

— Velocity of Blood flow :-

(1) Velocity :- Rate of displacement (linear) of blood per unit time within circulatory syst

(2) Blood flow :- Volume of blood flowing thru any area of circulatory system per unit time.

$$V \propto \text{Blood flow}$$

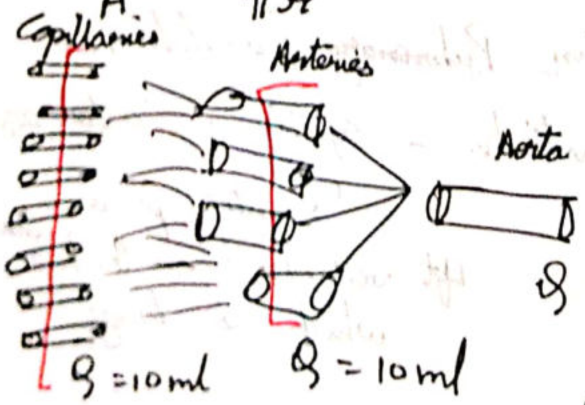
$$V \propto \frac{1}{A}$$

A – cross sectional area thru which blood is flowing

Total cross sectional area of capillaries > arteries > aorta

$$v = \frac{Q}{A} = \frac{Q}{\pi r^2}$$

eg :-



$Q = 10ml$ $Q = 10ml$ $Q = 10ml$

$A_3 = 100\,cm^2$ $A_2 = 10\,cm^2$ $A_1 = 1\,cm^2$

$\vartheta_1 = \frac{10}{1}$ $\quad \vartheta_2 = \frac{10}{10}$ $\quad \vartheta_3 = \frac{10}{100}$

$\vartheta_1 = 10$ $\quad \vartheta = 1$ $\quad \vartheta_3 = 0.1$

- Velocity is vv low in capillaries to allow proper exchange of nutrients and O_2. Work better as exchange areas.

- Relationship B/W blood flow, Pressure gradient and Resistance

① Pressure gradient :- It is the driving force that drives the blood from high pressure thru vessels to low pressure

② Resistance :- Impedement to the blood flow.

BF ∝ Pressure gradient

BF ∝ $\frac{1}{\text{Resistance}}$

$$\boxed{Q = \frac{\Delta P}{R}} \qquad R = \frac{\Delta P}{Q} \qquad \Delta P = P_1 - P_2$$

systemic circulation $\Delta P = 93 - 0 = 93$.
$= \text{MAP} - P$ in right atrium

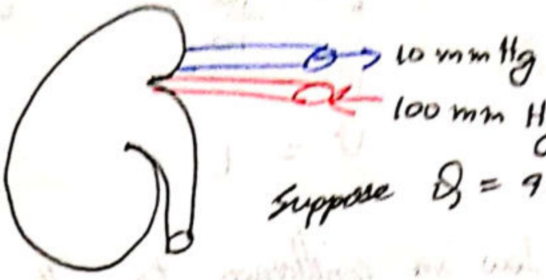
$$Q = \frac{\Delta P}{R} = \frac{93}{\text{Tot peripheral resistance}}.$$

$$\text{cardiac output} = \frac{\Delta P}{\text{TPR}}$$

$$5000 = \frac{93}{\text{TPR}} \Rightarrow \text{TPR} = \frac{93}{5000\ \text{ml}}.$$

Units of Resistance - mmHg/ml/min.

eg :-



Suppose $Q = 900$ ml/min

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

$$= \frac{100-10}{900\ \text{ml/min}} = \frac{\cancel{90}}{\cancel{900}_{10}}$$

$$R = 0.1\ \text{mm Hg/ml/min}$$

$$\boxed{\therefore TPR = \frac{\Delta P_{systemic}}{Q_{systemic}} \qquad R = \frac{\Delta P}{Q}}$$

$Q_{systemic}$ = Ca...

– Poiseuille's Equation :- sums up all the factors that determine resistance to blood flow.

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

η – viscosity of blood
- ↑ – ↑ Hemato...
- ↓ – Severe An...

$\Rightarrow \eta$ = viscosity

$\boxed{R \propto \eta}$

- ↑ η
  - ↑ RBC – Polycythemia
  - Multiple myeloma – abnormal prt are accummulated.
- ↓ η – Severe Anemia
  - ↓ RBC – blood becomes thin

=> l = length of blood vessels

$\boxed{R \propto l}$

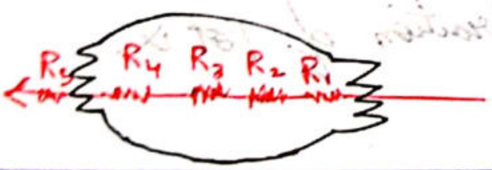
↑ R — ↑ l
↓ R — ↓ l

=> $\boxed{R \propto \frac{1}{r^4}}$ r = radius of the BV ★★

- When radius of the vessel is halved $\frac{r}{2}$
  Resistance becomes 16R
  - When radius become $\frac{1}{4} \times r$
    Resistance becomes 256R
  - When radius is doubled $2r$
    Resistance becomes $\frac{R}{16}$
  - Total peripheral Resistance depends mainly on diameter of Arterioles

- Resistance also depends on the arrangement of Vessels :-

① Series Parallel



$\boxed{\text{Total } R = \underset{\text{arteries}}{R_1} + \underset{\text{arterioles}}{R_2} + \underset{\text{capillary}}{R_3} + \underset{\text{venules}}{R_4} + \underset{\text{veins}}{R_5}}$

93 $\xrightarrow{R_1 \quad R_2 \quad R_3}$ 5

when blood is passing thru resistances P is dropping due to reasons

Some P is used to overcome resistance | Due to ↑ in cross sectional area.

If the R_2 is ↑ the pressure proximal becomes more near to MAP and press distal becomes more near to Caval pressure.

If the R_2 is ↓ – proximal P is dropped.
distal P is ↑.

When blood vessels are arranged in series blood flows from one to another sequentially.

Q is same at every pt.

eg :- vessels passing thru a single organ.

eg Renal artery → afferent arteriole → Glomerulus → eff arteriole → Peritub capill → Renal Venules → Renal Veins.

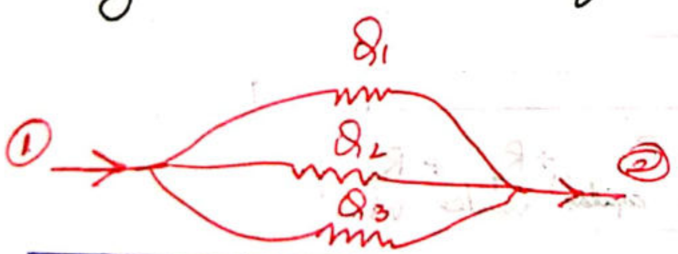
Pressure is progressively ↓ when blood passes from 1 to next resistor area.

(2) Parallel :- eg :- Blood vessels arranged in Sys circulation B/W Aorta & Vena Cava.

Tot blood flow [cardiac output] is simultaneously dist to the diff blood vessels arranged parallely.

Q is not same at all pts

Every vessel set has a fraction of Tot Q.



$$\boxed{Q = Q_1 + Q_2 + Q_3} \quad \text{-----} \quad (1)$$

Whenever a resistance is added parallel
Tot R ↓ ------ (2)

$$\boxed{\frac{1}{R} = \frac{1}{R_1} + \frac{1}{R_2} + \frac{1}{R_3}}$$

Pressures also do not change.

- Type of flow :- ① Laminar ② Turbulent

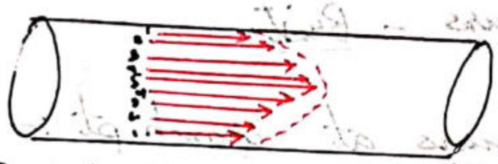
Normally in CVS - Laminar

- As layers of blood move from wall to center of the vessel the layers have progressively ↑ velocity.

Layers toward edge → slow → As they are subjected to More friction.

" in center → fast

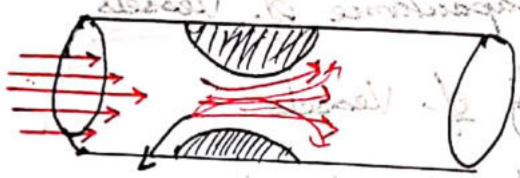
eg :-



Layer 5 - center - fastest.

Parabolic arrangement of layers - Laminar / Streamline.

-



Obstruction caused turbulence forming whirlpools ↓ eddy current

Blood moves radially & axially

More energy is lost than Laminar.

- Sometimes turbulence becomes severe → Murmurs. [Audible sounds] or Bruits.

- Reynold's number - helps to predict type of flow.

$R_n > 2000$
↳ Turbulence

$$\boxed{R_n = \frac{\rho v d}{\eta}}$$

ρ = density
v = vel
d = diameter of vessel
η = viscosity.

① Severe Anemia
- viscosity ↓
- cardiac output ↑ ⇒ ↑ vel

$$R_n = \frac{\rho \times \uparrow v \uparrow \propto d}{\eta \downarrow\downarrow\downarrow}$$

R_n is v high - Murmurs
Turbulent flow.

② Thrombosis / Stenosis – partial occlusion

Q is same at 1, 2, 3
A is less at 2

$V = \frac{Q}{A}$ $V \propto \frac{1}{A}$ Hence V (↑)
R_n (↑)

Turbulence occurs – Bruit

③ Turbulence also occurs at branch pt :-

– Concept of Compliance or Capacitance of Vessels
↳ distensibility of Vessels

Veins are more distensible due to this
↳ If some vol of blood is added to artery & veins ∴ Artery accommodates it with diffi and Vein easily.

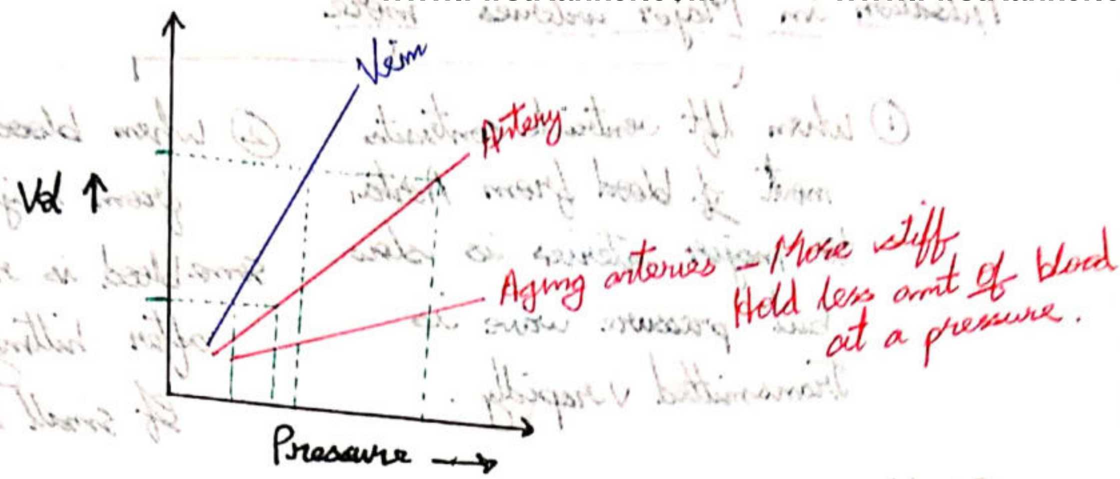
∴ Adding of vol of blood

Veins
Pressure ↑ is less
∴ More compliant

Artery
Press ↑ is more
Less compliant.

Constricted Veins are less compliant
↳ Hold less vol
Pressure (↑).

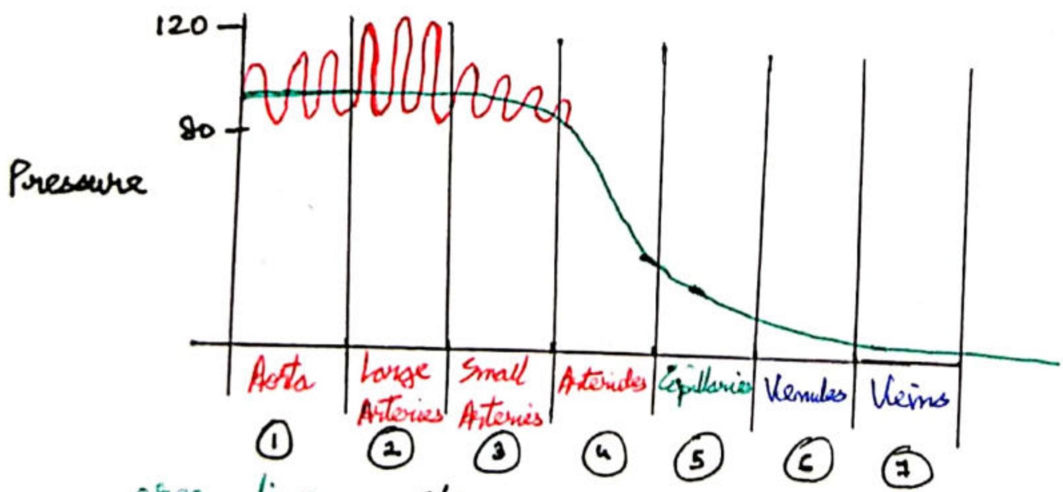
Veins (unstressed vol) $\xrightarrow{\text{Venoconstriction}}$ Arteries (stressed vol)
Veins $\xleftarrow{\text{Venodilatation}}$ Arteries



- For the same vol added Pressure inc is more in Artery than in vein.
- Aging arteries less compliant.

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

- Vessels are said to be compliant when large vol is accommodated with less inc in pressure.
- Pressures in CVS :-



green line - Mean pressure

Major drop in pressure - Arterioles - offers more resistance

Pulsations - prod due to systole & diastole of hrt

Pulsation in Major arteries more

① when left ventricle contracts most of blood from Aorta to major arteries is slow but pressure wave is transmitted v rapidly.

② when blood is from major to s some blood is reflected after hitting the of small arteries.

Pulsations disappear in arterioles — due to high resist

Best And Taylor

- **Hemodynamics** :- Refers to the principles that govern blood flow in the cardiovascular system
- These basic principles of physics are the same as those applied to the movt of fluids in generals.

- **Rheology** :- The science of rheology is concerned with investigation of the deformation and flow of matter.
- Blood plasma is a viscous fluid. The RBC's modify this property so that blood assumes anomalous viscous properties.

- **Forces acting on blood in circulation** :-

Main forces acting on blood in circulation are :-

① f_a ⇒ Inertial force — due to two types of acceleration
  ⓐ Time acceleration due to pulsatile ejection from hrt
  ⓑ Spatial acceleration — entry into constrict

② f_v ⇒ Viscous force — due to viscosity of blood.

③ f_g ⇒ Gravitational force

④ f_p ⇒ Pressure force — driving force for blood generated in the hrt

- According to Newton's law of Motion

$$\boxed{f_a = f_v + f_p + f_g}$$

→ This is the fundamental law governing the motion of blood

- f_a & f_g depend on density of blood
- Except for f_g all the other forces vary at diff places in the circulatory system.
- Reynold's number - estimate of relative magnitudes of inertial & viscous forces.

$$\boxed{R_N = f_a / f_v}$$

$N_R \approx 1000$ - Large arteries => f_a - highly impt
$N_R = 10^{-3}$ - Capillaries => f_a - negligible

- **Blood Viscosity :-**

- Throuout most of the circulation blood flow is laminar
- That is blood flows thru a vessel in an orderly manner with flow vel zero at the vessel wall and progressively ↑ towards the center of the wall
- Inspite of being laminar the streamlines are complex
- One of the most impt factors determining these streamlines is the Visious force.
- It arises in any fluid b/w two layers of fluid with diffe velocities experience a friction or resistance to slippage.
- Viscosity is a property of fluids.

Newtonian fluid	Non Newtonian fluid
A fluid which maintains a constant viscosity at any flow velocity	A fluid which changes its viscosity at different flow velocities
eg :- water	eg :- Blood.

- In a complex Non Newtonian suspension such as blood the viscosity is mainly determined by RBC's (which accounts for 40% of the vol of blood) and also by plasma proteins

- Viscosity of blood depends on
  - Velocity of blood flow
  - Hematocrit
  - Size of blood vessel.

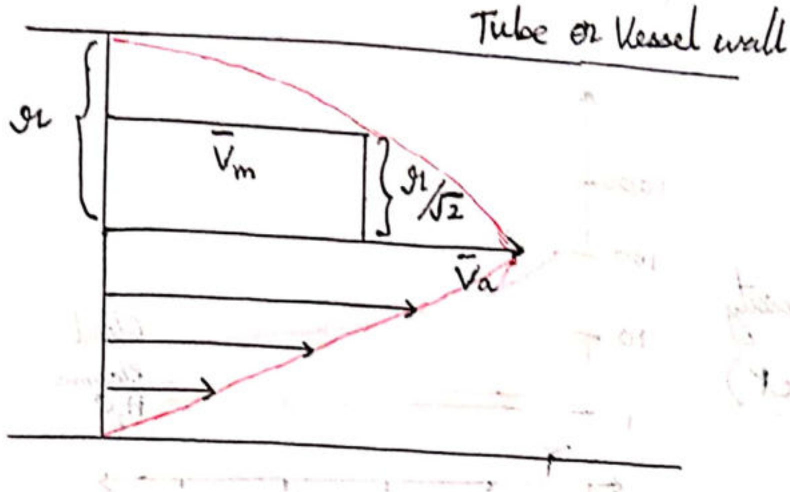
- Viscosity η :- It is defined as the ratio of shear stress (tangential force resisting / pushing blood) to shear rate (change of velocity ΔV B/w two neighbouring layers divided by their distance ΔX.

$$\boxed{\eta = \frac{\text{shear stress}}{\text{shear rate}} = \frac{F/A}{\Delta V / \Delta X}}$$

- Flow for a Newtonian fluid flowing in laminar manner yields a parabolic velocity profile across the channel with fastest vel in the center and vel 0 at the boundary with the wall. [Neglecting f_g and f_a]

- Laminar flow of blood in arteries and veins the profile is not strictly parabolic bcoz the inertial force is not negligible. Blood vessels are not strictly straight cylindrical tubes there are bifurcations, curvature & to a degree taper.

- At a distance of $r/\sqrt{2}$ from the center of the tube the flow velocity is half that along the central axis and equals mean flow velocity along the tube.

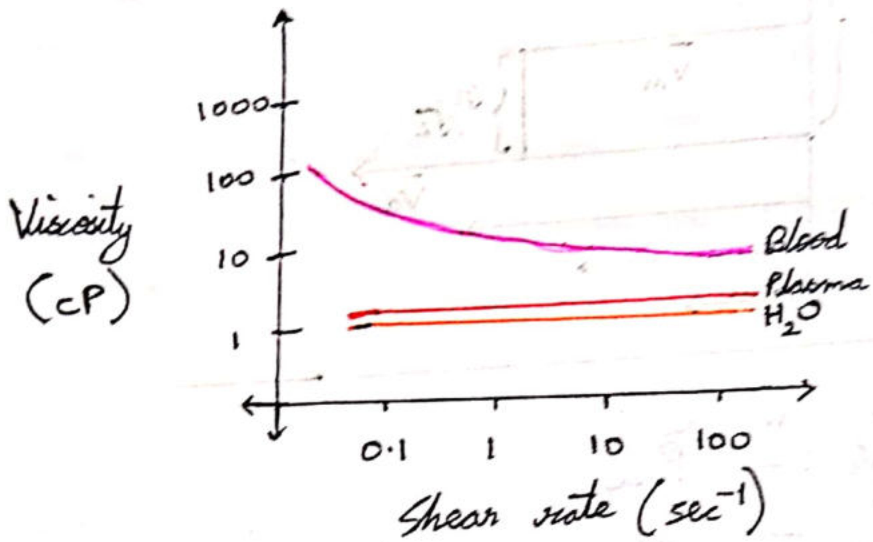


- Unit of Viscosity :- Poise
  water at room temperature has a viscosity of about 1 centipoise
  For convenience the η of various fluids is often related to that of water at the same temperature and this relation is termed the relative viscosity
- At normal hematocrit blood has a relative viscosity of 3 to 4

- Viscosity and Velocity of blood flow :-

- Blood plasma is a newtonian fluid but the presence of RBC's and other plasma pr changes its viscous properties.
- Changes in blood viscosity are non linear with changes in flow velocity. The shape of this viscosity - velocity relationship is determined entirely by the behaviour of individual RBC's.
- ↓ in flow vel => ↑ blood viscosity.
- At shear rates below 1 rouleaux formation occurs leading to ↑ blood viscosity.
- At shear rates > 1, less marked changes in viscosity occur which appear to relate to deformation of the red cells

- Red cell deformation is associated with reduced ... dissipation in the plasma, the red cell membrane and the Hb soln. This progressive ↓ in ~~velocity~~ viscosity is prevented by making red cells nondeformable.



- Blood shows Newtonian behaviour at high velocities with little change in viscosity with changing flow velocity. The curve is relatively flat.
  eg - large blood vessels.

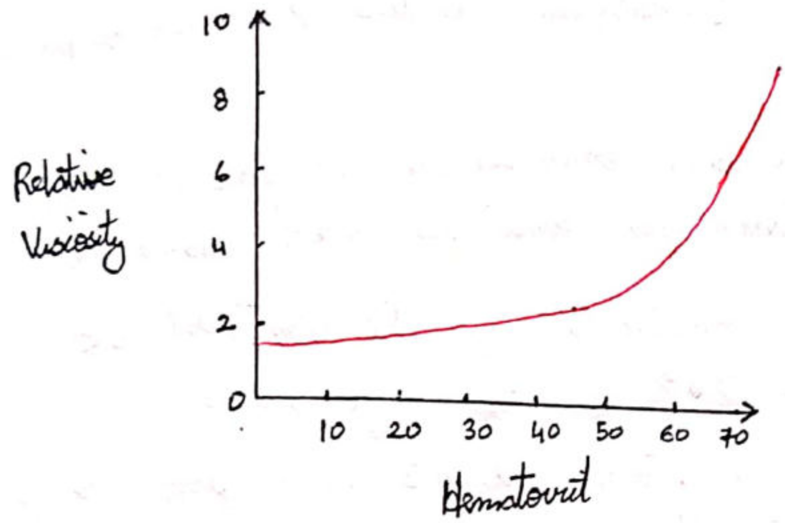
- At slower flow velocities the shear dependent ↑ in viscosity is marked eg Veins

- In certain diseases like sickle cell Anemia RBC's are non deformable due to polymerisation. — ↑ blood viscosity

- The η of blood is also markedly dependent upon the hematocrit.

- The effect of Hematocrit on blood viscosity ↑ sharply above a hematocrit of 50% eg:- Polycythemia vera where hematocrit is 60-70% or in individuals living at high altitudes.

- In such conditions substantial ↑ in the blood viscosity can occur with unfav effects on blood flow & work of hrt.



- Fahraeus Lindqvist effect :-

Decrease in viscosity occurs at tube radius below 0.5 - 1 mm and helps in flow thru small arterioles and capillaries.

- This effect may in large part explain the lower blood viscosity measured in vivo.
- Without this effect the overall slowing of blood velocity in the capillaries and adherence of red cells into aggregates at slow velocities would markedly ↑ the capillary blood viscosity.
- The reason for this effect is complex.
  But it has been related to a phenomenon causing the layers of blood next to the vessel wall be relatively cell free and leading to lower hematocrits in small blood vessels.
- There is a tendency for Red cells to move towards the higher velocity region in the center of tube ↑ the hematocrit there while the hematocrit decreases near the walls; the cell poor layer near the wall becomes proportionally larger as the vessel diameter becomes smaller.

- The rate of flow of plasma thru the blood vessels is lower than rate of most of RBC's as the central portion of the bloodstream contains a greater proportion of Red cells.
- In microvessels the side branches can ∴ have a much lower hematocrit, a phenomenon sometimes called Plasma skimming
- In The capillaries of skeletal for eg the hematocrit is <20% compared to 40-45% in the large vessels.

The lowering of hematocrit results in a blood viscosity reaching closer to that of plasma and undoubtedly it is an important component of the fall of viscosity in small vessels.

- The RBC's have immense deformability and can squeeze thru openings much smaller in diameter. This allows the Red cells to crowd readily thru complex narrow capillary networks with ~~only~~ less ↑ in vascular resistance
- Red cell bends and shears easily but cannot tolerate stretch. If the volume of the red cell is ↑ it becomes more spherical and breaks easily.
- In contrast white blood cells are stiffer and impose a higher resistance in capillaries when they have to undergo deformation for passage.
- In conditions of flow eg a shock; the larger white cells may not pass thru capillaries causing obstruction of Granulocyte capillary plugging has been determined to be the cause of the capillary no reflow phenomenon

with reperfusion after a period of arterial occlusion in the heart and in other organs.

- Limitations of Poiseuille's equation :-

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

- This equation does not take into account other factors that affect resistance to blood flow.

The additional factors are
1. Vessel distensibility
2. Aggregation of blood cells
3. Cell adhesion to the endothelium
4. local protrusions of endothelial cells.

- An impt difference B/W Poiseuille's experimental preparation and the circulation is the fact that blood vessels are distensible.

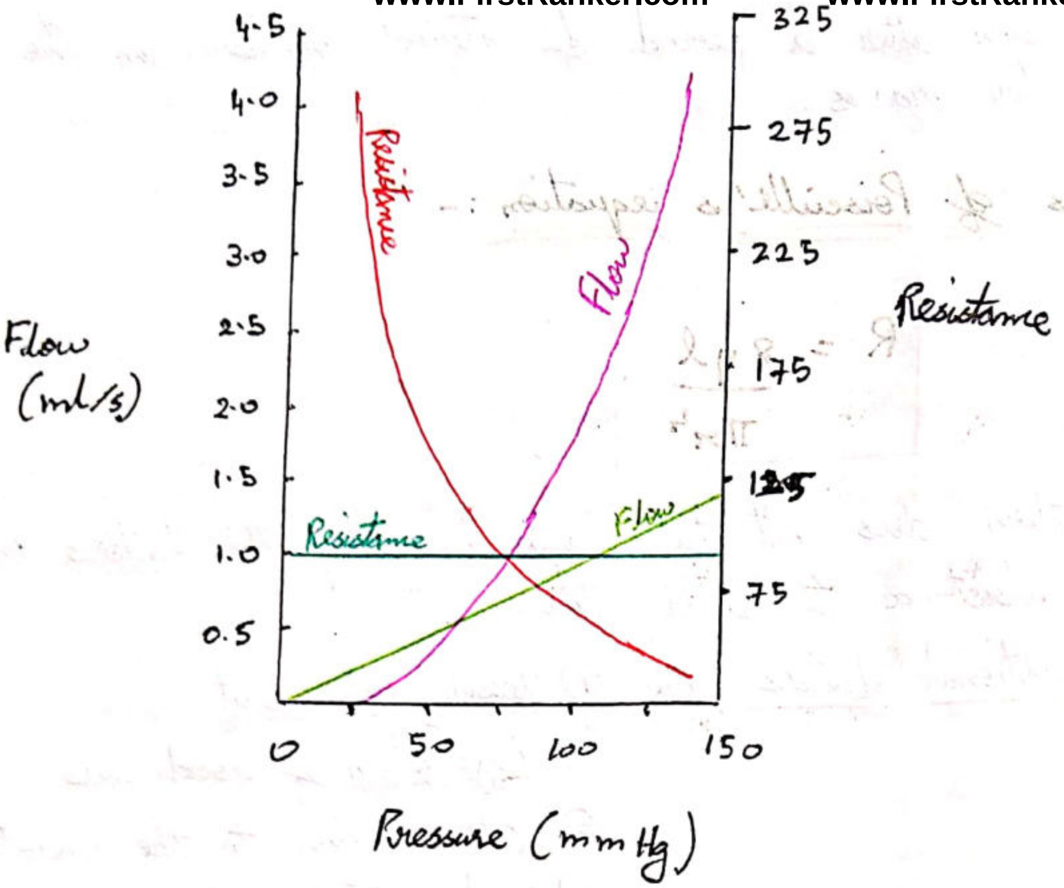
- In nondistensible tubes using Newtonian fluids as pressure ↑ flow increases linearly and resistance remains constant.

- In a distensible vascular bed the pressure flow relation is non linear, flow ↑ exponentially with ↑ in pressure as the vessel is distended

And as the pressure ↑ ⇒ resistance ↓

- More distensible the vessel the more the pressure flow curve shifts towards higher flows

- In beds with higher degree of vasomotor tone but without autoregulation the curve is shifted to the right & flattened



green colour – Newtonian fluid

– Laminar & Turbulent flow :-

- Laminar flow is silent
- Turbulent flow – Random pressure fluctuations – sounds occur can be heard by a steth
  1. Vascular bruits :- In a blood vessel beyond a constricting vascular lesion.
  2. Korotkoff sounds :- Tapping sounds beyond the cuff of sphygmomanometer.
  3. Systolic heart murmurs

→ Useful in assessing arterial blood flow in the traditional Auscultatory method for determining BP

4. Intense turbulence → Mech vibration → Thrills → felt by touch

- Tension in Vascular Walls :-

- Due to the transmural pressure blood vessel is stretched
- The higher the pressure difference → higher tension.
  If the wall is thicker → Tension less.

- Law of Laplace :-
  According to this law, the tension in the wall of a cylinder is eq to the product of the transmural pressure and the radius divided by the wall thickness

$$\boxed{T = \frac{Pr}{h}}$$

T => Tension
P => Transmural pressure
r => radius of Vessel
h => thickness of vessel wall

- Thin walled capillaries can withstand high internal pressures without bursting bcoz of their narrow lumen. This is explained by this law.

- In a thin walled blood vessel transmural pressure can be given by the formula

$$\boxed{P = T/r}$$

∴ The smaller the radius of a blood vessel lower is the tension in the wall to balance the distending pressure

- T_{aorta} is 12000 × $T_{capillaries}$

- In dilated vessel wall tension (↑) even when pressure remains constant.

- Capillary:-
i) Smaller the radius the greater the mechanical advantage in terms of the tension that must be borne by the wall.
ii) A capillary with its very small diameter (7 μm) and relatively thick wall (1μm) can support an intra vascular pressure of 25 mm Hg with a stress of only 2×10^4

- Arterioles :-
i) when the wall is relatively thick as in the arterioles a relatively lower wall tension exists and the smooth muscle bears a smaller load despite a relatively high intravascular pressure.
ii) This provides a Mech adv for changing blood vessel diameter in these small vessels.

- In an Aorta Aneurysm rise in BP → ~~rise~~ thinning of the aortic wall
↓
↑ in radius.

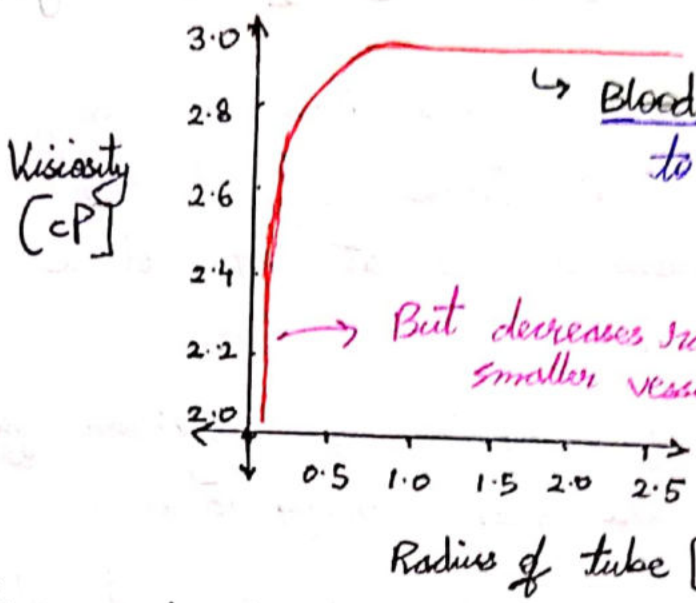
↑ BP → ↑ radius → Thinning of aortic wall

Further rise in wall stress σ.
This sequence leads to further stretching

The Aneurysm becomes unstable and expansion eventually leads to rupture of the Aorta in some individuals

- Note :-
  Ohm's law of hydrodynamics ($F = \Delta P / R$) applies to all vessels no matter how complicated.
  Poiseuille's equation applies to only rigid cylindrical tubes.

- Dependence of Viscosity on Vessel Radius :- Fahraeus Lindqvist effect



↳ Blood viscosity is relatively insensitive to changes in vessel radius for large vessels

→ But decreases rapidly with (↓) in radius for smaller vessels.

- Assumptions made by Poiseulle for his equation :-
  1. The fluid must be incompressible
  2. Tube must be straight, rigid, cylindrical, unbranched & have a const radius
  3. Vel of thin fluid layer at the wall must be zero i.e. no slippage
  4. The flow must be laminar
  5. The viscosity of fluid must be constant.

Bernoulli's Principle :-

It states that an ↑ in the speed of a fluid occurs simultaneously with a decrease in fluid's potential energy

$$\boxed{P_1 + \frac{1}{2}\rho v_1^2 + \rho g h_1 = P_2 + \frac{1}{2}\rho v_2^2 + \rho g h_2}$$

- This principle applies to the conservation of energy in flowing fluids and in simplest terms indicate that the lateral pressure in a stream flowing at high vel is less than when it flows velocity
- This occurs bcoz of dissipation of total energy as kinetic energy in the fast moving stream.
- When the diameter of vessel widens and vel slows the KE term is ↓ and the pressure is higher. whereas when the diameter of the vessel ↓ opposite happens.
- ↑ lateral pressure in regions of arteries that are abnormally enlarged may prod aneurysm formation,
- In Aorta → Lat pressure is dependent on KE
  In smaller arteries → KE component relatively unimpt
  Pressure drop due to frictional energy loss
- When blood is flowing thru large arteries & veins Total energy per unit of blood is given by.

$$E = P + \rho g h + \frac{1}{2}\rho v^2$$

- If all parts of the fluid sys are at same level → ρgh is ignored

- $KE \propto v^2$ is often ignored but becomes impt in some cases. When it is sufficiently high it causes flow of fluid from low pressure region to high pressure region. This is an impt mech for closure of heart valves.

- The driving forces for blood flow are not directly affected by the varying hts of diff circulatory components

- Like the **Principle of Siphon** :- it is only the difference in the tot pressure and energy at one pt in the circulation from that in the other that determines the flow. Not whether the flow is uphill or downhill.
  In a typical siphon the flow is uphill.

- **Applications of Bernoulli's effect** :-
  - It has impt implications for measurement of BP with open tip catheter
  - The press recorded with open tip facing the flow is higher than the actual pressure by an amount corresponding to the KE of the oncoming fluid.
    - Conversely the pressure recorded with open tip facing away from the flow is lower than the actual pressure by an eq amt
    - The measure pressure is correct only when the opening is on the side of the catheter, perpendicular to the flow of blood

- Resistance V/S Impedance :-

Resistance	Impedance
① It is an obstruction to constant non fluctuating current or non pulsatile flow	① It is an obstruction to fluctuating current or pulsatile flow.
② Obstruction by friction	② Impedance not only includes opposition to flow by friction but also due to vascular compliance and the blood mass.
③ Changing the compliance would not affect Resistance	③ Changing the compliance affects impedance clamping the Aorta (↑) impedance
④ Vasopressor drug results (↑) in Resistance	④. No effect.

- Windkessel effect :-

- The Lt ventricle pumps blood into the Aorta generating large phasic pressure changes
- The elastic prop of the Aorta & large arteries allow them to expand rapidly as the vol of blood enters the arterial blood
- This expansion allows them to store blood during systole
- the blood vol is then passed onward by recoil during diastole - the arteries act as an accessory pump.
- Phasic flow is made cont — distensibility / Progressive (↓) in diameter of Arteries
- This is called Windkessel effect