BIOMECHANICS

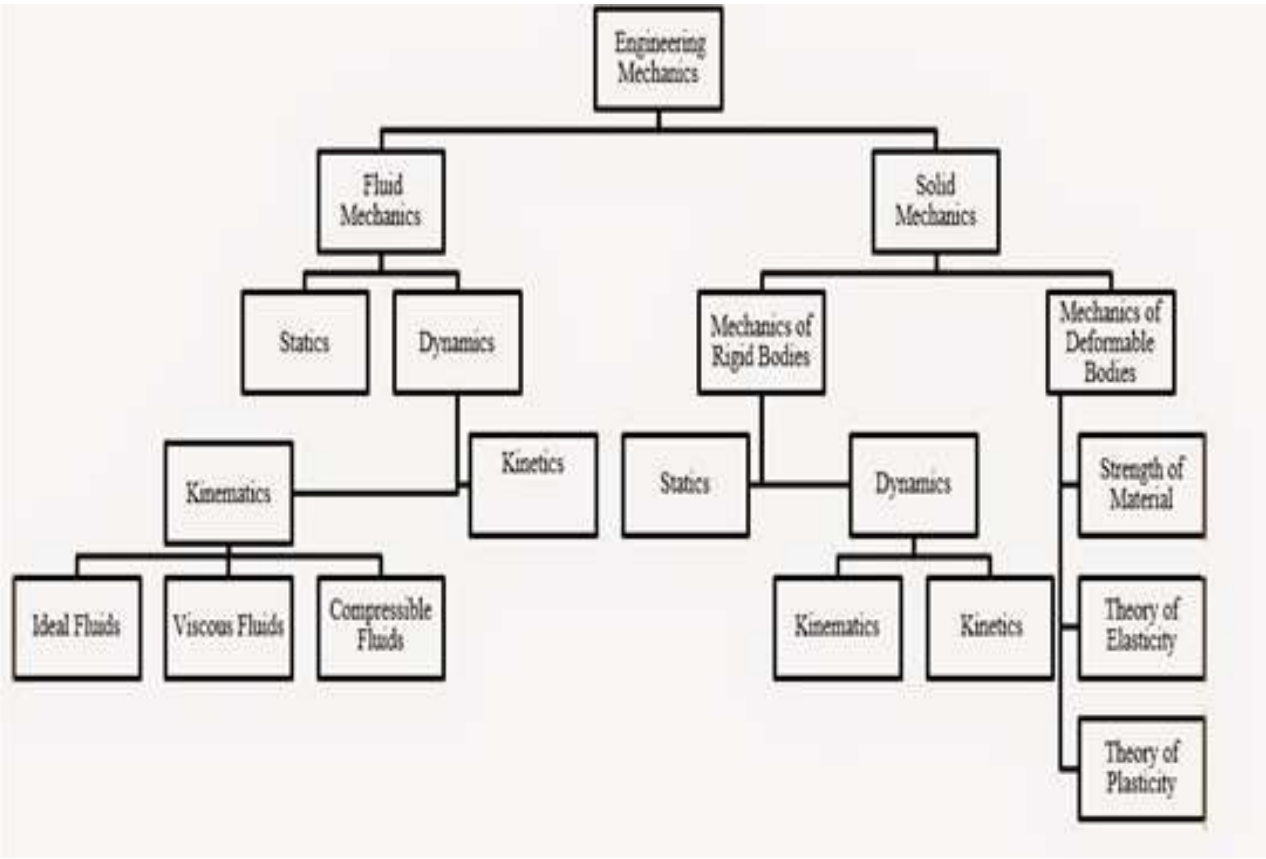
UNIT 1

INTRODUCTION

Scope of mechanics in medicine, mechanics of bone structure, determination of in-vivo elastic modulus. Biofluid mechanics, flow properties of blood.

Mechanics is a branch of physics that is concerned with the motion and deformation of bodies that are acted on by mechanical disturbances called forces. In general biomechanics is the study of the structure and the function of the mechanical aspects of biological system, at any level from whole organism to organs, cell and cell organelles using methods of mechanics.

Engineering mechanics or applied mechanics is the science of applying the principles of mechanics. Applied mechanics is concerned with both the analysis and design of mechanical systems. The broad band field of applied mechanics can be divided into three main parts.



In general, a material can be categorized as either solid or fluid. Solid materials can be rigid or deformable. A rigid body is one that cannot be deformed. In reality every object or material does

undergo deformation to some extent when acted upon by external forces. In some cases the amount of deformation is so small that does not affect the desired analysis. In such cases it is preferable to consider the body is rigid and carry out the analysis with relatively simple computations.

STATICS: It is the study of forces on rigid bodies at rest or moving with a constant velocity.

DYNAMICS: It deals with bodies in motion.

KINEMATICS: Is a branch of dynamics that deals with the geometry and time-dependent aspects of motion without considering the forces the motion.

KINETICS: It is based on kinematics and it includes the effects of forces and masses in the analysis.

The mechanics of deformable bodies deals with the relations between externally applied loads and their internal effects on bodies.

ELASTICITY: An elastic body is defined as one in which all deformation are recoverable upon removal of external forces.

PLASTICITY: A plastic body undergoes permanent deformation when external force is applied.

VISCOELASTICITY: It is the property of the material that exhibit both viscous and elastic characteristics when undergoing deformation. Viscoelastic material exhibits a time delay in returning the material to original shape. Some amount of energy is lost (eg. Honey).

FLUID MECHANICS: It is the study of liquids and gases in equilibrium or in motion.

SCOPE OF MECHANICS IN MEDICINE:

Most biomechanics work aims at increasing basic knowledge about living systems and any artificial interventions thereof. Any serious projects in bio engineering, except those which are well understood, need advanced basic understanding. For example, prosthetic heart valves are now commonplace; but a need exists for improving their service life, minimizing blood trauma, eliminating undesirable blood-artificial-material-interaction, and simplifying anticoagulation managements. To achieve these improvements in a second generation of valves, one must understand the flow of blood in the heart, through the valves, and in the aorta.

A. Clinical Problems in the Cardiovascular System

1. Prosthetic heart valve.
2. Heart assist devices, such as the left ventric1e assist pump, the aortic balloon pump, body acceleration synchronized with heart beat, peripheral cuffs, and diastolic counterpulsation.
3. Extracorporeal circulation. Heart-Iung machine. Hemodialysis machine.
4. Heart replacement.

5. Postoperative trauma, pulmonary edema, and atelectasis.
6. Arterial pulse wave analysis.
7. Ultrasound applications. Phonoangiography. Turbulent noise analysis. Pseudo-sound generation at artherosclerotic constrictions in arteries.

B. Quantitative Physiology

1. Systems analysis of physiology.
2. Rheology of biological tissues, such as blood, muscles, bones, connective tissues, and artificial implantable materials.
3. Analysis of fluid transfer across biological membranes and blood vessels.
4. Diffusion analysis such as pulmonary function, and indicator dilution method.
5. Interfaces. Surfactants in the lung. Thrombogenic tendency of blood on artificial implantable materials. Recent work shows that platelet .action and thrombosis on interfaces are shear stress dependent, thus projecting mechanics to the foreground of this important problem.
6. Microcirculation. Biomechanics has contributed to every aspect of microcirculation research. Perhaps it is a historical accident, but physiologists and mechanics researchers have cooperated throughout the development of this branch of science.

c. Additional Applications

Surgery. Biomechanics of injury and the healing of surgical wounds are topics of great importance. New surgical procedures such as arterio-venous reversal and attachment of implantable mechanical devices are studied.

Implantable Artificial Materials. Materials for prosthesis require a detailed study of their mechanical properties and biocompatibility.

Orthopedics, Orthosis, Orthodontics. Mechanics of bone and cartilage. Reaction of bone to stress resulting either in growth or reabsorption. Joint lubrication. Joint replacement. Orthopedic implants.

Artifical Limb. Design of artificial limb is a classical objective of biomechanics.

Artificial InternaiOrgans. Renal replacement. Artificial kidney. Artificial heart.

Wheelchairs and Beds. Chairs to enable disabled patients to function as normally as possible. Use of back muscle, eye movement, or voice to steer and control such chairs. Design of beds to minimize trauma of bed-bound patients.

Occupational Safety and H ealth. Examples are: mechanics of pulmonary function testing, detection of black lung disease, mechanics of exercises and athletics.

Highway Safety. Head injury research. Seat belt research.Impact analysis.

Safety of internal organs. Design of cars from the point of view of protecting the passengers.

Flight Safety. Vibration and impact of human bodies. Human tolerace to acceleration. Man's reaction to environment such as zero gravity, high heat, low heat, or low oxygen atmosphere.

Flying and Swimming in Nature. Flagellar motion. Microbiallocomotion. Swimming. Flight of insects and birds.

Design of Socially Acceptable Monitoring Instruments. Instruments to meter and record a variety of parameters such as blood pressure, heart rate, respiration rate, electrocardiograms, and

electromyograms, to be carried or worn by patients in a socially acceptable way over a long period of time.

MECHANICS OF BONE STRUCTURE:

A bone is a rigid organ that constitutes part of the vertebrate skeleton. Bones support and protect the various organs of the body, produce red and white blood cells, store minerals, provide structure and support for the body, and enable mobility.

Bone tissue (osseous tissue) is a hard tissue, a type of dense connective tissue. It has a honeycomb-like matrix internally, which helps to give the bone rigidity. Bone tissue is made up of different types of bone cells. Osteoblasts and osteocytes are involved in the formation and mineralization of bone; osteoclasts are involved in the resorption of bone tissue. Bones comes in a variety of shapes and have a complex internal and external structure . one of the types of tissue that makes up bone is the mineralized osseous tissue, also called as bone tissue, that gives rigidity and coral-like three-dimensional internal structure.Other types of tissue found in bones include bone marrow, endosteum, periosteum, nerves, blood vessels and cartilage.

In the human body at birth, there are over 270 bones, but many of these fuse together during development, leaving a total of 206 separate bones in the adult,not counting numerous small sesamoid bones. The largest bone in the body is the femur or thigh-bone, and the smallest is the stapes in the middle ear.

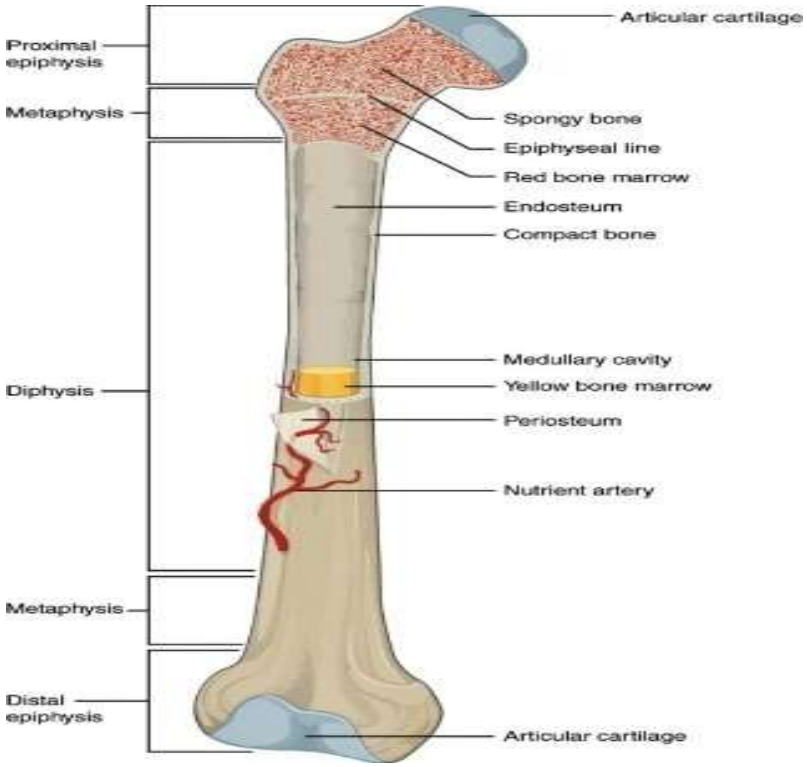
THE ANATOMY OF A LONG BONE:

A long bone has two parts: the diaphysis and the epiphysis. The diaphysis is the tubular shaft that runs between the proximal and distal ends of the bone. The hollow region in the diaphysis is called the medullary cavity, which is filled with yellow marrow. The walls of the diaphysis are composed of dense and hard compact bone.

The wider section at each end of the bone is called the epiphysis (plural = epiphyses), which is filled with spongy bone. Red marrow fills the spaces in the spongy bone. Each epiphysis meets the diaphysis at the metaphysis, the narrow area that contains the epiphyseal plate (growth plate), a layer of hyaline (transparent) cartilage in a growing bone. When the bone stops growing in early adulthood (approximately 18–21 years), the cartilage is replaced by osseous tissue and the epiphyseal plate becomes an epiphyseal line.

The medullary cavity has a delicate membranous lining called the endosteum (end- = “inside”; oste- = “bone”), where bone growth, repair, and remodeling occur. The outer surface of the bone is covered with a fibrous membrane called the periosteum (peri– = “around” or “surrounding”). The periosteum contains blood vessels, nerves, and lymphatic vessels that nourish compact bone. Tendons and ligaments also attach to bones at the periosteum. The periosteum covers the entire outer surface except where the epiphyses meet other bones to form

joints . In this region, the epiphyses are covered with articular cartilage, a thin layer of cartilage that reduces friction and acts as a shock absorber.



Flat bones, like those of the cranium, consist of a layer of diploë (spongy bone), lined on either side by a layer of compact bone. The two layers of compact bone and the interior spongy bone work together to protect the internal organs. If the outer layer of a cranial bone fractures, the brain is still protected by the intact inner layer.

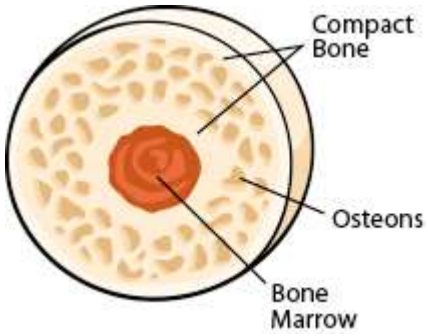
STRUCTURE:

Bone is not uniformly solid, but includes a tough matrix. This matrix makes up about 30% of the bone and the other 70% is of salts that give strength to it. The matrix is made up of between 90 and 95% collagen fibers, and the remainder is ground substance.Each bone in your body is made up of three main types of bone material: compact bone, spongy bone, and bone marrow.

COMPACT BONE:

Compact bone is the heaviest, hardest type of bone. It needs to be very strong as it supports your body and muscles as you walk, run, and move throughout the day. About 80% of

the bone in your body is compact. It makes up the outer layer of the bone and also helps protect the more fragile layers inside.



If you were to look at a piece of compact bone without the help of a microscope, it would seem to be completely solid all the way through. If you looked at it through a microscope, however, you would see that it's actually filled with many very tiny passages, or canals, for nerves and blood vessels. Compact bone is made of special cells called osteocytes. These cells are lined up in rings around the canals. Together, a canal and the osteocytes that surround it are called osteons. Osteons are like thick tubes all going the same direction inside the bone, similar to a bundle of straws with blood vessels, veins, and nerves in the center.

SPONGY BONE:

Spongy bone is found mostly at the ends of bones and joints. About 20% of the bone in your body is spongy. Unlike compact bone that is mostly solid, spongy bone is full of open sections called pores. If you were to look at it in under a microscope, it would look a lot like your kitchen sponge. Pores are filled with marrow, nerves, and blood vessels that carry cells and nutrients in and out of the bone. Though spongy bone may remind you of a kitchen sponge, this bone is quite solid and hard, and is not squishy at all.

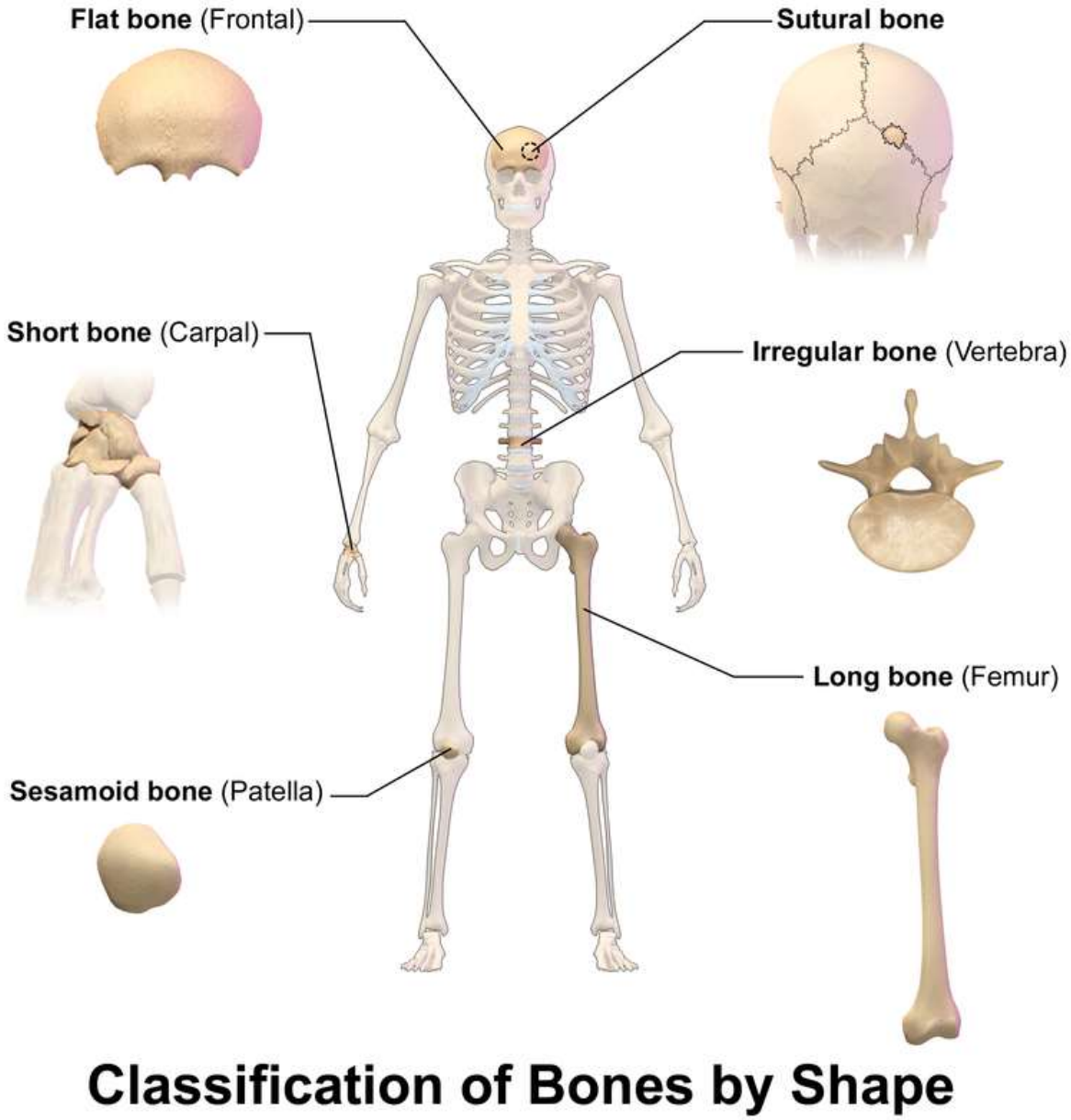
BONE MARROW:

The inside of your bones are filled with a soft tissue called marrow. There are two types of bone marrow: red and yellow. Red bone marrow is where all new red blood cells, white blood cells, and platelets are made. Platelets are small pieces of cells that help you stop bleeding when you get a cut. Red bone marrow is found in the center of flat bones such as your shoulder blades and ribs. Yellow marrow is made mostly of fat and is found in the hollow centers of long bones, such as the thigh bones. It does not make blood cells or platelets. Both yellow and red bone marrow have many small and large blood vessels and veins running through them to let nutrients and waste in and out of the bone.

When we were born, all of the marrow in your body was red marrow, which made lots and lots of blood cells and platelets to help your body grow bigger. As you got older, more and

more of the red marrow was replaced with yellow marrow. The bone marrow of full grown adults is about half red and half yellow.

TYPES OF BONES:



There are five types of bones in the human body: long, short, flat, irregular, and sesamoid.

- Long bones are characterized by a shaft, the diaphysis, that is much longer than its width; and by an epiphysis, a rounded head at each end of the shaft. They are made up mostly

of compact bone, with lesser amounts of marrow, located within the medullary cavity, and areas of spongy, cancellous bone at the ends of the bones. Most bones of the limbs, including those of the fingers and toes, are long bones. The exceptions are the eight carpal bones of the wrist, the seven articulating tarsal bones of the ankle and the sesamoid bone of the kneecap. Long bones such as the clavicle, that have a differently shaped shaft or ends are also called *modified long bones*.

- Short bones are roughly cube-shaped, and have only a thin layer of compact bone surrounding a spongy interior. The bones of the wrist and ankle are short bones.
- Flat bones are thin and generally curved, with two parallel layers of compact bones sandwiching a layer of spongy bone. Most of the bones of the skull are flat bones, as is the sternum.
- Sesamoid bones are bones embedded in tendons. Since they act to hold the tendon further away from the joint, the angle of the tendon is increased and thus the leverage of the muscle is increased. Examples of sesamoid bones are the patella and the pisiform.
- Irregular bones do not fit into the above categories. They consist of thin layers of compact bone surrounding a spongy interior. As implied by the name, their shapes are irregular and complicated. Often this irregular shape is due to their many centers of ossification or because they contain bony sinuses. The bones of the spine, pelvis, and some bones of the skull are irregular bones. Examples include the ethmoid and sphenoid bones.

FUNCTIONS OF BONE:

Bones form an important component of the skeletal system. They perform a wide range of important functions that can be classified into three categories:

Mechanical Functions of bones:

Protection:

At numerous places inside the body, bones serve to protect important and delicate organs. The best examples to be quoted here are those of brain (which is protected by the skull) and heart (which is protected by the ribcage).

Shape:

Because of their rigid nature, bones provide a framework around which the body is built. So bones are responsible for the shape and form of human body.

Movement:

Working with skeletal muscles, tendons, ligaments and joints, the bones form the moving machinery of human body. The major role of bones in movement is that they act as levers, which make use of the forces generated by skeletal muscles in a beneficial way.

Synthetic Functions of Bones:

Synthesis of blood cells:

The major synthetic role of bones is to produce blood cells. The bones themselves are not capable of doing this. Instead, they house the bone marrow, which contains Hematopoietic stem cells, capable of producing blood cells. In infants, bone marrow of all long bones is capable of this synthesis; however, as a person gets older, the red marrow turns into yellow fatty marrow, which is no more capable of hematopoiesis. The red marrow in adults and older individuals is restricted to vertebrae and heads of tibia and femur.

Metabolic Functions of Bones:

Mineral Storage:

Bones serve as an important store house of minerals such as calcium and phosphorus.

Fat storage:

The yellow bone marrow of long bones acts as a storage of fats.

Role in acid-base balance:

Bone buffers the blood against excessive pH changes by absorbing or releasing alkaline salts.

MECHANICAL PROPERTIES OF BONE:

Bone is hard and has a stress-strain relationship similar to many engineering materials. Hence stress analysis in bone can be made in a way similar to the usual engineering structural analysis.

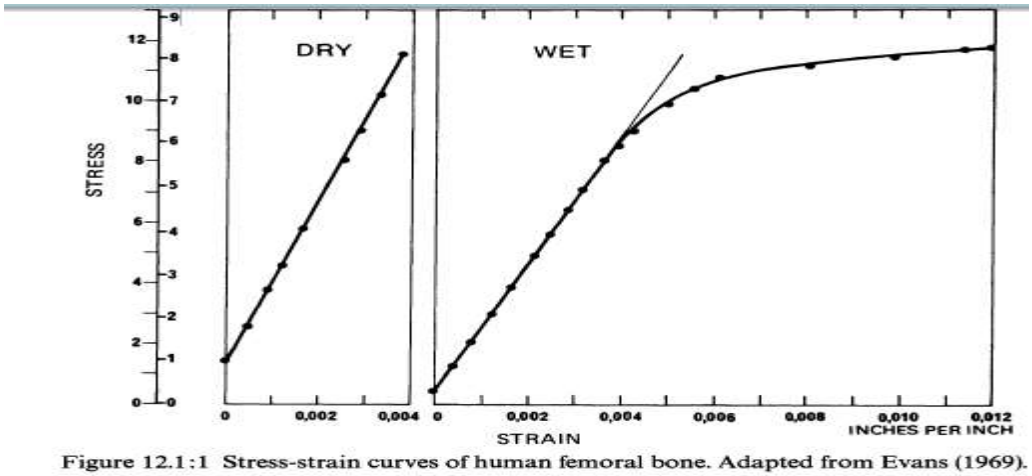


Figure 12.1:1 Stress-strain curves of human femoral bone. Adapted from Evans (1969).

Figure 12.1: 1 shows the stress-strain relationship of a human femur subjected to uniaxial tension. It is seen that dry bone is brittle and fails at astrain of 0.4%; but wet bone is less so, and fails at astrain of 1.2%. Figure 12.1: 1 suggests that Hooke's law is applicable for a limited range of strains. For uniaxialloading below the proportional limit, the stress *(J* is related to the strain 8 by*(J = E€,* where *E* is the Y oung's modulus.

Thus, for adult human femoral compact bone, the ultimate bending strength is 160 MPa (16 kgjmm2), and the ultimate shear strength in torsion is 54.1 ± 0.6 MPa, whereas the modulus of elasticity in torsion is 3.2 GPa (326 kgjmm2). It is weIl known also that the strength of bone varies with the age and sex of the animal, the loeation of the bone, the orientation of the load, the strain rate, and the test eondition (whether it is dry or wet).

DETERMINATION OF INVIVO ELASTIC MODULUS:

An elastic modulus (also known as modulus of elasticity) is a number that measures an object or substance's resistance to being deformed elastically (i.e., non-permanently) when a stress is applied to it.

$$\lambda \stackrel{\text{def}}{=} \frac{\text{stress}}{\text{strain}}$$

Invivo elastic modulus include analysis of elastic modulus within the body

Example:

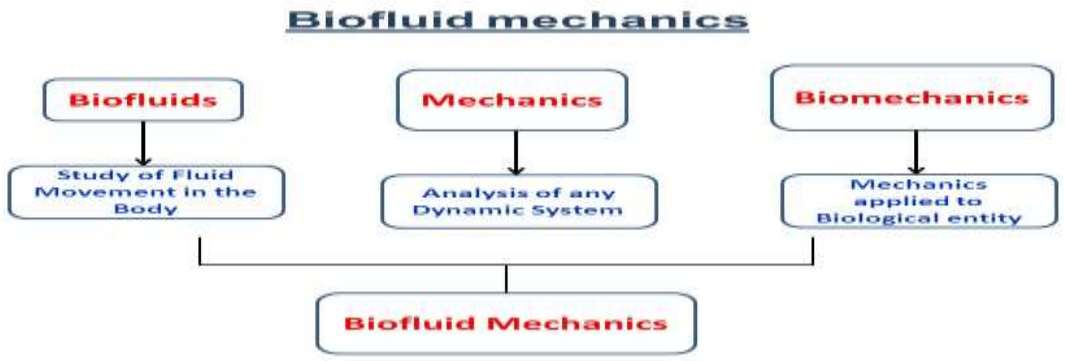
1) In vivo determination of young's modulus for the human cornea.

2) In Vivo Determination of Elastic Modulus of Canine Cardiac Muscle.

3) In vivo determination of the elastic response of bone.

BIOFLUID MECHANICS:

It is defined as the study of both liquid and gas fluid flows in or around biological organisms.



PROPERTY OF FLUID:

1) DENSITY(ρ):

Density, is defined as the mass of a unit volume of a material substance. The formula for density is ρ = M/V, where d is density, M is mass, and V is volume. Density is commonly expressed in units of grams per cubic centimetre. The symbol most often used for density is ρ

2) VISCOCITY(μ):

The viscosity of a fluid is a measure of its resistance to gradual deformation by shear stress or tensile stress. For liquids, it corresponds to the informal concept of "thickness"; for example, honey has a much higher viscosity than water. Unit for viscosity is poise, for biofluids it is centipoise.

3) SHEAR STRESS (τ):

A shear stress, often denoted τ , is the component of stress coplanar with a material cross section. Shear stress arises from the force vector component parallel to the cross section. Normal stress, on the other hand, arises from the force vector component perpendicular to the material cross section on which it acts.

$$S=F/A$$

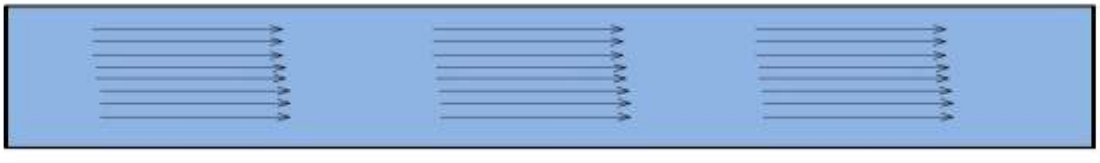
4) NEWTONIAN FLUID:

A Newtonian fluid is a fluid in which the viscous stresses arising from its flow, at every point, are linearly proportional to the local strain rate—the rate of change of its deformation over time. (ie) shear stess is proportional to the rate of deformation of the blood vessel.

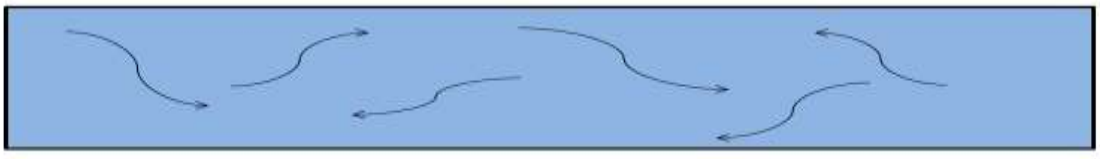
In large arteries the flow is Newtonian (laminar flow)

In small arteries and capillaries the flow is non-newtonian (turbulent flow).

LAMINAR AND TURBULANT FLOW:



Laminar Flow



Turbulent Flow

S.NO	LAMINAR FLOW	TURBULENT FLOW
1.	It is a fluid flow in which the fluid layers move parallel to each other and do not cross each other.	It is a fluid flow in which the fluid layers cross each other and do not move parallel to each other.
2.	The laminar flow generally occurs in the fluid flowing with low velocity.	The turbulent flow occurs when the fluid flows with high velocity.
3.	Laminar flow occurs in the small diameter pipes in which fluid flows with low velocity.	Turbulent flow occurs in large diameter pipes in which fluid flows with high velocity.
4.	The fluid flow is laminar when the value of Reynolds number (Re) is less than 2000.	The fluid flow is turbulent when the value of Reynolds number is greater than 4000.
5.	Shear stress in laminar flow depends only on the viscosity of the fluid and independent of the density.	Shear stress in the turbulent flow depends upon the density of the fluid.
6.	The fluid flow is very orderly i.e. there is no mixing of adjacent layers of the fluid and they move parallel to each other and also with the walls of the pipe.	The fluid flow is not orderly i.e. there is mixing of adjacent layers of fluid with each other and they do not move parallel to each other and also with the walls of the pipe.

FLOW PRINCIPLE:

The flow principle of fluid is based in two laws.

1) Conservation of mass

2) Conservation of energy

CONSERVATION OF MASS:

When the fluid flows at a constant rate in a tube the mass flow rate must be the same for all cross section along the length.

CONSERVATION OF ENERGY:

When the fluid flows at a constant rate in a tube if no energy is added to the system as work or heat then the total energy of the fluid is conserved and the tube is artery and vein.

REYNOLD'S NUMBER:

The Reynolds number is defined as the ratio of inertial forces to viscous forces and consequently quantifies the relative importance of these two types of forces for given flow conditions.

$$Re = \frac{\text{inertia forces}}{\text{viscous forces}}$$

If the viscous force dominates the inertia force then the flow is oscillatory or pulsatile or the inertia force dominates the viscous force then the flow is turbulence. So by Reynolds number one can identify the flow is laminar or turbulent.

Laminar flow: Re < 2000

Turbulent Flow: Re > 4000

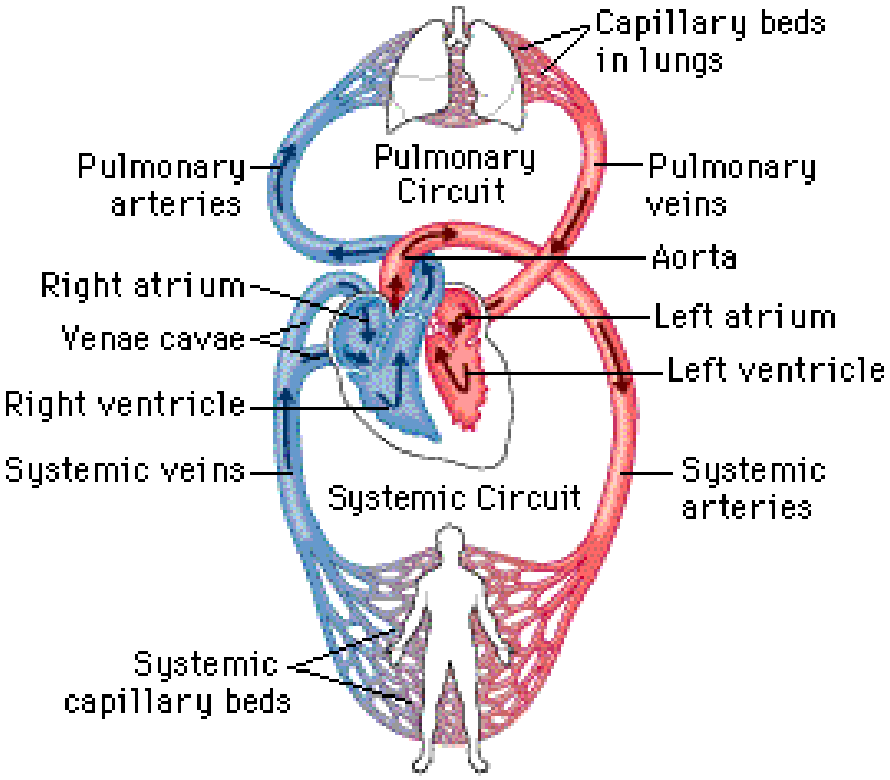
FLOW PROPERTIES OF BLOOD:

William Harvey was the first known physician to describe completely and in detail the systemic circulation and properties of blood being pumped to the brain and body by the heart. The main function of circulatory system is the transport of oxygen and nutrients to all part of the body. It consist of,

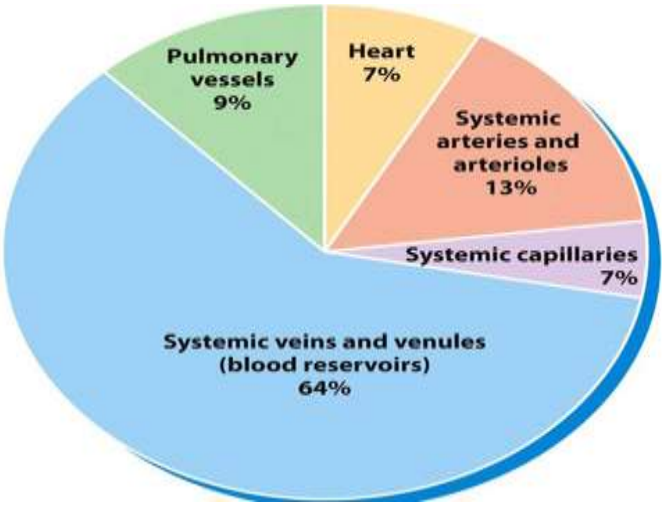
Heart-acts as pump

Blood vessels- delivery routes

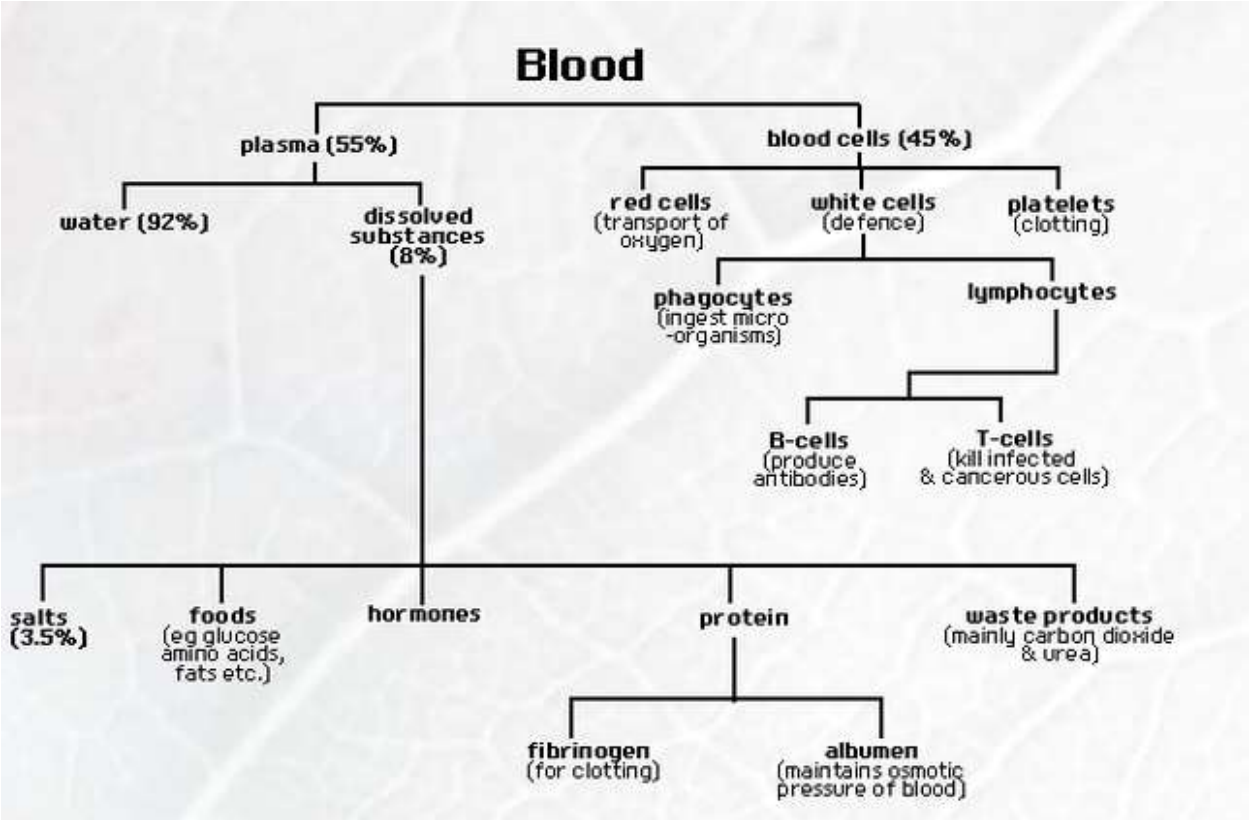
Blood –transport medium



In Circulatory system the total blood volume is uneventfully distributed,



COMPOSITION OF BLOOD:



HAEMATOCRIT:

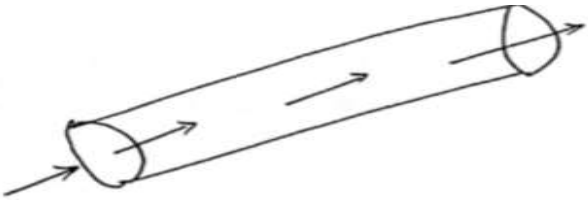
The ratio of the volume of red blood cells to the total volume of blood. It is normally 47% for men and 42% for women.

HAEMATOCRIT=volume of RBC/ volume of blood.

BLOOD FLOW IN ARTERY:

The aorta and artery have low resistance to blood flow the elastic property of the arteries help to convert the pulsatile flow of blood in to a more continuous flow. An accurate model of blood flow in arteries would include the following features.

1) Flow is pulsatile.
2) Arteries are elatic and tapered tubes.
3) The geometry of arteries is complex because it include tapered, curved and branching tubes.
4) In small arteries the viscocity depends up on the vessel radius and shear rate.
   These features are qualiative.
   The general time dependent governing equation of fluid flow in a straight cylindrical tube are given by contuinity and Navier's stoke equation.



$$\frac{\partial V}{\partial r} + \frac{V}{r} + \frac{\partial U}{\partial z} + \frac{U}{r} = 0$$

U and V – axial and radial components of fluid velocity.

r- radial coordinate in time axis.

$$\frac{\partial P}{\partial r} + \frac{P}{r} + \frac{\partial M}{\partial r} + \frac{M}{r} = 0$$

ρ- density of biofluids.

μ- viscocity of biofluids.

The simplest model of the steady laminar flow in a uniform circular cylinder is known as "Hagen-poiseuille flow".

For axis symmetric flow in a circular tube of internal radius R_0 and length (l) the boundary conditions are,

$$u(r = R_0) \quad \text{and} \quad \frac{\partial v}{\partial r}(r = 0) = 0$$

Consider the pressure is uniform along the tube, so poison's equation is obtained.

$$\mu(r) = \frac{-\Delta P}{4\mu l}(R_0^2 - r^2)$$

ΔP - change in pressure

For this flow the velocity is maximal and it is given by,

$$U_{max} = (R_0)^2 \frac{\Delta P}{4\mu l}, \; r = 0$$

So there will be resistance to flow and the resistamce is vascular resistance.
Vascular resistance (R) is the ratio of change in pressure to the flow.

$$R = \frac{\Delta P}{Q} \quad \frac{mmHg}{cm^3/s}$$

The arteries are composed of elastin, collagen fibers and smooth muscles. Because of this ability to expand as transmural pressure increases. Blood vessel may function to store blood volume under preesure. They function as capacitive elements similar to storge tanks. Therefore the vascular capacitance is given by,

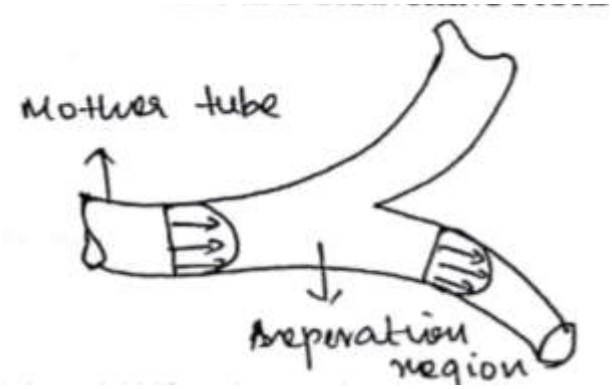
C=dv/dp

Capacitance increases, pressure increases with age. Veins have high capacitance than arteries.

FLOW IN CURVE TUBES:

Arteries are not generally straight uniform tubes it have some curve structures, when the stedy fluid enters the curved pipe in the horizontal plane all of its element are subjected to centripetal acceleration (force acting inwards). This centripetal acceleration direction will be normal to the original direction and directed towards the bend centre. The force is supplied by a pressure gradient, the plane of the bend so the net result is that the faster moving elements that occupy the core fluid near the centre of the tube, are swept towards the outside of the bend along the plane . And their place is occupied by the slower moving fluid located near the walls.

FLOW IN BIFUCATING AND BRANCHING SYSTEMS (flow is laminar):



A simplified arterial bifurcation may be represented by 2 curve tubes attached to a strong mother tube so the pattern of the blood flow in the downstream is in general similar to that of the flow in curve tubes. Centripetal acceleration will play a main role.

FLOW PROPERTIES IN VEIN:

Veins are thin walled tubular structure that may collapse when subjected to negative transmural pressure.

TRANSMURAL PRESSURE: Transmural pressure is the difference in pressure between two sides of a wall or equivalent separator.

$$\text{Transmural pressure} = P_{int} - P_{ext}$$

The collapsing of veins is classified in to 3 types based on the different pressure across the tube.

1) Pressure>0 – the tube (vein) is inflated, the cross section area increases and maintain circular shape.

2) Pressure<0- the cross section area of the tube collapse and maintain ellipse shape.

3) at certain negative transmural pressure the contact is obtained between the opposite walls thereby generating two lumens.

BLOOD RHEOLOGY:

Rheology is the branch of science that deals with the deformation of the flow of the blood in the blood vessels. Blood is the suspension of RBC, WBC, plasma and platelets. Blood plasma is an incompressible Newtonian fluid with the viscosity of about 1.2 centipoise at 37 degree Celsius. Normal human blood has a haematocrit of 40-45% so the rheological properties are strongly influenced by the red blood cells.

RBC:

RBC are considered as Newtonian fluid with the viscosity of about 6-7 centipoise at normal condition. The fluid interior of human RBC has low resistance to shear and bending deformation and due to the presence of excess surface area for its volume makes it highly deformable. Thus the RBC cells can easily pass through capillaries with the diameter lesser than its resting diameter.

WBC:

Leucocytes are lesser in number than RBC in normal blood. So they are less deformable and so it is neglected in rheological consideration.

PLATELETS:

Platelets are rheological un important in normal blood.

RHEOLOGY IN MICROVESSEL:

Rheological properties of blood flowing in micro vessels (arterioles, venules, capillaries) have been extensively studied by in vitro experiment. RBC suspension is Newtonian. Two key parameters are 1) apparent viscosity 2) the hematocrit of small tubes.

APPARENT VISCOSITY:

Apparent Viscosity is the shear stress applied to a fluid divided by shear rate. For a Newtonian fluid, the apparent viscosity is constant, and equal to the Newtonian viscosity of the fluid, but for non-Newtonian fluid, the Apparent Viscosity depends on shear rate.

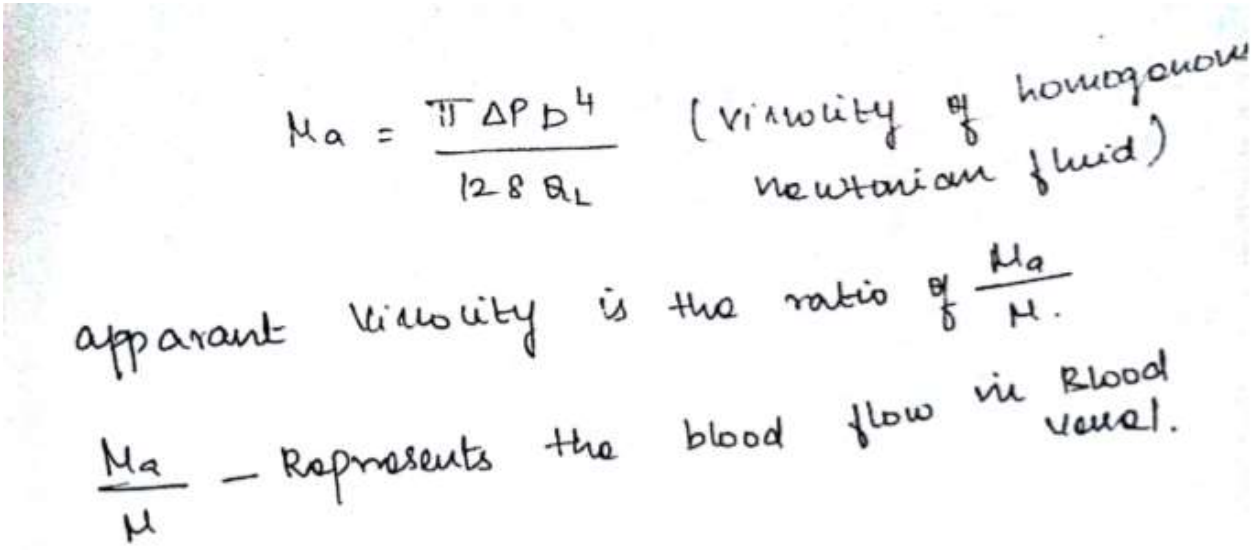
For a steady flow of a homogenous Newtonian fluid through a circular cylindrical tube the volume flow rate Q varies directly with pressure drop and also to the 4^{th} power of the tube diameter (d).

$$Q = \frac{\pi \Delta P D^4}{128 \mu l}$$

l- Length of the tube

μ- fluid viscosity

For micro vessels,



THE HEMATOCRIT OF SMALL TUBES

1) **Fahraeus effect:**

The Fahraeus effect is the decrease in average concentration of red blood cells in human blood as the diameter of the glass tube in which it is flowing decreases. In other words, in blood vessels with diameters less than 500 micrometers, the hematocrit decreases with decreasing capillary diameter.

2) **Fahraeus Lindquist effect:**

Resistance to flow through a small tube is smaller. The tube includes the micro vessels.

UNIT II MECHANICS OF PHYSIOLOGICAL SYSTEMS

Heart valves, power developed by the heart, prosthetic valves. Constitutive equations for soft tissues, dynamics of fluid flow in cardiovascular system and effect of vibration - shear stresses in extra-corporal circuits.

UNIT – 5
MECHANICS OF PHYSIOLOGICAL SYSTEMS

HEART VALVES:

Four cardiac valves help to direct flow through the heart. Heart valves cause blood to flow only in the desired direction. If a heart without these valves were to contract, it would compress the blood, causing it to flow both backward and forward (upstream and downstream). Instead, under normal physiological conditions, heart valves act as check valves to prevent blood from flowing in the reverse direction. In addition, heart valves remain closed until the pressure behind the valve is large enough to cause blood to move forward.

Each human heart has two atrioventricular (AV) valves which are located between the atria and the ventricles. The tricuspid valve is the valve between the right atrium and the right ventricle. The mitral valve is the valve between the left atrium and the left ventricle. The mitral valve prevents blood from flowing backward into the pulmonary veins and therefore into the lungs, even when the pressure in the left ventricle is very high. The mitral valve is a bicuspid valve, which has two cusps, while a tricuspid valve has three cusps. The other two valves in the human heart are known as semilunar valves. The two semilunar valves are the aortic valve and the pulmonic valve. The aortic valve is located between the aorta and the left ventricle, and when it closes, it prevents blood from flowing backward from the aorta into the left ventricle. An aortic valve is shown in Fig. The pulmonic valve is located between the right ventricle and the pulmonary artery, and when it closes, it prevents blood from flowing backward from the pulmonary artery into the right ventricle.

Clinical features:

Chordae tendineae rupture and papillary muscle paralysis can be consequences of a heart attack. This can lead to bulging of the valve, excessive backward leakage into the atria (regurgitation), and even valve prolapse. Valve prolapse is the condition under which the valve inverts backward into the atrium. Because of these valve problems, the ventricle doesn't fill efficiently. Significant further damage, and even death, can occur within the first 24 h after a heart attack, because of this problem.

Prosthetic Heart Valves

Prosthetic heart valves are devices used for replacing damaged or diseased natural valves of the heart. The natural valves are excised out and replacements are implanted

Need for Prosthetic heart valve:

- The heart valves, normally due to certain diseases, fail to function as unidirectional check valves; they either become too leaky in the closed state (Regurgitation) or very narrow and offer resistance to blood flow in the open state (Stenosis)
    - **Regurgitation** (or valvular insufficiency, incompetence, "leaky valve"), occurs when the leaflets do not close completely, letting blood leak backward across the valve. This backward flow is referred to as "regurgitant flow."
    - A regurgitant (incompetent, insufficient, or leaky) valve does not close completely, letting blood move backward through the valve.

Mechanical Valves

I. Caged ball valve:

The ball valve was the first mechanical heart valve used and designed by Charles Hufnagel. The Starr-Edwards ball valve was first used clinically as a mitral valve replacement in 1960. After the Starr-Edwards valve was established, several other design variations were created such as Magovern–Cromie, DeBakey–Surgitool, and Smeloff–Cutter ball valves.

Ball valves operate on the simple principle that the ball will be forced to one side of the valve or the other depending on which way blood is flowing. They were modeled after ball valves used in industrial applications to allow the flow of fluids on only one direction. When the pressure exerted by the heart onto the blood (and the ball) exceeds the pressure in the aorta, the ball is pushed away from the heart. This is the open position of the valve and blood can flow out of the heart into the aorta. After the heart ejects blood, the pressure inside the heart is greatly reduced so blood will try to flow back inside the heart. The negative pressure sucks the ball valve backwards. It fits over the opening of the heart and prevents backflow of blood. In a natural heart valve, blood flows directly through the center of the valve (central flow.) With a ball heart valve, the heart must work harder to push blood around the ball. There is no central flow with a ball valve and although it works in principle, it is not a good solution. Ball valves also are known to damage or kill blood cells due to colliding with the ball.

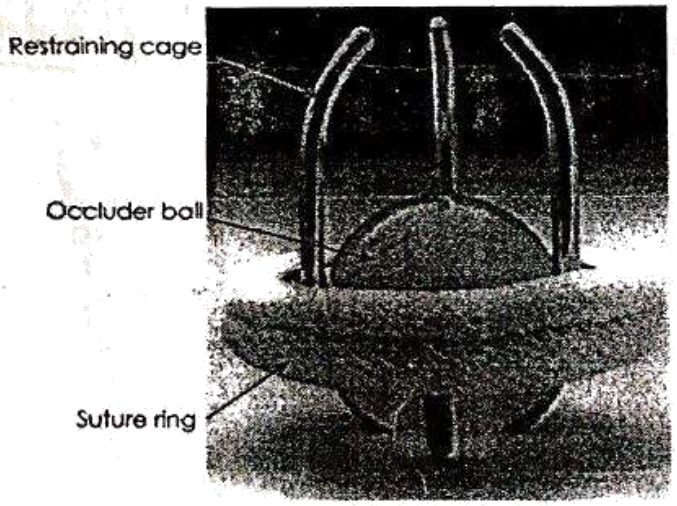
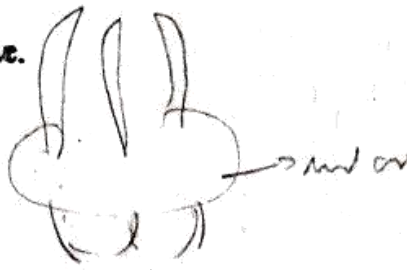


Fig 2: Caged ball valve.



Advantages

- Oldest
- durability up to 40 yrs

Disadvantages

- high profile
- hemolysis (rupture of RBC)
- high thrombogenecity (Blood clot)
- Poor hemodynamics in small sizes

Unique features

- Occluder travels completely out of the orifice
- Continuously changing points of contact of the ball reduces the wear & tear in any one area
- Thrombogenic risk 4-6% / year

II. Tilting disc valve

In the mid-1970s, a new valve was introduced: the tilting disc valve. A more modern tilting disc valve is showing in Figure 3 and some earlier models are shown in Figure 4. The purpose in creating the titling-disc valve was to restore the central blood flow that was lost with the ball valve design. These valves consist of a single circular disc restrained by two metal struts and a metal ring. The struts are attached to the metal ring. The struts prevent the disc from escaping the device in either direction. The disc opens and closes based on the same principles used in the ball valve design, except a disc is used instead of a ball.

Tilting disc valves can open at an angle of 60° and at a rate of 70 beats per minute. The angular opening of this valve reduces damage to blood cells. These are major improvements over the ball design but the struts of the tilting disc valves tend to fatigue and fracture over long periods of time.

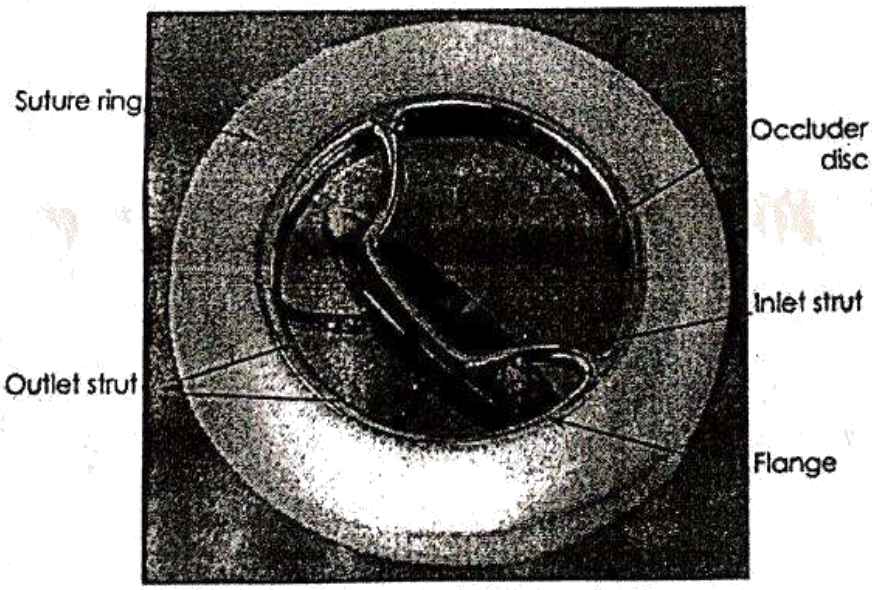


Figure 3: Over view of the titling-disc valve

Adv :

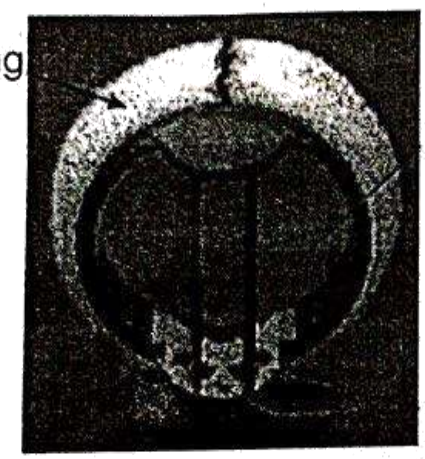
- low profile
- Good hemodynamics even in small sizes
- Excellent durability
- Permit central laminar flow

Disadv:

- Anticoagulation mandatory
- higher risk of thrombosis than cage ball valve
- sudden catastrophic valve thrombosis

Bileaflet valves

The first bileaflet valves were introduced in 1978. Some bileaflet valves are shown in Figures 5 and 6. The bileaflet design consists of two semicircular leaflets which pivot on hinges. Bileaflet valves have the best central flow – the leaflets open completely, allowing very little resistance to blood flow. These valves correct the problem of central flow and blood cell damage; however, they allow some backflow. This is a serious design flaw; many natural heart valves are replaced with mechanical valves because the valve became stiff and allowed backflow. Nevertheless, the majority of mechanical heart valves used today are bileaflet valves because they allow the least resistance to flow and the least blood damage.



Adv –

- Low bulk - flat profile
- Less thrombogenicy
- Central laminar flow
- two semicircular discs that pivot between open and closed positions
- No need for supporting struts
- Good hemodynamics even in small sizes
- 2 lateral ,1 central minor orifice , no chance of sudden catastropic thrombosis

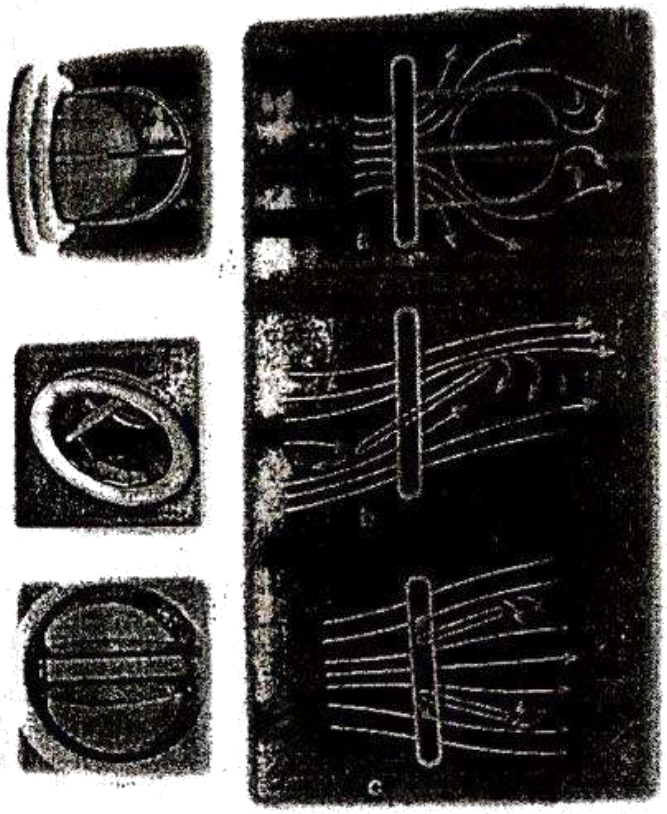
Disadv-

- Anticoagulation mandatory
- risk of thrombosis

Current mechanical valves are manufactured from a variety of materials,such as pyrolitic carbon and titanium. Structural failure of mechanical valves is rare, but, when it occurs, is usually catastrophic [Giddens et al., 1993]. One major disadvantage of the useof mechanical valves is the need for continuous, life-long anticoagulation therapy to minimize the risk of thrombosis and thromboembolic complications. Unfortunately, the anticoagulation therapy may lead to bleeding problems; therefore, careful control of anticoagulation medication is essential for the patient's well-being and quality of life. Another concern is the hemodynamic performance of the prosthesis. The hemodynamic function of even the best designs of mechanical valves differs significantly from that of normal heart valves.

Evolution – based on fluid dynamics

- Ball at the centre of fluid pathway made the caged ball design inferior leading to high pressure gradients and increased thromboembolic complications
- Substantially improved the gradients and provided improved flow characteristics
- Improved the hemodyanmics further providing a near central flow which also eliminated the complications due to the asymmetry in the flow characteristics of the tilting disc design



Tissue Valves or bioprosthetic valves

Tissue heart valves can come from a variety of sources: porcine (pig), bovine (cow) and homografts or allografts (human). The primary advantage of a tissue valve over a mechanical valve is that you do not need to be on lifelong blood-thinner medication.

1. Porcine and Bovine Tissues

Tissue valves are made with tissues from porcine (pig) heart valves or bovine (cow) cardiac tissue because they function like human heart valves. Once the animal tissue is removed, it is chemically treated to preserve the tissue and prevent immulogic reactions once it is placed.

2. Homografts or Allografts

A homograft or allograft is a human valve obtained from a human donor. This type of valve is particularly beneficial for pregnant women and children because it does not require long-term anticoagulation therapy. In addition, it provides near-native hemodynamic performance. Doctors use the term "hemodynamics" to describe the flow of blood through the heart valve. Because the availability of these valves is dependent upon donors, it is often limited.

Tissue prostheses gained widespread use during the mid-1970s. The major advantage of tissue valves compared to mechanical valves is that tissue valves have a lower incidence of thromboembolic complications [Butchart & Bodnar, 1992].
Therefore, most patients receiving tissue valves do not have to take anticoagulants long-term.

Advantages

The primary advantage of a tissue valve is that you do not need to be on lifelong blood-thinner medication (anticoagulation medication) unless you have another condition that makes it necessary.

Tissue heart valves from St. Jude Medical are supported by data and clinical experience. Two independent publications indicate superior durability to competitive valves out to 20 years. A valve's hemodynamic performance affects your quality of life immediately following the heart valve implant and throughout your lifetime. Tissue valves with good hemodynamic performance provide optimal blood flow and therefore allow for heart valve efficiency and activity.

Disadvantages

The main drawback with tissue heart valves is they are not as durable as mechanical valves. Various clinical studies indicate that tissue heart valves may last from 8 to 20 years depending on their position. Aortic valves have tended to last longer than mitral valves in these studies. The exact timing depends on the type of tissue valve, your age, lifestyle, medication requirements and other factors. The major disadvantages to tissue valves are large pressure drops compared to some mechanical valves (particularly in the smaller valve sizes), jetlike flow through the valve leaflets, material fatigue and/or wear of valve leaflets, and calcification of value leaflets, especially in children and young adults. Valve deterioration, however, usually takes place slowly with tissue valves, and patients can be monitored by echocardiography and other noninvasive techniques. The clear advantage of mechanical valves is their long-term durability.

Two major disadvantages with the use of mechanical valves is the need for life-long anticoagulation therapy and the accompanying problems of bleeding [Butchart & Bodnar, 1991]. Furthermore, the hemodynamic function of even the best designed valves differs significantly from that of the natural healthy heart valve. An obvious step in the development of heart valve substitutes was the use of naturally occurring heart valves. This was the basis of the approach to the use of antibiotic or cryotreated human aortic valves (homografts: from another member of the same species) removed from cadavers for implantation in place of a patient's own diseased valve.

The first of these homograft procedures was performed by Ross in 1962, and the overall results so far have been satisfactory. This is, perhaps, not surprising since the homograft replacement valve is optimum both from the point of view of structure and function. In the open position these valves provide unobstructed central orifice flow and have the ability to respond to deformations induced by the surrounding anatomical structure. As a result, such substitutes are less damaging to the blood when compared with the rigid mechanical valve. The main problem with these cadaveric allografts, as far as may be ascertained, is that they are no longer living tissue and therefore lack that unique quality of cellular regeneration typical of normal living systems. This makes them more vulnerable to long-term damage. Furthermore, they are only available in very limited quantities. An alternative approach is to transplant the patient's own pulmonary valve into the aortic position.

POWER DEVELOPED BY THE HEART:

The human heart is a pump that is made of muscle tissue. It has four chambers: the right atrium and the left atrium, which are located at the top, and the right ventricle and left ventricle, which are located at the bottom. A special group of cells called the sinus node is located in the right atrium. The sinus node generates electrical stimuli that make the heart contract and pump out blood. Each contraction represents a heartbeat. When the heart contracts it is in a systolic phase and when it rests it is in a diastolic phase. It takes blood about a minute to circulate through the cardiovascular system and pump oxygenated blood throughout the body.

The power of the heart can be calculated by multiplying the pressure by the flow rate. An average person has six liters of blood that circulates every minute, making the flow rate 10^{-4} m^3/s (cubic meters per second). The pressure of the heart is about 10^4 pascal, making the heart's power about one watt. This is the power of a typical human heart, but it's different for everyone.

The average heart beats about 75 times per minute, which is about five liters of blood per minute. Although this isn't much, it enables the heart to complete a tremendous amount of work in a person's lifetime. The human heart beats about 40 million times a year, which adds up to more than 2.5 billion times in a 70-year lifetime. This results in approximately 2 to 3 billion joules of work in a lifetime, which is a huge amount.

Constitutive equations for ligament and other soft tissue

ABSTRACT

Ligaments, tendons and other soft tissues are nonlinearly viscoelastic. To discriminate among various constitutive equations which may be used to describe the tissue, appropriate experimental modalities are requisite. Ideally, testing should span physiologic ranges for load (or strain), load history (recovery and reloading), and load onset and duration, and a robust model will fit all data. Methods to expand the experimental window of time for relaxation and creep are presented and evaluated. The role of ramp, relaxation and recovery protocols is studied in the context of viscoelasticity describable by linear, quasilinear (QLV), nonlinear superposition, Schapery, and multiple integral formulations. The advantages associated with testing protocols that expand the time windows for creep or relaxation are presented.

INTRODUCTION

Like other soft tissues in the body, tendons and ligaments exhibit viscoelastic, or time-dependent, behavior. When these tissues are held at a constant strain level, stress in the tissues decrease, a phenomenon called stress relaxation. Conversely, when held at a constant stress level, strain in the tissues increase, known as creep. Creep and relaxation are important components of tissue behavior, and investigation of such behavior takes careful consideration. An optimal experiment extracts the maximum amount of useful information from the specimen being tested. This may often require performing multiple phases in the experiment, such as testing at various strain levels or strain rates, to robustly capture the true behavior. As an example, performing a single stress relaxation test on a specimen at one specific strain level will give information about the time-dependent nature of the specimen, but it fails to give any insight about linearity. Performing relaxation tests at multiple strain levels would be required to determine any nonlinear viscoelastic properties. It may also be necessary to perform viscoelastic tests at various strain rates, as any stress-strain curve generated will depend on the strain rate utilized during data collection. While a concave-up stress-strain curve is an indication of nonlinear behavior, a concave-down curve could be due to nonlinearity or to time dependence or both.

Careful planning is also required in order to determine which constitutive equation provides the best representation of the data. The purpose of this paper is not only to present constitutive equations for the modeling of soft tissues such as tendon and ligament, but also to suggest the means by which to evaluate the best choice of constitutive model by experimentation. In addition, methods to expand the time window of observation are discussed.

CONSTITUTIVE EQUATIONS

Several constitutive equations have been utilized in the analysis of soft tissue mechanics. Of the most popular are quasi-linear viscoelasticity (QLV) and nonlinear superposition. It is also possible to use linear superposition (over a restricted range of load or strain), Schapery nonlinear equations, and other single or multiple integral models. Determining which model to use to fit data requires careful thought and cogent experimental design.

A stress-strain curve in response to a constant strain rate reveals the stiffness of a material by its slope, and the material strength by the maximum load achieved. Such a plot also can reveal material nonlinearity. In study of biological tissue such as ligament, stress relaxation testing is frequently done in order to determine viscoelastic properties. A single relaxation test reveals viscoelastic behavior, if any exists. The shape of the relaxation function at a particular strain level is a quantitative measure of the viscoelastic response. The relaxation function is given by:

$$E(t) = \sigma(t) / \varepsilon_0 , \tag{1}$$

where ε_0 is the constant strain level to which the ligament is pulled and $\sigma(t)$ is the stress, defined by:

$$\sigma(t) = F(t)/a_0. \tag{2}$$

Here, F(t) is the force and a_0 is the undeformed area. It has been shown [1] that the shape of the curve can be approximated by the power law described as:

$$\frac{\sigma(t)}{\varepsilon_0} = At^{-n}. \tag{3}$$

where A has units of Pa and t has units of seconds. This indicates that E(t) can be approximated by a straight line on a plot of $\log[\sigma(t)/\varepsilon_0]$ vs. log[t]. The slopes of the straight lines of the relaxation data on these plots indicate the power, or n value, of the data, and thus the magnitude of n indicates how rapidly relaxation or recovery occurs in time. Power law damping such as this fits experimental data well, and the nonlinear behavior (single parameter n) is easier to distinguish than the three spectral damping commonly used with QLV.

A single relaxation curve does not adequately describe tissue behavior, however, as it does not reveal whether the material is nonlinear or linear, and gives no insight regarding the proper model to use. Any number of models can be made to fit a single relaxation curve. Model selection then requires the fitting of multiple curves to begin eliminating some options.

A protocol consisting of a stress-strain curve and a single relaxation test can reveal that a tissue is nonlinear and that it is viscoelastic, and can therefore falsify a linear elastic model (Fig. 1). Results of this type are common in the biomechanics literature. However such a protocol cannot falsify a viscoelastic model including QLV and nonlinear superposition. By performing multiple relaxation tests over a wide range of applied strain levels (relative to a state with preload which may approximate that in nature) and plotting the resulting stress relaxation curves, any nonlinear viscoelasticity of the tissue becomes apparent and one can begin to discriminate among possible models. Also, relaxation rate changes at each strain may become apparent with multiple strain levels. This allows comparison of models since a constant relaxation rate, independent of the level of applied strain, is implied in QLV (equation 7), whereas a changing relaxation rate is allowed by the nonlinear models such as the nonlinear superposition and Schapery models. This is demonstrated in a rat ligament study in which stress relaxation was performed at a range of sub-damage strains (0.82%, 1.74%, 2.38%, and 3.74%). The resulting curves (Fig. 2) demonstrated a dependence of rate on strain which was not captured by a QLV model [1]. Similarly, in cornea [2], creep becomes more pronounced as stress increases. The behavior therefore does not follow quasi-linear viscoelasticity (QLV). Also, tendon [3] does not follow QLV because relaxation rate depends on strain level. Multiple relaxation tests at different strain levels can therefore verify or falsify linear or QLV models, but cannot falsify nonlinear models such as nonlinear superposition or the Schapery model. The presence of a varying relaxation rate is not enough to distinguish whether the nonlinear superposition or Schapery model (for example) is the strongest model; further examination is necessary.

A powerful way to further discriminate between models is to apply a more complex loading history. A simple method is to perform a multi-level stress relaxation test involving two different strains (Fig. 3a,b). For example, a relatively large strain can

be imposed for a given length of time, followed by an immediate lowering or increase of the strain. Such step functions allow for easier model calculations, and their implications will be discussed in following sections.

As nonlinear superposition [4], QLV [5, 6, 7], and Schapery [4, 8] models have been used to describe tissue behavior, they will be further described here.

The basic form of the nonlinear superposition is:

$$\sigma(\varepsilon, t) = \int E[t-\tau, \varepsilon(\tau)] \frac{d\varepsilon(\tau)}{d\tau} d\tau, \tag{4}$$

but with the use of the discrete step strain function, the nonlinear superposition prediction for stress response to a relaxation recovery protocol becomes:

$$\sigma(t) = \varepsilon_a E(t, \varepsilon_a) - (\varepsilon_a - \varepsilon_b) E(t - t_1, \varepsilon_b), \tag{5}$$

where t is the time from the start of stress relaxation at the first step strain, t_1 is the time at which recovery begins, ε_a denotes the first strain level and ε_b denotes the second strain level. Because the relaxation modulus in Equation 5 is a function of both time and strain, if a series of relaxation experiments is done at different strain levels, a material which obeys nonlinear superposition can exhibit relaxation curves which differ in magnitude and in shape as a function of strain.

The basic equation for stress in QLV is:

$$\sigma(t) = \int_0^t E_t(t-\tau) \frac{d\sigma}{d\varepsilon} \frac{d\varepsilon(\tau)}{d\tau} d\tau. \tag{6}$$

QLV is a special case of nonlinear superposition in which the kernel is separable into a product, $E(t, \varepsilon) = E(t)g(\varepsilon)$, where $g(\varepsilon)$ represents the nonlinear strain dependence which is independent of time. With the kernel defined as such, the rate of stress relaxation (reflected in the definition of E(t)) is thus independent of strain level, demonstrated in the gray fit lines in Fig. 3a. Since we are using discrete step strain functions, the integral reduces to:

$$\sigma(t) = (\varepsilon_a) E_t(t) g(\varepsilon_a) - (\varepsilon_a - \varepsilon_b) E_t(t - t_1) g(\varepsilon_b) \tag{7}$$

to describe the stress behavior predicted by QLV. The relaxation modulus in Equation 7 is a product of functions of time and strain. Therefore if a series of relaxation experiments is done at different strain levels, a material which obeys QLV must exhibit relaxation curves which may differ in magnitude but have the same shape (time dependence). Also, recovery (the second term in equation 7) must follow the same time dependence as relaxation in a material obeying QLV, because the time dependence is unchanged by multiplication by a strain-dependent number.

The general form of the Schapery nonlinear stress relaxation is given by:

$$\sigma(t) = h_e E_e + h_1 \int_{0^-}^{t} \Delta E(\rho - \rho') \frac{dh_2}{d\tau} d\tau, \tag{8}$$

where h_e, h_1, and h_2 are strain-dependent material properties, ΔE is the transient component of the modulus (defined by $\Delta E \equiv E(t)-E_e$), E_e is the equilibrium or final value of the modulus (defined by $E_e = E(\infty)$), and ρ and ρ' are defined as follows:

$$\rho \equiv \int_0^t dt' / a_\varepsilon[\varepsilon(t')] \quad (a_\varepsilon > 0) \tag{9}$$

$$\rho' \equiv \rho(\tau) = \int_0^\tau dt' / a_\varepsilon[\varepsilon(t')] \tag{10}$$

where a_ε is an additional strain-dependent material property [9]. Physically, ρ can be regarded as an internal clock time which can depend on strain. Again, the use of step functions enables us to simplify the integral equation to

$$\sigma(t) = \left[h_e^{\upsilon} E_e + h_1^{\upsilon} h_2^{\upsilon} \Delta E \left(\frac{t-t_a}{a_\varepsilon^{\upsilon}} - \left| \frac{h^b}{h^a} \right|_{\varepsilon_b} \right) \right] \varepsilon_b - \left[\Delta E \left(\frac{t-t_a}{a_\varepsilon^{\upsilon}} \right) - \Delta E \left(\frac{t}{a_\varepsilon^{a}} + \frac{t-t_a}{a_\varepsilon^{\upsilon}} \right) \right] \varepsilon_a \,, \tag{11}$$

where "a" denotes the first strain level and "b" denotes the second, and t_a denotes the time at which the second step is invoked. The Schapery model's strain-dependent properties h_e, h_1, and h_2 are related to Helmholtz free energy (specifically, 3rd order and higher strain effects), and strain property a_ε is related to strain influences in free energy and entropy production. Further simplification of this equation is possible in tendon and ligament, since it has been determined that, for fibrous composite materials in isothermal testing conditions, $h_1 = a_\varepsilon = 1$ [9]. This leaves the equation for the two-step model as:

$$\sigma(t) = \left[h_e^{b} E_e + h_2^{b} \Delta E(t - t_a) \right] \varepsilon_b - h_2^{a} \left[\Delta E(t - t_a) - \Delta E(t) \right] \varepsilon_a \,. \tag{12}$$

As with nonlinear superposition, a material obeying the Schapery model can exhibit relaxation curves which differ in shape as a function of strain. Moreover the recovery behavior need not follow the shape of the relaxation curve at any strain.

All three of these single-integral equations are able to fit data from a single relaxation test at one strain of tendon or ligament well when the parameters in the model are taken from the data which they are fitting [4]. To determine which of these is the best model for the tissue, a more comprehensive testing protocol and corresponding predictions are necessary. The two-step protocol presented here is a good start towards determining more robust viscoelastic behavior. When the second strain level is lower than the first ($\varepsilon_b < \varepsilon_a$), the researcher gains information about both the relaxation behavior and the recovery behavior of the tissue. The recovery response is highly relevant to the function of tissue in the body because tissues are naturally subject to load–unload cycles. Further modifying the experiment to incorporate more complex loading histories, such as the use of a sinusoidal strain input, can determine if any of these models proves to accurately represent tissue behavior, but does so with additional complexity in

calculations (as we no longer have step functions). It may also indicate that more complex models (i.e. multiple integral models) are required to capture the true behavior.

Previous experiments show that stress relaxation in ligament and other soft tissues occurs at different rates depending on strain level [1, 10]. Similarly, the rate of creep depends on stress level [11]. Creep predictions based on relaxation are poor [1, 12] if QLV is assumed. So to gain a more complete understanding of the viscoelastic properties of a tissue, both stress relaxation and creep tests must be performed, and these must be carried out in strategic fashion.

DYNAMICS of FLUID FLOW in CARDIOVASCULAR SYSTEM

The physiology of the human blood circulation can be divided into two distinct but remarkably harmonized processes; (1) the pumping of blood by the heart, and (2) the transport of blood to all body tissues via the vasculature, or blood vessels. Blood supplies all body tissues with the substances needed for survival, so it is vital that blood delivery is amply for tissue demands.

Dynamics of fluid flow in CV System is concerned with the forces generated by the heart and the resulting motion of blood through the cardiovascular system.
So its required to study the Interrelationships between

- Pressure
- Flow
- Resistance
- Blood flow pattern through vessels (velocity)
- Diameter of vessels
- Control mechanisms that regulate blood pressure

Interrelationships Among Pressure, Flow, and Resistance
Blood flow through a blood vessel is determined by two factors: (1) *pressure difference* of the blood between the two ends of the vessel, also sometimes called "pressure gradient" along the vessel, which is the force that pushes the blood through the vessel, and (2) the impediment to blood flow through the vessel, which is called *vascular resistance.* Figure (1) demonstrates these relationships, showing a blood vessel segment located anywhere in the circulatory system.

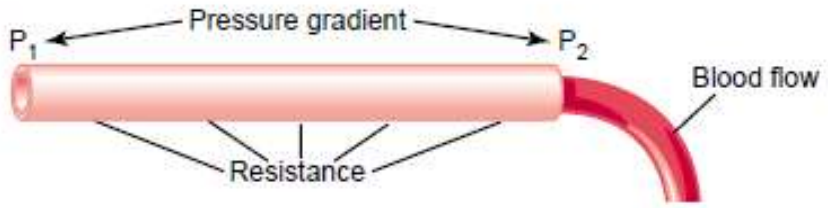


Fig 1. Interrelationships among pressure, resistance, and blood flow.

P1 represents the pressure at the origin of the vessel; at the other end, the pressure is P2. Resistance occurs as a result of friction between the flowing blood and the intravascular endothelium all along the inside of the vessel. The flow through the vessel can be calculated by the following formula, which is called *Ohm's law (Darcy's law* :

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

in which F is blood flow, ΔP is the pressure difference (P1 - P2) between the two ends of the vessel, and R is the resistance. **This formula states, in effect, that the blood flow is directly proportional to the pressure difference but inversely proportional to the resistance.**

Note that it is the *difference* in pressure between the two ends of the vessel, not the absolute pressure in the vessel, that determines rate of flow. For example, if the pressure at both ends of a vessel is 100 mm Hg and yet no difference exists between the two ends, there will be no flow despite the presence of 100 mm Hg pressure.

Blood Flow

Blood flow means simply the quantity of blood that passes a given point in the circulation in a given period. Ordinarily, blood flow is expressed in *milliliters per minute* or *liters per minute,* but it can be expressed in milliliters per second or in any other unit of flow. The overall blood flow in the total circulation of an adult person at rest is about 5000 ml/min. This is called the *cardiac output* because it is the amount of blood pumped into the aorta by the heart each minute. Organs differ in their requirements from moment to moment, and blood vessels constrict or dilate to regulate, blood flow to various areas in response to the tissues immediate needs. Consequently, blood flow can increase to some regions and decrease to other areas at the same time.

Resistance to Blood Flow

Resistance is the impediment to blood flow in a vessel, but it cannot be measured by any direct means. Instead, resistance must be calculated from measurements of blood flow and pressure difference between two points in the vessel. If the pressure difference between two points is 1 mm Hg and the flow is 1 ml/sec, the resistance is said to be 1 *peripheral resistance unit*, usually abbreviated *PRU*.

Total Peripheral Vascular Resistance and Total Pulmonary Vascular Resistance.

The rate of blood flow through the entire circulatory system is equal to the rate of blood pumping by the heart—that is, it is equal to the cardiac output. In the adult human being, this is approximately 100 ml/sec. The pressure difference from the systemic arteries to the systemic veins is about 100 mm Hg. Therefore, the resistance of the entire systemic circulation, called the *total peripheral resistance,* is about 100/100, or 1 PRU.

In conditions in which all the blood vessels throughout the body become strongly constricted, the total peripheral resistance occasionally rises to as high as 4 PRU. Conversely, when the vessels become greatly dilated, the resistance can fall to as little as 0.2 PRU. In the pulmonary system, the mean pulmonary arterial pressure averages 16 mm Hg and the mean left atrial pressure averages 2 mm Hg, giving a net pressure difference of 14 mm.Therefore, when the cardiac output is normal at about 100 ml/sec, the *total pulmonary vascular resistance* calculates to be about 0.14 PRU (about one seventh that in the systemic circulation).

"Conductance" of Blood in a Vessel and Its Relation to Resistance:

Conductance is a measure of the blood flow through a vessel for a given pressure difference. This is generally expressed in terms of milliliters per second per millimeter of mercury pressure, but it can also be expressed in terms of liters per second per millimeter of mercury or in any other units of blood flow and pressure. It is evident that conductance is the exact reciprocal of resistance in accord with the following equation:

$$\text{Conductance} = \frac{1}{\text{Resistance}}$$

Very Slight Changes in Diameter of a Vessel Can Change Its Conductance Tremendously!

Slight changes in the diameter of a vessel cause tremendous changes in the vessel's ability to conduct blood when the blood flow is streamlined. This is demonstrated by the experiment illustrated in Figure

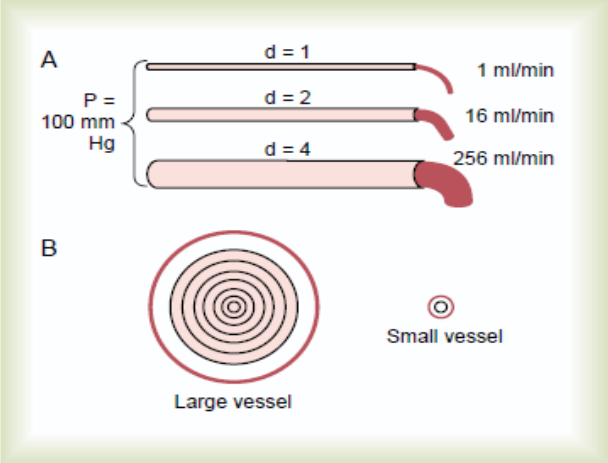


Figure 14–9

A, Demonstration of the effect of vessel diameter on blood flow. *B*, Concentric rings of blood flowing at different velocities; the farther away from the vessel wall, the faster the flow.

which shows three vessels with relative diameters of 1, 2, and 4 but with the same pressure difference of 100 mm Hg between the two ends of the vessels. Although the diameters of these vessels increase only fourfold, the respective flows are 1, 16, and 256 ml/mm, which is a 256-fold increase in flow. Thus, the conductance of the vessel increases in proportion to the *fourth power of the diameter*, in accordance with the following formula:

$$\text{Conductance} \propto \text{Diameter}^4$$

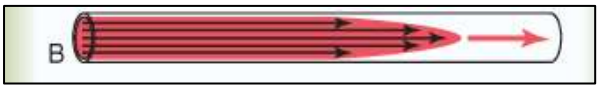
Poiseuille's Law. The cause of this great increase in conductance when the diameter increases can be explained by referring to Figure 14–9*B*, which shows cross sections of a large and a small vessel. The concentric rings inside the vessels indicate that the velocity of flow in each ring is different from that in the adjacent rings because of *laminar* flow. That is, the blood in the ring touching the wall of the vessel is barely flowing because of its adherence to the vascular endothelium. The next ring of blood toward the center of the vessel slips past the first ring and, therefore, flows more rapidly.The third, fourth, fifth, and sixth rings likewise flow at progressively increasing velocities. Thus, the blood that is near the wall of the vessel flows extremely slowly, whereas that in the middle of the vessel flows extremely rapidly. In the small vessel, essentially all the blood is near the wall, so that the extremely rapidly flowing central stream of blood simply does not exist. By integrating the velocities of all the concentric rings of flowing blood and multiplying them by the areas of the rings, one can derive the following formula, known as Poiseuille's law:

$$F = \frac{\pi \Delta P r^4}{8 \eta l}$$

in which F is the rate of blood flow, ΔP is the pressure difference between the ends of the vessel, r is the radius of the vessel, l is length of the vessel, and h is viscosity of the blood.

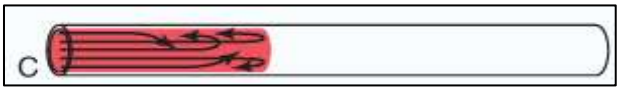
Velocity- Modes of vessel flow:

Laminar Flow (decreased velocity)



- When blood flows through a long smooth vessel it flows in straight lines, with each layer of blood remaining the same distance from the walls of the vessel throughout its length
- When laminar flow occurs the different layers flow at different rates creating a parabolic profile
- The parabolic profile arises because the fluid molecules touching the walls barely move because of adherence to the vessel wall.

Turbulent Flow (increased velocity)



- When the rate of blood flow becomes too great, when it passes by an obstruction in a vessel, when it makes a sharp turn, or when it passes over a rough surface, the flow may then become turbulent
- Turbulent flow means that the blood flows crosswise in the vessel as well as along the vessel, usually forming whorls in the blood called eddy currents. When eddy currents are present, the blood flows with much greater resistance than when the flow is streamline because eddies add tremendously to the overall friction of flow in the vessel.

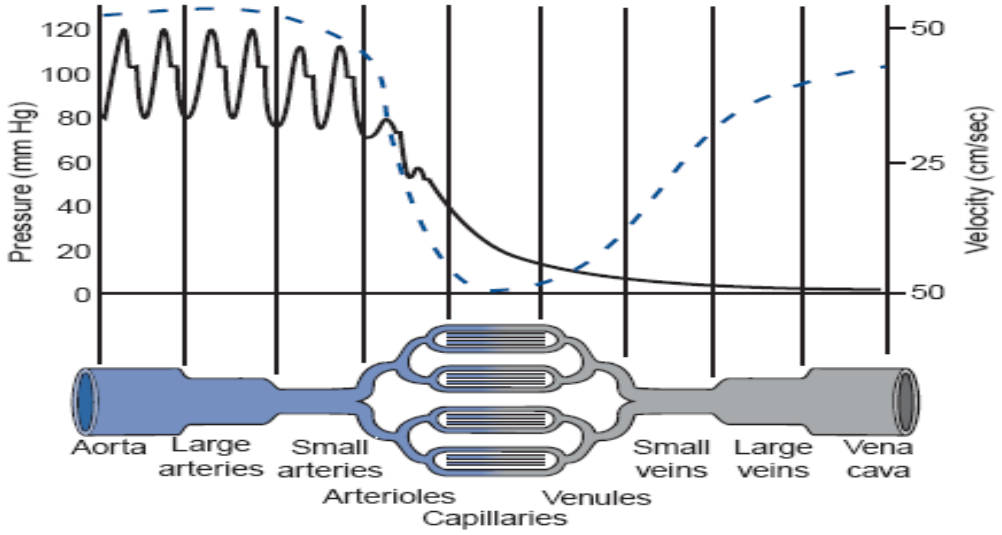


FIGURE 11.9 Pressure and flow velocity profile in the systemic circulation. The arterial portion of the circulation is characterized by high, pulsatile pressure and high flow velocity. This profile changes to one of low pressure and velocity without pulsatile character in the veins. The largest drop in mean arterial pressure occurs across the arteriolar segment of the circulation, indicating that this is the sight of highest vascular resistance in the cardiovascular system.

Vascular Compliance (or Vascular Capacitance)

In hemodynamic studies, it usually is much more important to know the total quantity of blood that can be stored in a given portion of the circulation for each millimeter of mercury pressure rise than to know the distensibilities of the individual vessels. This value is called the compliance or capacitance of the respective vascular bed; that is, Compliance and distensibility are quite different. A highly distensible vessel that has a slight volume may have far less compliance than a much less distensible vessel that has a large volume because compliance is equal to distensibility times volume. Compliance is total quantity of blood that can be stored in a given portion of the circulation. So, can also express as Vascular capacitance. Veins are more compliant because they have more blood during normal resting state.

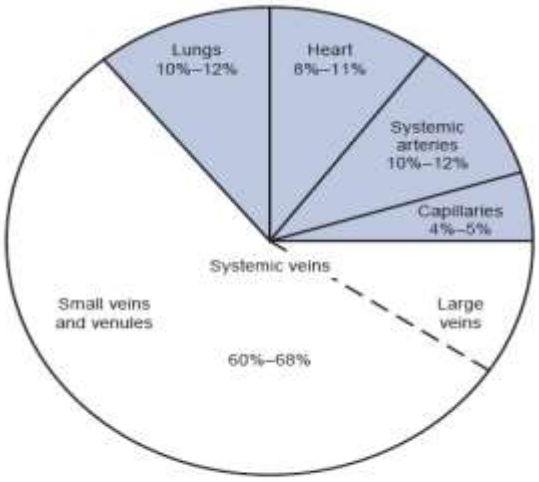
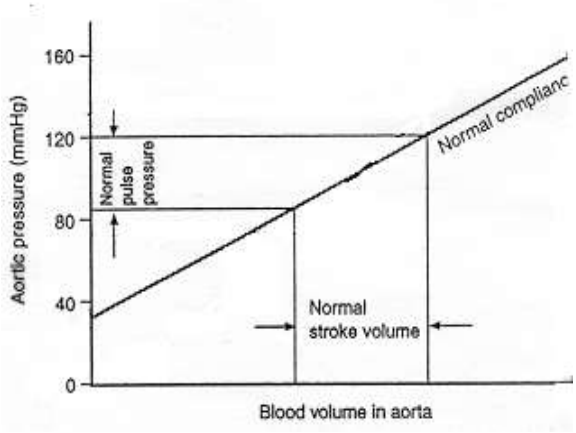
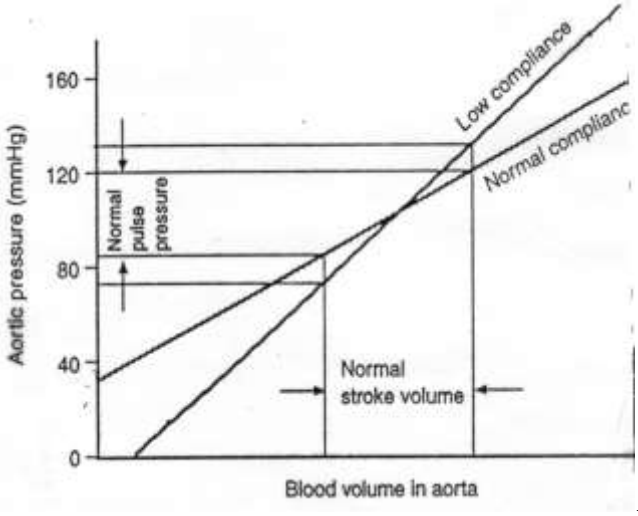


FIGURE 11.8 Blood volumes of various elements of the circulation in a person at rest. The majority of the blood volume is in systemic veins.

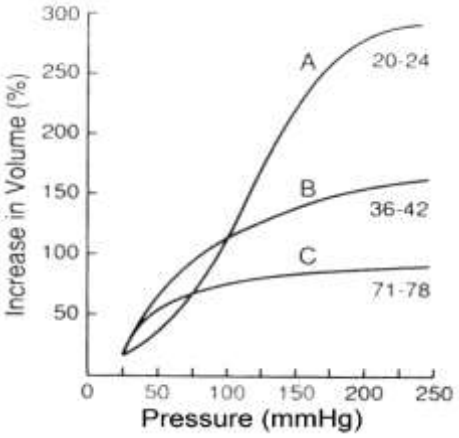
Aortic pulse pressure depends on arterial compliance:



With each ejection the aortic volume increases by one stroke volume

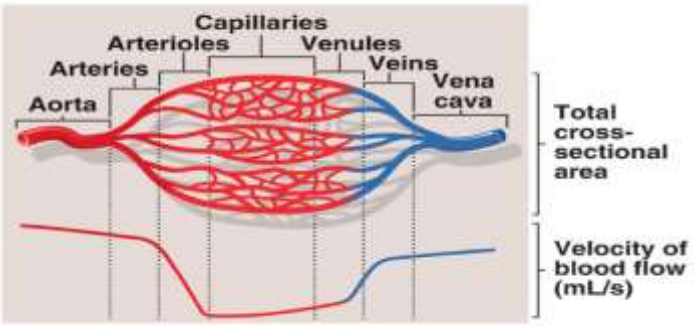


If aortic compliance were to decrease, pulse pressure will increase



Aging reduces aortic compliance
Pulse pressure naturally increases with age
Systolic hypertension >140

Cross Sectional Area :
As diameter of vessels decreases, the total cross-sectional area increases andvelocity of blood flow decreases.

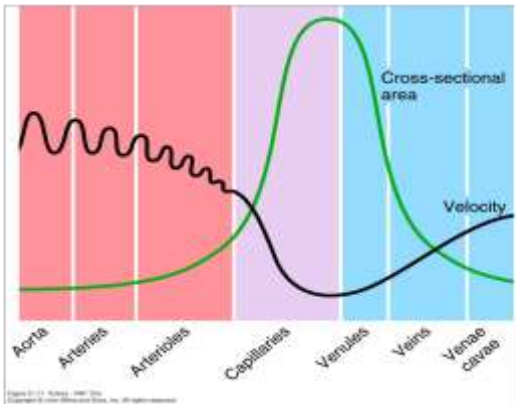


Controlling **blood vessel radius** (one-half the diameter) is the principal method of blood flow control. This is accomplished by contracting or relaxing the smooth muscle within the blood vessel walls. To see why radius has such pronounced effect on blood flow, we need to explore the physical relationship between flood and the vessel wall. Blood in direct contact with the vessel wall flows relatively slowly because of the friction, or drag, between the blood and the lining of the vessel. In contrast, fluid in the center of the vessel flows more freely because it is not "rubbing" against the vessel wall. When we contrast large- and small-radius vessels, we see that proportionately more blood is in contact with the wall of small vessels, hence blood flow is notably impeded in small-radius vessels.
Although **vessel length** does not ordinarily change in a healthy person, any increase in vessel length causes a corresponding flow decrease. This effect is principally caused by friction between blood and the vessel wall. Consequently, given two blood vessels of the same diameter, the longer vessel will have more resistance, and thus a reduced blood flow.
If all the *systemic vessels* of each type were put side by side, their approximate total cross-sectional areas for the average human being would be as follows:

Vessel	Cross-Sectional Area (cm^2)
Aorta	2.5
Small arteries	20
Arterioles	40
Capillaries	2500
Venules	250
Small veins	80
Venae cavae	8

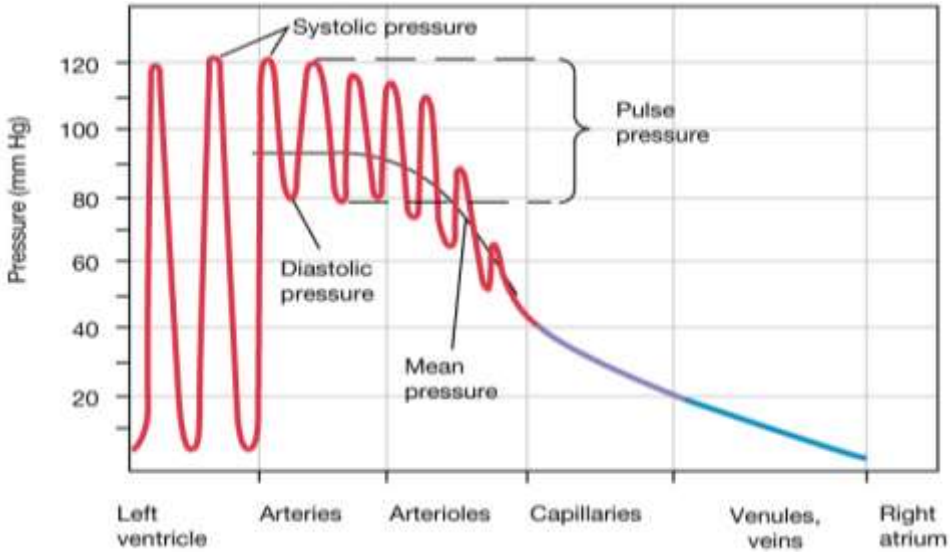
Because the same volume of blood must flow through each segment of the circulation each minute, the velocity of blood flow is inversely proportional to vascular cross-sectional area. Thus, under resting conditions, the velocity averages about 33 cm/sec in the aorta but only 1/1000 as rapidly in the capillaries, about 0.3 mm/sec.

Relationship between Velocity of Blood Flow and Total Cross-sectioned area in Different Types of Blood Vessels



Blood Pressure:
Force exerted by the blood against any unit area of the vessel wall

-Contraction of ventricles generates blood pressure
-Systolic BP – highest pressure attained in arteries during systole
-Diastolic BP – lowest arterial pressure during diastole
-Pressure falls progressively with distance from left ventricle
-Blood pressure also depends on total volume of blood



Pressure throughout the systemic circulation

Pulse pressure:
Mean arterial pressure:

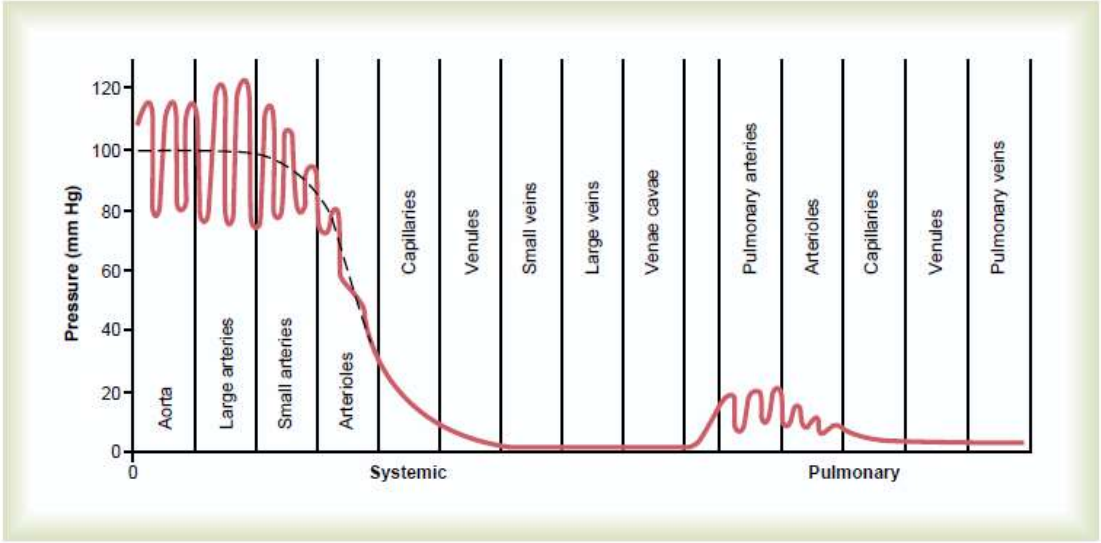


Figure 14–2

Normal blood pressures in the different portions of the circulatory system when a person is lying in the horizontal position.

Effect of vibration – shear stresses in extra-corporeal circuits

Extracorporeal Circuit

Blood when taken from a patient's circulation and sent an external circuit, to have a process applied to it before it is returned back to body. All of the apparatus carrying the blood outside the body is termed the **extracorporeal circuit.**

Examples: Heart Lung machine (Cardiopulmonary bypass), hemodialyzer

Therapeutic functions of the extracorporeal circuit

The extracorporeal circuit is designed to remove blood from the patient's circulation, deliver the blood to some form of purification device, and the return the purified blood to the patient. These tasks must be performed without damaging blood components, without exposing the patient to potentially harmful contaminants from the extracorporeal circuit or the environment.

Components of the Extracorporeal Circuit

Three basic blood containing elements of the circuit are a means of accessing the circulation, blood tubing set and a blood purification device.

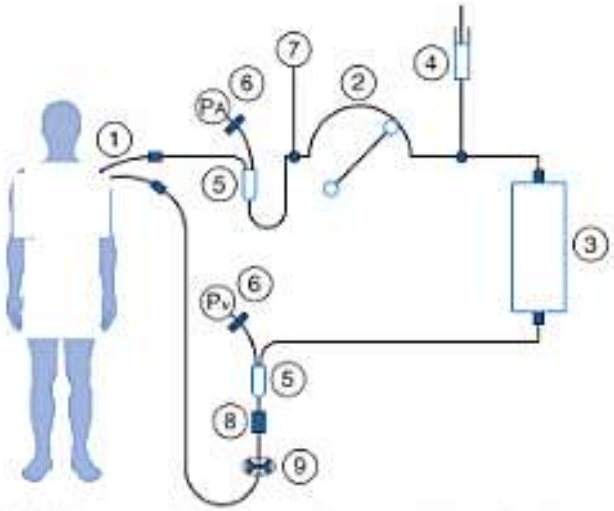


FIGURE 211-1. Typical extracorporeal circuit for hemodialysis. Convective therapies, such as hemodiafiltration and hemofiltration, use a similar circuit with the addition of lines for the infusion of replacement solution before or after the hemodialyzer or hemofilter. The major components of the extracorporeal circuit are as follows: 1, a blood access device (shown as a central venous catheter); 2, a blood pump; 3, a blood purification device (hemodialyzer, hemofilter, or sorbent cartridge); 4, an anticoagulant infusion pump; 5, air-capture chambers; 6, pressure-monitoring systems (shown as a pressure transducer isolated from the blood path by a pressure-transmitting sterile barrier); 7, a side line for priming the extracorporeal circuit with saline; 8, an ultrasonic air and foam detector; and 9, a line clamp.

1. Blood Access

Patients receiving renal replacement therapy (RRT) in an acute setting do not have access to blood. In such cases catheter is placed in to the blood vessel.

2. Blood Tubing

Blood is conveyed to and from blood purification device by disposable blood tubing set. Tubing has two segments: an arterial segment that connects the outflow from blood access to the inlet port of blood purification device, and a venous segment that connects the outlet port of the blood purification device to the return blood access. Tubing is generally made of polyvinylchloride and sterilized with ethylene oxide or gamma irradiation.

3. Blood pumps

The pressure at the inlet to the catheter or needle used to withdraw blood from the patient into extracorporeal circuit is usually insufficient to provide the desired flow rate of blood through the extracorporeal circuit. Blood pump is generally used to provide a controlled flow of blood to the blood purification device. These pumps are called as peristaltic pumps, also known as roller pumps and diaphragm pumps.

4. Blood purification component

Most blood purification devices used in critical care nephrology are hollow-fiber membrane devices that allow exchange of solutes using diffusion and /or convection and removal of water by ultra filtration.

5. Ancillary components

Extracorporeal circuits include various other components, depending, in part, on the nature of the therapy being performed. For example anticoagulation unit etc

The extracorporeal circuit is a thrombogenic device. In order to initiate cardiopulmonary bypass, systemic heparinization is required in order to establish a safe level of anticoagulation. The current dosing regimen is 200- 300 units of heparin per kilogram of patient weight. It's administration is usually targeted to reach and maintain an activated clotting time of 350-500 seconds (normal range is defined as ≤130 seconds). It is intended to inhibit the formation of clots within the ECC and oxygenator, as well as prevent thrombotic events from occurring in the patient. This represents a significantly high level of anticoagulation that must then be appropriately reversed upon termination of bypass.

Turbulent flow, shear stress, changes in viscosity, blood-oxygen interface, and blood-artificial surface interface effect proteins and the formed elements in the blood. Trauma to the cellular elements of the blood during cardiopulmonary bypass results in destruction or alteration

to red cells and platelets. Clot-promoting and heparin-neutralizing factors released from these damaged cells result in a state of hypercoagulability. Intravascular clotting can subsequently occur, resulting in an abnormal consumption of clotting factors. These developments in turn stimulate production of antithrombin activity and the release of plasminogen activator which results in fibrinolysis. The first response of blood to any extracorporeal circuit is given below.

2. Biological Response to CPB

Blood circulating in the human body is in constant contact with a continuous luminal endothelial cell (EC) layer known as the endothelium which coats the interior of all blood vessels in the body. This EC layer is the barrier between the tissue cells of the blood vessel wall and the blood itself. The endothelium is capable of producing, secreting, and binding proteins or soluble factors to achieve hemostasis [5], and it plays a major role in regulating membrane permeability, lipid transport, vasomotor tone, coagulation, fibrinolysis, and inflammation [6]. This regulation is achieved by the expression of endothelial-derived surface proteins or secretion of biologically active soluble factors. During CPB, blood leaves the circulatory system and enters the extracorporeal circuit where it comes into contact with synthetic surfaces. This contact leads to activation of the blood which triggers coagulation effects and inflammatory response.

2.1 Hemostasis

In the human body the balance between thrombosis and bleeding is kept by the endothelium. The endothelial cells produce up to nine procoagulants and as many anticoagulants depending on the circumstances [7]. The process of stopping bleeding with clot formation and then restoring blood flow is known as hemostasis and it is a very complex process.

Upon injury to the blood vessel, the endothelium is disturbed and subendothelium proteins are released into the blood. These proteins, most notably von Willebrand factor, activate blood platelets which then aggregate at the site of the injury forming a plug to stop the bleeding. Activated platelets also release the contents of their stored granules which triggers a protein cascade to induce more coagulation and activate more platelets [8]. This process occurs almost immediately after disturbance of the endothelium and is known as primary hemostasis. The incisions made during cardiac surgery with CPB disrupt blood vessels and so initiate this chain of events.

The interaction of blood components with artificial surface and the resulting reactions are mentioned below:

3. Blood-material interaction

3.1 Protein Adhesion

The first event that takes place when blood comes into contact with the synthetic surfaces of the extracorporeal circuit is rapid protein adsorption onto these surfaces. This adhesion makes these proteins what blood components interact with when flowing through the circuit and so the changes in blood composition and activation of blood can be attributed to these adhered proteins [13]. For this reason protein adsorption to biomaterial surfaces is of much interest for mitigating the deleterious effects of CPB.

The two proteins of major importance regarding adhesion to the biomaterial surface are fibrinogen and albumin. Fibrinogen is the precursor to fibrin, which plays a critical role in thrombosis. Fibrinogen also interacts with platelets. In 1969 Zucker et al. showed that platelets attach to surface adsorbed fibrinogen [14]. This reaction partially explains the thrombocytopenia, or low platelet presence in blood, observed during and after clinical cardiac surgery. Activation of platelets in the circuit can also disrupt the hemostatic balance in the circulatory system and if platelets bind to fibrinogen in the circuit they are not available in the blood vessels for clotting. Albumin is a very water soluble protein whose adherence to the biomaterial surface is important because it inhibits adherence of leukocytes and platelets [15]. With the role that platelets play in hemostasis and leukocytes in the inflammatory response it is ideal in terms of biocompatibility if these cells have minimal interaction with the biomaterial. The effects of albumin on thrombus formation and inflammatory response have lead to its utilization in the preparation of biomaterials aimed at improving biocompatibility [13].

Biomaterial characteristics that influence protein adhesion are surface roughness, surface area and surface chemistry. The impact that these factors have on protein adhesion and biocompatibility of the material will be investigated in a later section.

3.2 Hemolysis and Platelet Reaction

Hemolysis is the breaking open of erythrocytes, or red blood cells, and the release of hemoglobin. Erythrocytes are the blood cells responsible for carrying oxygen and delivering it to the body. Hemolysis is of major concern in CPB because if erythrocytes are damaged then the body does not receive adequate oxygen during cardiac surgery leading to hypoxia and major complications. Hemolysis during CPB is caused by erythrocytes impacting the biomaterial surface or by elevated shear stress in the shear flow caused by surface roughness [16].

Platelets adhere to and aggregate on the biomaterial surface via proteins adhered to the surface. Platelets that adhere to the surface also release constituents that can activate other platelets and increase aggregation [17]. Platelet rupture can also occur in the extracorporeal circuit due to the same factors as hemolysis: surface impact and shear stress. Platelet rupture is of major consequence to the hemostatic balance because platelet activation factors are released upon rupture and ruptured platelets cannot aggregate to form clots in blood vessels. Therefore, platelet reaction to the extracorporeal circuit can cause thrombocytopenia and aid in SIRS. The surface roughness is the critical biomaterial characteristic for hemolysis and platelet rupture during CPB and will be discussed below.

4. Biomaterial Considerations

Since protein adhesion, hemolysis, and platelet rupture have been identified as the main factors in alteration of blood components during CPB, it is logical to now investigate what aspects of the biomaterial affect these activities. Surface roughness has been directly correlated to an increase in shear stress within the blood flow. With shear stress being the primary cause of hemolysis and platelet rupture this surface characteristic is of critical importance to the biocompatibility of extracorporeal circuit surfaces. Surface area has a direct relationship with protein adhesion because increased surface area allows more space for proteins to interact with and adhere to as well as more opportunity for proteins to be in the proper orientation for surface adhesion. The chemical composition also has a major influence on protein adhesion as this dictates protein structure and presentation of bonding sites.

4.1 Surface Roughness and Shear Stress

Hemolysis occurring within the extracorporeal circuit during CPB has two main causes: erythrocytes bursting upon impact with the synthetic surfaces of the CPB tubes and pump and shear stresses large enough to cause rupture [18]. Studies conducted by Maruyama et al. have shown that the primary cause of hemolysis is rupture due to shear stress and identified the shear stress level required for hemolysis to be between 719 Pa and 903 Pa [16,18].

Surface roughness of the biomaterial surface in CPB circuitry is characterized by sharp peaks and valleys. These peaks increase the turbulence of the flow around them which results in elevated shear stress levels [16]. With the ultimate goal of producing more biocompatible materials for CPB circuitry Maryuma et al. conducted experiments to determine the threshold of surface roughness, R_a, which produces shear stress levels large enough to cause significant hemolysis. This level is between $R_a = 0.6$ μm and $R_a = 0.8$ μm. Measuring the level of hemoglobin in the blood allows for quantification of hemolytic activity. Hemoglobin concentrations indicating significant hemolytic activity were found between 0.6 μm and 0.8 μm

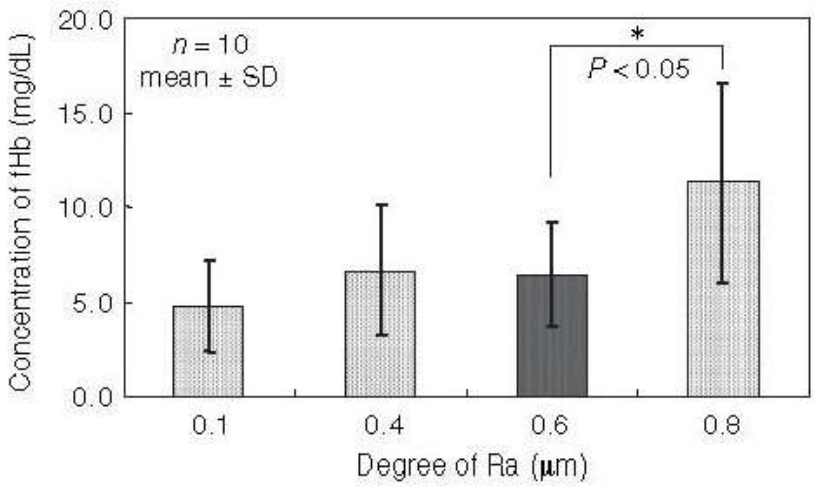


Figure 7. Hemolysis due to surface roughness ranging from R_a 0.1μm (without roughened surface) to 0.8 μm. fHb = free hemoglobin released upon rupture of red blood cells [16].

and with surface roughness values less than these producing normal amounts of hemoglobin in the blood (Fig. 7). The level of hemoglobin concentration is also proportional to the square of the shear rate indicating that hemolysis increases with shear stress (Fig. 8).

With the knowledge of this threshold range for the point at which surface roughness causes shear stress sufficient for hemolysis biomaterial producers can attempt to manufacture extracorporeal circuit tubing and other CPB apparatuses with R_a levels below it. This will reduce hemolysis and platelet rupture during cardiac surgery with CPB leading to a reduction in the deleterious effects of the procedure.

4.2 Surface Area

The surface area of the tubing in extracorporeal circuitry has a large influence on protein adsorption to the surface of the biomaterial. The increased surface area allows more space for the adherence of blood proteins like fibrinogen and albumin. As previously discussed, adherence of these proteins can disrupt or promote the hemostatic balance upheld by the blood vessel endothelium. For this reason, many studies have been conducted to measure the effect that varying material surface area has on inflammatory activity and found a direct link between reduction of platelet activation and leukocyte activity and reduced surface area. This biomaterial characteristic represents a relatively simple way of improving the biocompatibility of CPB circuitry.

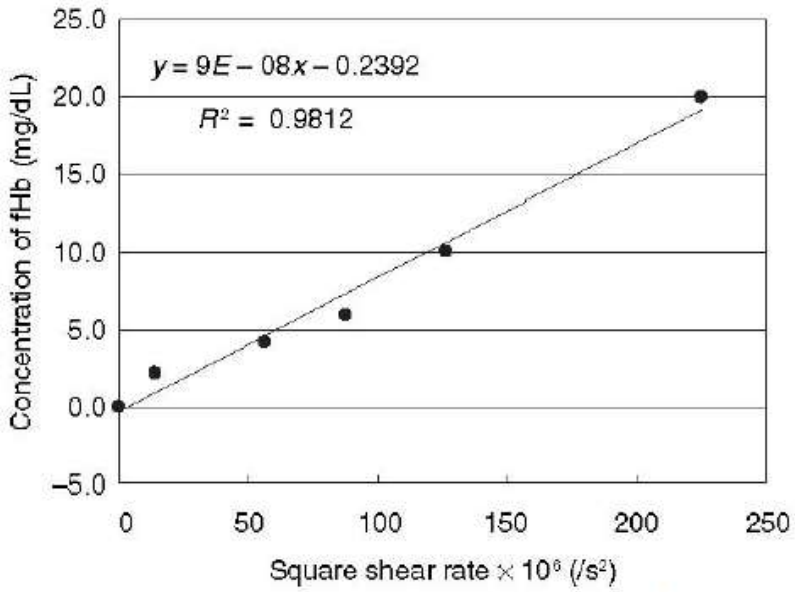


Figure 8. Relationship between square shear rate and hemolysis level. The hemolysis level increased approximately in proportion to the square shear rate [16].

4.3 Surface Chemistry

The surface chemistry of the biomaterial surface in cardiopulmonary bypass procedures can have a major influence on the proteins that adhere to the surface. Many studies have been devoted to developing chemical coatings for CPB circuitry that will provoke adherence of specific proteins intended to prevent blood activation or at least reduce it. There has been interest in low molecular weight heparin, platelet aggregation inhibitors, platelet-preserving agents, and inhibitors for complement, kallikrein and leukocyte sequestration [19]. The most popular method of surface chemistry alteration is heparin-coated circuits [20]. Heparin is a highly-sulfated glycosaminoglycan and it is widely used as an injectable anticoagulant. Coating CPB circuitry with this chemical has been shown to significantly reduce complement and leukocyte activation in patients undergoing cardiac surgery with full systemic anticoagulation [21]. Essentially, the presence of heparin on the biomaterial surface presents the activation of certain complement cofactors and also inhibits the signaling process for leukocyte activation.

Another surface chemistry modification procedure is performed with surface-modifying additive (SMA). The mechanism of antithrombogenicity of the SMA is mainly based on the effect of limiting platelet interaction with the surface and subsequently reducing platelet activation. This is in contrast to the heparin-coated material which still allows platelet binding. Complement activation assessed by the terminal complement complex is not influenced by SMA [22].

Other surface chemistry modification methods involve pre-coating of the material with albumin or an albumin attractant. This is because albumin does not allow attachment of leukocytes or platelets and inhibits their adherence to the material surface [23]. This method has been shown to reduce blood activation in some cases but results are variable due to the many other factors involved [24].

Modifying the surface chemistry of the extracorporeal circuit has profound effects on blood activation. Coating the material with antithrombogenics can reduce thrombosis in some cases and the inflammatory response in others but usually not both at once. Developing methods for selective protein adherence to the material surface or pre-coating with proteins that inhibit blood activation is another approach that has had some success [15]. It would seem that a greater understanding of the processes involved with blood activation, thrombosis, and the inflammatory response is needed before surface chemistry modification techniques can completely alleviate all of the deleterious effects of CPB.

UNIT III ORTHOPAEDIC MECHANICS

Mechanical properties of cartilage, diffusion properties of articular cartilage, mechanical properties of bone, kinetics and kinematics of joints, Lubrication of joints.

Mechanical properties of bone

Introduction

Although an organic material, bone can often be considered in the same way as man-made engineering materials. However, due to the nature of its synthesis it is likely to show more variation in measured properties than typical engineering materials. Factors include:

- Age
- Gender
- Location in the body
- Temperature
- Mineral content
- Amount of water present
- Disease, e.g. osteoporosis

These variables can to an extent be dependent on each other. For example, the mineral content will vary according to the bone's location in the body, and with the age of the patient.

As humans age, their bones typically become less dense and the strength of these bones decreases, meaning they are more susceptible to fracture. Osteoporosis is a disease involving a marked decrease in bone mass, and it is most often found in post-menopausal women.

These variables mean that there is a range of measured properties for bone, and so values given in tables will always be an average, with quite a considerable spread possible in the data.

In addition, the anisotropic structure of bone means that its mechanical properties must be considered in two orthogonal directions:

- Longitudinal, i.e. parallel to osteon alignment. This is the usual direction of loading
- Transverse, i.e. at right-angles to the long axis of the bone

Young's Modulus

Young's modulus, also known as the **tensile modulus** or **elastic modulus**, is a measure of the stiffness of an elastic material and is a quantity used to characterize materials. It is defined as the ratio of the stress along an axis over the strain along that axis in the range of stress in which Hooke's law holds

Bone can be considered to consist primarily of collagen fibres and an inorganic matrix, and so in a simple level it can be analysed as a fibre composite.

Composites are materials that are composed of two or more different components. They are commonly used in engineering and industry where the combination of the two materials creates a composite with properties that are superior to those of the individual components.

The Young's Modulus of aligned fibre composites can be calculated using the Rule of Mixtures and the Inverse Rule of Mixtures for loading parallel and perpendicular to the fibres respectively.

RULE OF MIXTURES

$E_{ax} = f E_f + (1 - f) E_m$

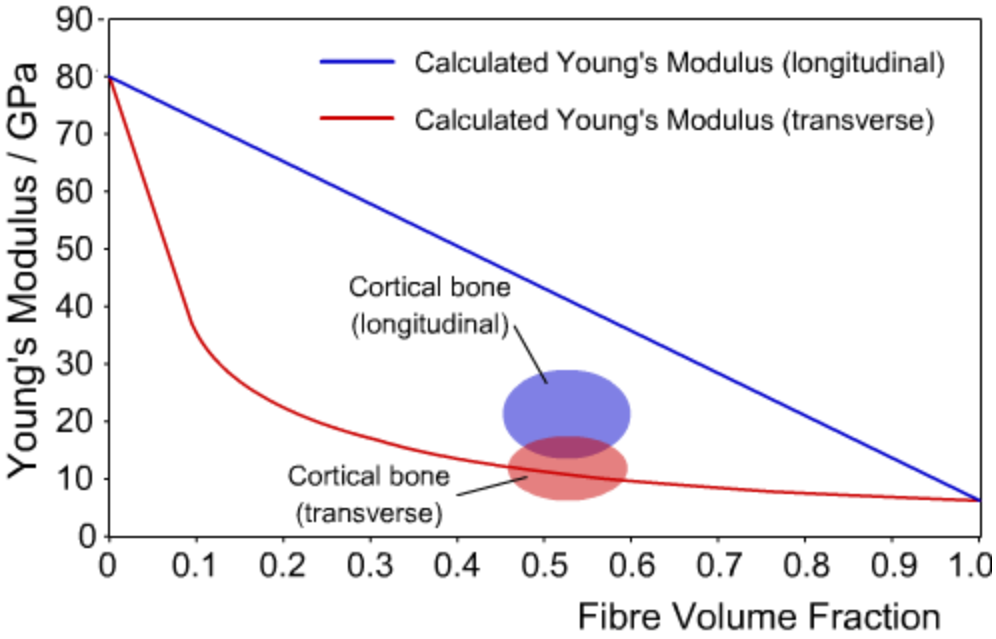
INVERSE RULE OF MIXTURES

$Eax=[fEf+(1-f)Em]-1$
Where

E_f = Young's Modulus of fibres
E_m = Young's modulus of matrix
E_{ax}, E_{trans} = Young's Modulus of composite in axial and transverse directions
f= volume fraction of fibres

These formulae predict that the composite will be stiffer in the axial direction than the transverse, so cortical bone will be stiffer in the direction parallel to the osteons (i.e. parallel to the long axis of the bone).

The chart below shows calculated values for the Young's Modulus of bone in both the longitudinal and transverse directions, for a range of fibre volume fractions, as well as the actual values.



Calculated and experimental values of Young's Modulus for cortical bone

We can see that for the transverse direction, the composite model closely agrees with experimental values. However, in the longitudinal direction the difference is large, indicating the model does not give an accurate picture of the behavior of bone.

This difference occurs because the composite model of the microstructure of bone is highly simplified, since the collagen fibres are not aligned parallel to the axis of the osteons, and the bone mineral exists as discrete crystals, rather than forming a continuous matrix.

A better approximation would be to model bone as a two level composite. One level is provided by hydroxyapatite-reinforced collagen in a single osteon, and the second level is obtained by the approximately hexagonal packing of osteons in a matrix of interstitial bone.

The actual values for the Young's modulus of bone, compared to collagen and hydroxyapatite, are shown in the table below. The measured value of Young's Modulus also depends on temperature, decreasing with an increase in temperature, and the strain rate, increasing in value with an increase in strain rate.

Material	**Young's Modulus, E (GPa)**
Collagen (dry)	6
Bone mineral (Hydroxyapatite)	80
Cortical bone, longitudinal	11-21

Cortical bone, transverse	5-13

Tensile and Compressive Strength

Bones such as the femur are subjected to bending moments during normal loading. These create both tensile and compressive stresses in different regions of the bone.
There is a large variation in measured values of both the tensile and compressive strength of bone. Different bones in the body need to support different forces, so there is a large variation in strength between them. Additionally, age is an important factor, with strength often decreasing as a person gets older.

	Longitudinal direction	**Transverse direction**
Tensile strength (MPa)	60-70	~50
Compressive strength (MPa)	70-280	~50

Elasticity

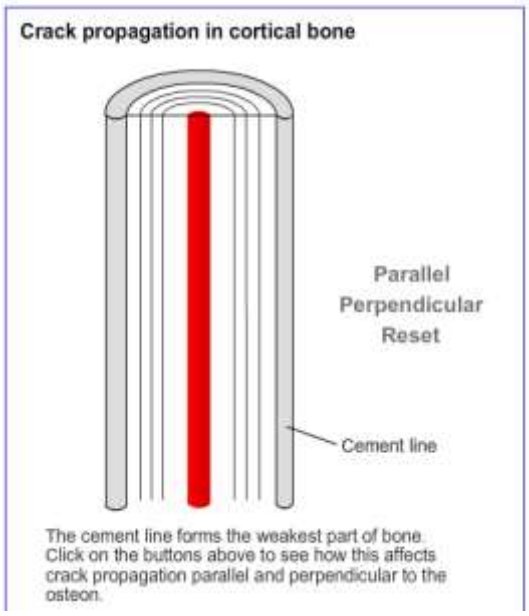
Bone mineral is a ceramic material and exhibits normal Hookean elastic behaviour, i.e. a linear stress-strain relationship. In contrast, collagen is a polymer that exhibits a J-shaped stress-strain curve.
Typical stress-strain curves for compact bone, tested in tension or compression in the wet condition, are approximately a straight line. Bone generally has a maximum total elongation of only 0.5 - 3%, and therefore is classified as a brittle rather than a ductile solid.

- Adult femoral compact bone,
- Ultimate bending strength= 160MPa
- Ultimate shear strength= 54.1±0.6MPa
- Elasticity modulus= 3.2GPa
- The strength & elasticity modulus of spongy bone are much smaller than compact bone

Fracture Toughness

In contrast to the findings for tensile and compressive strength and modulus, the values of toughness in the transverse direction are generally higher than those in the longitudinal direction. This is due to the presence of the cement lines in the microstructure. These are narrow regions around the outermost lamellae in the osteons, and they form the weakest constituent of bone. Crack propagation parallel to the osteons can occur much more easily

through these regions and this significantly decreases the fracture toughness of cortical bone in the longitudinal direction. If a crack is propagating perpendicular to an osteon it will change direction when it reaches a cement line, thus blunting the crack. This is illustrated in the animation below.



As a result, although bone is classified as a brittle material (with the major component being mineral), its toughness is excellent. Bone's fracture energy, G_c, is approximately 1.5 kJ m^{-2}, which is comparable to steel at low temperatures and wood when measured parallel to the grain. This is much tougher than man-made ceramics due to the presence of the collagen fibres in bone. Since the stress-strain curves for loading and unloading are different the elasticity is therefore time-dependent, a common feature of fibrous proteins.

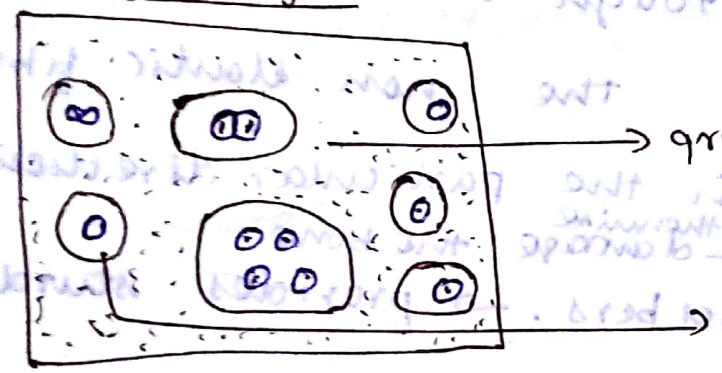
- Bone is a biomaterial with a complex hierarchical structure, which gives it some impressive material properties.
- It is primarily composed of a bioceramic (similar to hydroxyapatite) and collagen, a fibrous protein.
- At a microscopic level, it can be seen that the collagen-bone mineral composite forms concentric lamellar structures known as osteons, which are the main structural element of bone. The osteons are densely packed together in cortical bone and their long axes tend to run parallel to the long axis of the bone.
- In common with many biomaterials, bone is anisotropic: its mechanical properties differ depending on the orientation of the sample being tested.
- Hip replacements are a very common surgical procedure, particularly among older people, as bones can become more brittle with age.

Orthopedic mechanism:

Cartilage: → Bearing and linking Articular surface with bone, tough, semi transparent, elastic and flexible → glyco-protein material - chondrin → No nerves / blood vessels.

Types: 1) Hyaline cartilage.
2) Fibro "
3) Elastic "

Hyaline cartilage:



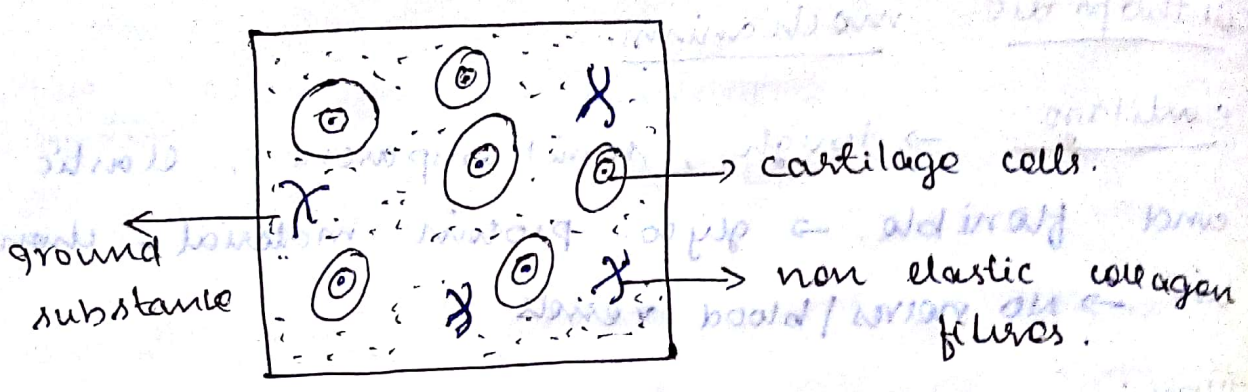
Eg:
→ Trachea → Larynx → tip of the nose

It is semi transparent and bluish white in colour → very flexible and elastic → consist of living cells called chondrocytes.

Functions: → Reduces friction at joints - provides sliding → movement → support → growth.
↓ ↳ (trachea and bronchi to open)
Both the tightly fixed it will allow.
movement

Growth: → in new region it is responsible for longitudinal growth.

Fibro cartilage:



Eg: → discs b/w vertebrae. → edges of the articular cavities.

→ it is too extremely tough tissue.

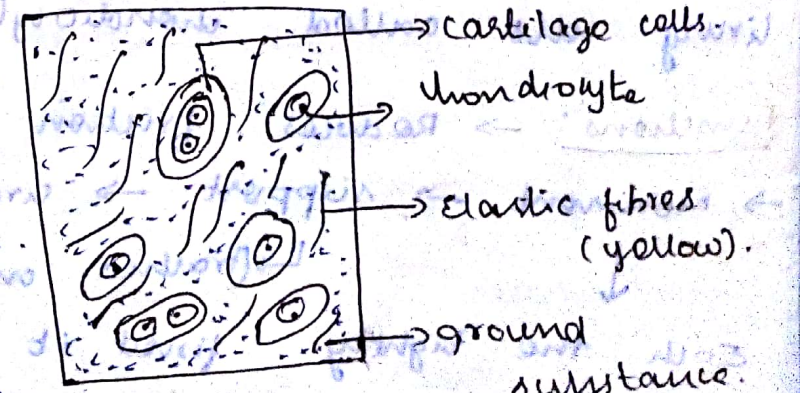
→ If stress is applied the non elastic fibre will allign in the particular direction otherwise damage the bone

Function: → shock absorbers. → provides sturdiness without impedance movement

→ deepens sockets. → prevent dislocation.

Elastic cartilage: (chondrocytes)

Eg: 1) Lobe of the ear.
2) Epiglottis.
3) part of the larynx.



Function:
1) maintain shape.
2) support

Temporary cartilage - later turns to bone

Permanent cartilage - remain the same

→ cartilage acts as the model and is gradually replaced by bone as the embryo grows. such cartilage is temporary cartilage. The process by which bone tissue follows the cartilage model and slowly replaces it is called as ossification.

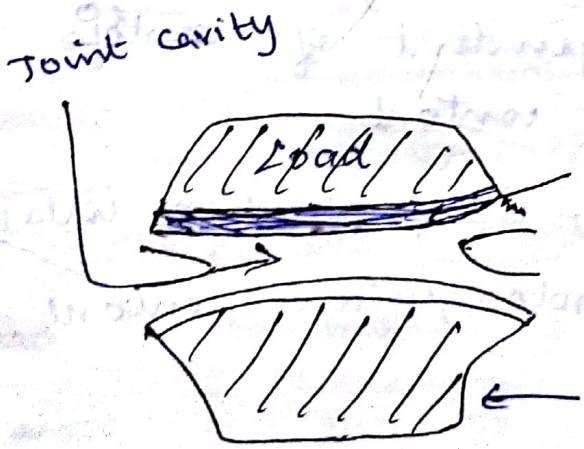
Permanent cartilage: → which does not become ossified eg: found in tip of the nose, external ear

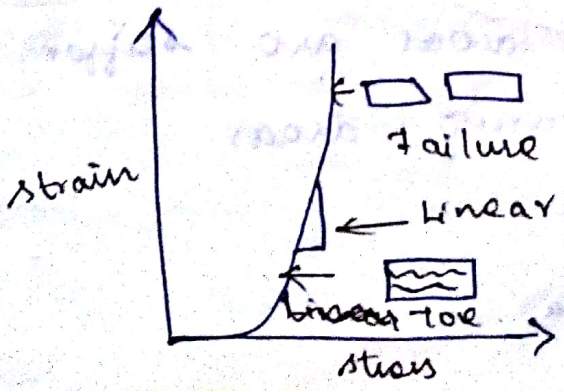
Contribution of mechanical properties:

→ collagen → elastin → proteoglycans

→ chondrocytes.

composition: → Density: 1.3 → water content: 75%.





Stress - strain relationship

Effect of squeeze: → ~~when~~ veins subjected to cartilage squeeze, it loses it water content and so lose volume get deformed. on the removal of squeeze water is taken back by cartilage it can be easily deformed.

→ mechanical property based on location, size, subject and age

→ It is Anisotropic due to the presence of collagen content → it is load bearing.

→ material used for the replacement of cartilage would depend on the analysis of stress experienced at that site.

→ Tensile behaviour: — cartilage has low tensile strength when compared to tendons and ligament. It is independent of ~~protein~~ ~~glycan~~ proteoglycan content

→ compressive behaviour - independent of collagen and depends on proteoglycan content.

It is elastic.

→ High weight bearing areas are stiffer than lower weight bearing areas.

→ Tensile modulus: 1-10mpa

→ compressive modulus: 1MPa.

3 zones: 1) superficial zone. → High collagen content
2) middle zone. → Low collagen content
3) Deep zone. → calcified cells.

strength and young's modulus ↓ when collagen ↓.

→ Articular cartilage experience creep at an equilibrium pt under compression. equilibrium is not reachable until about 30 sec after the load and creep is recoverable.

Diffusional properties of articular cartilage:

Biphase theory: the movement of interstitial fluid within the cartilage and across the articular surface are the two fundamental phenomena that governs the following.

1) mechanical response
2) Bio-mechanical fn
3) Biological integrity of tissue

Fluid transport mechanism: The interstitial fluid is transported to the matrix because of the pr gradient across the tissue with increase in Ps there is a increase in transport. The apparent Permeability is gn by.

$$Q = \frac{A k_{app} P_A}{h}$$

Q - volume flow rate.

A - Area of cartilage.

P_n - pr drop.

h - thickness.

k_{app} - apparent permeability.

the avg. value of $k_{app} = 0.58 \times 10^{-15} m^4/Nsec$

this small value indicates very high frictional resistance for the flow of fluid

it also found that permeability ↓es with depth the fluid transport is accomplished by deformation of cartilage matrix. the exudation is accomplished by squeezing the tissue b/w 2 rigid plates when this is done compression of solid matrix causes a rise of internal pr acting on the fluid this favours the exudation.

This mechanism is called as flow due to consolidation. the compression ↓es the Permeability of the tissue

Experiment.

the cartilage is bounded b/w

2 specimen holders. precise amount of strain is applied to the tissue the displacement of tissue is measured by LVDT. There is a release of fluid during compression so there is a change in the height of fluid in the capillary tube in this experi the bovine cartilage was used. more it was found that permeability decreases with increase strain the decrease was due to compaction of tissue which decreases the avg pore size of the tissue. thereby making it more difficult for the flow of fluid

$k_{app} = A(PA) \exp[\alpha(PA\epsilon)]$ the formula relating permeability, strain, stress and poisson is given.

Instrinsic Permeability.

It is defined as the Permeability for of the tissue that depends only on uniform strain

LUBRICATION OF JOINTS

Synovial fluid and its functions:

- The lubricant in a bone joint is called synovial fluid.
- Syn means like and ovial means egg white.
- The lubricant is therefore similar to egg white.
- It is non-newtonian in nature.
- This non-newtonian behavior is provided by a molecule known as Hyaluronic acid containing a chain of the COO^- group approximately 1.5 nm apart.
- In a healthy joint, the molecular weight of the hyaluronic acid is approximately 5×10^5 and its concentration is 0.1-5 mg/ml.
- The hyaluronic acid of diseased joints gets depolymerized and the fluid does not have non-newtonian behavior.

Normal synovial fluid is clear, pale yellow, viscid, and does not clot. Synovial fluid is a plasma dialysate modified by constituents secreted by the joint tissues. The major difference between synovial fluid and other body fluids derived from plasma is the high content of hyaluronic acid (mucin) in synovial fluid. The exact source of the hyaluronic acid has been the subject of debate. It is generally assumed, however, that both fibroblasts beneath the synovial membrane intima and synovial membrane-lining cells produce this mucopolysaccharide constituent of synovial fluid. Hyaluronic acid is a nonsulfated polysaccharide composed of equimolar quantities of D-glucuronic acid and *N*-acetyl-*D*-glucosamine residues. It was first identified in joint fluid by acetic acid precipitation. The normal viscosity of synovial fluid is due to the hyaluronic acid.

Functions:

Synovial fluid is believed to have two main functions: to aid in the nutrition of articular cartilage by acting as a transport medium for nutritional substances, such as glucose, and to aid in the mechanical function of joints by lubrication of the articulating surfaces. Articular cartilage has no blood, nerve, or lymphatic supply. Glucose for articular cartilage chondrocyte energy is transported from the periarticular vasculature to the cartilage by the synovial fluid. Under fasting conditions, the glucose concentration of synovial fluid is usually approximately equal to that of blood. A decreased amount of synovial fluid glucose may be associated with articular diseases, particularly septic and immune-mediated arthritis.

The normal volume of synovial fluid obviously varies from joint to joint. In the dog, the average is 0.24 ml (0.01 ml- 1.0 ml). The pH ranges from 7.0 to 7.8. Experimental work with dogs has shown that the pH is lowered by exercise and returns to a higher value at rest. As noted above, the viscosity of synovial fluid is due to the hyaluronic acid. Precipitation of synovial fluid mucin with weak acetic acid (mucin clot test) leaves a fluid with a viscosity similar to water. The lubricating ability of synovial fluid is often equated with its normal viscosity. However, experimentally induced tryptic digestion of hyaluronic acid will destroy the synovial fluid lubricating abilities without lowering its viscosity. It has also been shown that hyaluronic acid can be depolymerized without altering its lubricating capacity. Almost all of the protein

constituents of synovial fluid are derived from plasma. The passage of plasma proteins to synovial fluid is related to the size and shape of the protein molecule. The proteins of coagulation are not found in normal synovial fluid, while proteins of the plasmin system may be found in variable quantities. Normal synovial fluid does not clot but may exhibit thixotropy, the property of certain gels to become fluid when shaken. On standing at room temperature, normal synovial fluid may assume a gelatin-like appearance. When shaken, it will resume its normal fluid nature.

Cellular Constituents Cell counts of canine synovial fluid vary from joint to joint but normally are low. Average counts range from 0 to approximately 3000 cells/mm3.(28) Lymphocytes are seen in the greatest numbers, and both B and T lymphocytes have been identified. Monocytes and neutrophils are also present normally, while macrophages are seen only occasionally. Differentiation between mononuclear cells of bone marrow origin and those derived from local tissues may be difficult, since reactive macrophages may assume the characteristics of type A or B synovial cells. Occasionally, aspiration produces clusters of synovial membrane cells that are more readily identifiable. In pathologic fluids, chondrocytes, osteoblasts, and osteoclasts may be seen. Fragments of articular cartilage may contain chondrocytes within lacunae. Exposed subchondral bone may give osteoblasts and multinucleated osteoclasts access to the synovial fluid.

Despite being subjected to varying high loads, the articular surfaces in the synovial joints undergo minimal wear & tear. This is due to the lubrication process occurring within the joints.

Lubrication of Joints

Lubrication is a process of reducing friction and/or wear (or other forms of surface damage) between relatively moving surfaces by the application of a solid, liquid, or gaseous substance (i.e., a lubricant).

The main function of a lubricant is to keep the surfaces apart so that interaction (e.g., adhesion and shear) between the solids cannot occur; thus, friction and wear can be reduced or controlled. Lubrication reduces frictional resistance between bearing surfaces by keeping them apart. Friction and the resulting wear of two un-lubricated surfaces sliding on each other are due to the interaction or contact between the opposing surfaces. In many mechanical bearings lubricated by oil, the relative continuous motion of the surfaces produces a wedge of lubricant that keeps the surfaces apart. This phenomenon is defined as hydrodynamic lubrication and requires uninterrupted motion in the same direction to maintain the integrity of the wedge. Because joints oscillate and change direction of motion, pure hydrodynamic lubrication is not the mechanism by which synovial fluid functions as a lubricant.

Many theories based on extensive investigation of the physical properties and abilities of synovial fluid to act as a lubricant have been presented to explain the mechanisms of joint lubrication. It appears that the low frictional resistance to joint motion is due to a combination of mechanisms. Each mechanism complements the other and depends on the tissues involved and the load imparted to the joint.

Resistance to joint motion comes from the stretching of surrounding soft tissues (ligaments, tendons, muscle) and frictional resistance of the joint parts that must slide across each other (cartilage, synovium, tendons in sheaths). Surfaces that contact each other during joint motion and therefore give rise to frictional resistance have been defined as (1) a soft tissue interface-synovium on synovium or synovium on cartilage-and (2) a cartilage-on-cartilage type.

The different types of lubrication are

1. Boundary lubrication
2. Fluid film lubrication
    a. Hydrostatic lubrication
    b. Hydrodynamic lubrication
3. Mixed Lubrication

Boundary lubrication:

It involves adsorption of a single monolayer of lubricant on each surface. This type of lubrication prevents direct surface to surface contact at an articulation and therefore minimizes the friction of the articulating surfaces.

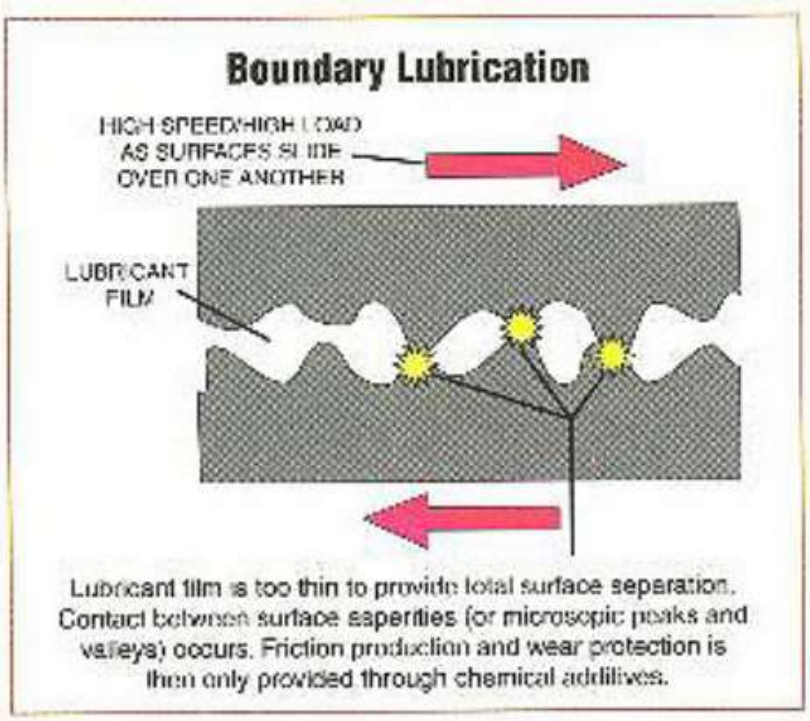
It is independent of the properties of lubricating surface and the mechanical properties of the lubricant. In synovial joints, the glycoprotein which is found in synovial fluid is believed to be the adsorbed molecule. The thickness of this layer of the adsorbed molecules is between 1-100 nm the articular surfaces.

Lubrication of synovial surfaces by synovial fluid requires hyaluronate and is due to a boundary phenomenon. Boundary lubrication occurs when each bearing surface is coated or impregnated with a thin layer of lubricant that keeps the sliding surfaces apart, allowing ease of motion with a low coefficient of friction between the sliding surfaces. Hyaluronate sticks to the synovial surfaces. The lubricating properties of synovial fluid in a soft tissue system are directly related to the concentration and molecular weight of the hyaluronate, which is also determined by viscosity. However, it is not the viscosity of synovial fluid that is responsible for lubrication of this system but the stickiness or boundary phenomena exhibited by the fluid. Viscous solutions containing no hyaluronate do not lubricate a soft tissue system nearly as well as solutions containing hyaluronate of equal or even lower viscosity.

The lubricating properties of synovial fluid on articular cartilage were originally attributed to its viscosity, which in turn is due to the presence of hyaluronate or mucin. However, viscosity or the resistance of a fluid to shearing forces is not the same as lubricating effectiveness. Digestion of synovial fluid hyaluronate by hyaluronidase, which totally destroys the viscous nature of the fluid, does not decrease the lubricating properties of synovial fluid on articular cartilage when compared with a nonviscous buffer. This is in contrast to the finding that proteolytic digestion of synovial fluid decreases its lubricating abilities. A glycoprotein has been isolated from synovial fluid and removal of this fraction from the fluid deprives it of its lubricating properties.

The mechanisms of cartilage-on-cartilage lubrication have been attributed to boundary effects and the presence of a fluid film. The boundary effect of synovial fluid in a cartilage-on-cartilage

system is similar to that in a soft tissue system in that synovial fluid readily adheres to the cartilage surfaces, helping to keep them apart and decreasing frictional forces. Unlike the soft tissue system, however, the boundary effect of synovial fluid is not due to the hyaluronate but to the lubricating glycoprotein fraction of synovial fluid. It is this fraction that sticks firmly to the articular cartilage surfaces. Although hyaluronate does not directly decrease the coefficient of friction in a cartilage-on-cartilage system, it may enhance the longevity of the lubricating ability of the protein fraction and act as a spreading factor. Articular cartilage is quite resistant to shear forces but very sensitive to impact loading. Boundary lubrication of articular cartilage is extremely effective in preventing wear due to motion but loses its protective abilities under high loads. Therefore, other lubricating mechanisms must be at work.



Fluid-film lubrication:

Fluid-film lubrication is a class of mechanisms of lubrication in which a film of fluid separates the opposing sliding surfaces. The fluid may be introduced intentionally, as the oil in the main bearings of an automobile, or unintentionally, as in the case of water between a smooth rubber tire and a wet pavement. Although the fluid is usually a liquid, it may also be a gas. The gas most commonly employed is air. For a fluid film to lubricate moving surfaces effectively, it must be thicker than the roughness of the opposing surfaces. The thickness of the film depends on the viscosity of the fluid, the shape of the gap between the parts, and their relative velocity, as well as the stiffness of the surfaces.

Types:

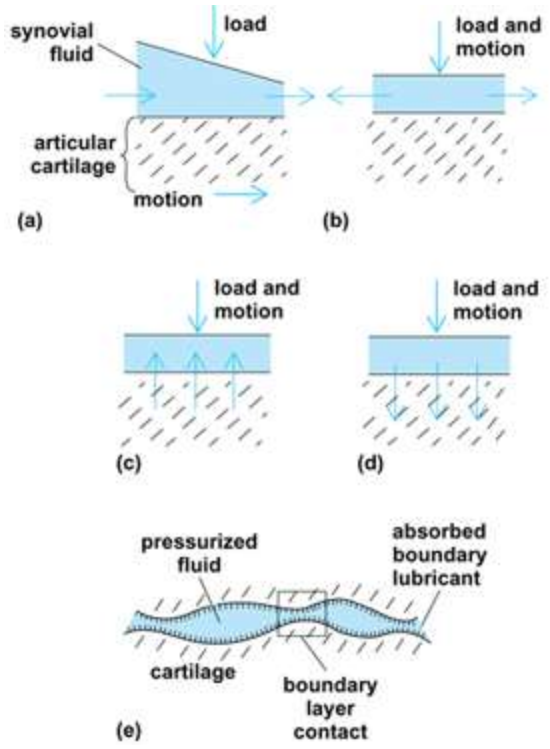
a. Hydrodynamic lubrication
b. Squeeze film or hydrostatic lubrication

Hydrodynamic lubrication:

This occurs when rigid bearing surfaces which are not parallel and are separated by fluid film, slight tangentially in relation to each other. A converging wedge of the fluid forms the lubricating surface. The viscosity within this wedge of fluid produces a lifting pressure between the two surfaces. Articular cartilage is elastic fluid-filled and backed by a relatively impervious layer of calcified cartilage and bone. This means that load-induced compression of cartilage will force interstitial fluid to flow laterally within the tissue and to surface through adjacent cartilage. As that area in turn becomes load bearing it is partially protected by the newly expressed fluid above it. This is a special form of hydrodynamic lubrication so-called because the dynamic motion of the bearing areas produces an aqueous layer that separates and protects the contact points.

Squeeze film lubrication:

Squeeze-film lubrication is a form of lubrication in which the approaching surfaces generate pressure in the lubricant as they squeeze it out of the area of impending contact. The resulting pressure keeps the surfaces apart, and the lubricant film that forms in the area of impending contact is referred to as the squeeze film. Electromicroscopically, articular cartilage is shown to have depressions and irregularities on its surface. In the early phases of loading these depressions may trap fluid. With increasing load, the articular cartilage surface may deform and the irregularities disappear. The surface deformation and intrinsic elasticity of articular cartilage will tend to make the space of impending cartilage contact narrower at its margin than at its center. More fluid will be trapped in the center of the contact area where it may help to form a squeeze film. This mechanism of lubricant trapping has been called "boosted" lubrication. Compression of articular cartilage produces a watery film on its surface. This wept fluid is composed mainly of water and small ions. Pore size in articular cartilage has been measured to be approximately 60 nm, although occasional large pores (l000 nm) are present. This small pore diameter restricts the passage of large molecules such as the mucopolysaccharides of the cartilage matrix and hyaluronate and synovial fluid protein while allowing passage of interstitial fluid of the cartilage.



(a) hydrodynamic; (b) squeeze film; (c) hydrostatic weeping; (d) boosted. (e) A combination of boundary layer lubrication at points of contact and fluid-film lubrication (mixed)

Weeping lubrication:

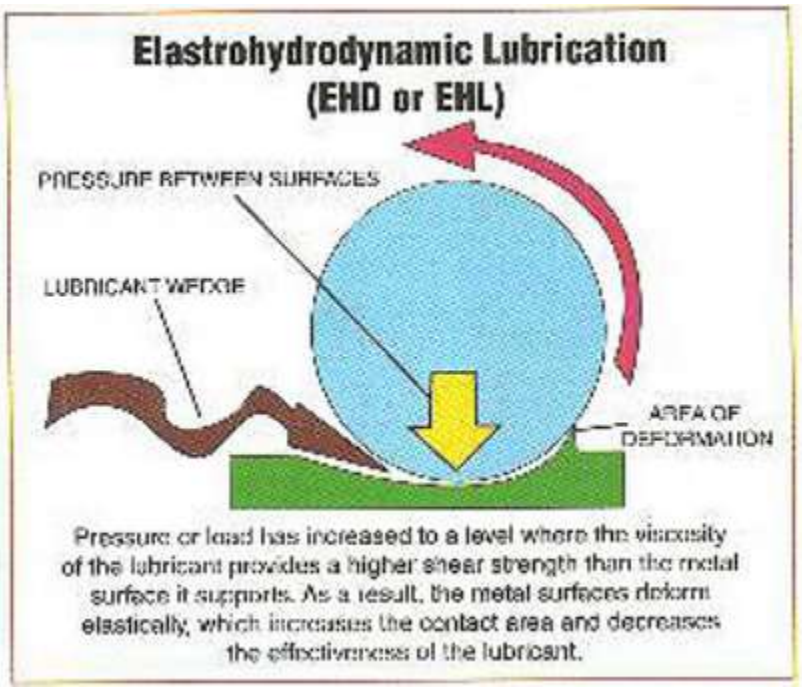
It has been suggested that in a highly loaded joint this wept fluid creates a lubricating fluid filmy referred to as "weeping" or self-pressurized hydrostatic lubrication. The fluid flow onto the cartilage surface probably occurs at the periphery of the area of impending contact where the pressure is lower rather than at the center of the contact area where the pressure is highest. Fluid flow out of the cartilage toward the subchondral area is blocked by the subchondral plate, and sideways flow is retarded by the relatively poor perfusion characteristics of cartilage.

Elastohydrodynamic lubrication :

Another consideration in the mechanism of decreased friction between articular cartilage surfaces is the intrinsic elasticity of the cartilage. The compliance of articular cartilage may allow its uneven surface to flatten under loading, thus lowering the pressure at impending junction sites. *Elastohydrodynamic lubrication* has been defined as a form of fluid lubrication occurring when bearing surfaces are sufficiently elastic for the lubricant pressure generated by motion under a given load to depress the surfaces a distance greater than their highest peaks, thus facilitating maintenance of a fluid film.

In summary, synovial joints contain two systems that require lubrication: a soft tissue system and a cartilage-on-cartilage system. Lubrication of the soft tissue system is of the boundary type, requiring the hyaluronate of the synovial fluid to stick to the sliding surfaces of the system, thus keeping them apart. In contrast, the cartilage-on-cartilage system is independent of hyaluronate

and dependent on a glycoprotein fraction of synovial fluid. At low loads the lubricating action of the glycoprotein is of the boundary type. At high loads the cartilage surfaces are kept apart by fluid film composed of fluid and interstitial fluid wept from the articular cartilage itself. The elasticity of articular cartilage may potentiate the fluid-film lubricating mechanisms at high loads.



UNIT - 4

Introduction to Finite Element Analysis:

In the past few decades the finite element modelling has been developed as an effective tool for modelling and simulation of the Biomedical Engineering System.

- Finite element modelling (FEM) is a computational technique which can be used to solve the biomedical engineering problems based on the theories of continuum mechanics.
- Applications of Finite Element modelling in the four areas of bone biomechanics,

  (i.e)., - Analysis of stress and strain
  - Determination of mechanical properties.
  - Fracture Fixation design (Implants).
  - Fracture load prediction.

The finite elements method was first used in bone biomechanics for analysis of mechanical behaviour of skeletal parts in 1972. Steadily this method has become very popular in biomechanics field.

Finite Element Modelling (FEM) has 3 major stages to analyse the human bones (i.e).

i). pre - processor

ii). Solution

iii). The post process stage.

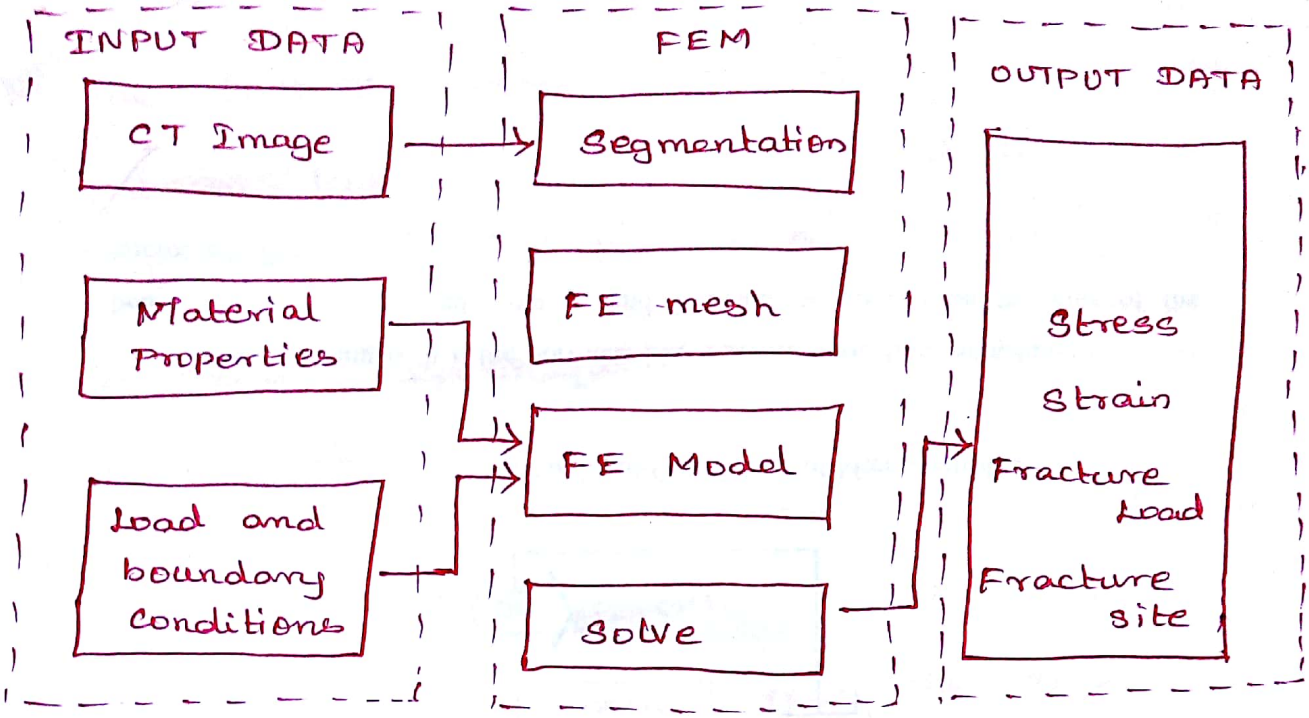
→ In pre - process stage a CAD model is required to be generated.

→ The Geometry and material properties of bone can be acquired from computed tomography (CT).

→ The Geometry of fracture fixation (implant) is usually developed on CAD software like CATIA, Solid works, Pro/E, etc.

→ once the bone model is developed the mesh generation is carried out. The material properties to each model is assigned and finally the boundary conditions are applied.

→ It is essential to apply the correct boundary condition in FEA to get accurate results.



Finite Element Modelling of bone based on CT images.

(7). Stress and strain Analysis:

The stress analysis of bones using finite element modelling is a key to understand the bone remodelling, assessment of fracture risk and designing of fracture fiocation.

- The Geometry of bone was obtained from the CT Scan.
- Due to the irregular stress distribution at the contact region (joint) of bones the fracture is more likely to occur at the joints

in case of impact (falling of human.

→ The contact stress between the bones of human knee joints (femur and tibia) is determined using the FEM software NASTRAN.

→ In many experiments the compact and spongy bone are modelled as isotropic and homogenous but in real conditions both type of bones have different material properties.

→ It has been observed that the strain rate affects the human bone toughness.

② Mechanical Properties of Bone :

It is essential to predict the mechanical property (strength, stiffness, toughness) of bones in order to estimate the fracture risk.

→ In humans usually after thirty years of age, the mass of bone starts to decrease.

→ This phenomenon is known as osteoporosis.

→ Due to osteoporosis the mechanical properties of bones are negatively affected.

→ Quantitative Computed Tomography (QCT) based geometry of bone provides more accurate mechanical properties of bone than the Dual Energy X-ray Absorptiometry (DXA).

③. Fracture Fixation Design (Implant):

There are 2 types of fracture fixation in bone,

i). External Skeletal Fixation
(POP, clamp fixators, ring fixators).

ii). Internal Fixations.
(Plate and screws, intramedullary nails)

→ Analysis of Internal Fixation Configuration using FEM uses the material for the plate used ~~is their~~ are titanium Alloy.

④. Fracture Load Analysis:

When load in a particular region of a bone exceeds the ultimate strength of bone, then fracture occurs.

- Fracture means the continuity of bone being disrupted.

The finite element modelling has been developed as an effective tool in the bone biomechanics has some limitation. The most important limitation in application of FEM for bone biomechanics are,

- Lack of Anatomical detail in the modelling phase.
- Lack of information about the material properties of bone.
- Bone structure also hampers its accuracy.

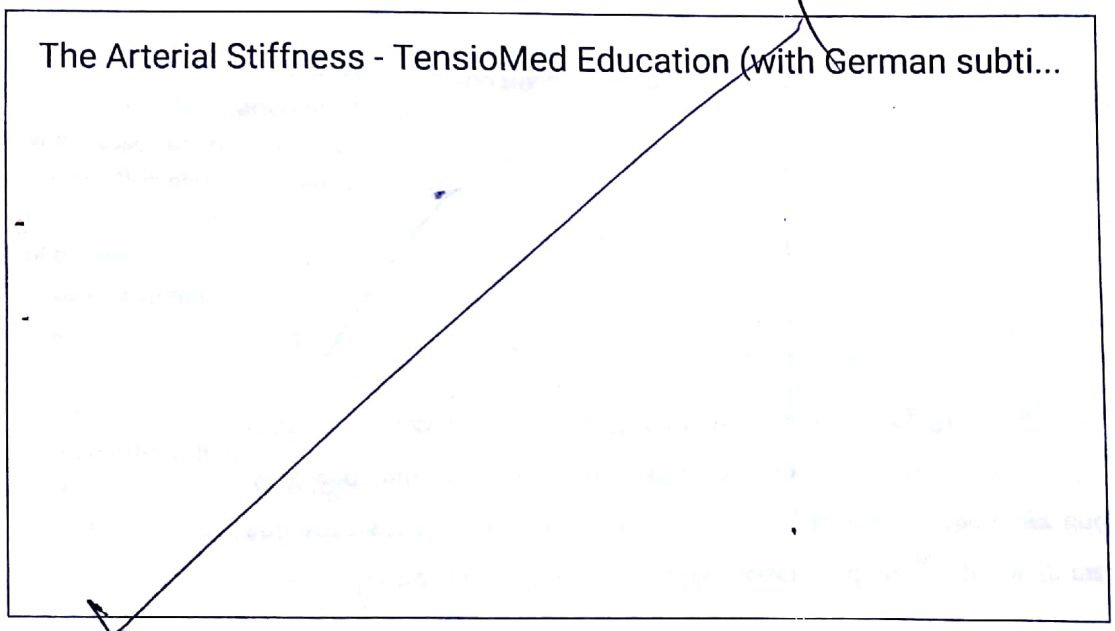
With the recent advances in computer tomography, some of these limitations have been overcome upto some extent.

Arterial Stiffness

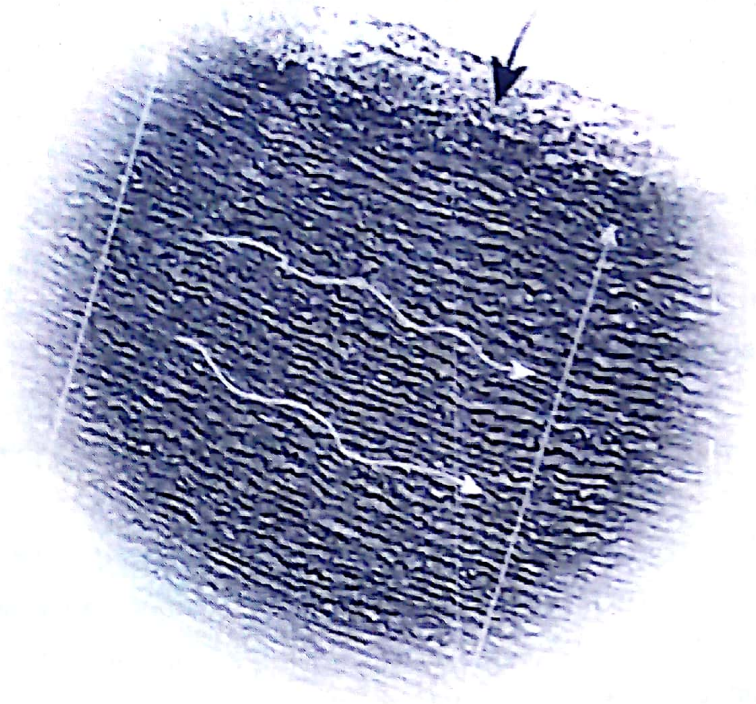
Why Should We Measure Arterial Stiffness through Pulse Wave Velocity?

The arterial stiffness refers to the actual arterial wall characteristics, namely to its elastic our rigid properties. The aortic/arterial stiffness is mainly determined by the structure of the aortic wall furthermore (with less extent) by and other factors, like blood pressure, stroke volume and cardiac output.
The aorta represents the typical type of elastic artery. As it can be seen on the pictures below, the aorta has a very thick elastic layer, with less amount of smooth muscle, while in the muscular arteries the smooth muscle cells are dominating in the tunica media.

Education video with German subtitle



Aorta – elastic artery



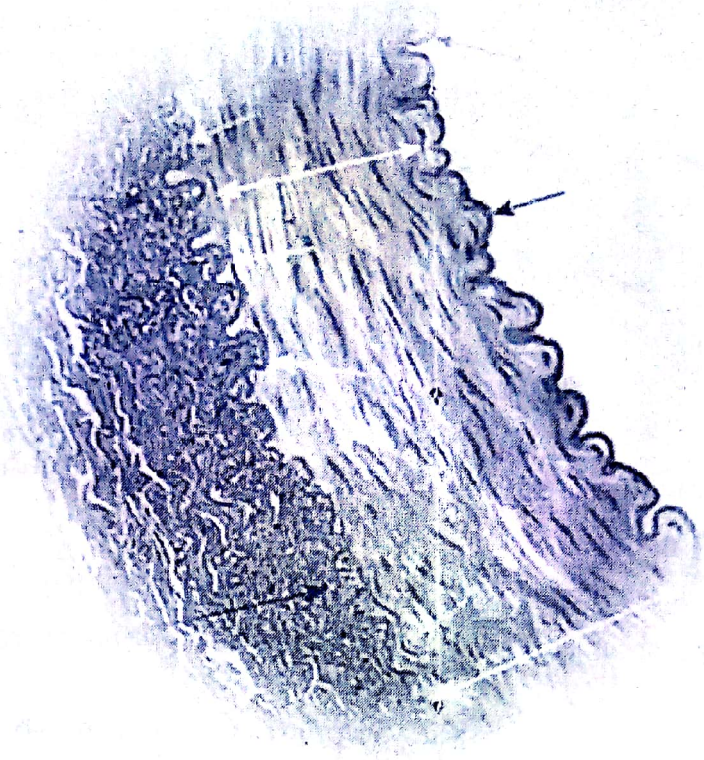
During ageing the aortic/arterial stiffness (rigidity) increases. The stiffening of the aortic (arterial stiffness) wall plays a pivotal role in the deterioration of a fundamental physiological function of the aorta, namely in the "windkessel" function. The windkessel function of the aorta means that in normal situation the soft, elastic aortic wall easily and largely dilates to due to the ejection of the left ventricle pushing the stroke volume into the aorta. This way the intermittent ventricular flow/pressure becomes a continuous (albeit pulsatile) flow during the whole cardiac cycle, and the systolic energy created by the ejection of the left ventricle will be stored for the diastole, as well.

In case of stiffened aortic wall (diminished windkesel function) the vast majority of the pulse wave energy will be driven towards the periphery during systole and poor perfusion condition will develop during diastole. The reason why the diastolic pressure is so important for the blood and oxygen supply to the myocardial tissue is that the left coronary artery is almost entirely perfused during the diastole.

However the stiffening of the aortic wall (arterial ageing) differs individually. Many subjects exhibit earlier development of the increased aortic/arterial stiffness (early vascular or arterial ageing), while the luckier subjects have slower or missing occurrence of the aortic/arterial stiffening. Those who belong to the early arterial ageing group have poorer life expectancy due to higher cardiovascular risk.

Indeed we have a large body of evidence about the increased aortic/arterial stiffness and adverse cardiac outcome. Several longitudinal trials pointed out, that the increased aortic/arterial stiffness measured as increased pulse wave velocity (/en/pulse-wave-velocity) predicts cardiovascular diseases (coronary heart disease, myocardial infarction, stroke) furthermore cardiovascular and all cause mortality. This relationship was proven in hypertension, end-stage renal disease, diabetes and in general population.

Muscular artery



The basic relation between the aortic/arterial stiffness and the aortic pulse wave velocity is simple; the more rigid, the stiffer the aortic wall, the higher the aortic pulse wave velocity (PVW) (/en/pulse-wave-velocity) is. Nevertheless the blood pressure plays also an important role influencing the aortic stiffness and consequently, the pulse wave velocity. If the blood pressure is increased the lateral tension towards the aortic wall increases, the elastic lamellae of the aortic wall will be stretched, thus finally the aortic wall will be stiffer, i.e. the aortic pulse wave velocity will increase. In a very elegant study Guerin, P., et al (Circulation, 2001;103;987-992) pointed out among ESRD subjects that if the blood pressure was lowered, the aortic pulse wave velocity (/en/pulse-wave-velocity) also decreased in those patients who had favorable survival during the follow-up period. However in those subjects whose aortic pulse wave velocity has not decreased together with the blood pressure reduction had very poor survival. Consequently the aortic pulse wave velocity should always be judged together with the blood pressure and the changes due to the antihypertensive therapy,play prognostic role.

On the basis of the mentioned circumstances the recent 2013 ESH/ESC Guidelines for the management of the arterial hypertension (Journal of Hypertension 2013, 31:1281–1357) advocates the measurement of aortic pulse wave velocity in order to reveal organ damage, and to improve cardiovascular risk assessment, because patients in intermediate risk could be reclassified into lower or higher risk group by the value of the measured aortic pulse wave velocity. In the former Guidelines the threshold of the abnormal aortic pulse wave velocity was given at 12 m/s, however due to the overestimation of the distance measurement using site-to-site (direct distance between carotid measuring site and femoral measuring site) method the Guidelines suggested to use a lower threshold that can be calculated according to the next formula; 12m/s*0,8, which is 9,6 m/s, however 10 m/s was finally given as the threshold due to an easier adaptation of the value in the daily practice.

It is worth mentioning that the pulse wave velocity can be measured locally or regionally (including the whole aortic length). Local arterial stiffness is most frequently measured on the common carotid artery. Today we have the most relevant scientific evidence with regional (aortic) pulse wave velocity (PWV) (/en/pulse-wave-velocity). The value of the local arterial stiffness regarding prognostic significance is scarce.

Finally, the answer for the title question, why should we measure arterial stiffness through pulse wave velocity can be given with sufficiently based scientific evidence; the aortic pulse wave velocity (/en/pulse-wave-velocity) is an independent predictor of adverse cardiac outcome, furthermore cardiovascular and all cause mortality. It means that the patient

UNIT - 4

Pulse Wave Velocity

In the arterial system the pulse wave travels with different velocity

Basically, the softer, the more elastic the aortic wall, the lower the pulse wave velocity (PWV) is. Thus the lowest pulse wave velocity (PWV) can be measured on the aorta, because this is typical elastic, "reservoir" artery. Towards the periphery the arterial wall contains more muscular elements, the wall will be stiffer, and consequently the pulse wave will be higher and higher.

Education video with German subtitle

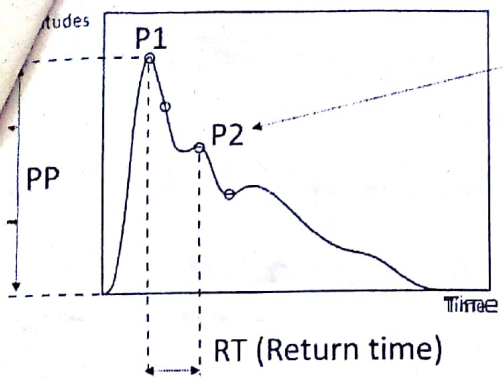
Pulse Wave Velocity - TensioMed Education (with German subtitle)

The physical entity of arterial pulse wave velocity (PWV) cannot be confused with the flow velocity. It is a rather common misunderstanding when one explains and talks about arterial (aortic) pulse wave velocity our partner often answers, "I used to measure arterial pulse wave velocity with echo". The Doppler ultrasound measured velocity is always a flow velocity measurement, that determined by the moving, travelling speed of the corpuscular elements of the blood, inside the artery. If the direction of the blood flow moves towards the Doppler probe, the emitted ultrasound frequency will increase and if the blood flow moves away from the probe, the frequency will decrease.

However the pulse wave velocity (PWV) is the speed of the propagation of the pressure wave (energy) generated by the left ventricle. During the propagation of the pressure wave the elements of the fluid give the energy to each other, like in the "Newton's cradle" (see the animation). The velocity of the pulse pressure wave is the velocity of the „tsunami" generated by the contraction of the heart.

We have large body of evidence that the increased aortic pulse wave velocity (PWVao) has prognostic role regarding adverse

Normal Aix PWVao and SBPao



Low peripheral arterial resistance

cardiac outcomes and deaths in end-stage renal disease, hypertension, coronary artery disease, diabetes mellitus and general population. Increased aortic pulse wave velocity (PWVao) refers to the rigid stiff aorta that increases the systolic blood pressure reduces the reservoir function of the aorta. Increased aortic pulse wave velocity is considered to be an organ damage, and a marker of high cardiovascular risk, independently from the traditional risk factors according to the 2013 Guidelines for the management of arterial hypertension issued by European Society of Hypertension and the European Society of Cardiology.

The arterial pulse wave velocity, of course, can be measured not only regionally on the aorta, but also on other arteries, such as femoral or brachial. However several studies pointed out that only the aortic pulse wave velocity (PWVao) has independent prognostic value, while the brachial and femoral pulse wave velocity (PWV) has not. Thus, as generally agreed, the aortic pulse wave velocity (PWVao) is considered to be the most relevant marker.

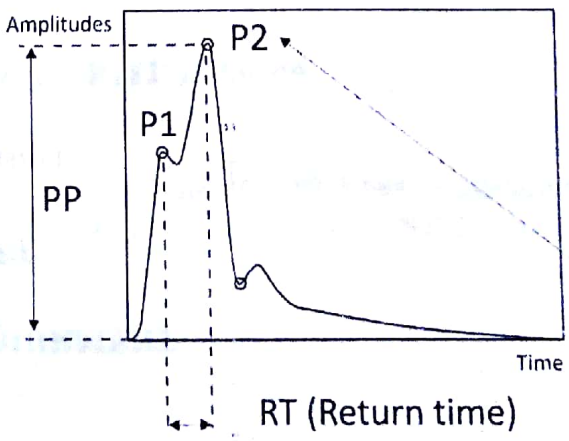
Regarding the measurement of aortic PWV the carotid-femoral method is considered to be the standard, however it suffers some limitations;

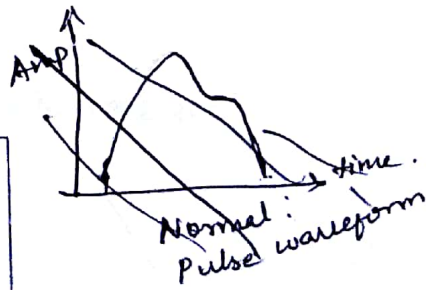
- The characteristics of carotid, iliac and femoral arteries are also influencing the measured PWV, however the wall of these arteries has more muscular part, thus these arteries are stiffer and their PWV is higher than the aorta, which is finally the point of interest.
- The simultaneous measurement of the pulse wave at the carotid and femoral site with two sensors overestimates the aortic pulse wave velocity (the higher the true aortic PWV, the higher the error is), because of the opposit direction of the pressure wave propagation from the heart.
- The other method with sequential measurement of pulse wave on the carotid and femoral artery with ECG gating and with one probe, helps to treat this problem, however because of the isovolumetric contraction time varies on beat-to-beat basis, larger variance and poorer reproducubility can be achieved.

Indeed comparing the above mentioned methods to the oscillometric one the carotid-femoral methods showed poorer variance and reproducibility (Baulmann, J., et al.: A new oscillometric method for assessment of arterial stiffness: comparison with tonometric and piezo-electronic methods. J Hypertens, 2008, 26:523–528).

It has to be mentioned also, that the aortic PWV is influenced by other factors, as well, mainly by the blood pressure and cardiac output. The relationship between these entities and the pulse wave velocity (PWV) is obvious; the more

Abnormal Aix PWVao and SBPao





High peripheral arterial resistance

pressure/volume load dilates the aortic wall the stiffer the arterial wall will be. For this reason the blood pressure and volume/cardiac output issues should always be judged together with the actual aortic PWV. In case of high heart rate or elevated systolic blood pressure the aortic pulse wave velocity (PWVao) measurement should be repeated in normal (normalised) blood pressure and heart rate situation. If the aortic PWV remains to be high even after the normalisation of the mentioned parameters, the cardiovascular risk of the patient is very high. This unpleasant situation refers to a very probable scenario; the aortic wall damaged morphologically, the arteriosclerotic and atherosclerotic structural changes stiffened the aortic wall in such an extent, that the decrease of the lateral tension of the wall does not help, i.e. the morphologically damaged arterio and atherosclerotic aortic wall is rigid, stiff, independently from the decrease of the lateral tension of the wall.

Could the aortic pulse wave velocity to be a specific target of intervention (e.g. specific drugs), it is still a question. Taking into consideration that this parameter is generally low in young age, it seems that the increase of the aortic pulse wave velocity (PWVao) is a consequence of different noxae. Consequently we have to detect and manage (treat) those factors that can contribute to the aortic stiffening in an early time, like high blood pressure, dyslipidaemia, diabetes, obesity, sedimentary lifestyle.

Aortic pulse wave velocity (PWVao) can be measured throughout the day with lately occurred new, oscillometric technologies. However we have no prospective data and cut-off value of the mean 24h aortic pulse wave velocity (PWVao) regarding to hard cardiac outcomes, thus this method, although is very promising, actually is still remain for research purposes.

PWV ↑ses when arteries stiffness or rigid with aging & disease

How to measure aortic pulse wave velocity?

Several methods are available to measure aortic pulse wave velocity. The really true aortic pulse wave velocity can only be measured via invasive catheterization positioning the catheters to the aortic root and to the aortic bifurcation and to measure the time difference of the pressure signals on identical heart beats. For the non-invasive measurement of aortic pulse wave velocity different methods are available;

carotid femoral pulse wave velocity measurement with

→ Effect generates electrical charge when force applied.
– measures changes in pressure.

- piezoelectric sensors
- applanation tonometry
- Doppler ultrasound
- oscillometric cuffs

Aortic pulse wave velocity measurement with

- magnetic resonance
- one single upper arm cuff (Arteriograph)

UNIT - 4

11

Mechanics of Blood Vessels

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11.1 Assumptions

This chapter is concerned with the mechanical behavior of blood vessels under static loading conditions and the methods required to analyze this behavior. The assumptions underlying this discussion are for *ideal* blood vessels that are at least regionally homogeneous, incompressible, elastic, and cylindrically orthotropic. Although physiologic systems are *nonideal*, much understanding of vascular mechanics has been gained through the use of methods based upon these ideal assumptions.

11.1.1 Homogeneity of the Vessel Wall

On visual inspection, blood vessels appear to be fairly homogeneous and distinct from surrounding connective tissue. The inhomogeneity of the vascular wall is realized when one examines the tissue under a low-power microscope, where one can easily identify two distinct structures: the media and adventitia. For this reason the assumption of vessel wall homogeneity is applied cautiously. Such an assumption may be valid only within distinct macroscopic structures. However, few investigators have incorporated macroscopic inhomogeneity into studies of vascular mechanics [1].

11.1.2 Incompressibility of the Vessel Wall

Experimental measurement of wall compressibility of 0.06% at 270 cm of H_2O indicates that the vessel can be considered incompressible when subjected to physiologic pressure and load [2]. In terms of the mechanical behavior of blood vessels, this is small relative to the large magnitude of the distortional strains

that occur when blood vessels are deformed under the same conditions. Therefore, vascular compressibility may be important to understanding other physiologic processes related to blood vessels, such as the transport of interstitial fluid.

11.1.3 Inelasticity of the Vessel Wall

That blood vessel walls exhibit inelastic behavior such as length-tension and pressure-diameter hysteresis, stress relaxation, and creep has been reported extensively [3,4]. However, blood vessels are able to maintain stability and contain the pressure and flow of blood under a variety of physiologic conditions. These conditions are dynamic but slowly varying with a large static component.

11.1.4 Residual Stress and Strain

Blood vessels are known to retract both longitudinally and circumferentially after excision. This retraction is caused by the relief of distending forces resulting from internal pressure and longitudinal tractions. The magnitude of retraction is influenced by several factors. Among these factors are growth, aging, and hypertension. Circumferential retraction of medium-caliber blood vessels, such as the carotid, iliac, and bracheal arteries, can exceed 70% following reduction of internal blood pressure to zero. In the case of the carotid artery, the amount of longitudinal retraction tends to increase during growth and to decrease in subsequent aging [5]. It would seem reasonable to assume that blood vessels are in a nearly stress-free state when they are fully retracted and free of external loads. This configuration also seems to be a reasonable choice for the reference configuration. However, this ignores residual stress and strain effects that have been the subject of current research [6–11].

Blood vessels are formed in a dynamic environment that gives rise to imbalances between the forces that tend to extend the diameter and length and the internal forces that tend to resist the extension. This imbalance is thought to stimulate the growth of elastin and collagen and to effectively reduce the stresses in the underlying tissue. Under these conditions it is not surprising that a residual stress state exists when the vessel is fully retracted and free of external tractions. This process has been called *remodeling* [7]. Striking evidence of this remodeling is found when a cylindrical slice of the fully retracted blood vessel is cut longitudinally through the wall. The cylinder springs open, releasing bending stresses kept in balance by the cylindrical geometry [11].

11.2 Vascular Anatomy

A blood vessel can be divided anatomically into three distinct cylindrical sections when viewed under the optical microscope. Starting at the inside of the vessel, they are the intima, the media, and the adventitia. These structures have distinct functions in terms of the blood vessel physiology and mechanical properties.

The intima consists of a thin monolayer of endothelial cells that line the inner surface of the blood vessel. The endothelial cells have little influence on blood vessel mechanics but do play an important role in hemodynamics and transport phenomena. Because of their anatomical location, these cells are subjected to large variations in stress and strain as a result of pulsatile changes in blood pressure and flow.

The media represents the major portion of the vessel wall and provides most of the mechanical strength necessary to sustain structural integrity. The media is organized into alternating layers of interconnected smooth muscle cells and elastic lamellae. There is evidence of collagen throughout the media. These small collagen fibers are found within the bands of smooth muscle and may participate in the transfer of forces between the smooth muscle cells and the elastic lamellae. The elastic lamellae are composed principally of the fiberous protein elastin. The number of elastic lamellae depends upon the wall thickness and the anatomical location [12]. In the case of the canine carotid, the elastic lamellae account for a major component of the static structural response of the blood vessel [13]. This response is modulated by the smooth-muscle cells, which have the ability to actively change the mechanical characteristics of the wall [14].

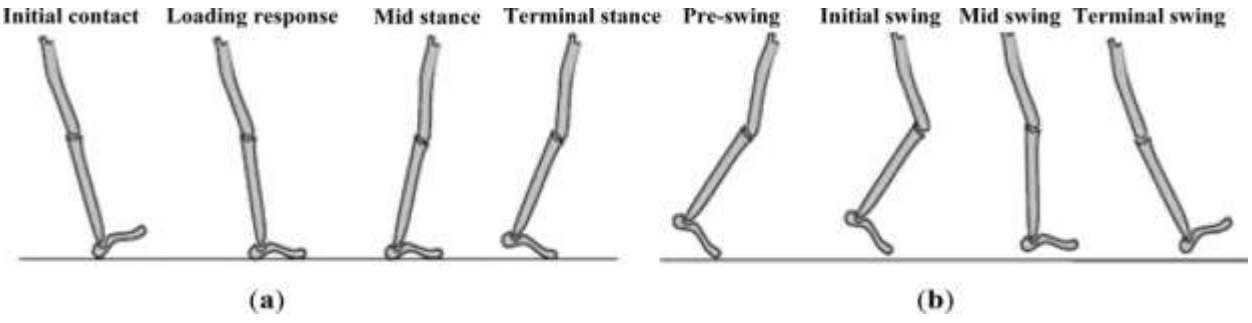
UNIT V ORTHOPAEDIC APPLICATIONS

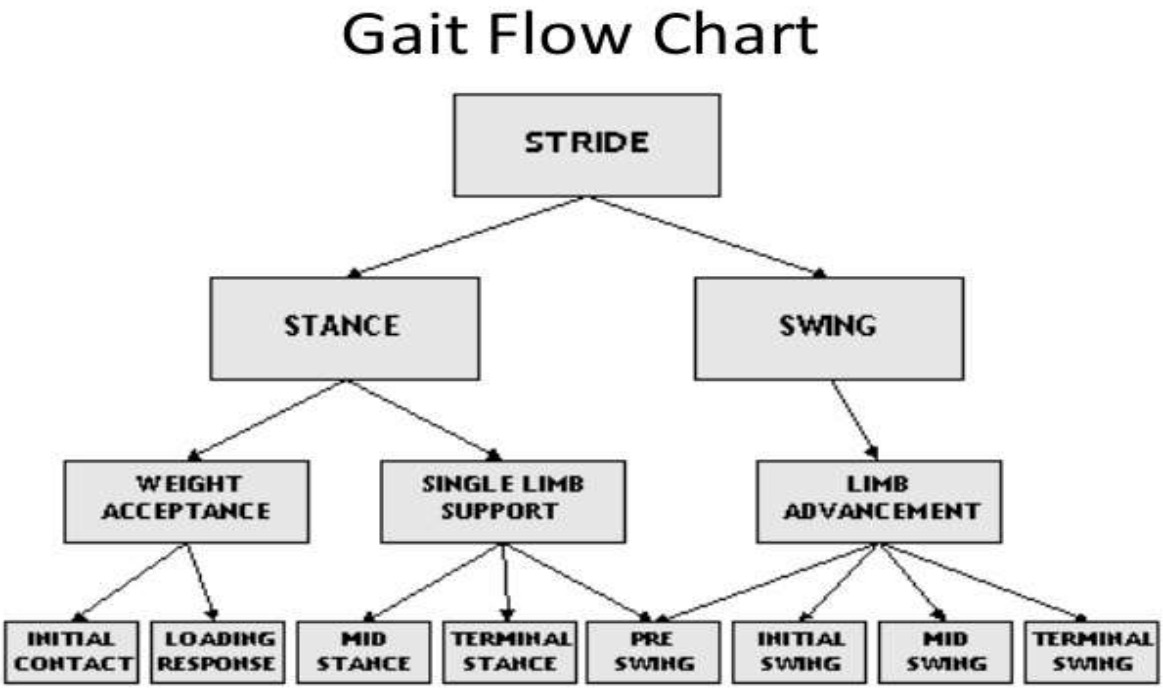
Dynamics and analysis of human locomotion - Gait analysis (determination of instantaneous joint reaction analysis), occupant response to vehicular vibration. Mechanics of knee joint during standing and walking

GAIT ANALYSIS:

Gait analysis is a highly complex study of the biomechanics of locomotion. It is important to recognize important phases of the gait cycle when fitting and examining shoes. Understanding the customer's gait will provide a foundation for a proper fitting of shoes and orthotics. When an individual walks, the foot acts as a lever. The muscles in the back of the leg pull up against gravity. Weight is transferred in rapid order from the anklebone to the heel bone, down the outer longitudinal arch, across the metatarsal heads, and off the big toe. Thus the body weight is propelled forward toward the ball of the foot. The center of gravity of the body is shifted to a few inches in front of the toes at a point not yet occupied by the person. At the same time, the other foot is moved forward by the thigh and hip. The muscles in the front of the leg contract the inner longitudinal arch and prepare it to accept the weight of the body as the heel comes down in a forward motion. The muscles in the feet themselves raise the arch and act as a spring that helps support the inner longitudinal arch and increase the stability of the foot. Movement of the human foot is a coordinated action and effort by each part of its structure. The gait cycle is divided into different phases. The following information applies to walking gait phases. However, running gait is similar to walking gait in that the same phases exist, though the percentages of time spent in each phase and stage of the phase differ between the two. Running also has an additional "float phase."

Phases of the Gait Cycle:





Weight Acceptance

1) Initial Contact: The movement when the foot strikes the ground

a) Muscle Involvement: Anterior Tibialis (AT)

b) Muscle Function: Anterior Tibialis (AT) is a dorsiflexor and serves to decelerate plantarflexion following heel strike

2) Loading Response: Shock is absorbed as forward momentum is preserved. A foot-flat position is achieved. AT is dorsiflexor and serves to decelerate plantarflexion following heel strike

a) Muscle and function: Posterior Tibialis works to decelerate pronation and leg rotation.

Single Limb Support

1) Mid-Stance: The body progresses over the foot in a controlled manner. The contralateral swing limb provides the momentum

a) Muscles and functions:
    i) Peroneus Longus (PL): Stabilizes and plantarflexes the first ray when foot supinates in late mid-stance and during heel lift at terminal stance
    ii) Peroneus Brevis (PB): Assists PL to transfer weight from lateral to medial side
2) Terminal Stance: The body progresses past the forefoot
a) Muscle and function: Posterior Tibialis accelerates subtalar joint supination and external leg rotation

Swing Limb Advancement

1) Pre-Swing: The foot remains on the floor. The knee rapidly flexes while weight is shifted to the other limb
a) Muscle and function: Gastrocnemius works to provide heel lift
2) Initial Swing: The thigh begins to advance; the knee continues to flex and the foot clears the ground
a) Muscles and functions: Anterior Tibialis, extensor hallucis, and digitorum longus; work to dorsiflex the foot so it can clear the ground. The hamstrings work to flex the knee and lift the foot off of the ground
3) Mid-Swing: The thigh continues to advance as the knee begins to extend. Foot clearance is maintained
a) Muscle and action: Quadriceps muscles work to swing leg forward
4) Terminal Swing: The leg reaches out to achieve step length
a) Muscles and actions:
    i) Hamstrings: Deceleration of leg and foot
    ii) Quadriceps: Lift and swing leg forward. Stabilize knee at heel contact

Examples of common problems in gait and the gait cycle:

1) Contact occurs directly on forefoot or flatfoot during weight acceptance. Weak tibialis posterior and quadriceps muscles can cause excessive forefoot contact and lead to excessive pressure on metatarsal heads

    Corrective action: Accommodative orthotic and rocker sole

2) Foot slap: usually there is some nerve involvement; muscles involved include tibialis anterior, calf and quadriceps. Occurs during weight acceptance and results in high forefoot pressure

   Corrective action: Rocker sole

3) Excessive inversion/supination: due to weak peroneals, most evident in single limb acceptance and support. Causes high pressure and ulcers on base of fifth metatarsal shaft and head

   Corrective action: Outflare sole, lateral post

4) Excessive eversion/pronation: weak posterior tibialis and sometimes anterior tibialis or soleus causes this in weight acceptance and single limb support phase. Associated with first and second metatarsal head breakdown and in advanced stages may include dropped navicular and associated ulcers

   Corrective Action: Straight last shoe, strong heel counter and medial post on foot orthotic

5) No heel lift: caused by weak calf muscles during single limb support phase

   Corrective action: Rocker sole

MECHANICS OF KNEE JOINT DURING STANDING AND WALKING:

The knee joins the thigh with the leg and consists of two joints: one between the femur and tibia (tibio femoral joint), and one between the femur and patella (patellofemoral joint). It is the largest joint in the human body. The knee is a modified hinge joint, which permits flexion and extension as well as slight internal and external rotation. The knee is vulnerable to injury and to the development of osteoarthritis.

It is often termed a *compound joint* having tibiofemoral and patellofemoral components.

STRUCTURE:

The knee is a modified hinge joint, a type of synovial joint, which is composed of three functional compartments: the patellofemoral articulation, consisting of the patella, or "kneecap", and the patellar groove on the front of the femur through which it slides; and the medial and lateral tibiofemoral articulations linking the femur, or thigh bone, with the tibia, the main bone of the lower leg. The joint is bathed in synovial fluid which is contained inside the synovial membrane called the joint capsule. The posterolateral corner of the knee is an area that has recently been the subject of renewed scrutiny and research.

The knee is the largest joint and one of the most important joints in the body. It plays an essential role in movement related to carrying the body weight in horizontal (running and walking) and vertical (jumping) directions.

At birth, the kneecap is just formed from cartilage, and this will ossify (change to bone) between the ages of three and five years. Because it is the largest sesamoid bone in the human body, the ossification process takes significantly longer.

Articular bodies:

The main articular bodies of the femur are its lateral and medial condyles. These diverge slightly distally and posteriorly, with the lateral condyle being wider in front than at the back while the medial condyle is of more constant width. The radius of the condyles' curvature in the sagittal plane becomes smaller toward the back. This diminishing radius produces a series of involute midpoints (i.e. located on a spiral). The resulting series of transverse axes permit the sliding and rolling motion in the flexing knee while ensuring the collateral ligaments are sufficiently lax to permit the rotation associated with the curvature of the medial condyle about a vertical axis.

The pair of tibial condyles are separated by the intercondylar eminence composed of a lateral and a medial tubercle.

The patella also serves an articular body, and its posterior surface is referred to as the trochlea of the knee. It is inserted into the thin anterior wall of the joint capsule. On its posterior surface is a lateral and a medial articular surface, both of which communicate with the patellar surface which unites the two femoral condyles on the anterior side of the bone's distal end.

Articular capsule:

The articular capsule has a synovial and a fibrous membrane separated by fatty deposits. Anteriorly, the synovial membrane is attached on the margin of the cartilage both on the femur and the tibia, but on the femur, the suprapatellar bursa or recess extends the joint space proximally The suprapatellar bursa is prevented from being pinched during extension by the articularis genus muscle. Behind, the synovial membrane is attached to the margins of the

two femoral condyles which produces two extensions similar to the anterior recess. Between these two extensions, the synovial membrane passes in front of the two cruciate ligaments at the center of the joint, thus forming a pocket direct inward.

Bursae:

Numerous bursae surround the knee joint. The largest communicative bursa is the suprapatellar bursa described above. Four considerably smaller bursae are located on the back of the knee. Two non-communicative bursae are located in front of the patella and below the patellar tendon, and others are sometimes present.

Cartilage:

Cartilage is a thin, elastic tissue that protects the bone and makes certain that the joint surfaces can slide easily over each other. Cartilage ensures supple knee movement. There are two types of joint cartilage in the knees: fibrous cartilage (the meniscus) and hyaline cartilage. Fibrous cartilage has tensile strength and can resist pressure. Hyaline cartilage covers the surface along which the joints move. Cartilage will wear over the years. Cartilage has a very limited capacity for self-restoration. The newly formed tissue will generally consist of a large part of fibrous cartilage of lesser quality than the original hyaline cartilage. As a result, new cracks and tears will form in the cartilage over time.

Menisci:

The articular disks of the knee-joint are called menisci because they only partly divide the joint space. These two disks, the medial meniscus and the lateral meniscus, consist of connective tissue with extensive collagen fibers containing cartilage-like cells. Strong fibers run along the menisci from one attachment to the other, while weaker radial fibers are interlaced with the former. The menisci are flattened at the center of the knee joint, fused with the synovial membrane laterally, and can move over the tibial surface.

The menisci serve to protect the ends of the bones from rubbing on each other and to effectively deepen the tibial sockets into which the femur attaches. They also play a role in shock absorption, and may be cracked, or torn, when the knee is forcefully rotated and/or bent.

Ligaments:

The ligaments surrounding the knee joint offer stability by limiting movements and, together with the menisci and several bursae, protect the articular capsule.

BLOOD SUPPLY:

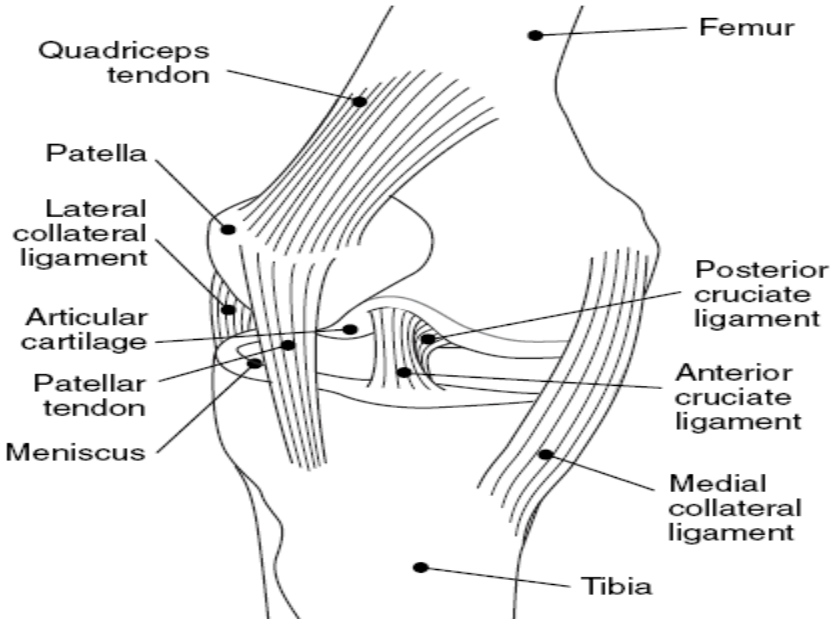
The femoral artery and the popliteal artery help form the arterial network or plexus, surrounding the knee joint. There are six main branches: two superior genicular arteries, two inferior genicular arteries, the descending genicular artery and the recurrent branch of anterior tibial artery.

The medial genicular arteries penetrate the knee joint.

MOVEMENT:

The knee permits flexion and extension about a virtual transverse axis, as well as a slight medial and lateral rotation about the axis of the lower leg in the flexed position. The knee joint is called

"mobile" because the femur and lateral meniscusmoveover the tibia during rotation, while the femur rolls and glides over both menisci during extension-flexion.



The knee permits flexion and extension about a virtual transverse axis, as well as a slight medial and lateral rotation about the axis of the lower leg in the flexed position. The knee joint is called "mobile" because the femur and lateral meniscusmove over the tibia during rotation, while the femur rolls and glides over both menisci during extension-flexion.

The center of the transverse axis of the extension/flexion movements is located where both collateral ligaments and both cruciate ligaments intersect. This center moves upward and backward during flexion, while the distance between the center and the articular surfaces of the femur changes dynamically with the decreasing curvature of the femoral condyles. The total range of motion is dependent on several parameters such as soft-tissue restraints, active insufficiency, and hamstring tightness.[23]

Extended position:

With the knee extended both the lateral and medial collateral ligaments, as well as the anterior part of the anterior cruciate ligament, are taut. During extension, the femoral condyles glide and roll into a position which causes the complete unfolding of the tibial collateral ligament. During the last 10° of extension, an **obligatory terminal rotation** is triggered in which the knee is rotated medially 5°. The final rotation is produced by a lateral rotation of the tibia in the non-weight-bearing leg, and by a medial rotation of the femur in the weight-bearing leg. This terminal rotation is made possible by the shape of the medial femoral condyle, assisted by contraction of the popliteus muscle and the iliotibial tract and is caused by the stretching of the anterior cruciate ligament. Both cruciate ligaments are slightly unwinded and both lateral ligaments become taut.

Flexed position:

In the flexed position, the collateral ligaments are relaxed while the cruciate ligaments are taut. Rotation is controlled by the twisted cruciate ligaments; the two ligaments get twisted around

each other during medial rotation of the tibia — which reduces the amount of rotation possible — while they become unwound during lateral rotation of the tibia. Because of the oblique position of the cruciate ligaments at least a part of one of them is always tense and these ligaments control the joint as the collateral ligaments are relaxed. Furthermore, the dorsal fibers of the tibial collateral ligament become tensed during extreme medial rotation and the ligament also reduces the lateral rotation to 45-60°.

EFFECTS OF VIBRATION :

There are 2 types of Vibrations,

1). Whole Body Vibration

2). Hand - Arm Vibration.

1). Whole Body Vibration (WBV) :

WBV caused by poorly designed or poorly maintained vehicles, platforms or machinery cause or other health effects such as,

- lower back pain [damage to vertebrae & discs, ligaments loosened from shaking].
- Motion Sickness
- Bone Damage
- Varicose Veins / Heart conditions (Variation in blood pressure from Vibration).
- Stomach and digestive conditions.
- Respiratory, Endocrine & metabolic changes.
- Impairment of Vision.
- Reproductive organ damage.

The longer a worker is exposed to Whole body Vibration, the greater the risk of health effects and muscular disorders.

2). Hand Arm Vibration [HAV] :

HAV long term exposure from using hand held tools such as pneumatic tools (eg. concrete breakers), chainsaws, grainders, etc. causes a range of conditions and diseases, including,

- White finger (also known as "dead finger") - damage to hands causing whiteness and pain in the fingers.
- carpel tunnel syndrome [Numbness or tingling in hands]
- Sensory nerve damage.
- Muscle and joint damage in the hands and arms.

These conditions and diseases can have very serious consequences for people. The effects can be permanently disabling even after a few years of uncontrolled exposure.

amage

Damage to the body from exposure to vibration depends on,

- Length of Exposure time.
- Frequency (rate at which the surface or tool vibrates, measured in vibrations/s or Hertz).
- Amplitude (the size of vibration). Amplitude can measure acceleration, speed or distance covered.

⇒ The main findings of the report were,

- Young workers were more likely to report vibration exposure than old workers.
- The industries workers had the highest exposure to vibration ~~were~~ than agriculture, forestry, fishing, transport and storage.
- 43 % of vibration - exposed workers were exposed to hand - arm vibration only, 38 % were exposed to whole body vibration only and 17 % were exposed to both hand arm and whole body vibration.