

pharmacology

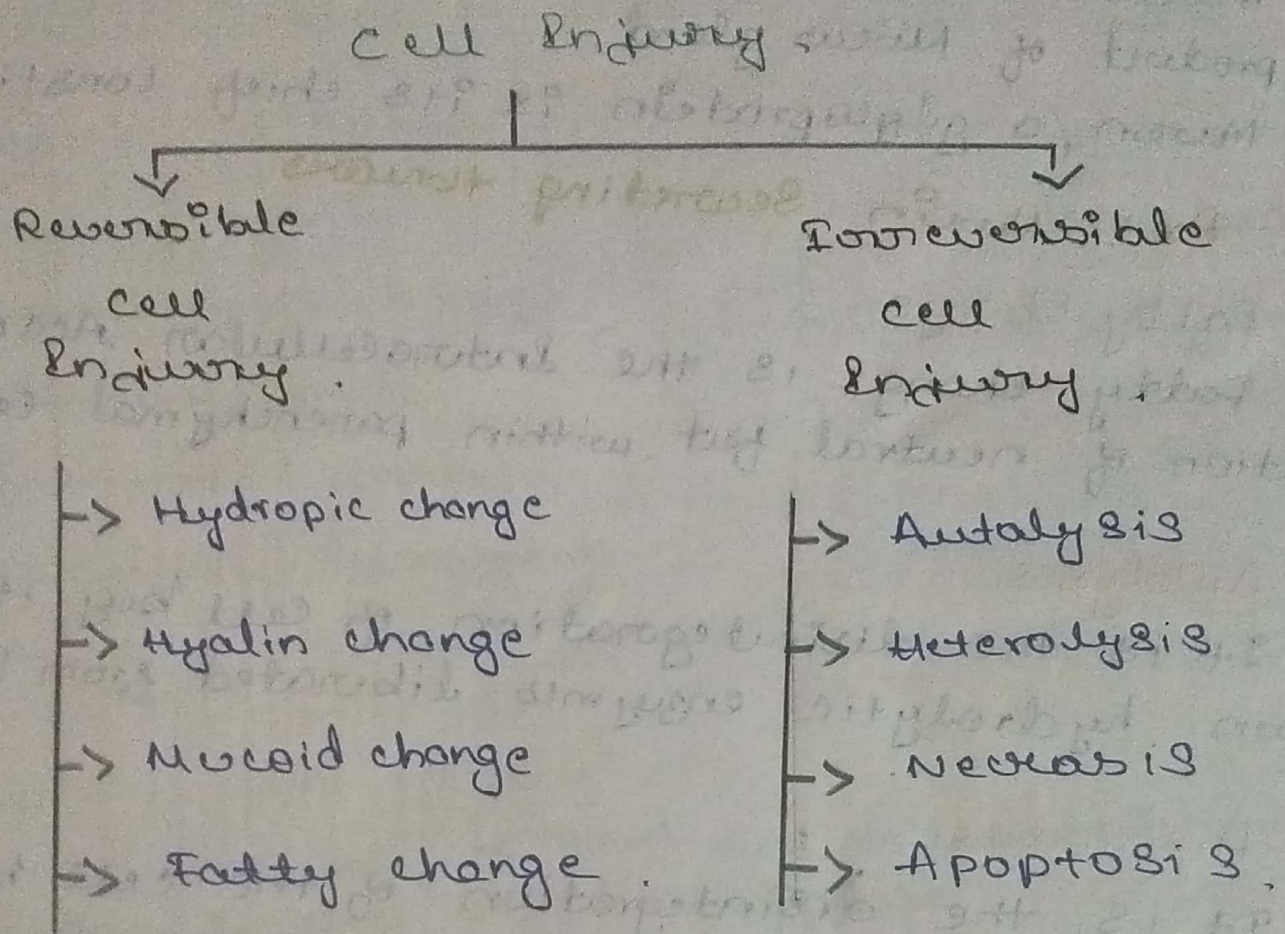
Roll No. _____ Subject PATHOLOGY School/College _____

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10 mark important concepts



- ① Explain the various types of cell injury with examples & discuss the Etiology of cell injury



→ Hydropic change

* Hydropic change means accumulation of water within the cytoplasm of cell

* Failure in sodium pump results in Accumulation of sodium which leads to increase in intracellular water to maintain iso-osmotic condition.

→ Hyalin change

* The word Hyalin means glassy, It doesn't refer to any specific substances.

* It is seen in Epithelial & Connective tissue
Ex: Hyalinised old scar of Fibrocollagenous tissue

→ mucoid change

* mucoid means mucus. It is a secretory product of Mucus gland.

* Mucin, a glycoprotein is its chief constituent

Ex: Mucin secreting tumours

→ Fatty change

* Fatty change is the Intracellular Accumulation of neutral fat within parenchymal cells

→ Autolysis

* It is the disintegration of cell by its own hydrolytic enzymes liberated from lysosomes

→ Heterolysis

* It is the disintegration of the cell by hydrolytic enzymes liberated from Inflammatory mediators like neutrophils.

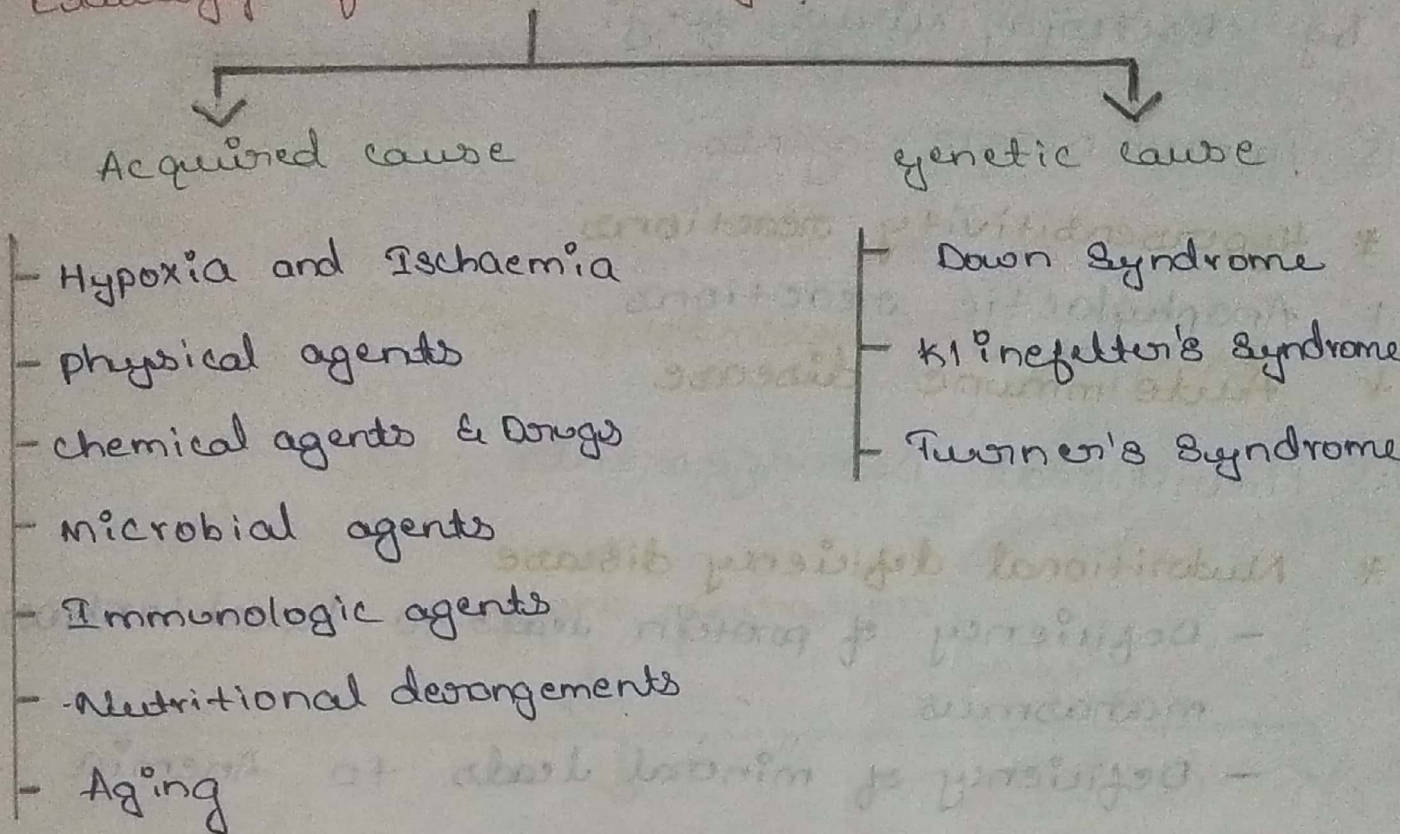
→ NECROSIS

* It is death of tissue followed by degradation of tissue by hydrolytic enzymes liberated from dead cells.

→ APOPTOSIS

* It is a programmed cell death

of cell injury - variety of stresses a cell encounters as a result of internal and external environmental changes
Etiology of Cell Injury / RCI / ICI.



① Hypoxia and Ischaemia

- * Reduced supply of blood due to interruption
- * Heart disease and lung disease
- * Disorder of oxygen carrying RBC (Anaemia, CO poisoning)

② Physical agents

- * Mechanical trauma (Road accidents)
- * Thermal trauma (by heat and cold)
- * Electricity
- * Radiation

③ Chemicals and drugs

- * Chemical poisons such as cyanide, arsenic, mercury
- * Strong acids and alkalis
- * Environmental pollutants
- * Insecticides and pesticides

④ Microbial agents

* Microbial Injury Includes Infection caused by Bacteria, virus, fungi

⑤ Immunologic agents

- * Hypersensitivity reactions
- * Anaphylactic reactions
- * Autoimmune disease

⑥ Nutritional Derangements

- * Nutritional deficiency disease
 - Deficiency of protein leads to Kwashiorkor, marasmus
 - Deficiency of mineral leads to Anemia
- * Nutritional Excess disease
 - It leads to heart disease, hypertension, atherosclerosis, obesity

⑦ AGING

- * Senescence leads to Impaired ability of the cells to undergo replication and repair and ultimately lead to cell death.

⑧ Down Syndrome

- * Down Syndrome is caused by trisomy 21 results in Intellectual disability.

⑨ Klinefelter's Syndrome

- * It is caused by sex chromosome trisomy (XXY) results in low testosterone level

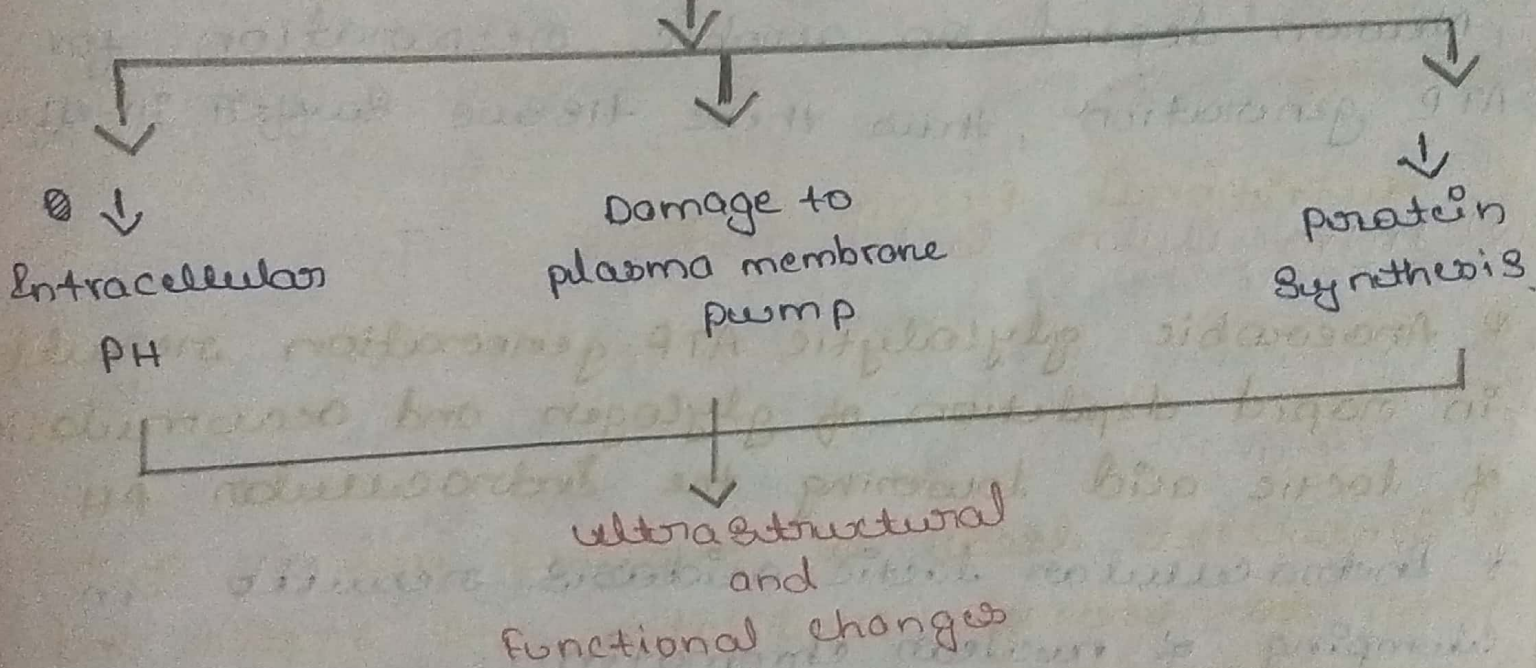
⑩ Turner's Syndrome

* It is caused by sex chromosome monosomy (XO), which results in Infertility, delayed puberty.

② Discuss the pathogenesis of reversible cell injury induced by hypoxia / Ischaemia

Ischaemia / Hypoxia.

↓
↓ ATP generation.



Reversible cell injury.

* If Ischaemia or hypoxia is of short duration, the effects may be reversible on rapid reoxygenation of circulation.

① Decreased ATP generation

- * In Ischaemia, aerobic respiration as well as glucose availability are both compromised resulting in cell injury.
- * Ischaemic cell injury also causes accumulation of metabolic waste.
- * In Hypoxia anaerobic glycolytic ATP generation continues, thus cell injury is less severe.
- * But highly specialised cells such as myocardium, Neuron depend on aerobic respiration for ATP generation, thus these tissues suffer ill-effects.

② Intracellular lactic acidosis

- * Anaerobic glycolytic ATP generation results in rapid depletion of glycogen and accumulation of lactic acid lowering the intracellular pH.
- * Intracellular lactic acidosis results in clumping of nuclear chromatin.

③ Damage to plasma membrane pump

- * ATP is required for generation of phospholipids from cellular fatty acids which are required for continuous repair of membranes.
- * Lack of ATP results in damage to membrane pump.

* Failure of Sodium potassium pump.

- Sodium pump allows active transport of Sodium out of the cell and diffusion of potassium into the cell.
- lowered ATP results in Intracellular accumulation of Sodium.
- accumulation of Sodium leads to Increase in Intracellular water to maintain Iso-osmotic condition. (i.e. hydropic swelling).

* Failure of Calcium pump.

- Calcium pump involves in exchange of Calcium across the cell membrane.
- Due to membrane damage Excess of Calcium move into the cell and causes swelling.

④ Reduced protein synthesis

- * As a result of continuous hypoxia, membrane of endoplasmic reticulum swells up.
- * Ribosomes are detached from rough endoplasmic reticulum results in Inactivation of their function.

⑤ Discuss the pathogenesis of Irreversible cell injury induced by hypoxia/ischaemia?

Irreversible cell injury.

- * persistent Ischaemia or hypoxia results in Irreversible damage to structure and function of the cell.

① Calcium Influx

- * As a result of continued hypoxia, large influx of calcium ions occurs
- * Calcium ions collect in the mitochondria thus disable its function

② Activated phospholipase

- * Increased calcium in the cell activates endogenous phospholipase, which in turn degrades phospholipids which is the main constituent of cell membrane

③ Activated proteases

- * Cytoskeleton which anchors the cell membrane is damaged by activated protease

④ Activated Endonucleases

- * DNA are damaged by the activated lysosomal enzyme such as endonucleases
- Irreversible damage can be in three forms

- pyknosis :- condensation & clumping of nucleus
- karyorrhexis :- Fragmentation of nucleus
- karyolysis :- Dissolution of the nucleus

⑤ Lysosomal hydrolytic enzymes

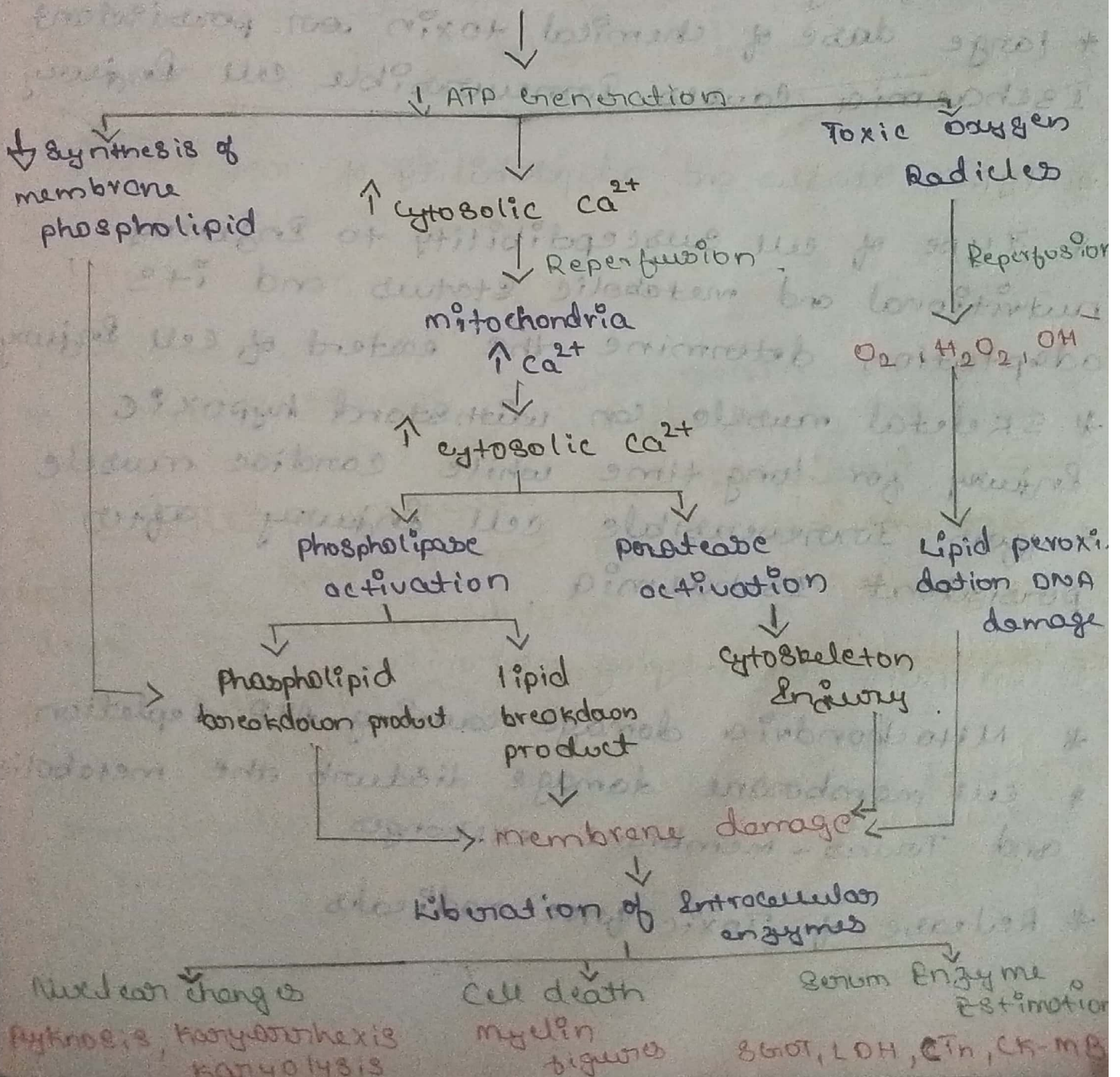
- * Lysosomal hydrolytic enzymes are activated due to lack of oxygen in the cell and acidic pH

* Hydrolytic enzymes on activation brings about digestion of cellular components & results in cell death.

* The dead cell is eventually replaced by masses of phospholipids called myelin figures

* Liberated Enzymes such as SGOT, LDH, cTn, CK-MB from membrane damage is used as clinical parameters of cell death.

ISCHAEMIA | HYPOXIA



④ Explain the pathogenesis of Cell Injury / RCT / IRI, factors influencing cell injury

① Factors pertaining to etiological agent & host

① Type, duration and severity of injurious agent

* The Extent of cell injury depend upon Type, duration and severity of the stimulus

* Small dose of chemical toxin on short duration of Ischaemia causes reversible cell injury.

* large dose of chemical toxin on persistent Ischaemia causes Irreversible cell injury

② Type, status and adaptability of target cell

* Type of cell, susceptibility to injury, nutritional and metabolic status and its adaptation determine the extent of cell injury

* skeletal muscle can withstand hypoxic injury for long time while cardiac muscle suffers Irreversible cell injury after persistent Ischaemia.

③ Common underlying mechanisms

* Mitochondria damage causing ATP depletion.

* cell membrane damage disturb the metabolic and Trans-membrane exchanges

* Release of Toxic free radicals

③ usual morphologic changes

- * morphologic changes of reversible cell injury appear earlier eg: hydropic swelling
- * later morphologic changes of Irreversible cell injury i.e cell death are seen eg. myocardial infarction

④ Functional Implications

- * The Interruption of blood supply (i.e. Ischaemia) and Impaired oxygen supply to the tissues i.e hypoxia are most common forms of cell injury in human beings

⑤ Explain the morphology of Reversible cell injury?

① Hydropic change

- * Hydropic change means accumulation of water within the cytoplasm of the cell
- * Swelling results from impaired regulation of sodium and potassium at the level of cell membrane.
- * It results in Intracellular accumulation of sodium.
- * Accumulation of sodium leads to increase in intracellular water to maintain isotonic condition.

② Hyaline change

* The word 'hyaline' means glassy. It does not refer to any specific substances.

* Intracellular hyaline: It is mainly seen in epithelial tissue.

Ex: Russell's bodies representing excessive immunoglobulins in the RER of plasma cells.

* Extracellular hyaline: It is mainly seen in connective tissue.

Ex: Hyalinised old scar of fibrocollagenous tissue.

③ MUCOID CHANGE

* Mucoid means mucus. It is a secretory product of mucous gland.

* Mucin, a glycoprotein, is its chief constituent.

* Mucin is produced by epithelial cells of mucous gland as well as by connective tissue, such as ground substance in umbilical cord.

Ex: Mucin secreting tumours (stomach, bowel)

④ Fatty change

* Fatty change is the intracellular accumulation of neutral fat within parenchymal cells.

Fatty liver.

* It is the common site for accumulation of fat because it plays central role in fat metabolism.

Etiology .

① condition Both Excess fat

① obesity .

① Diabetes mellitus .

② Liver cell damage

① Alcoholic liver disease

① Starvation .

* Microscopically characteristic feature is the presence of numerous lipid vacuoles in the cytoplasm of hepatocytes

⑥ Explain pathogenesis of acute inflammation ?

VASCULAR EVENTS

① Haemodynamic changes .

① Transient Vasoconstriction.

→ It is an Immediate Response characterized by vasoconstriction of only arterioles which decreases the amount of blood flowing through them

② progressive vasodilation.

→ It is seen in arterioles. It results in Increased blood flow which is the main reason behind redness and warmth.

③ changes in hydrostatic pressure.

* Due to persistent vasodilation, local hydrostatic pressure within the blood vessel increases

* This Results in escape of fluid from vessels into interstitial spaces, thereby causing Swelling .

(iv) Slowing of Microcirculation.

* Due to Exudation and transudation into the Interstitial spaces, blood becomes more concentrated. This increases its viscosity which leads to Slowing of microcirculation.

(v) Leucocyte Margination and Emigration.

* Leucocytes align along the periphery of blood vessels by a process known as margination.
* Leucocytes move into the extravascular space through the gaps present between the endothelial cells by the process known as emigration.

(2) Altered vascular permeability.

* vasodilation and elevated hydrostatic pressure are responsible for the escape of vascular fluid.
* However, later vascular permeability also causes oedema.
* The normal fluid balance is governed by
 (i) Force leading to outward movement
 (ii) Force leading to inward movement.
* Under normal condition, both inward and outward movement of the fluid is balanced.
* However, in case of Inflammation, the intra-vascular osmotic pressure decreases resulting in oedema.

CELLULAR EVENTS

(i) Exudation of WBC

(i) Margination and pavementing

* Exudation of plasma makes the peripheral layer to become thin and central layer to become wider

* Due to such redistribution, neutrophils migrate from centre towards the periphery of the endothelium. This process is known as pavementing.

(ii) Rolling & Adhesion of WBC's

* The neutrophils at the periphery roll along the endothelial lining of the blood vessels. This is known as Rolling phase

* These leucocytes adhere to endothelial lining by adhesion molecule, such as

(i) Selectins.

(ii) Immunoglobulins.

(iii) Emigration & Diapedesis.

* The adhered neutrophils destroy the basement membrane by releasing collagenases

* The neutrophils then move into the extravascular space through the damaged basement membrane. This process is known as Emigration.

* Due to increased hydrostatic pressure, RBCs also seep passively through the endothelial cell gaps. This is known as diapedesis.

(iv) Chemotaxis.

* The process of migration of leucocytes from the extravascular space to the site of injury along chemical gradient is known as chemotaxis.

* The agents which act as chemoattractants for WBC's are called chemotactic agents.

* Various chemotactic agents are

- Bacterial products
- Cytokines
- Platelet factor 4 (PF4)

(2) PHAGOCYTOSIS

* It is the process of engulfment of solid particles and cell debris by phagocytes.

* Phagocytic cells are two types

(i) Polymorphonuclear neutrophils (PMN's)
(microphages)

(ii) Monocytes. (macrophages)

(a) Recognition and Attachment stage

* Initial step is the recognition of bacteria by the phagocytes due to chemotactic substances released by bacterial products.

* Circulating phagocytes and bacteria are negatively charged, hence repel each other, to overcome this bacteria is coated by opsonins present in circulating serum.

⑥ Engulfment Stage.

* phagocytes engulf the opsonized bacterium by forming pseudopods around it.

* cell membrane of lysosome fuses with cell-membrane of phagocytes to form phagosome

⑦ Secretory Stage

* In this stage preformed granules which are stored in phagocytes are secreted into the phagosome, as well as Extracellularly.

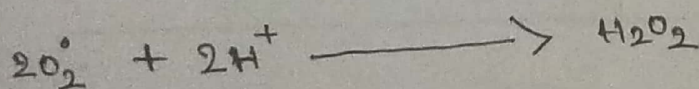
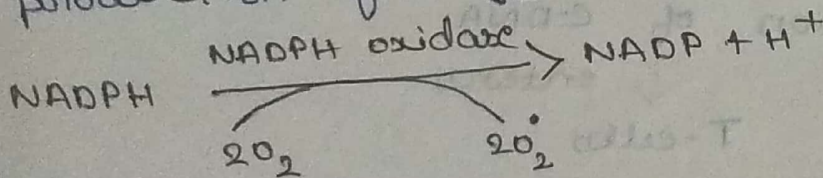
⑧ Degradation Stage.

* The engulfed bacteria is killed and degraded by antibacterial agents and hydrolytic enzymes

Mechanism of antimicrobial agents

(i) Oxygen - Dependent mechanism.

-> production of oxygen free radical.



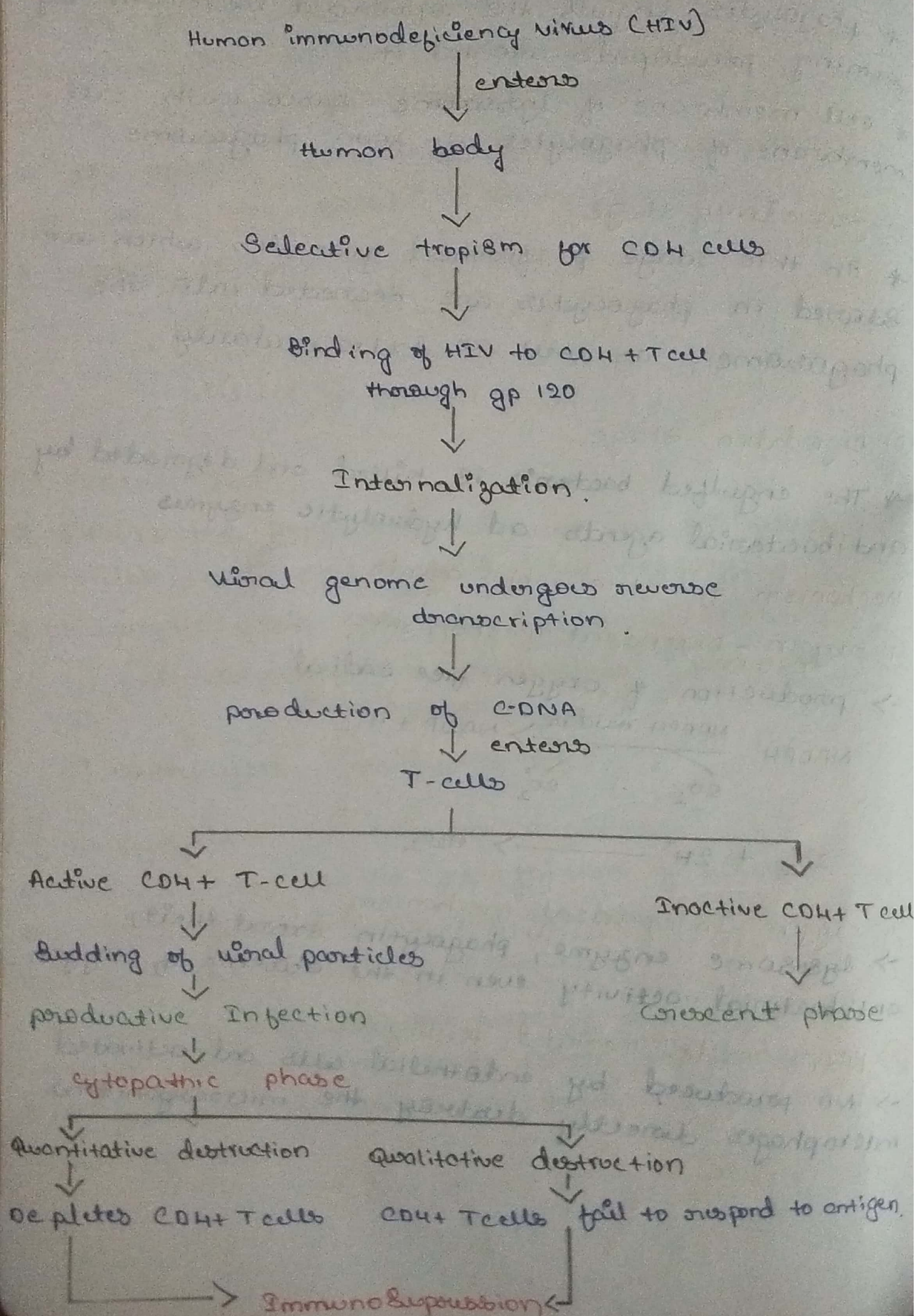
(ii) Oxygen - Independent mechanism

-> lysosome enzyme, phagocytin exert their antibacterial activity even in the absence of oxygen.

(iii) Nitric oxide mediated killing.

-> NO produced by endothelial cells and activated macrophages directly destroy the microorganisms.

① Discuss in detail the pathogenesis of HIV Infection



① what are the various chemical mediators of Inflammation?

CELL DERIVED MEDIATORS VLPEN

1. vasoactive amines

① Histamine

* Histamine is stored in the granules of mast cells, basophils and are released in response to substance which induce acute inflammation.

* Action of Histamine includes

- vasodilation
- Increased vascular permeability.
- Itching and pain

② Serotonin

* Serotonin is present in chromaffin cells of GIT, mast cells

* Action of serotonin includes

- vasodilation
- Increased vascular permeability.

2. Lysosomal components

* Two inflammatory leucocytes, neutrophils and monocytes contain lysosomal granules. These granules are released in response to injurious stimuli.

* Action of granule enzyme includes

- Destruction of engulfed bacteria.

3. Platelet Activating Factor

* It is released from mast cells, basophils, endothelium & platelets.

* Action of PAF Includes

- vasodilation
- Increased vascular permeability
- Bronchoconstriction
- Acts as chemotactic agent

4. Cytokines

* They are produced by lymphocytes and monocytes

(i) Interleukin-1 (IL-1) and $\text{TNF-}\alpha$

- $\uparrow$ Leucocyte adherence
- $\uparrow$ PGI Synthesis
- Fever, Anorexia, neutrophilia, shock

(ii) Tumour necrosis factor- β (TNF- β)

- $\uparrow$ Leucocyte adherence
- $\uparrow$ PGI Synthesis
- Fever, Anorexia, neutrophilia, shock

(iii) Interferon (IF)

- Activates macrophages and neutrophils
- Generates NO Synthase

(iv) chemokines

- Acts as chemottractant for Neutrophils, Monocytes

5. Nitric oxide and oxygen metabolites

* Nitric oxide is produced by action of nitric oxide Synthase on amino acid, arginine

* Function of Nitric oxide Includes

- vasodilation
- Antiplatelet activity
- microbicidal action

Arginine $\xrightarrow{\text{NO Synthase}}$ Nitric oxide

* Exposure of leucocytes to microbes results in extracellular release of superoxide anion (O_2^-), H_2O_2 & OH^-

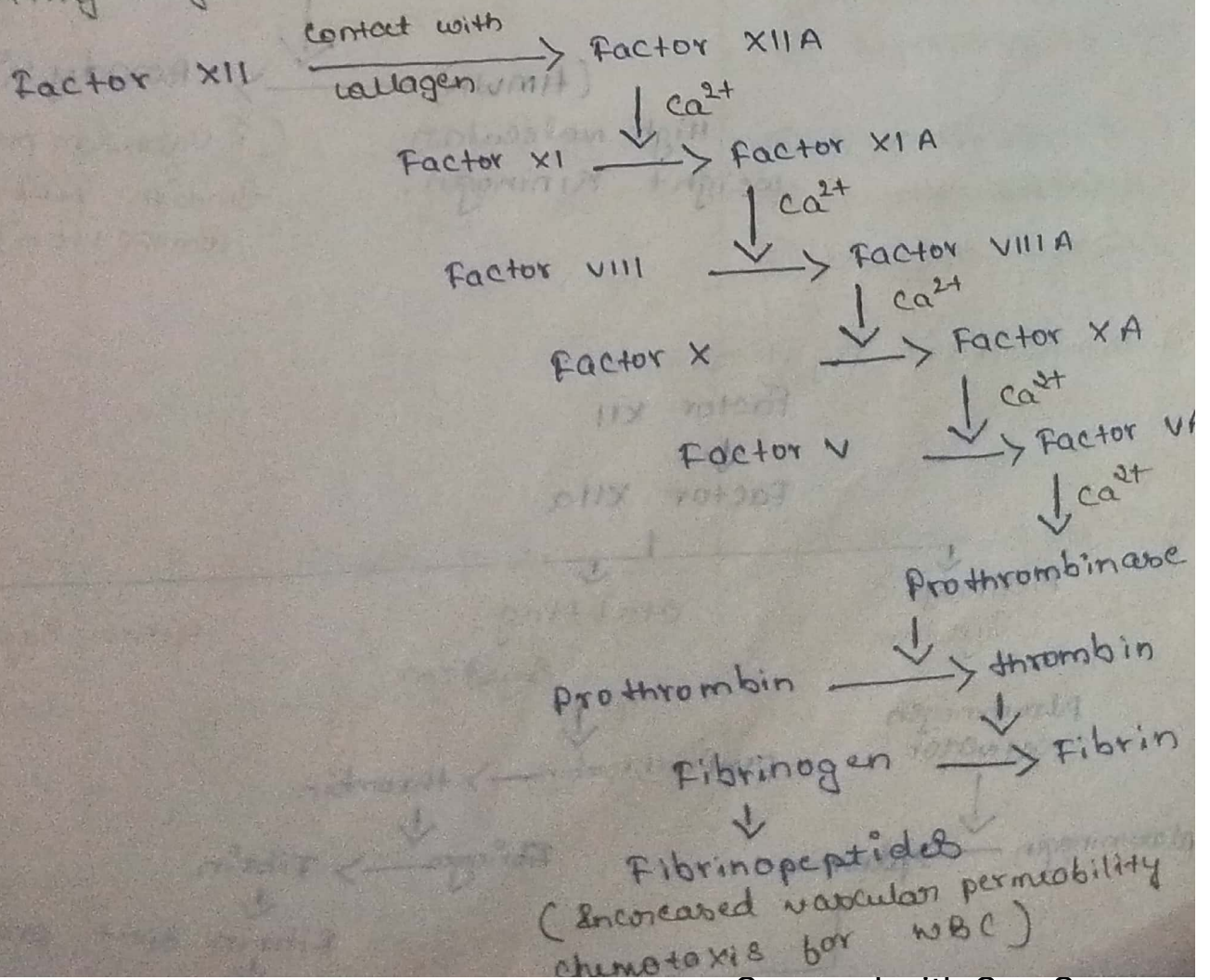
* These radicals bring about following inflammatory responses.

- > Increased vascular permeability.
- > Endothelial damage
- > Damages other cells.

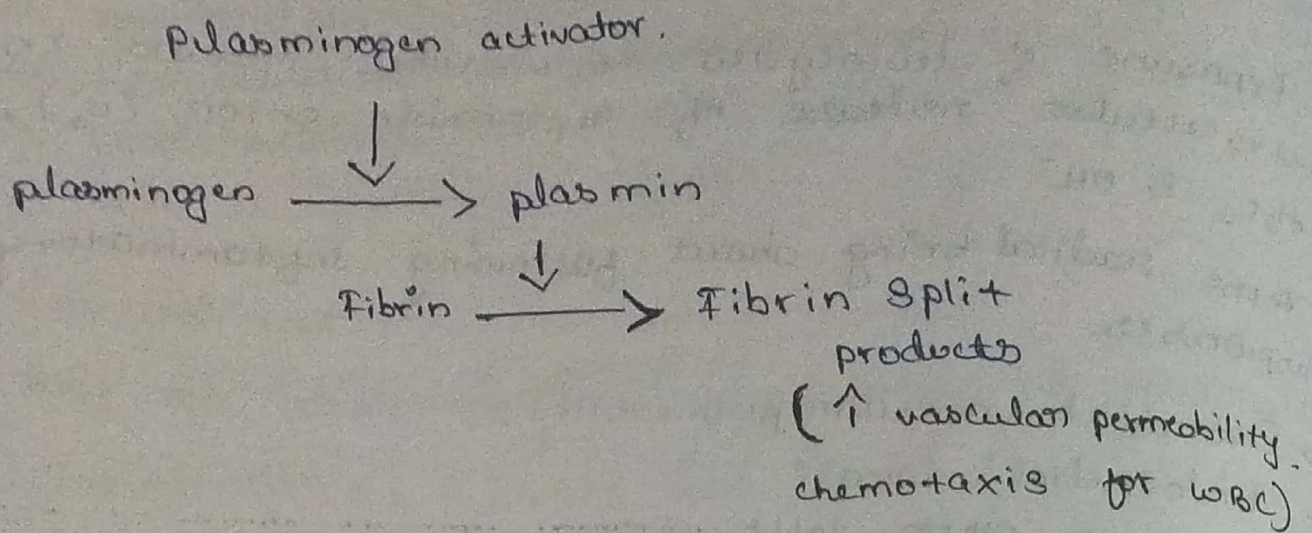
PLASMA DERIVED MEDIATORS

* These include various products derived from activation and interaction of factor linked system.

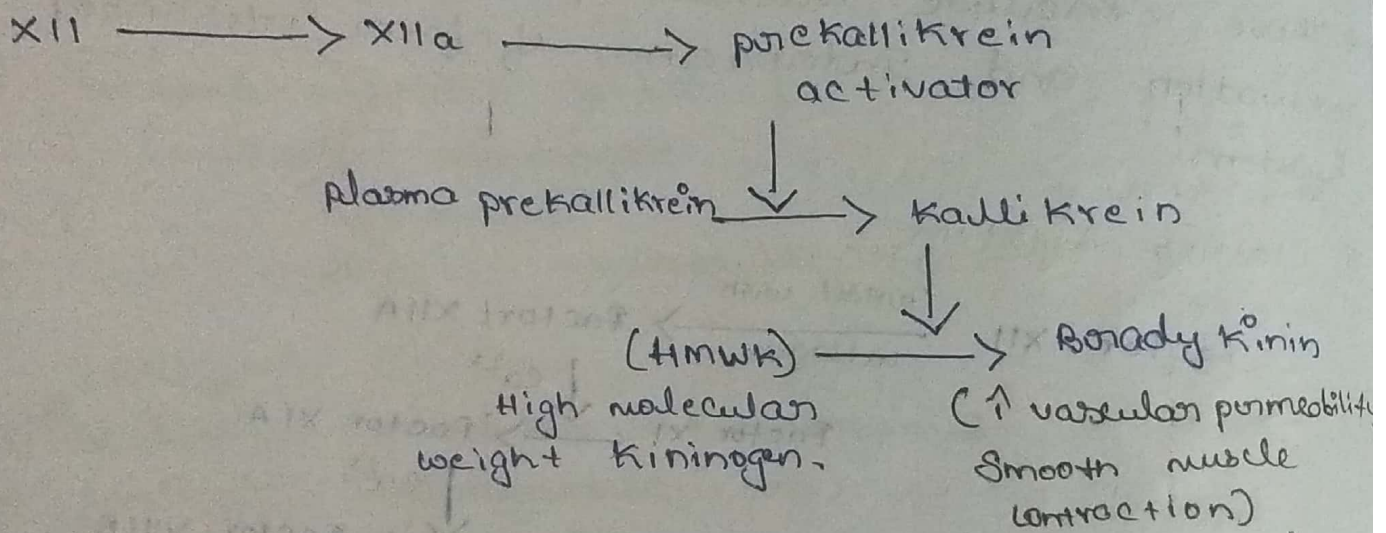
① Clotting system.



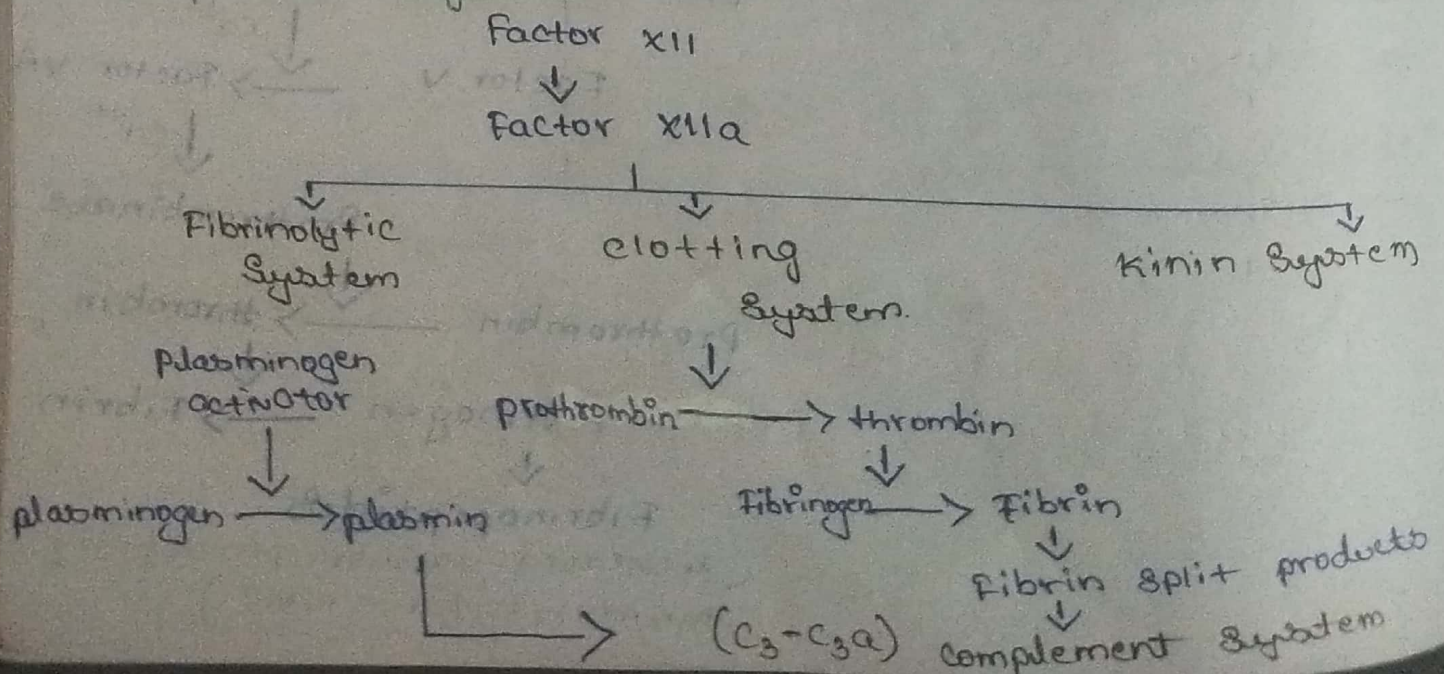
② Fibrinolytic System



③ Kinin System



④ Complement System



- * C₃ a being about following Inflammatory Responses
- ↑ vascular permeability and
 - causes Tissue oedema.
 - Facilitate phagocytosis
 - Induces mast cells to release histamine

C² MOOSO.

② Difference b/w Acute & Chronic Inflammation

Features	Acute Inflammation	Chronic Inflammation
① Causative agent	① pathogens ② Injured tissues	① Acute Inflammation ② Foreign bodies
② cells involved	① Monocytes ② macrophages	① monocytes ② macrophages ③ lymphocytes
③ mediators	① vasoactive amines ② Eicosanoids	① cytokines ② growth factors ③ oxygen species
④ onset	well defined	vague
⑤ Duration	Few days	months or years
⑥ Symptoms	prominent	Insidious
⑦ outcomes	Healing Chronic Inflammation	Tissue destruction Fibrosis

③ Explain the process of wound healing?

Healing.

It is the physiological response of the cell to injury to recreate the normal structure and function of the damaged tissue.

① Healing by primary Intention.

Healing by primary Intention occurs in wounds that are

- * Not prone to any Infection.
- * less tissue loss.

Steps involved.

① Haemorrhage.

- * Filling of Injured area with blood followed by clotting
- * Formation of blood clot is essential to protect the wound from Infection as well as dehydration.

② Inflammation.

- * Invasion of polymorphs to the site of Injury.
- * macrophages become active and replace the polymorphs

③ Epithelial cell proliferation.

- * Epithelial cells from the margin and from the base continue to divide and invade the Injured area until gap b/w the wound is filled.
- * Meanwhile, the granulation tissue formation begins and start filling the gap b/w the wound
- * This phase ends with stratification and keratinization of newly formed epidermal layer.

④ Granular Tissue formation

* Fibroblast proliferation and angiogenesis are the two events involved in formation of granular tissue.

⑤ Scar formation.

* The final step is the formation of scar and thinning of epidermis.

② Healing by Secondary Intention.

Healing by Secondary Intention occurs in wounds that

* Infected and open.

* Extensive tissue loss.

Steps involved

① Haemorrhage.

* Filling of injured area with blood followed by clotting

* Formation of blood clot is essential to protect the wound from infection as well as dehydration.

② Inflammation

* Invasion of polymorphs to the site of injury.

* Macrophages become active and replace the polymorphs.

③ Epithelial cell proliferation.

* Epithelial cells from the margin and from the base continue to divide and invade the injured area until gap b/w the wound is filled.

* meanwhile, the granulation tissue formation begins and start filling the gap b/w the wound.

* This phase ends with stratification and keratinization of newly formed epidermal layer

① Granular tissue formation
* Fibroblast proliferation and angiogenesis are the two events involved in formation of granular tissue

② Contraction of wound

* Contraction of wound is the characteristic feature of secondary union

* Wound contraction reduces the space by $\frac{1}{3}$ rd level.

* Wound contracts due to presence of myofibroblasts in the granulation tissue.

③ Scar formation

* The final step is the formation of scar and thinning of epidermis.

④ What are different types of hypersensitivity reaction? Describe all

- ① IgE mediated Hypersensitivity. Type-I
- ② Antibody mediated Hypersensitivity. Type-II
- ③ Immune-complex mediated Hypersensitivity. III
- ④ Delayed Type Hypersensitivity. Type-IV

① IgE mediated Hypersensitivity

* It is also referred as anaphylactic hypersensitive response

* Attack of Allergens results in production of antibody secreting plasma cells and memory cells

* Mast cells and basophils contain F_c receptors to which IgE binds.

* Exposure to allergen results in degranulation of mast cells & basophils

* Inflammatory mediators released by degranulation diffuse into the surrounding tissue

Components Involved in Type-1 hypersensitivity

② Allergens

* Some of the common allergens causing type 1 hypersensitive response include pollen, drugs

Eg: penicillins, Sulphonamides
Bee venom, Dust.

③ Reaginic Antibody

* These are IgE class of immunoglobulins formed against an allergen.

④ Mast cells and Basophils

* Mast cells and basophils contain F_c receptors to which IgE binds.

* Exposure to allergen results in degranulation of mast cells & basophils

* Inflammatory mediators released by degranulation diffuse into the surrounding tissue

Anaphylactic Reactions and their treatment

* The effect of inflammatory mediators is anaphylaxis, results from abnormal allergic response to a substance, resulting in itching, nausea, vomiting, sudden drop in blood pressure, wheezing.

* Anaphylactic reaction is treated by

→ prednisone (corticosteroid) in treatment of Asthma

→ Antihistamines are given to patients with Rhinitis & conjunctivitis

→ epinephrine (adrenaline)

② Antibody mediated hypersensitivity

* It is also known as cytotoxic hypersensitivity, and is mediated by IgM or IgG class of antibodies.

* Some examples of Type-II hypersensitivity are

ABO Transfusion reaction, Rh haemolytic disease.

ABO Transfusion Reaction.

* Blood Typing and transfusions are based on the alloantigens of human erythrocytes.

* Humans exhibit four different kinds of blood types A, B, AB, O.

* During blood transfusion blood matching must be accurately done

Ex: If a person with blood group A is given with blood group B, then B antibodies will stick to B antigen. Then RBCs of donated blood will clump together and clog the blood vessels.

Rh factor

- * Additional antigens present on surface of RBCs are called 'Rh' factor.
- * When Rh^{-ve} person receives blood from Rh^{+ve} person 'Rh' antigen of ' Rh^{+} ' blood triggers the production of 'Rh' antibodies.
- * A person with Rh^{+ve} blood can safely receive blood from Rh^{-ve} person.

③ Immune - complex mediated hypersensitivity

- * Immune complexes formed as a result of interaction b/w antigen and antibody.
- * Sometimes they are retained in the body and are deposited in tissues causing Immune complex disease.

④ Serum sickness

- * When a foreign antigen is injected, it binds with antibodies to form larger particles that are deposited in various tissues causing inflammation.
- * Serum sickness is characterized by fever, urticaria, lymphadenopathy and splenomegaly.
- * Allopurinol, barbiturates, captopril, quinidine, Sulphonamides associated with this type of Reaction.

⑤ glomerulonephritis

- * It is a type of kidney disease characterized by the inflammation of glomeruli of kidney.
- * This leads to impairment in the kidney's ability to filter urine.

③ Rheumatoid Arthritis

- * It is an autoimmune disease characterized by the presence of rheumatoid factor, IgM and IgG antibody.
- * The binding of antigen and antibody forms immune complexes that deposit on synovial membrane causing inflammation.
- * Treatment involves non-steroidal anti-inflammatory drugs like aspirin & immunosuppressive drugs.

④ Delayed Type Hypersensitivity

- * Antigen presenting cell (APC) ingest the antigen processes it and displace to class II MHC protein.
- * The T-helper cell binds to APC, gets activated and begins to produce cytokines.

⑤ Contact Hypersensitivity

- * Exposure of certain individuals to simple chemicals like nickel, nitrophenol, drugs like Sulphanamides and soaps results in contact hypersensitivity.
- * All these molecules penetrate the skin and act as haptens.
- * APC in the epidermal layer of skin take up the antigens & are carried to the lymph nodes for activation of T-cells.
- * T_H -cells secrete cytokines that result in flushing, itching and necrosis.

⑥ Tuberculin Type Hypersensitivity.

* Another familiar example of delayed type hypersensitivity is tuberculosis.

* The bacterial protein, tuberculin is injected intradermally and acts as antigen.

* patient suffering from tuberculosis or those who recovered from infection develop reddish swollen lesions at the site of injection caused by infiltration of macrophages.

⑤ Define Autoimmunity and classify autoimmune disease and describe the mechanism of Autoimmunity

Autoimmunity is the immune response towards body's own cells and tissues as a result of which autoimmune disease arise.

Types of Autoimmune disease.

① organ specific Diseases.

* In these type of diseases, autoantibodies formed react with antigens in a specific organ, and thereby causes its chronic inflammatory destruction.

② organ Non-specific Disease.

* In these type of diseases, autoantibodies formed react with antigen in many organs and thus cause systemic lesion.

Mechanism

① Defects in Immune System.

Ⓐ Cell Based defects.

- Reappearance of deleted lymphocytes that are capable of reacting against self antigens.
- Inactivation of suppressor T-cells
- Defects in functioning of helper-T-cells that can escape suppressor T-cells.

Ⓑ Foreign antigen complex with self-antigens, so that later it gets destroyed by the antibodies

Ⓒ Defects in MHC System.

② Defects in Somatic System.

- These includes production of abnormal antigens in normal cells due to cell injury.

③ Combined Immune and Somatic defects

- Autoimmune diseases arises as a result of defect in both autoantigens and abnormal immune reactivity.

④ What is allograft? what are various mechanism involved in the rejection of allograft.

Ans: In allograft, donor and recipient are of same species but are genetically non-identical. Due to genetic difference grafts undergo rejection.

① Direct pathway .

* In this pathway, CD8 and CD4 T-cells ~~of the~~ recipient recognize the alloantigenic MHC molecules ~~on APCs~~ of graft via their T-cell receptors.

* class I MHC antigens ~~on the APC's~~ of the graft are recognized by CD8⁺ cytotoxic T-cells of host and differentiate into CTLs. Activated CTL so formed damage graft cell.

* class II MHC antigens ~~on the APC's~~ of the graft are recognized by the CD4⁺ T cells which undergo proliferation and result in the production of cytokines like IFN- γ which increases vascular permeability and eventually damage graft tissue.

② Indirect pathway .

* In this pathway APC's of host pick up MHC molecule from the graft and presents to T-lymphocytes.

* It generates CD4⁺ T cells which gain access to the graft.

* In the graft these CD4⁺ T cells interact with the antigens.

* Activated CD4⁺ T cells in turn stimulates B lymphocytes which generate antibodies against the graft.

Direct pathway

Indirect pathway

MHC molecule

(class I)

MHC molecule

(class II)

MHC molecule

$CD8^+$

$CD4^+$

$CD4^+$

CTLs

T

Cytokines

Hypersensitivity
Reaction

Activated
Macrophages

Apoptosis

Necrosis

Endothelial cell injury.

B lymphocytes

Recognition
of
antigens

Antibodies
released

5 mark important concepts 

① Discuss various stages of shock / mechanism involved

(stages of shock)

- ① non progressive compensated shock
- ② progressive decompensated shock
- ③ Irreversible decompensated shock

① non-progressive compensated shock

* In the early stage of shock, an attempt is made to maintain adequate cerebral and coronary blood supply by redistribution of blood. So the vital organs are adequately perfused and oxygenated. This is achieved by)

i) widespread vasoconstriction. ✓

* In response to reduced blood flow and tissue anoxia, the neural and humoral factors for eg:- baroreceptors, chemoreceptors, catecholamines, and Angiotensin-II are activated. All these bring about vasoconstriction.

ii) Fluid conservation by the kidney. ✓

* In order to compensate the fall of blood volume in hypovolemic shock following factors assist in restoring the volume.

- Release of Aldosterone by activation of RAAS
- Release of ADH
- Reduced GFR due to arteriolar constriction

iii) Stimulation of adrenal medulla. ✓

* In response to low cardiac output adrenal medulla is stimulated to release excess of catecholamines (epinephrine & non-epinephrine) which increases heart rate.

② progressive decompensated shock

- * This the stage where patient suffer from pre-existing cardiovascular and lung disease beside persistence of the shock
- * Its effects due to tissue hypoperfusion includes the following -

① pulmonary hypoperfusion ✓

- * Decompensated shock worsens pulmonary perfusion and increases vascular permeability resulting in tachypnoea and adult Respiratory distress syndrome (ARDS).

② Tissue Ischaemia ✓

- * Tissue Anoxia causes metabolic lactic acidosis due to anaerobic glycolysis.
- * Lactic acidosis lowers tissue pH which inturn makes vasoconstrictor response ineffective this results in vasodilation and peripheral pooling of blood.

③ Irreversible decompensated shock

- * when the shock is so severe that inspite of compensatory mechanism despite therapy no recovery take place, It is called as Irreversible shock. Its effects due to widespread cell injury include the following

① progressive vasodilation ✓

- vasodilation results in peripheral pooling of blood which further deteriorate the effective circulating blood volume.

(ii) Increased vascular permeability. ✓

* Anoxic damage to tissue releases inflammatory mediators which causes increased vascular permeability.

* This results in escape of fluid from circulation into the interstitial tissue, thus deteriorate effective circulating blood volume.

(iii) Myocardial depressant factor (MDF). ✓

* Release of Myocardial depressant factors results in further depression of cardiac function, reduced cardiac output and decreased blood flow.

(iv) Hypercoagulability of blood. ✓

* Tissue damage in shock activates coagulation with release of clot factor, thromboplastin and ADP, which leads to slowing of blood stream and vascular thrombosis.

Clinically at this stage patient has features of coma, worsened heart function, and renal failure due to acute tubular necrosis.

② Discuss the etiology and pathogenesis of shock

Etiology of shock

① Hypovolaemic shock. ✓

* Acute haemorrhage ✓

* Dehydration from vomiting, diarrhoea.

* Burns ✓

* Excessive use of diuretics ✓

② Cardiogenic shock ✓

(i) Deficient emptying

- * Myocardial Infarction ✓

- * Cardiomyopathies ✓

- * Rupture of heart, ventricles ✓

- * Cardiac arrhythmias ✓

(ii) Deficient filling

- * Cardiac tamponade ✓
from haemopericardium

(iii) Obstruction to outflow

- * Pulmonary embolism ✓

- * Aortic valve thrombosis ✓

③ Septic shock ★

Mechanism: Release of inflammatory mediators

(i) Gram negative Septicaemia ✓

Infection with E. coli and pseudomonas

(ii) Gram positive Septicaemia ✓

Infection with streptococci, pneumococci

④ Other types

④ Traumatic shock ✓

- * Severe Injury

- * Surgery with marked blood loss

⑤ Neurogenic shock ✓

- * Cervical spinal cord injury

- * Head Injury

⑥ Hypoadrenal shock ✓

- * Administration of high doses of glucocorticoids

- * Secondary adrenal insufficiency

pathogenesis.

All forms of shock involves following 3 derangements

① Reduced effective circulating blood volume

* by actual loss of blood volume as occurs in hypovolaemic shock.

* by decreased cardiac output without actual loss of blood (normovolaemia), as occurs in cardiogenic shock and septic shock.

② Impaired tissue oxygenation.

* Reduction in effective circulating blood volume decreases venous return to the heart resulting in decreased cardiac output.

* consequently it causes reduced supply of oxygen to the organs and tissues and hence tissue anoxia, which results in cellular injury.

③ Release of inflammatory mediators.

* In response to cellular injury, Innate Immunity of the body gets activated as a body defense mechanism and release inflammatory mediators but eventually these agents themselves become the cause of cell injury.

③ Explain the mechanism of cardiogenic shock

- * Acute circulatory failure with sudden fall in cardiac output from acute disease of the heart without actual reduction of blood volume (normovolaemia) results in cardiogenic shock

Etiology of cardiogenic shock

(i) Deficient emptying

- * Myocardial Infarction.
- * Cardiomyopathies
- * Rupture of heart ventricle.
- * Cardiac arrhythmias

(ii) Deficient filling.

- * Cardiac tamponade from haemopericardium

(iii) Obstruction to outflow.

- * pulmonary embolism.
- * Ball valve thrombus.

pathogenesis

- * cardiogenic shock results from a severe left ventricle dysfunction from various causes

- * The resultant decreased cardiac output has its effects in the form of decreased tissue perfusion and movement of fluid from pulmonary vascular bed into pulmonary interstitial space and alveolar space.

Cardiogenic shock



↓ cardiac output



↓ blood flow



↓ supply of oxygen



Anoxia.



Inflammatory
mediators



SHOCK

- (i) Deficient emptying
- (ii) Deficient filling
- (iii) obstruction to outflow

(ii) Discuss the mechanism of Hypovolemic shock and its management.

* It is a form of shock results from inadequate circulatory blood volume due to loss of red cell mass and plasma from haemorrhage.

Etiology of Hypovolemic shock.

- * Acute haemorrhage
- * Dehydration from vomiting, diarrhoea
- * Burns.
- * Excessive use of diuretics

Pathogenesis

* The major effects of hypovolaemic shock are due to decreased cardiac output and low intracardiac pressure.

* Severity of clinical features depends upon degree of blood volume lost

$\leq 1000 \text{ ml}$:- compensated

$1000 - 1500 \text{ ml}$:- mild

$1500 - 2000 \text{ ml}$:- moderate

$> 2000 \text{ ml}$:- Severe

* Clinical features Includes tachycardia, hypotension, low urinary output, alteration in mental state.

Hypovolaemic shock, $\longrightarrow$ $\downarrow$ Effective circulating Blood volume

Haemorrhage

Trauma

Surgery

Burns

Dehydration

$\downarrow$ venous return

$\downarrow$ cardiac output

$\downarrow$ Blood flow

$\downarrow$ Supply of oxygen

Anoxia

Inflammatory mediators

Shock

management

- ① Resuscitation
- ② Immediate control of bleeding
- ③ Extracellular fluid replacement
- ④ Drugs

- * Chronotropic agents
- * Inotropic agents

⑤ Explain the end organ changes involved in shock

① Hypoxic Encephalopathy

* If blood pressure falls below 50 mmHg, Brain suffers from severe Ischemic damage with loss of cortical function, coma & vegetative state.

② Heart in shock

* Haemorrhage and necrosis :- There may be large Ischaemic areas located in subepicardial and subendocardial region.

* Zonal lesions :- These are opaque contraction bands in cardiac myocytes.

③ Shock Kidney

* Irreversible renal injury

* Renal Ischaemia

* Acute tubular necrosis

* End result is Anuria & death

④ Adrenals in shock

- * Release of aldosterone in response to hypoxic kidney.
- * Release of glucocorticoids from adrenal cortex and catecholamines like adrenaline from adrenal medulla.

⑤ Shock lung

- * Adult Respiratory Distress Syndrome (ARDS) are seen
- * changes include congestion, interstitial and alveolar edema, etc.

⑥ Mechanism of Free radical induced cell injury.

① Lipid peroxidation

- * Oxygen free radicals attack the polyunsaturated fatty acids (PUFA) of the cell membrane leading to the formation of extremely destructive PUFA radical such as lipid hydroperoxy & lipid hypoperoxide radical

- * Lipid peroxides propagated to other sites and causes membrane damage

- * Lipid peroxides are destroyed by transition metals like Iron

② Oxidation of proteins

- * Free radical bring about degradation of cytosolic proteases
- * cell destruction by inducing oxidation of cellular protein, cross linking amino acids and by fragmentation of polypeptide chains.

③ DNA Damage

- * Free radical damage the nuclear and mitochondrial DNA by breaking the double stranded DNA into single strands resulting in cell injury.

④ Cytoskeleton Damage

- * Free radical react with cytoskeletal element and interfere with ATP generation causing in mitochondria. This results in ATP depletion & cell damage.

⑦ What are the various biochemical intra cellular accumulation found in cell injury?

① Accumulation of lipids

- * Fatty change is the intracellular accumulation of neutral fat within parenchymal cells
- * In Fatty liver, liver is the common site for accumulation of fat because it plays central role in fat metabolism.

② Accumulation of protein

- * In proteinuria, there is excessive renal tubular reabsorption of proteins by proximal tubule which show pink hyaline droplets in cytoplasm.
- * The cytoplasm of plasma cells shows pink hyaline inclusions called Russell bodies representing immunoglobulins.

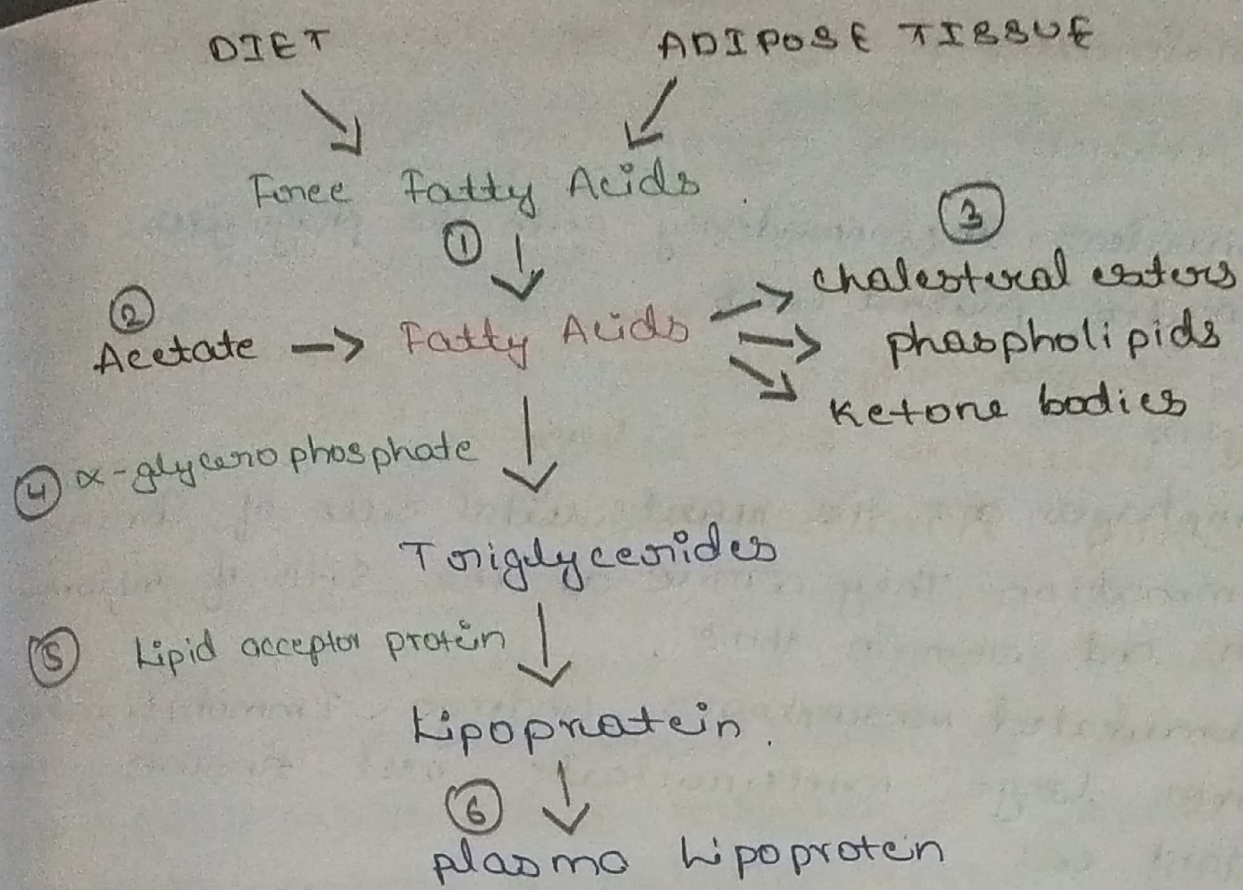
③ Accumulation of glycogen

- * In diabetes mellitus, there is intracellular accumulation of glycogen because normal cellular uptake of glucose is impaired.
- * In glycogen storage disease there is defective metabolism of glycogen due to genetic disorders.

④ Accumulation of pigments

- * Exogenous pigments like carbon, coal dust when inhaled, it is picked up by alveolar macrophages and transported to lymph nodes.
- * Endogenous pigments are derivatives of haemoglobin and amino acids.

⑤ Explain the role of lipid metabolism in the pathogenesis of fatty liver



→ In Fatty liver, Intracellular accumulation of triglycerides can occur due to following 6 steps

- * Increased entry of free fatty acids into liver.
- * Increased synthesis of Fatty acids by the liver.
- * Decreased conversion of fatty acids to ketones.
- * Increased α -glycerophosphate causing increased esterification of Fatty acids to Triglycerides.
- * Decreased synthesis of 'lipid acceptor protein' resulting in decreased formation of lipoprotein from triglycerides.
- * Block in the Excretion of lipoprotein from liver to plasma.

⑨ Explain the pathogenesis of chronic Inflammation

① Infiltration with mononuclear cells

* Mononuclear inflammatory cells are phagocytes
lymphocytes, plasma cells.

phagocytes

* Macrophages are the most vital cells of chronic inflammation. They arrive at the site of inflammation and remain there.

* Accumulated macrophages undergo immobilization to form large multinucleate cell known as giant cell

* Activated macrophages secrete acids, complement components (C_1 - C_5), coagulation factors

* All these components are involved in tissue destruction, repair which comprises fibrosis, neo vascularization etc.

Lymphocytes

* During chronic inflammation T & B lymphocytes migrate to site of inflammation in response to chemotactic agents

* Substance released from activated macrophage activates T-lymphocytes. This process leads to excessive tissue destruction.

plasma cells

* These are end product of B cell activation. Plasma cells produce antibodies which are directed against antigens at the site of inflammation.

② Necrosis

* It is the characteristic feature of chronic inflammation

* It occurs due to activated macrophages which releases tissue destroying substance like acids, complement components, coagulation factor reactive oxygen species

③ Proliferative changes

* In order to overcome the effect of necrosis body initiates the proliferation of blood vessels and fibroblasts. This results in formation of inflammatory granulation tissue which eventually gets healed.

⑩ Explain pathology of obesity?

* Adipose tissues are found in subcutaneous layer of the skin, around kidneys, heart, joints

* Functions include storage of fat, reduce heat loss shape to body organs, support & protect organs from injury.

* Adipocytes are also involved in releasing endocrine regulating molecules like leptin (energy regulating hormone), angiotensin, TNF- α and Interleukin-6

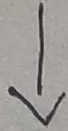
Increase in the number of adipocytes

or

Increase in the size of adipocytes

or

Increase in Intracellular lipid deposition



ob gene and its protein product leptin

&

db gene and its protein product leptin receptor
have been identified



These genes play a role in altering
eating and physical activities that leads
to obesity -

① Etiology and metabolic changes of obesity?

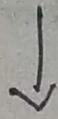
Etiology

- * Sedentary lifestyle
- * Diet rich in fat, carbohydrates
- * Excess eating
- * Genetic factors
- * Hypothyroidism
- * Insulinoma

metabolic changes

① Type-2 Diabetes mellitus

Excess fat



Antilipolytic action of Insulin
is inhibited

High level of
free fatty acid



Development of Insulin
resistance in liver & muscle



Increased gluconeogenesis
& decreased glucose uptake



Increased blood glucose level.

② Hypertension

* Hypertension in obese people is perhaps due to sodium retention, Increased blood vol, vascular resistance & cardiac output.

③ Cholesterol

- * hypercholesterolemia increases with increase in BMI
- * obese peopl have high level of LDL, HDL etc.

④ cholelithiasis

Due to Increase in total cholesterol, the incidence of cholelithiasis (gall stones) in obese pepl is 6 times higher than normal weighed pepl.

① Explain ^{other} granulomatous inflammation

- * chronic granulomatous inflammation is characterized by the formation of granulomas which are small lesions composed of epithelioid cells at centre and lymphoid cells at periphery.
- * Epithelioid cells are modified macrophages

other components of granuloma.

- * giant cells
 - granuloma also show the presence of giant cells
 - giant cells are formed by the fusion of epithelioid cells and contain many nuclei.
 - giant cells have weak phagocytic activity but produce certain secretory products which aid in expelling foreign agents.

* Necrosis

- Necrosis is the common feature of certain granulomatous condition.

* Fibrosis.

- This results due to proliferation of fibroblasts at the periphery of granulomas

Factors favouring formation of granulomas

- ① presence of irritants such as talc particles or organisms like m. tuberculosis, which are rarely digested.
- ② Hypersensitivity.

② discuss the factors affecting wound healing?

① Local factors

- * presence of Infections.
- * foreign agents
- * Ionizing Radiations.
- * Movement of Injured area.
- * poor blood Supply.

② Systemic factors.

- * Age.
- * glucocorticoids.
- * uncontrolled diabetes
- * malignant disease.
- * obesity.
- * malnutrition.
- * presence of Infection.
- * haematological disturbances
- * anaemia.
- * vitamin deficiency.
- * Steroids.

③ what is healing by granulation tissue formation
In this phase following events takes place.

① By the end of third day.

- * Fibroblasts start invading the Injured area
- * presence of collagen fibrils at Injured area
- * thickening of epithelial layer of epidermis.

- (i) By the end of fifth day.
- * Filling of Enclosed area by granulation Tissue.
 - * Angiogenesis.
 - * Bridging of collagen fibrils
 - * Restoration of normal thickness of epidermis.
 - * Keratinisation & stratification of epidermis.

- (ii) By the end of second week.
- * Replication of fibroblasts
 - * Disappearance of vascular channels.

- (iii) By the end of first month.
- * Formation of Scar filled with connective tissue.
 - * gradual Increase in wound strength.

- (4) write a note on MHC antigen
- * Histocompatibility molecules are glycoproteins expressed on the surface of all vertebrate cells
 - * No two individuals will have similar set of histocompatibility molecules, This causes rejection of transplants.
 - * In humans histocompatibility molecules are called as Human Leukocyte antigen.

Types of MHC molecules.

- (i) class - I histocompatibility molecule
- * It is expressed on surface of all nucleated cell except RBC and cells of CNS.

② class-II histocompatibility molecule.

* These molecules are expressed on only certain types of cells like macrophages, B lymphocytes, dendritic cells

* $CD8^+$ T-cells can bind only to class I histocompatibility molecule, $CD4^+$ T-cells can bind only to class-II histocompatibility molecule.

Role of MHC proteins

① organ Transplantation.

* MHC antigen cause very strong Immune response, during transplant rejection.

② Regulation of Immune System

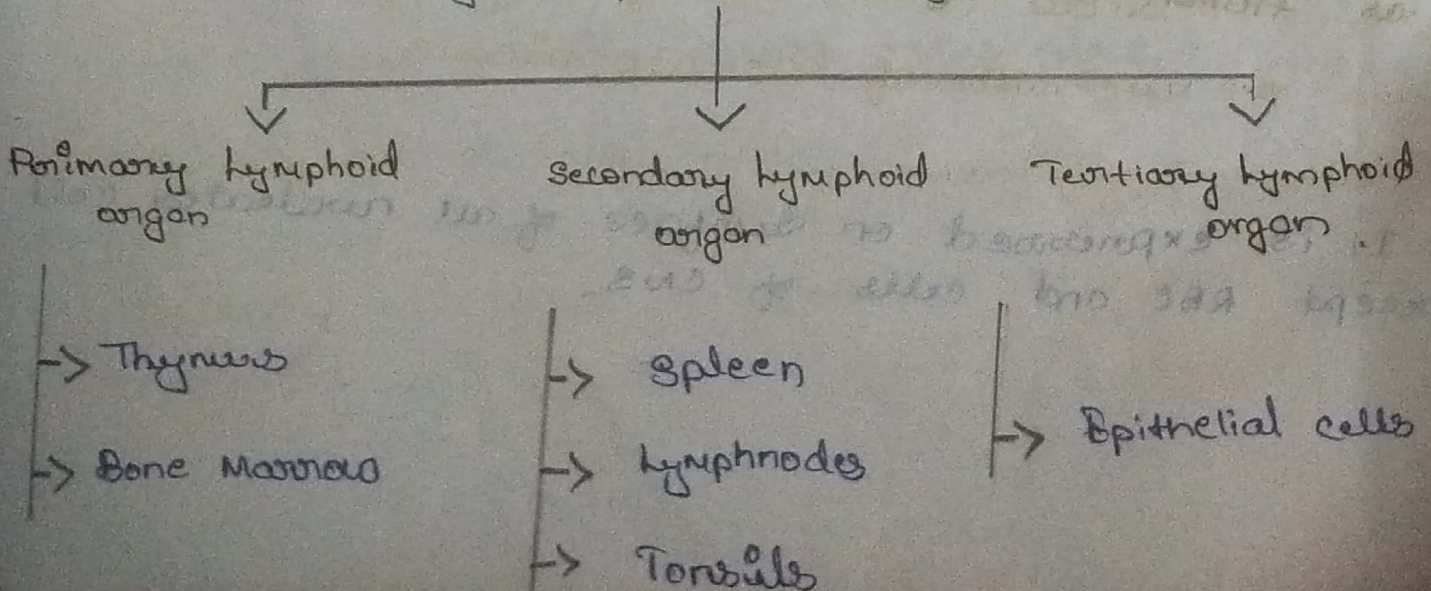
* Class I and class II MHC antigen play vital role in regulating both cellular and humoral Immunity.

③ MHC Associated Diseases

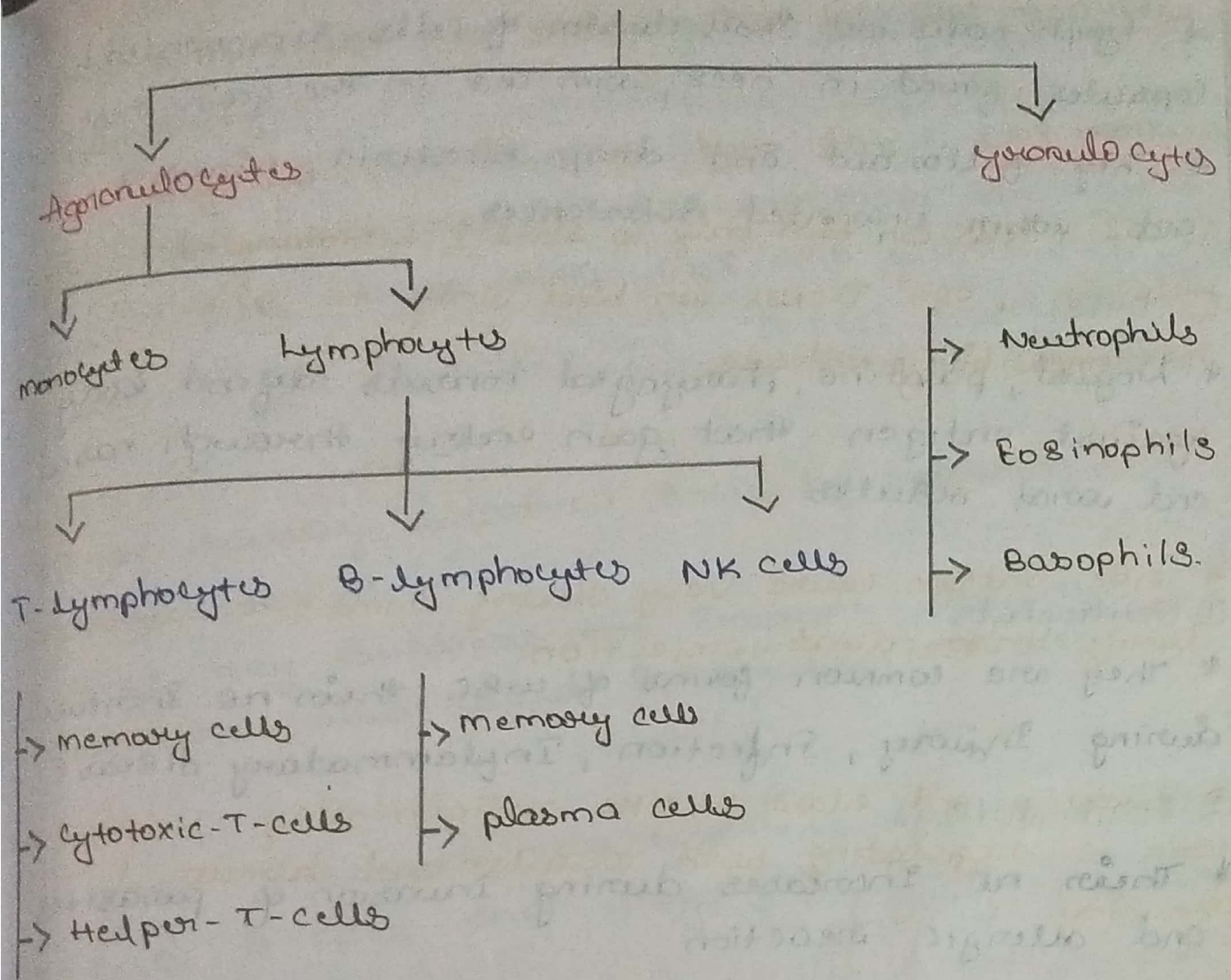
* Diseases associated with MHC include auto-Immune diseases, Inflammatory disease.

⑤ Briefly discuss components of Immune System

organs of Immune System



cells of Immune System



① Thymus

* Immature Thymocytes produced in the bone marrow migrate via blood stream to the thymus and mature there.

② Bone marrow

* Bone marrow is the site of origin of all the cells by the process of haematopoiesis.

③ Spleen

* Spleen takes foreign antigens from the blood passing through it
* old RBC's are destroyed in this organ

④ Lymph nodes

- * Lymph nodes are small clusters of cells, surrounded by capsules found in neck, arm and in the groin region
- * They filter out and trap bacteria, viruses and other unwanted substances.

⑤ Tonsils

- * Lingual, palatine, pharyngeal tonsils defend body against antigen that gain entry through nasal and oral routes.

① Neutrophils

- * They are common forms of WBC, their no increases during injury, infection, inflammatory disease.

② Eosinophils

- * Their no increases during invasion of parasites and allergic reaction.

③ Basophils

- * Their no increases during allergic reaction.

① Monocytes / Macrophages

- * Macrophages are referred as Antigen presenting cells (APC)
- * Their job is to ingest foreign material and present to T cells & B cells

② T-lymphocytes

- * T-lymphocytes are responsible for cell-mediated immunity.
- * In thymus, cell develops both CD4 & CD8 markers
- * The cell which retain CD4 markers are called helper T-cells, those with CD8 are cytotoxic-T-cells
- * Both T_H and T_C cells have T-cell receptor.

③ Cytotoxic - T cells

- * These destroy abnormal cells by releasing powerful toxins.
- * Since they suppress the immune response, they are also called suppression - T-cell.

④ Helper - T-cells

- * These increases no of cytokines to activate T-cells, B-cells and phagocytic cells.

① B-lymphocytes

- * These are involved in antibody production and provides humoral immunity.

② plasma cells

- * These produces antibody which are released into blood, lymph.

③ Memory cells

- * These remain in circulation, whenever there is second infection, they divide and produce more memory cells

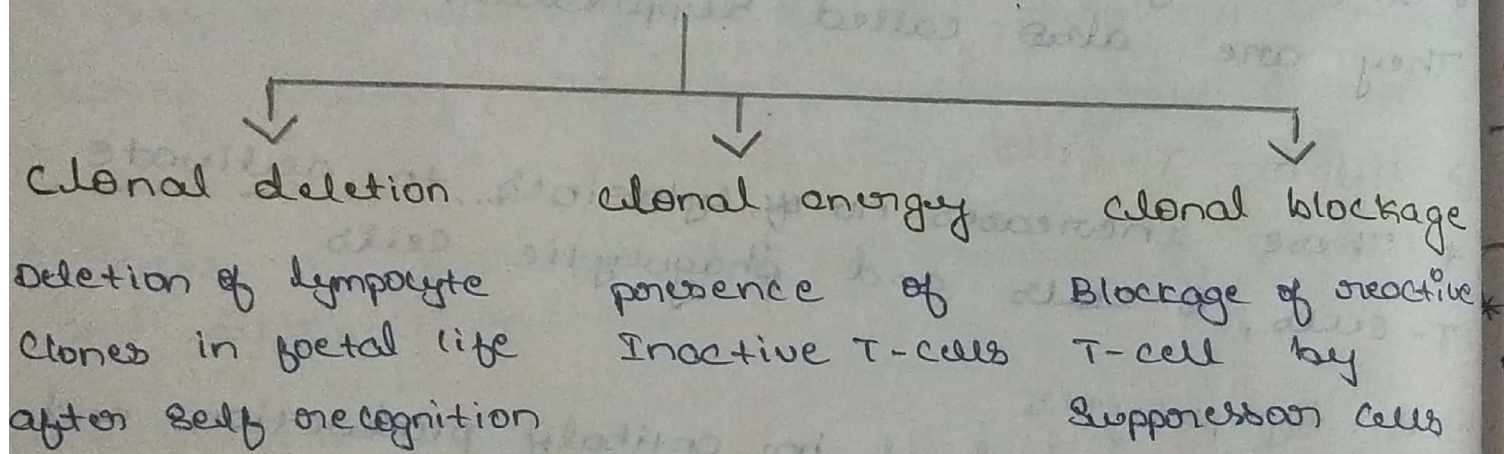
① NK cells

* All normal body cells contain class I MHC proteins on their plasma membranes, the complementary receptors are located on these NK cells.

* These cells bring about destruction of only those cells which lack or exhibit reduced amount of MHC proteins.

⑥ Briefly explain the mechanism of Immune tolerance.

Immune Tolerance



① Clonal deletion.

* During embryonic life, mature T cells & B cells form colonies. These colonies upon activation respond against body own antigen.

* Such lymphoid cells are selectively deleted, consequently, a state of immune tolerance develops.

② clonal Anergy.

- * Mature lymphocytes carry receptors on their surface that helps to identify antigens.
- * when surface receptors are lost, they remain inactive.

③ clonal Blockage.

- * Lymphoid T-cells neither deleted nor rendered inactive, but rather they are blocked by suppression T-cells

⑦ Difference b/w cell mediated & humoral Immunity.

Humoral

- * Adoptive Immunity mediated by antibodies produced by B lymphocytes
- * mediated by B cells
- * Antibody mediated Immunity
- * B cells secrete antibodies
- * Rapid onset
- * Antibodies travel through circulation
- * Acts on Extracellular microbes

cell mediated

- * Adoptive Immunity mediated by cytotoxic lymphocytes without involvement of antibodies
- * mediated by T cells
- * cell mediated Immunity
- * T cells secrete cytokines
- * Delayed onset
- * Direct cell to cell contact
- * Acts on Intracellular microbes

*Antigen detection is done by antibodies

* Antigen detection is done by receptors on the cell

⑧ Define and classify gangrene. D/w wet and dry gangrene.

It is the most often coagulative necrosis due to Ischaemia associated with putrefaction.

- Dry gangrene
- wet gangrene
- gas gangrene

Dry gangrene

- ① commonly occurs in limbs
- ② Slow loss of blood supply.
- ③ organs are dry shrunken and black
- ④ Line of demarcation present
- ⑤ Bacteria fails to survive

wet gangrene

- ① most common in bowel
- ② Sudden loss of blood supply
- ③ Swollen, red and dark
- ④ No clear line of demarcation
- ⑤ Numerous present

⑥ Atherosclerosis is the cause

⑥ Embolic, trauma are the cause

⑦ Limited amputation is sufficient

⑦ Higher amputation is often needed

⑨ How UV radiation affects biological system

① Skin Cancer.

① melanoma.

* It is the most serious form of skin cancer seen in young adults

* UV exposure and sunburns are risk factors for the disease

* other influences include genetic factors and immune system deficiencies

② Non-melanoma skin cancer

* Non-melanoma skin cancers are less deadly than melanomas

* Basal cell carcinomas appear as small fleshy lumps

* Squamous cell carcinomas appear as red scaly patches

② premature aging and other skin damages

* Chronic exposure to the sun also causes premature aging, overtime can make the skin thick, wrinkle

* Actinic keratosis are skin growths that occur on body areas exposed to the sun

* It is characterized by reddish, rough textured growth

③ Cataract & other eye damage

* cataracts are a form of eye damage in which there is loss of transparency in the lens of the eye.

* If untreated cataracts leads to blindness

④ Immune Suppression

* Overexposure to UV radiation may suppress proper functioning of body's immune system and skin natural defense

* Skin normally defends against foreign invaders such as cancers and infections, over-exposure to UV radiation, reduces skin ability to protect against invaders

⑩ Explain various effects of radiation?

① Haematopoietic Syndrome

* Haematopoietic^{stem} cells found in spleen, bone marrow, lymphatic tissue represents most radiosensitive cells

* Exposure to doses exceeding 200 rads impairs their functioning

* Affected stem cells results in reduced production of RBC, WBC & platelets which leads to Haematopoietic Syndrome such as anaemia, fatigue, lower immunity.

② Gastrointestinal System Syndrome

- * GIT cells are less actively dividing and relatively less radiosensitive
- * Exposure to doses exceeding 700 rads results in breakdown of gastrointestinal mucosal lining enables bacteria to access the vasculature
- * Symptoms include Nausea, vomiting, diarrhoea, bleeding ulcers etc.

③ Central Nervous System Syndrome

- * It involves destruction of least radiosensitive cells i.e. nerve cell
- * Exposure to doses exceeding 5000 rad results in central nervous system syndrome.
- * Symptoms include Nausea, vomiting, seizures, coma, loss of consciousness etc.

④ Genetic Effects

- * Damage to reproductive cell (egg or sperm) may appear as genetic mutations, birth defects or any other condition in offspring
- * Mutations caused by radiation cannot be distinguished from those caused by agents such as chemicals, viruses.

⑤ Somatic effects

- * Somatic effects occur due to damage caused by the radiation to somatic cell
- * It generally occurs due to occupational exposure and typically results in cancer or leukaemia

⑥ In-utero effects

- * In-utero effect is mistakenly considered to be genetic effect because it becomes pronounced in the embryo
- * Embryonic cells are rapidly dividing and are also rich in oxygen & blood supply, these are highly radiosensitive

⑪ Source, function, deficiency disorder of fat soluble vitamins

① vitamin A (Retinol).

Source.

- * dairy products, egg, liver oil,
- * green leafy vegetables, yellow fruits, tomato, carrots

Function

- * helpful for vision in dim light
- * Maintenance of Immune System
- * growth & development of the body

Deficiency Disorder

- * Night blindness
- * Conjunctival xerosis
- * Corneal xerosis
- * Bitot's spots

② Vitamin D (Calciferol)

Source

- * Egg, liver oil, vegetable oil, animal fat

Functions

- * It maintains homeostasis of calcium & phosphate

Deficiency Disorder

- * Rickets (It involves reduced calcification of growing bone characterized by bone deformity & muscular hypertonias).

③ Tocopherol (Vitamin E)

Source

- * Dairy products, egg, liver oil ✓
- * Green leafy vegetables, walnut oil, vegetable oil ✓
Sunflower oil, corn oil.

Functions

- * It acts as antioxidant and works to protect the cell of the body

④ Vitamin K (Naphtho quinone)

Source .

- * leafy vegetables ,
- * vegetable oil , coconut oil , olive oil , cotton seed oil

Function

- * Controls haemorrhagic disorders by activating blood clotting factors

Deficiency disorder

- * prothrombin content is reduced & clotting time is prolonged.

12. Source , function , deficiency disorder of water soluble vitamins

① Vitamin B₁ (Thiamine)

Source

- * Milk products
- * green vegetables , pork

Function

- * It is involved in citric acid cycle and acts as prosthetic group for decarboxylases

Deficiency disorder

- * Beriberi
- * Wernicke's encephalopathy

② Vitamin B₂ (Riboflavin)

Source

- * milk products.
- * Egg, meat

Functions

- * Assists in functioning of enzyme cytochrome P450
- * They are component of electron transport chain

Deficiency Disorder

- * cheilosis
- * glossitis

③ Vitamin B₆ (Pyridoxine)

Source

- * Egg, meat.
- * green vegetables

Functions

- * It acts as co-enzyme during biosynthesis of haem & haemoglobin.

Deficiency disorder

- * cheilosis
- * glossitis
- * Megaloblastic anaemia

④ Vitamin B₁₂

Source

- * Dairy products
- * Meat, Egg, Fish

Functions

- * Concerned with storage of folic acid in erythrocytes
- * Involved in formation of normal RBC.

Deficiency disease

- * Megaloblastic anaemia
- * Infertility.

⑤ Vitamin C (Ascorbic acid)

Source

- * Fresh raw fruits
- * Orange, lemon
- * Green vegetables, potato, tomato

Functions

- * Cellular respiration
- * Formation of haemoglobin, erythrocyte maturation
- * Immunological reactions
- * Healing of bone fractures

Deficiency disorder

- * Scurvy, characterized by bleeding gums, anaemia, weakness

1.4 Causes & metabolic changes of starvation?

Causes

- * poverty
- * famine
- * deliberate fasting
- * person with anorexia nervosa.
- * other condition which impairs digestion & assimilation

Metabolic changes

- ① Starvation initiates process of glycogenolysis as glucose store dry out.
- ② glycogenolysis takes place in the liver, It is a process of breakdown of stored glycogen to glucose to maintain normal blood glucose level
- ③ when glycogen store dry out, process of gluconeogenesis gets initiated
- ④ gluconeogenesis takes place in the liver, It is process where non-carbohydrate source such as amino acid, lactate, pyruvate & glycerol from fats are used for synthesizing glucose
- ⑤ Amino acids are sourced from skeletal muscle while fats from adipose tissue
- ⑥ In liver fatty acids are converted ^{into} ketone bodies which are consumed as fuel by most of the cells and partly by the brain

① Different types of angina & functions?

① Stable angina

- * It is a typical & common angina
- * occurs during emotional stress, heavy exercise
- * Easy to predict
- * It occurs due to atherosclerosis
- * pain lasts for 1-15 min and relieved by Rest / administration of nitroglycerin

② variant angina

- * It is ~~an~~ atypical & uncommon angina
- * occurs at rest
- * Difficult to predict
- * occurs due to Spasm of coronary artery
- * pain lasts of 20 min and Relieved by nitroglycerin and CCB's to reduce the events

③ unstable angina

- * It is atypical and uncommon
- * occurs during exercise and at rest
- * Difficult to predict
- * occurs due to ~~spasm of coronary artery~~ atherosclerotic progression, there is high chance of getting MI.
- * pain lasts for more than 20 min

15. complications of Diabetes mellitus

Short term

① Diabetic ketoacidosis ?

- * It is a secondary condition occurs due to deficiency of Insulin
- * usually associated with type I rarely with type II

Fatty Acids



Acetyl CoA



Ketone bodies

Ketone bodies

- * Excess amount of ~~Insulin~~ is released into circulation due to deficiency of Insulin

long term complication

① macrovascular disease

② CVD Disease

- * Risk of CVD is increased by smoking, hyperlipidemia, nephropathy

- * Atherosclerosis, MI, Hypertension are common in both Type-1 & Type 2 DM, occurs due to dyslipidaemia, obesity

③ PVD

- * In this arteries of the legs are affected leading to claudication and cramping pain while walking
- * Iliac vessel may also be affected leading to buttock pain & erectile dysfunction.

Microvascular disease

(a) Retinopathy

- * Retinopathy associated with DM enhances the chance of blindness in pt below 60 yr
- * Symptoms are slow in onset hence regular screening is required
- * glycaemic control, blood pressure control helps to reduce the risk.

(b) Nephropathy

- * Nephropathy can be evaluated by detecting the presence of albuminuria i.e. presence of small amount of albumin in urine and proteinuria i.e. presence of large amount of albumin

(c) Neuropathy

- * progressive loss of peripheral nerve fibres leading to nerve dysfunction is called peripheral neuropathy.

Complications of wound healing

- * Infection
- * Implantation :- epidermal cyst
- * pigmentation
- * Scar formation
- * hernia
- * Extensive contraction
- * Neoplasia.
- * ulcers

⑥ Pathophysiology of DM II

genetic susceptibility



multiple genetic defects



dysregulation of β -cell
insulin secretion



hyperglycemia



β -cell ~~dys~~ dysfunction



DM

CRF.

ARF, Disorders, Infection, Toxicity.



Damage of nephron



Loss of nephron more than 75%.



Deterioration of kidney function.



Hypertrophy of nephron.



Increase water & salt load
on each nephron



Stimulation of compensatory
mechanism



Decreased reabsorption of salt & water



Retention of fluid in
interstitial space



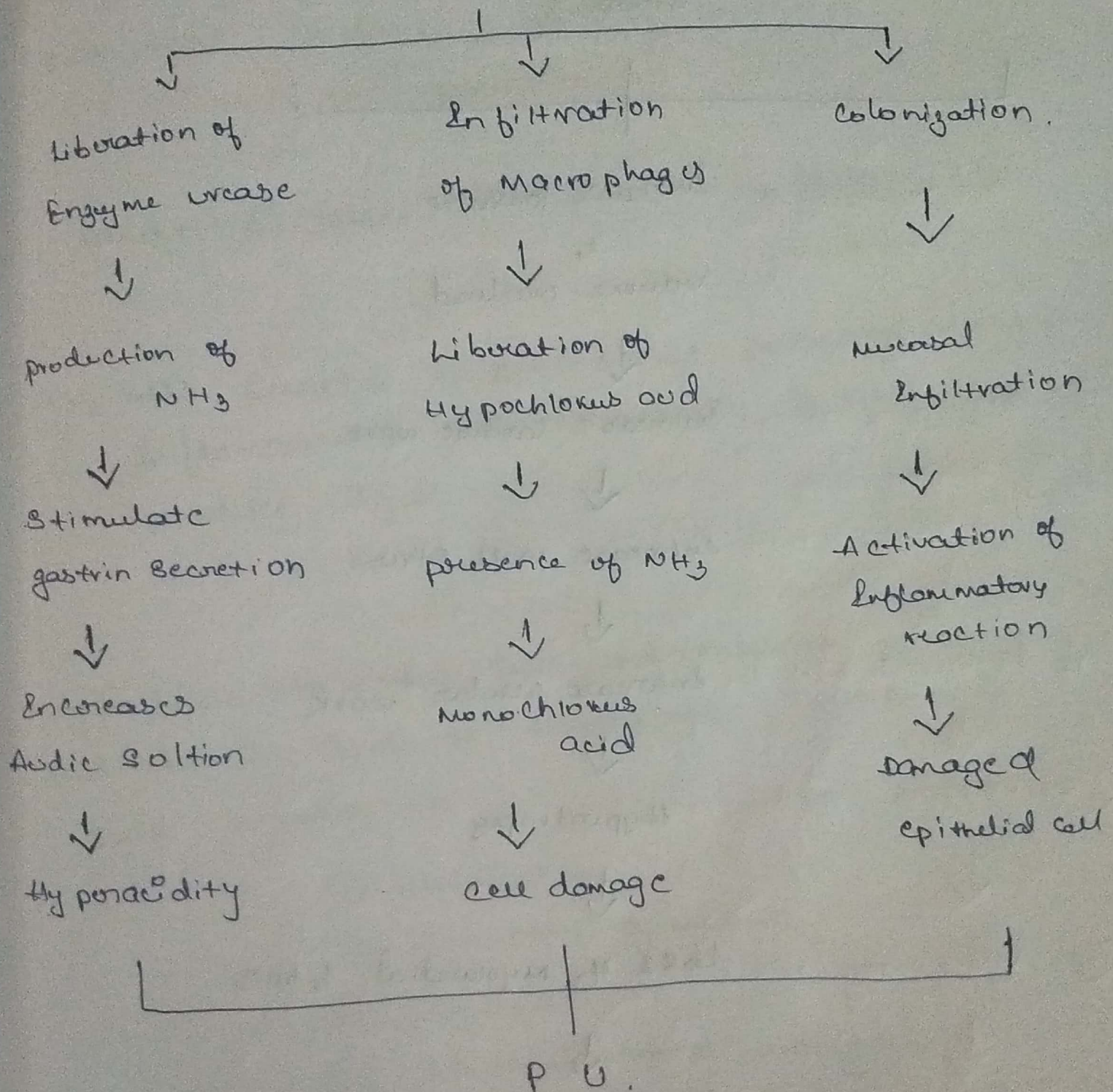
Edema.



Chronic Renal failure.

peptic ulcer.

Infection by *H. pylori*

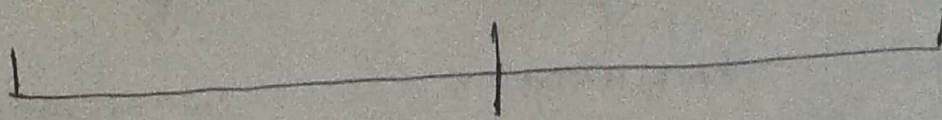


CAF

HTN.

valvular
stenosis

MI



Increase pressure
&
volume overload



Increase cardiac work.



Increase wall stress



Increase cellular work



Hypertrophy



Loss of myocardial fibres



Systolic & diastolic dysfunction



Cardiac dysfunction



CHF

Asthma

Airway epithelium and submucosa contain dendritic cells that capture & process antigens.



Dendritic cells stimulate TH₂ cells



TH₂ cells secrete cytokines that promote allergic inflammation and stimulate B cells to produce IgE



IgE binds to mast cells and releases primary mediators such as histamines, cytokines

This results in

- (i) Bronchoconstriction
- (ii) Secretion of mucus
- (iii) Vasodilation
- (iv) Accumulation of leukocytes

Parkinsonism

Substantia nigra & basal ganglia
of midbrain

Dopaminergic
pathway

cholinergic pathway

↓
Depigmentation
of dopaminergic
neuron damage

↓
Excitation of
caudate nucleus

↓
↓ Stimulation of dopamine

↓
↑ Secretion of Ach

↓
↓ Secretion of dopamine

↓
Excitation

↓
↓ voluntary movement

↓
↓ voluntary movement

↓
Affects the dopamine
& Ach balance

↓
Parkinsonism

Stroke

Hypotension
vasospasm

HTN
Atherosclerotic
Embolism
Thrombosis

Syphilis
TB

Atherosclerotic
Cardiac
embolism

Rupture of plaque
trauma
Loss of elasticity

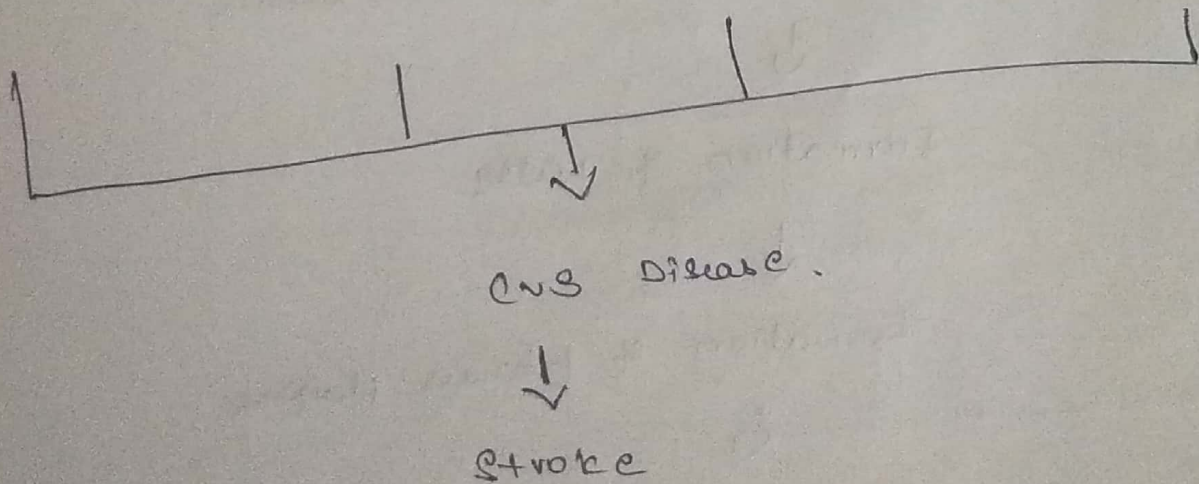
↓
↓
↓
Cerebral
perfusion
↓
Cerebral
Ischemia

↓
occlusion
of cerebral
blood vessel
↓
↓ cerebral
blood flow
↓
Cerebral
Ischaemia

↓
Inflammatory
Response
↓
vasculitis
↓
Cerebral
Ischaemia

↓
Vascular
morphological
changes

↓
loss of
Cerebrovascular
Responsiveness
↓
Cerebrovascular
damage
↓
Hemorrhage



Atherosclerosis

Endothelial Injury



Release of Inflammatory compounds



Increased permeability at the site of injury



Ingestion of Lipoprotein



Release of oxygen free radical



oxidation of lipoprotein



proliferation of Smooth muscle



Formation of fatty



Formation of fibrous plaque



progression of lesions



Atherosclerosis

Angina

Atherosclerosis

Arterial spasm

Atherosclerosis

+
plaque split
+
thrombus



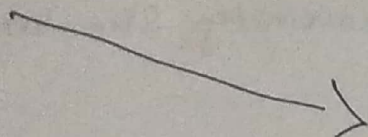
obstruction



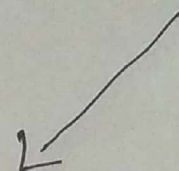
Sudden Reversible
obstruction



occlusion



Ischoemia



Hypoxia



Reduced
oxygen supply

→ Angina



Thrombolytic

→ unstable
angina

IBD

Entry of allergens



Inappropriate exposure to antigen



~~For~~ Secretion of toxins

& bacterial cell wall products



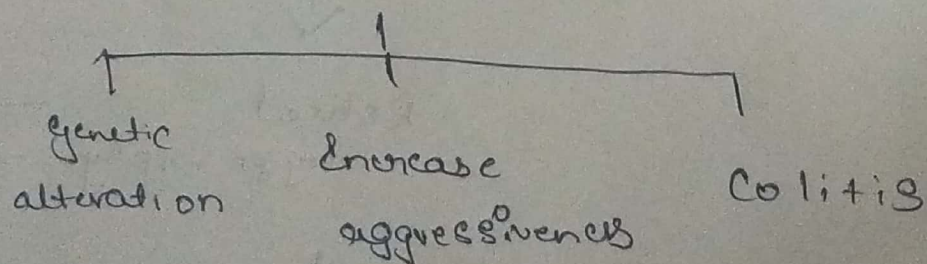
Host mucosal Immunity Stimulation



over expression of Immune cells



Fail to downregulate Immune
Reaction



IBD

2 mark important concepts



① Define shock ? Mention the types of shock

Ans shock is a life-threatening clinical syndrome of cardio-vascular collapse

Types.

- ① Hypovolaemic shock
- ② Cardiogenic shock
- ③ Septic shock
- ④ Traumatic shock
- ⑤ Neurogenic shock
- ⑥ Hypoadrenal shock

② Enlist the clinical features of decompensated shock

- ① Decreased cardiac output
- ② Mental confusion
- ③ Decreased urinary output
- ④ Tachypnoea
- ⑤ Brain : Hypoxic encephalopathy
- ⑥ Heart : Myocardial necrosis
- ⑦ Lungs : Adult Respiratory Distress Syndrome (ARDS)
- ⑧ Kidney : Acute Tubular necrosis
- ⑨ Adrenals : Necrosis

③ Write the mechanism of Septic Shock?

* Septic shock results most often from gram negative bacteria entering the body from urinary tract, alimentary tract, respiratory tract or skin.

* In Septic shock, there is Immune System activation and Systemic Inflammatory response to infection as follows

Activation of macrophage-monocyte

* Lysis of gram negative bacteria releases endotoxins, a lipopolysaccharide into circulation, where it binds with LBP to form LPS-LBP complex.

* Then the complex binds to CD14 molecule on the surface of macrophage which stimulates cytokines.

Effect of cytokines are

① Altering Endothelial cell adhesiveness: This results in recruitment of more neutrophils which liberate free radicals that cause cell injury.

② Promoting nitric oxide synthase: It stimulates increased synthesis of nitric oxide which is responsible for vasodilation & hypotension.

Q Define atrophy with ex

Ans It refers to reduction in size and no of parenchymal cells of organ due to reduction in their cellular organelles

Ex': Atrophy of uterus after parturition
Atrophy of Brain with age

Q Non - glycogen's disease

- * It is a glycogen storage disease due to defect in the enzyme glucose-6-phosphatase
- * generally liver, kidney & intestine is involved
- * It is characterized by accumulation of glycogen in hepatocytes and renal cells

Q pompe's disease

- * It is a glycogen storage disease due to defect in the enzyme Acid maltase.
- * generally all organs are involved
- * It is characterized by accumulation of glycogen in lysosome of all tissues.

Q Hydropic Swelling

- * It is the first and foremost morphological change in cell injury
- * It occurs when energy dependent pumps are impaired, leads to intracellular accumulation of Ca^{2+} , Na^{+} and water, to maintain osmotic condition

⑧

Karyolysis

It is the dissolution of nuclear chromatin.

pyknosis.

It is the reduction in the nuclear size of the affected cells.

Karyorrhexis

It is the fragmentation of completely or partially pyknotic nucleus.

⑨

Hypertrophy

It is the increase in size of the parenchymal cell without change in number due to increased synthesis of cellular components.

Physiological Hypertrophy

Hypertrophy of uterus during pregnancy.

Pathological Hypertrophy

Hypertrophy of nephrons of one kidney when another kidney is removed.

10.

List out complication of wound healing

*

pigmentation

*

Granulation tissue

*

Epidermal cyst formation

*

Excessive wound contraction

*

Neoplasia

11. Cardinal Signs of Inflammation

- (1) Rubor
- (2) calor
- (3) dolor
- (4) Tumor
- (5) Functio laesa

12. Regeneration

It is the replacement of injured cell with the same cell by cell division.

Repair

It is the replacement of injured tissue with fibrous tissue.

13. Autograft

* It is the graft where donor & recipient are same.

* The graft is harvested from one site of individual to another site of same individual.

Isograft

* It is the graft where donor & recipient are different, but genetically identical (Monozygotic twins).

Allograft

- * It is the most common type of graft where donor and recipient are of same species but are genetically non-identical.

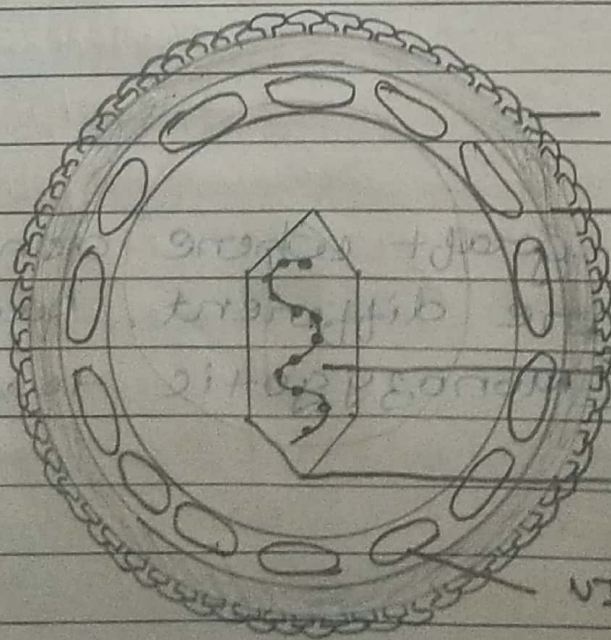
Xenograft

- * It is the graft where donor and recipient belongs to different species hence they exhibit maximum genetic difference

14. Sjogren Syndrome

- * It is a long term autoimmune disease that affects body's moisture producing glands.
- * It is characterized by dry eyes, mouth, skin, chronic cough etc.

15. Diagram of HIV Virus



ENVELOPE PROTEIN

ENVELOPE

VIRAL GENOME

NUCLEOCAPSID

VIRAL TEGUMENT

16 Define Amyloidosis ?

* It is the term used for group of diseases characterized by Extracellular deposition of fibrillar Insoluble proteinaceous substance called amyloid

17 Mc. Ardle's Syndrome ?

* It caused due to the deficiency of muscle phosphorylase

* It is an important substance to break down glycogen in muscle cells, This condition leads to muscle pain & fatigue during exercise

18 Apoptosis

Necrosis.

① programmed & Co-ordinated cell death ① cell death & degradation of tissue by hydrolytic enzymes

② physiological & pathological process is

② Hypoxia & toxins

③ No Inflammatory reaction

③ Inflammatory reaction present

19 Metaplasia - Give ex

* It is the reversible change of one type of epithelial cell to another type, usually in response to abnormal stimuli, often reverts back on removal of stimulus

Intestinal metaplasia
gallbladder metaplasia

20. Hypertrophy ? give ex

* Hypertrophy is an increase in the size of parenchymal cells resulting in enlargement of the organ or tissue

Ex Enlarged size of uterus in pregnancy

21. Hyperplasia ? give ex

* Hyperplasia is an increase in the no of parenchymal cells resulting in enlargement of the organ or tissue

Ex! Hormonal hyperplasia
Hyperplasia of female breasts.

22. Dysplasia ? give ex

* Dysplasia means 'disordered cellular development' accompanied with hyperplasia & metaplasia

Ex!:- Increased no of layers of epithelial cells

23. Types of necrosis

- * Coagulative necrosis
- * Fat necrosis
- * Fibrinoid necrosis
- * Caseous necrosis.

24. Role of histamines in acute inflammation

- * vasodilation
- * Increased vascular permeability
- * Itching & pain

25. Cardinal signs of Inflammation

- (i) rubor (redness)
- (ii) calor (heat)
- (iii) dolor (pain)
- (iv) tumor (swelling)

26. Transudate

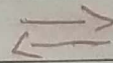
Exudate

- * Filtration of blood plasma without change in vascular permeability.
- * Filtration of blood plasma with increased vascular permeability
- * Non-inflammatory oedema
- * Inflammatory oedema

27. Prostaglandins

PGI_2 , PGF_2 , $PGF_{2\alpha}$, PGI_2

28. Regeneration



Repair

- * Restoration of tissue structure & function after injury
- * new growth of tissue to their normal state
- * Heals major types of damages
- * Heals minor type of damages
- * Form Scar tissue
- * Does not form Scar tissue

29. Autoimmunity

- * It is the state in which the body's immune system fails to distinguish between self & nonself and react by formation of autoantibodies against one's own tissue antigens.

30. Biological Significance of Inflammation

- * enables neutrophils migrate to the destroyed area & engulf invaders
- * localizes the invading pathogens
- * plasma protein (fibrinogen) $\rightarrow$ blood clot
- * excess tissue fluid $\rightarrow$ dilute toxic irritants

31. T cells

- * T-cells originate in the bone marrow & mature in the thymus
- * occur inside the lymphnode
- * Bear TCR Receptor
- * Involved in cell mediated immunity

B cells

- * B-cells originate & mature in the bone marrow
- * occur outside the lymphnode
- * Bear BCR Receptor
- * Involved in humoral mediated immunity

32. Myasthenia gravis.

- * It is an autoimmune disorder caused by an antibody mediated blockade of nicotinic acetylcholine receptors at neuromuscular junction of skeletal muscle.

33. SLE (Systemic Lupus Erythematosus)

- * SLE is an autoimmune disease, where immune system of the body mistakenly attacks healthy tissue.
- * It can affect the skin, joints, kidney, brain and other organs.

34. Rheumatoid Arthritis.

- * Rheumatoid Arthritis is an autoimmune & inflammatory disease, which means that your Immune System attacks healthy cells in our body by mistake, causing inflammation in the affected part of the body.
- * It mainly affects joints in the hands & feet.

35. Neoplasia (Tumour)

- * "Neoplasia is a mass of tissue formed as a result of abnormal, excessive, unco-ordinated, purposeless proliferation of cells even after cessation of stimulus for growth."

36. How does radiation causes carcinogenesis?

- * Ionizing radiations causes cancer in animals and in man.
- * It causes damage to DNA and results in mutation.
- * It causes chromosomal breakage, translocation or point mutation.
- * The effect depends on dose, frequency, age, individual susceptibility.

37. Write four contrasting features of benign & malignant tumours.

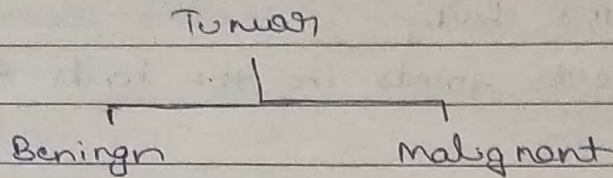
Benign	Malignant
* usually slow	* usually rapid
* Compress surrounding tissue without invading them	* Invades the surrounding tissue.
* Metastasis absent	* Metastasis present
* Local complication	* Metastatic complication

38. Carcinogenicity

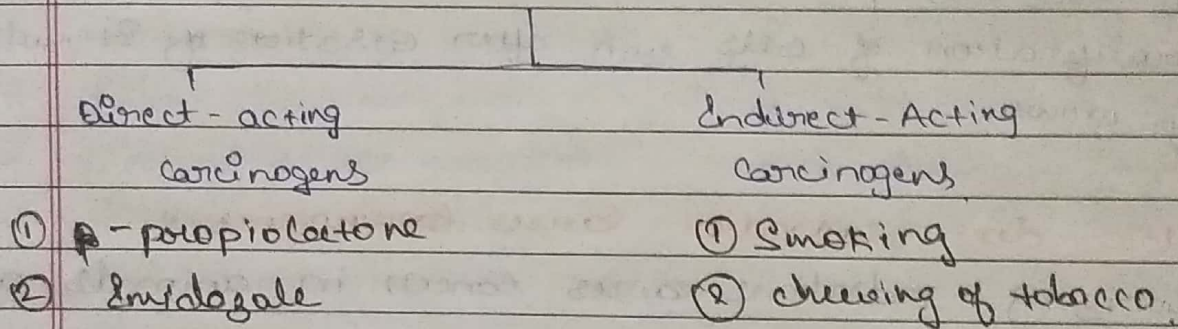
ability of

* The term Carcinogenicity denotes a chemical substance or mixture of chemical substances to induce cancer

39. What is Tumor & classify



40. Two ex for Direct & Indirect Acting Carcinogens



41. Human cancer associated with viral infections

* Renal cancer

* Cervical cancer

42. Grading of cancer

grade I $\rightarrow$ cancer cells that resemble normal cells and aren't growing rapidly

grade II $\rightarrow$ cancer cells that don't look like normal cells and are growing faster than normal cells

43 Stages of cancer

- * Early Stage
- * Localized
- * Regional spread.
- * Distant spread

44 Radiation Induced malignancies

- * Skin cancer
- * Leukaemias
- * Thyroid
- * Lung, breast cancer
- * uterus, ovary

45 Ames test

- * Ames test is a widely employed method that uses bacteria to test whether a given chemical can cause mutations in the DNA of the test organism

46 Tumour markers

Bladder cancer	BTA (Bladder Tumor Antigen)
Lung cancer	Alk gene
B-cell lymphoma	B-cell Immunoglobulin gene

47 Ionizing

gamma rays

x rays

UV rays

non-ionizing

visible

IR, microwaves

Radiowaves

48. Effects of Radiation

- * Genetic effects
- * CNS Syndrome
- * Somatic effects
- * En-tero effects

49. Radiation Sickness

- * Radiation sickness is damage to our body caused by large dose of radiation often received over a short period of time

50. Therapeutic applications of Radiation?

- * ^{131}I is used for treatment of thyroid cancer
- * ^{60}Co are used in treatment of cervical cancer
- * Radioisotopes have role in management of malignancies
- * Tumor tissues are attacked by beam of radiation

51. What is biological effects of radiation?

- * Skin Cancer
- * premature aging & other skin damages
- * Cataract & other eye damage
- * Immune Suppression.

52. Kwashiorkor

Marasmus

- | | |
|---|---|
| <ul style="list-style-type: none"> * observed b/w 1-5 yr old children * caused due to low protein diet * high-protein foods are required | <ul style="list-style-type: none"> * common in 1st yr of life. * caused due to lack of protein, carbohydrate & fat diet. * Nutritious, well balanced diet is required |
|---|---|

53. Complications of obesity

- * Sleep apnea & Snoring
- * Lung disease
- * Heart disease
- * Stroke
- * Liver disease
- * Pancreatitis
- * Arthritis

54. Air pollutants & its effects

- ① Carbon compound (CO & CO_2)
 - Respiratory problems
 - Green house effect
- ② Nitrogen compound (NO & NO_2)
 - Irritation in eye or lungs
 - Acid rain damages
- ③ Sulphur compounds
 - Respiratory prob in humans

55. Scurvy

Scurvy is a condition characterized by weakness, bleeding gums, anemia caused by prolonged deficiency of vitamin C (ascorbic acid) in the diet.

56. Symptoms of CO poisoning

- * Chest tightness
- * Head ache
- * Fatigue
- * Dizziness
- * Drowsiness

57. Effects of Cigarette Smoking

- * causes atherosclerosis
- * lung cancer
- * chronic bronchitis
- * stroke, tremors, Irritability
- * Smoking causes intestinal cramps, diarrhoea, Constipation.

58. Effects of SO_2

- * laughing, choking -
- * sneezing, running nose
- * bronchitis
- * itching, rashes on skin

59. Toxic effects of NO

- * chest tightness
- * Shortness of breath
- * Headache
- * fatigue
- * dizziness
- * drowsiness

60. Megaloblastic anemia

- * Megaloblastic anemia is a condition in which bone marrow produces usually large, structurally abnormal, immature RBC

61. causes, sign, symptoms of rickets
causes

* vitamin D deficiency

Signs & Symptoms

* bone pain or tenderness

* curved legs

* muscle weakness

* Soft bones

* large forehead on abdomen

* wide joints in elbows & ribs

* dental cavities

62. Effects of protein - Energy malnutrition

* Reduced bone weight

* muscle wasting & decreased strength

* Reduced Respiratory & cardiac muscle capacity

* Skin thinning

* edema

* Immuno deficiency

63. Symptoms of Schizophrenia

* Hallucinations

* Delusions

* Disorganized thinking

* Agitation, Anxiety

* Euphoria

64. peptic ulcer
- * Formation of sores in the lining of stomach and duodenum are referred as peptic ulcers
 - * usually peptic ulcers occurs due to Excess acid production.

65 Depression

Mania

- * Feeling of Sadness
- * faulty or negative thinking
- * Social withdrawal.
- * Appetite & weight changes
- * Irritability
- * loss of focus & attention
- * overactive, talkative
- * decreased need of sleep

66 Angina ? its types

- * Angina is the classical Symptom of myocardial Ischaemia. It refers to severe pain in the middle of the chest during exercise or effort due to decreased supply of oxygenated blood to the heart

- (i) Stable Angina.
- (ii) unstable Angina.
- (iii) variant Angina
- (iv) Microvascular Angina

67 complications of DM

- * Cardiovascular Disease
- * peripheral vascular disease
- * Microvascular disease (Neuro, Nephro, Retinopathy)
- * Formation of Advanced glycation end product.

68. Atherosclerosis

* It is characterized by building up of fatty material in the arterial walls, leading to decreased blood flow.

Risk factors-

- Age, Sex, history.
- Excess Lipid Deposition
- Smoking
- DM, lack of exercise, Sedentary life style

69. IBD, Give two ex

* It is an inflammatory disorder of the alimentary tract commonly affecting the terminal ileum & the rectum. It can affect entire length of alimentary tract.

Ex: ulcerative colitis
Crohn's disease

70. Symptoms of parkinsonism

- * Tremor
- * Rigidity
- * Bradykinesia
- * postural instability

71. What is CHF?

* Congestive cardiac failure is defined as the inefficiency of the heart to pump sufficient amount of oxygenated blood to all organs of the body to meet their metabolic demands.

- Types
- (i) Acute heart failure
 - (ii) Left-sided heart failure
 - (iii) Chronic heart failure
 - (iv) Right sided heart failure

72. Causes of Acute Renal failure

- (i) Renal Ischaemia
- (ii) glomerular disease
- (iii) Kidney stones.
- (iv) NSAID's Antibiotics

73. different stages of Alcoholic liver disease

- (i) Alcoholic Steatosis
- (ii) Alcoholic Hepatitis
- (iii) Alcoholic Cirrhosis

74. COPD

* It is a chronic obstructive lung disease in which airflow obstruction occurs due to narrowing of airway. It mainly involves lung parenchyma, small bronchioles, large airways

78. Signs & Symptoms

Hyperthyroidism	Hypothyroidism
* weight loss	* weight gain
* heat intolerance	* cold intolerance
* moist skin	* Dry skin
* weakness, fatigue	* weakness, fatigue
* muscle cramps.	* muscle cramps

79. Goiter ? clinical symptoms

* A goiter is an enlargement of your thyroid gland. That's the gland at the front of your neck just below Adam's apple

- * A hoarse voice
- * Tightness in throat
- * Swollen neck veins
- * Coughing
- * Trouble breathing & swallowing

80 Thyroiditis?

- * Thyroiditis is the inflammation of thyroid gland, the butterfly shaped gland in the neck,
- * Drugs, radiation, virus, bacteria causes thyroiditis

81 Lung function test

- (i) Spirometry
- (ii) Lung volume test
- (iii) Gas diffusion test
- (iv) Exercise stress test

82 Name the causative organism for Amoebic & bacterial dysentery

Amoebic $\rightarrow$ Entamoeba histolytica
 Bacterial $\rightarrow$ Shigella species

83 Causative organism for UTI

- (1) E. coli
- (2) Pseudomonas aeruginosa
- (3) Streptococcus faecalis
- (4) Staphylococcus epidermidis

84. List the type of pneumonia?

- * Bacterial pneumonia
- * viral & mycoplasma pneumonia
- * Legionella pneumonia
- * Aspiration pneumonia
- * Lipid pneumonia

85. pneumonia

causative agent

- * Streptococcus pneumoniae
- * Staphylococcus aureus
- * Rhino virus, Influenza, coxsackie virus
- * Legionella pneumonia

Signs & Symptoms.

- * Fever, Headache
- * muscle pain
- * cough with expectoration.

86. Amoebic dysentery

bacterial dysentery

- * caused by Entamoeba histolytica
- * caused by Shigella species