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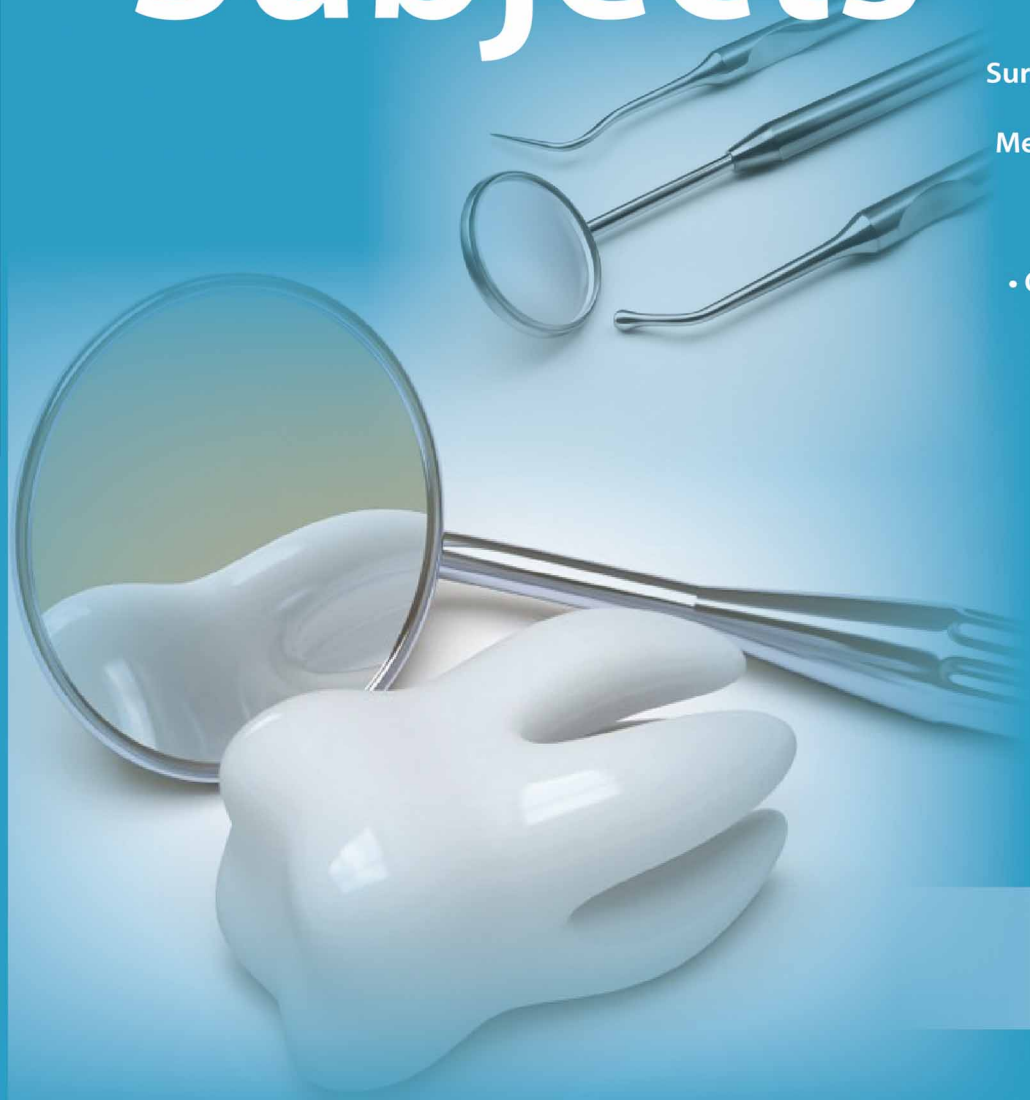
ROADS Review of All DENTAL Subjects



• Anatomy • Biochemistry • Physiology • General Pathology • Microbiology • Pharmacology • General Medicine and Surgery • Dental Materials • Dental Anatomy and Histology • Oral Pathology and Oral Medicine • Complete Dentures • Removable Partial Dentures • Fixed Partial Dentures • Periodontics • Radiology • Pedodontics • Conservative Dentistry • Endodontics • Oral Surgery • Orthodontics • Community Dentistry • Miscellaneous Topics • AIDS



Fully Explained Topics for Last 10 years

- **AIPGDEE**
- **AIIMS**
- **PGI**
- **COMED-K**
- **BHU**

Neha Sethi

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CHAPTER 4

General Pathology

Objectives

- General pathology
- Wound healing and repair
- Inflammation
- Fluid and hemodynamic pathology
- Thrombosis, embolism, infarction and aneurysm
- Neoplasia
- Various disorders and their important features
- Pathological deposits
- Immunology and genetics
- Pathology of blood cells
- Cardiovascular pathology
- Transfusion disorders
- Respiratory pathology
- Environmental pathology
- Miscellaneous

GENERAL PATHOLOGY

- ***Adaptation***: Is the cell's response to prolonged stress
- ***Cell injury***: Reversible (hydropic change) or irreversible (necrosis)
- ***Cell death***: Necrosis (due to irreversible injury) and apoptosis (programmed cell death).

General Mechanisms of Cell Injury

- Oxygen derived free radicals.
- Increased intracellular calcium
- ATP depletion
- Defects in membrane permeability

Changes seen in both reversible and irreversible cell injury(AIPG 2005)	Changes seen only in irreversible cell injury
• Cellular swelling • Mitochondrial swelling • Dilatation and degranulation of RER • Autophagocytsosis	• Cell membrane rupture, lysosomal rupture • ***Nuclear changes*** – pyknosis (nuclear condensation), karyolysis (loss of nuclear chromatin, karyorrhexis (nuclear fragmentation). ***(AIPG 2007))*** • ***Amorphous densities*** in mitochondria • Ca^{2+} influx capase activation

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Degeneration

- Reaction of cells to injury and represents retrograde change caused by a disturbance in cell metabolism.
- ***Degeneration*** is ***reversible*** cell injury while ***cell death*** is a state of irreversible cell disorganization.
- ***Seven types*** of degeneration are present
  - Fatty
  - Atheromatous,
  - Mucinous,
  - Zenker.
  - Albuminous
  - Hyaline,
  - Calcerous

Fibrinoid degeneration	• Tissue accumulates deposits of acidophilic homogoneous material resembling fibrin.
	• Seen in immunopathologic tissue injury, e.g., Autoimmune diseases, immune complex vasculitiis, Arthus reaction.
Cloudy swelling	• Most common degeneration (albuminous, hydropic).
	• Reversible.
	• Most common sites for cloudy swelling are kidneys, liver and myocardium
Hyaline degeneration	• Non-specific degeneration mainly affecting the collagenous connective tissue and fibrous tissue is hyaline degeneration.
Intracellular hyaline degeneration	• Mallory stain is used in case of hyaline degeneration. Mallory's hyaline seen in alcoholic liver cell injury.
	• Hyaline degeneration in voluntary muscle is called as Zenker degeneration.
	• Zenker degeneration is seen in typhoid fever specifically in striated muscle.
	• Russel bodies – Excess immunoglobulins in plasma cells
Extracellular hyaline degeneration	• Hyalinised old scar, hyaline arteriosclerosis and hyalinised glomeruli
Mucinous degeneration	• Excessive production of mucus
	• Two types
	– Epithelial
	– Mesenchymal
Fatty degeneration	• Cholesterol crystals are seen in cells undergoing autolysis in **lipoid degeneration**.

Necrosis

Types of Necrosis

Coagulation necrosis	• Most common type of necrosis.
	• Conversion of normal cells into their **tombstones** is hallmark of coagulative necrosis.
	• General architecture is preserved except for some nuclear change.
	• Most common results from interruption of blood supply, commonly seen in the end organs.
	• E.g.: **Infarct of heart, kidney and spleen etc**
Liquefaction necrosis	• Also known as **colliquative necrosis.**
	• There is enzymatic digestion of the tissue.
	• Architecture is lost. Necrotic tissue is soft and liquefied.
	• Tissue is softened/liquefied due to enzyme action (autolysis and heterolysis)

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	• It is commonly seen in CNS resulting from interruption of blood supply. It also occurs in area of bacterial infection.
	• Seen in ***brain, abscesses***
Caseous necrosis	• Induced by cell mediated immunity (T lymphocytes, macrophages, cytokines)
	• Tissue appears cheesy; histologically consists of granular material surrounded by epithelioid cells and multinucleated giant cells
	• Seen in ***tuberculosis, fungal granulomas***
Fat necrosis	• Necrosis in adipose tissue induced by lipases (derived from injured pancreatic cells in ***acute pancreatitis***) or trauma to adipose tissue (***breast trauma***)
Fibrinoid necrosis	• Free fatty acids bind with calcium to form calcium soaps.
	• Typically seen in ***arteries, arterioles or glomerular capillaries*** damaged by autoimmune diseases
	• Blood vessels are impregnated by fibrin and other serum proteins and appear magenta-red on histology
Gangrenous necrosis	• A clinical term for ***ischemic necrosis accompanied by bacterial infection, which*** leads to partial liquefaction of tissues.
	• *'Dry gangrene' (mummification)* refers to noninfected ischemic necrosis accompanied by drying of the tissues.

Apoptosis

Programmed cell death ***(NEET 2013, AIPG 2012)***

- ***Mitochondria*** plays a central role-***Increased mitochondrial outer-membrane permeability*** is the major trigger of the ***intrinsic apoptosis pathway.***

Morphological Changes	• Cell shrinkage
	• Chromatin condensation
	• Formation of cytoplasmic blebs and apoptotic bodie
	• Phagocytosis of apoptotic cells or bodies
Biochemical Feature	• Protein cleavage
	• DNA breakdown
	• Phagocytic recognition
Pro-apoptotic molecules	• ***Bak, Bim, Bax***
Anti-apoptotic molecules	• ***Bcl-2, Bcl-x*** ***(AIPG 2004)***
Examples of Apoptosis	• During embryogenesis (programmed destruction of cells)
	• Endometrium, prostate in adults (hormone dependent involution of tissues)
	• Cell deletion in multiplying cell populations (intestinal crypt epithelium), tumours and lymphoid organs.
	• Cell death by cytotoxic T cells, cell injury in certain viral diseases, atrophy in organs after duct obstruction.
Functions of Apoptosis	• Elimination of cells in embryological development (e.g. motor neurones).
	• Induction of tolerance to self-antigens by removal of autoreactive T lymphocytes.
	• Removal of virally infected cells.
	• Removal of cells, which have undergone DNA damage.

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Detection of apoptosis	• Terminal deoxynucleotidyl transferase biotin – dUTP nick end labeling (TUNEL) mostly used for in vivo apoptosis detection
	• DNA fragmentation assay – observed by electrophoresis of genomic DNA
	• Annexin V/Propidium iodide assay by flow cytometry or fluorescent microscopy
	• Caspase activity assay ***(AIPG 2008)***

- The mechanisms by which ***Diabetes Mellitus*** enhances apoptosis are:
  - Activation of the polyol pathway
  - Formation of advanced glycation end products
  - Formation of reactive oxygen species
  - Higher levels of TNF-α expression

Differences between Apoptosis and Necrosis

	NECROSIS	APOPTOSIS
Etiology	Acute cell injury due to extracellular stimuli	Various intracellular or extracellular stimuli
Character	Pathologic	Physiologic/pathologic
Distribution	Groups of cells or patches of tissues	Widely scattered isolated cells
Energy requirement	Passive (ATP independent)	Active (ATP dependant)
Cell size	Enlarged (swelling)	Reduced (shrinkage)
Nucleus	Pyknosis karyohexis-karyolysis	Fragmentation into nucleosome sized fragments
Cellular contents	Enzymatic digestion, contents may leak out of the cell	Intact, maybe released as apoptotic bodies
Plasma membrane	Disrupted	Intact – altered structure
Adjacent inflammation	Frequent	Not present
DNA changes	Random DNA fragmentation	Chromatin condensation with DNA fragmentation

Apoxemia

- Reduction in the amount of oxygen (O) dissolved in plasma (Pao_2, where the lowercase letter "a" means arterial). ***(NEET 2013)***
- It can be ***secondary*** to
  - Respiratory acidosis, which is secondary to retention of CO_2 in the lungs (e.g., chronic obstructive pulmonary disease).
  - Ventilation defects, in which air does not reach the respiratory unit for gas exchange (e.g., atelectasis, or the collapse of alveoli).
  - Perfusion defects characterized by blockage of blood flow through the pulmonary capillaries (e.g., pulmonary embolus).
  - Diffusion defects, which interfere with gas exchange at the alveolar-capillary interface (e.g.. fluid in patients with left heart failure}.
  - As long as the capillary PO_2 is higher than that in the tissue, 0: will move into the tissue by the process of diffusion. ***(AIPG 2012)***

- **Hb-related abnormalities** encompass those associated with a reduction in Hb concentration; decreased O; saturation (SaO_2). representing the percentage of heme groups occupied by O_2; and decreased release of 0_2 from Hb at the tissue level.
- ***The o_2 content is the total amount of O_2 carried in the blood and is equal to 1.34 (Hb g/dL) X SaO_2 + PaO_2.***
  - In anemia, there is a reduction in the Hb concentration, which reduces the total O_2 content (1.34 Hb g/dL] x SaO_2 + PaO_2) without altering the SaO_2 or the PaO_2, since there is normal O_2 exchange in the lungs.
  - Methemoglobin is heme iron in the ferric state, which is unable to bind with O_2, iron must be in the ferrous to bind with O_2.
  - Oxidizing agents such as nitrites (e g., nitroglycerin). nitrates converted into nitrites in the gut (e.g., from nitrate-rich well water), and sulfur-containing drugs increase the formation of metHb.
  - Patients are cyanotic and do not respond to administration of O_2, since the heme iron is in the ferrous state.
  - ***Methylene blue*** is the treatment of choice, since it enhances the conversion of iron to the ferrous condition by acting as an artificial electron carrier in the NADPH dependent metHb reductase system, white ascorbic acid, a reducing agent, assumes an ancillary role in treatment.

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- **In carbon monoxide (CO) poisoning**, CO competes with 0_2 for binding sites on heme iron in place of oxygen, thereby decreasing the O_2 content by decreasing the SaO_2
  - The patient will have a **cherry red discoloration** of the skin owing to the combination of CO with myoglobin.
  - Administration of o_2 is the treatment of choice in CO poisoning.
  - Factors moving the O, dissociation curve to the left (high affinity of Hb for O2,), such as decreased 2,3-bisphosphoglycerate, CO, metHb, hypothermia, and HbF (fetal Hb), decrease the release of 0_2 to tissue.

- Abnormalities in ***oxidative phosphorylation*** include [Those which block the oxidative pathway (e.g., inhibition of cytochrome oxidase by CO and cyanide) and those which produce abnormalities In phosphorylation by damaging mitochondrial membranes (e.g., uncoupling agents such as alcohol and aldehydes).
  - The O_2 content is normal, but ATP synthesis is decreased
  - O_2 an electron acceptor, is the last reaction in the electron transport chain, which underscores why tissue hypoxia virtually shuts down ATP synthesis.

Ultrastructural and biochemical alterations In hypoxic cell Injury

Reversible consequences directly related to a reduction In ATP synthesis.	• Loss of the ATP-dependent sodium/potassium pump • Cloudy swelling • Increased Anaerobic glycolysis • Intracellular lactic acidosis • Reduced protein synthesis • EM findings -swelling of the smooth endoplasmic reticulum (first alteration) and mitochondria.
Irreversible consequences of hypoxic cell injury.	• Free radical damage • Cell membranes are damaged • Formation of large amorphous densities in mitochondria • Increased cytosolic concentration of calcium • Nuclear abnormalities - pyknosis -(ink dot appearance), karyorrhexis (nuclear fragmentation), and karyolysis (dissolution of nuclear chromatin – ***AIIMS May 2010)***. • Release of intracellular enzymes from damaged cells is an excellent marker of cell injury

Free radicals

- Unstable chemical species that have a single, unpaired electron in their outer orbit.
- Produced by ionizing radiation, damaged mitochondria, oxidase reactions, drugs (e.g., acetaminophen) and chemicals (e g. carbon tetrachloride [CCI_4,]).
- Oxygen-derived FRs include superoxide, hydroxyl ions, and peroxide.
- Cause cell injury by lipid peroxidation, in which lipid FRs combine with molecular oxygen
- FRs are neutralized by superoxide dismutase: catalase; glutathione peroxidase (which generates glutathione) and antioxidants, such as vitamin E, vitamin C, and selenium
- ***Examples***:
  - Retrolental fibroplasia and blindness in newborns secondary to o_2. FR injury results from administration (damage occurs to tissue in Iron overload states (hemochromatosis), since iron helps generate FRs by fenton reaction)
  - Acetaminophen hepatotoxicity is associated with the formation of acetaminophen FRs in the-cytochrome P450 system, leading to liver necrosis and failure.
  - CCI4 hepatotoxicity is related to its conversion to CCI, FRs in the cytochrome system, leading to liver cell necrosis and fatty change

Cytoskeletal abnormalities

- Microtubules are polymers composed of the protein tubulin that are capable of undergoing rapid assembly and disassembly in the cytosol.
- The Chediak Higashi syndrome (CHS) is an autosomal recessive cytoskeletal abnormality in which the main defect is in the assembly (polymerization) of microtubules in the cytoplasm.
- Alterations occur in certain of the intermediate function when damaging stimuli cause cells to release stress proteins (also called heat shock proteins) that are vital to survival of cell.
  - ***Ubiquitin*** aids in the removal of old or damaged proteins, such as intermediate filaments by first binding to the protein and then delivering it to proteases for degradation.
  - **Mallory's bodies** are masses of keratin Intermediate filaments in hepatocytes of patients with alcoholic liver disease that have been "ubiquinated," or marked for destruction by proteases.
- The membrane skeleton provides structural integrity to the cell membrane. It is composed of spectrin, actin, and protein 4.1.
- Congenital spherocytosis, an autosomal dominant disease, is an example of a defect in spectrin resulting in a hemolytic anemia

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Microscopic Examination of Cerebral Ischemia (AIPG 2012)

After the first 12 hours	Upto 48 hours
• Both cytotoxic and vasogenic edema predominate	• Neutrophilic emigration
• Endothelial and glial cells, mainly astrocytes swell and disintegrate	• Phagocytic cells, derived from circulating monocytes and activated microglia are evident.
	• The macrophages become stuffed with the products of myelin breakdown or blood and may persist in lesion for months to years
	• Reactive astrocytes can be seen as early as 1 week after the insult.

Pathophysiology of growth alterations

Atrophy	• Shrinkage in cell size (organ size and weight as well) with the loss of cell substance and reduction in metabolic activity. • May be a physiologic (e.g., involution of the thymus with advancing age) or a pathologic process (e.g., cerebral atrophy in atherosclerosis of the carotid artery). • In **autophagy:** cell organelles are encompassed by membranes to form vacuoles wherein they are progressively degraded until only undigestable, lipid rich material, called ***lipofuschin*** remains behind in residual bodies. • In H-and E-stained tissue, lipofuschin often' called the "***wear and tear pigment***, has yellow brown granular appearance, Which If extensive enough, grossly discolors the tissue, producing a condition called ***brown atrophy.*** • Clinical examples include skeletal muscle atrophy in a cast and atrophy of target organs in hypopituitarism as a result of the loss of trophic hormone stimulation
Agenesis	• Absence of an organ resulting from failure to develop any discernible primordial tissue (anlage) during embryonic development (e.g., renal agenesis).
Aplasia	• The primordium of life organ is present, but there Is no further development of the tissue (e.g , aplasia of the adrenal cortex).
Hypoplasia	• Incomplete or partial development ol an organ or tissue (e.g., hypoplastic left heart) and differs from atrophy In that the latter begins with a normal-sized organ that subsequently becomes smaller as ceil mass is lost.
Hypertrophy	• Increase in cell size due to an increase In the synthesis of structural components and organelles • Examples of physiologic hypertrophy is hypertrophy of the heart in a well-trained athlete
Hyperplasia	• Increase In the number of cells • Both hyperplasia and hypertrophy are characterized by an increased expression of the protooncogenes, which are growth-controlling genes ***(AIPG 2005)*** • Same factors can stimulate both processes such as growth hormones, estrogen, protocooncogenes, growth factors ***(AIPG 2005)*** • Clinical examples of physiologic hyperplasia is hyperplasia of endometrial glands during the proliferative phase of the menstrual cycle • An example of approximately equal amounts of hypertrophy and hyperplasia is smooth muscle hypertrophy/ hyperplasia in the uterus in pregnancy.
Metaplasia	• Reversible replacement of one adult cell type by another adult cell type, usually as an adaptive process instigated by the host tissue as a response against chronic irritation (e.g., smoking, Infection). • Clinical examples include the presence of increased goblet cells in the terminal bronchioles of a smoker
Dysplasia	• Reversible atypical hyperplasia • Disorderly proliferation of cells with nuclear variation in size and shape and increased mitotic activity with normal mitotic spindles. • Arises from a metaplastic or hyperplastic process and may progress to cancer through a series of steps if the irritating stimulus is not removed. ***(AIPG 2001)*** • Using the bronchial mucosa in a smoker as an example, the following progression occurs: normal pseudostratified ciliated columnar epithelium—goblet cell hyperplasia and basal cell - hyperplasia— squamous metaplasia—> squamous dysplasia (mild, moderate, severe)— squamous carcinoma in situ (limited to the full thickness of the epithelium) —• invasive squamous carcinoma (invades through the basement membrane and has the ability to metastasize)
Anaplasia	• Lack of differentiation. The malignant cells resemble more primitive, undifferentiated cells than those from which they arose. ***(AIPG 2007)***

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Also know

- In the cell division cycle, the main cell cycle regulators are ***kinases*** (e.g., p34 kinase), which regulate the progression front one phase to the next, and small proteins called cyclins whose main function is to activate these kinases
- Cells in the ***G phase*** (G = gap), or resting phase of the cell cycle, enter the cycle when one or more of the above-mentioned growth promoter mechanisms are invoked.
- In the ***G_1 phase*** (most variable phase of the cycle) structural proteins are synthesized.
- Chromosomal replication occurs in S (synthetic) phase.
- In the ***G_2 phase***, the mitotic spindle (composed of microtubules) is assembled.
- ***Mitosis (M phase)*** results in division of the cell into two daughter cells; the latter then enter the G1, phase, which determines whether the cells enter the G1„ phase, remain in the cycle to divide again, or become terminally differentiated and unable to divide again.

- *Cell growth by paracrine stimulation refers to the production of a growth peptide in one cell that attaches to peptide receptors on nearby cells without having to enter the blood stream.*
- *Cell growth by autocrine stimulation occurs when the same cell produces growth factors and receptors.*

"Wound Healing and Repair

The healing process may be of three types

- Healing by first intention (***primary healing***)
- Healing by second intention (***secondary healing***)
- Healing by third intention (***delayed primary closure***)

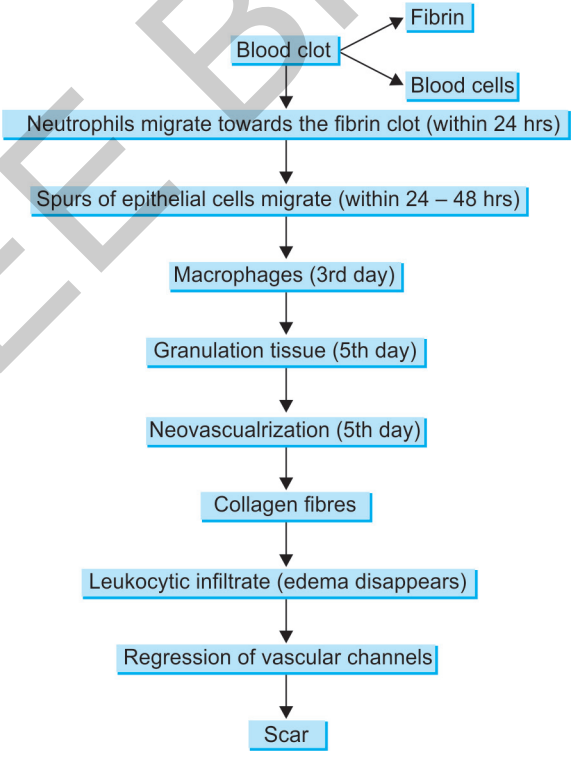

Fig.4.1 **Healing** by first intention

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"DIFFERENCE BETWEEN HEALING BY PRIMARY AND SECONDARY INTENTION

Primary Intention	Secondary Intention	Third intention
Wound is clean, uninfected and surgically incised	Wound is open with a large tissue defect, at times infected	Delayed primary closure
No much loss of tissues and cells	Extensive loss of tissues and cells	
Edges of wounds are approximated by surgical sutures	Wound is not approximately by surgical sutures.	Wound is left open to be closed at a later day (4 – 7 days) using a primary closure technique
Healing is rapid	Healing is slow	Slow
Large scar is formed	Neat scar of primary union is not formed	
Less granulation tissue	More granulation tissue	
Wound contraction is not present	Wound contraction present	Is less

Wound Healing

- By ***24-48 hour***: Epithelial closure takes place.
- ***On day 3***: Neutrophils are replaced by macrophages.
- ***By Day 5***: Collagen fibrils begin to appear and epithelial proliferation is maximal.
- At the end of ***first week***: Wound strength is approximately 10%.
- By 3 ***months***: Recovery of tensile strength is 70-80%.

Recovery of Tensile Strength Results from

- Increased collagen synthesis exceeding collagen degradation during the first 2 months.
- Structural modifications of collagen fibres when collagen synthesis ceases at late times.

Cells involved in wound healing may be classified into three types on the basis of their potential to divide

Labile cells	• These cells divide continually and have considerable division potential throughout postnatal life. • They are necessary for regeneration as these cells can populate the wound, divide, differentiate and restore the original tissue architecture and composition. • Eg. Surface epithelial cells of epidermis, alimentary tract, respiratory tract urinary tract, hemopoietic cells of bone marrow and cells of lymph node and spleen.
Stable cells	• These cells can populate the wound site and produce a scar, which is a repair tissue. • They have limited division potential • Eg. Fibroblasts, Endothelial cells, osteoblasts and chondrocytes
Permanent cells	• These cells have exited the cell cycle, differentiated and do not divide. • Injuries to tissues containing permanent cells can only heal by repair because these cells cannot divide and are replaced by cells that produce only scar tissue • Eg. Neurons of the nervous system, skeletal muscle and cardiac muscle cells

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Mechanical Physical Factors

Cell-To-Cell Interaction	• Cell movement and differentiation are related to cell-to-cell interactions as has been noted in the healing of a wound in which epidermis and connective tissue have a complex interaction.
Cell-To-Matrix Interaction	• Cell migration and differentiation are influenced by the surrounding matrix. The presence of fibrin appears to stimulate fibroblast activity as well as angiogenesis. • LAMININ inhibits epidermal cell migration. • INTEGRINS are the transmembrane glycoproteins on the cells that acts as receptors for matrix proteins.
Growth Stimulators And Inhibitors	• Growth factors are a class of natural biologic mediators that regulate crucial cellular events involved in tissue repair such as • DNA synthesis • Chemotaxis • Differentiation • Matrix synthesis

Cells in tissue culture continue to move on a surface until they establish contact with similar cells, at which point movement stops. This is termed 'contact inhibition'. This appears to be dependent on the density of cells pressed and has been termed 'density dependent regulation of growth'.

INFLAMMATION

Vascular Events	Leukocyte Events (Cellular Events)
• Increased Blood Flow	• Margination
• From relaxation of terminal arterioles in the inflammatory lesion	• Diapedesis
• Produces local erythema (rubor) and heat (calor)	• Chemotaxis
• Increased Vascular Permeability	• Phagocytosis
• From contraction of capillary endothelial cells	• Degranulation
• Extravasation of water, low and high molecular weight solutes and blood cells	
• Leads to local swelling (tumor)	

Chemical mediators of inflammation

Vasodilation	Histamine, serotonin, bradykinin
Increased vascular permeability	Vasoactive amines, C3a and C5a, bradykinin, leukotriene C4
Chemotaxis, leucocyte recruitment and activation	C5a, leucotriene B4, IL-1, TNF
Fever	IL-1, TNF, prostaglandin
Pain	Prostaglandin (PGE_2)
Tissue damage	Neutrophil and macrophage lysomal enzymes, oxygen Metabolites ***(NEET 2013)***

Opsonins and Opsonic Receptors

Target	Opsonin	Opsonic receptor	Cells possessing Opsonic Receptors
Gram negative bacteria	LPS-binding protein (LBP) or septin	CD14	Neutrophils (if primed with TNF- α), Macrophages

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Target	Opsonin	Opsonic receptor	Cells possessing Opsonic Receptors
Any particle or cell	C3b	CR3 (Mac I)	Neutrophils
Any Particle or cell	IgG1, IgG2, IgG3	Fcγ RII	Neutrophils Macrophages

Acute Phase Reactants

Positive	Negative
CRP	Albumin
Mannose binding protein ***(MAH 2012)***	Transferrin
Complement factors	Transthyretin
Ferritin	Transcortin
Cerruloplasmin	Retinol Binding Protein
Serum amyloid A	
Haptoglobin ***(MAH 2012)***	
Alpha 1 antitrypsin	
Alpha2 macroglobulin	

Cytokines

- **Soluble proteins** produced by a wide variety of hematopoietic and non hematopoietic cell types
- Involved in the regulation of the growth, development, and activation of immune system cells and in the mediation of the inflammatory response.
  - **Immunoregulatory cytokines** involved in the activation, growth, and differentiation of lymphocytes and monocytes, **e.g., IL-2, IL-4, IL-10, , and transforming growth factor (TGF)**
  - **Proinflammatory cytokines --IL-1, TNFα and IL-6** and the chemokine family of inflammatory cytokines, within which are included IL-8, monocyte chemotactic protein (MCP)-1, MCP-2, MCP-3, macrophage inflammatory protein and regulation-upon-activation, normal T expressed and secreted (RANTES)
  - Cytokines that regulate **immature leukocyte growth and differentiation,** e.g., **IL-3, IL-7, and GM-CSF.**

Prostaglandins

LTA4	Produced in leucocytes, platelets, mast cells, vascular tissue.
LTC4, LTD4, LTE4	• Contraction of smooth muscle • Bronchoconstriction • Vasoconstriction • ↑vascular permeability • Components of SRSA. ***(PGI 1995)***
LTB4	• ↑Chemotaxis • Adhesion of WBC ***(COMED 2006)*** • Release of lysosomal enzymes
Thromboxanes	• Produced mainly in platelets • Promotes platelet aggregation • Vasoconstriction • Smooth muscle contraction
Prostacyclins	• Produced by endothelium of vessels • Vasodilation • Inhibits platelet aggregation ***(PGI 1992)***

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FLUID AND HEMODYNAMIC PATHOLOGY

Oedema

Factors which cause oedema formation		Factors which prevent oedema formation
Vascular factors	**Tissue factors**	
• Decreased plasma oncotic pressure of intestinal fluid	• Increased oncotic pressure of interstitial fluid	• Increased blood colloidal osmotic pressure
• Increased capillary hydrostatic pressure	• Decreased tissue tension	• Decreased capillary hydrostatic pressure
• Increased capillary permeability	• Lymphatic obstruction (or) decreased lymphatic drainage	• Decreased capillary permeability
	• Increased sodium and water retention	• Decreased tissue oncotic pressure
		• Increased tissue hydrostatic pressure (or) tissue tension
		• Increased lymphatic drainage

Shock

- Shock results in the hypoperfusion of tissue with subsequent impaired tissue oxygenation ***(NEET 2013)***
- The ***four types*** are
  - ***Hypovolemic shock*** (e.g.. due to hemorrhage or excessive sweating).
  - ***Cardiogenic shock*** (e.g.. pump failure secondary to an acute myocardial infarction),
  - ***Septic shock*** (gram-negative endotoxic shock), and
  - ***Neurogenic shock*** (loss of vasomotor tone in venules and small veins as In fainting, spinal cord injury or use of autonomic blocking agents).

- Loss of 20% of the blood volume (about 1000 mL) results in hypovolemic shock.
  - In blood loss, the hemoglobin (Hb) and hematocrit (Hct) may remain normal for 1-3 days owing to the equal loss of plasma and RBCs and to vascular contraction around the reduced volume of blood.
  - Laboratory abnormalities include metabolic acidosis secondary to retention of lactate from tissue hypoxia and hyperglycemia due to glycogenolysis from the release of cortisol, glucagon, and catecholamine.

- ***Cardiogenic shock*** can arise from an acute myocardial infarction, valvular disease, and cardiomyopathies.
- ***Septic (endotoxic) shock*** may be associated with endotoxin-containing gram-negative bacteria (e.g.. Escherichia coli), exotoxin-producing gram-positive organisms, and fungi.
  - The ***acute phase*** is characterized by
    - Peripheral arteriolar vasodilatation (warm skin, reduced oxygen exchange in tissue leading to tissue hypoxia and then to lactic acidosis and hypotension).
    - High-output cardiac failure (dilated arterioles shunt more blood through the microcirculation back to the heart), and
    - Sinus tachycardia

- Endotoxins primarily bind to CD14 receptors on leukocytes, endothelial cells, and other cells, resulting in direct injury or the release of the following chemical mediators.
  - ***Interleukin 1 (IL-1)*** and ***tumor necrosis factor*** (TNF) are released from macrophage activation (the increased neutrophil adhesion to vessels leads to vessel damage). ***(AIIMS May 2013)***

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Review of All DENTAL Subjects (ROADS)

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- Review of All Dental Subjects (ROADS) is a sincere effort of the author to give a new approach and horizon to AIPGDEE preparation.
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