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INI-CET

The Next Level of Preparation

REVIEW OF AIIMS (2015-2019) and INI-CET

A Valuable Drop in the Ocean

2020-2022

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3rd Edition

JAYPEE

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CHAPTER 4

Pathology

GENERAL PATHOLOGY

Cellular Responses to Injury

Free Radicals

Properties	$O_2^{\bullet -}$	H_2O_2	•OH	$ONOO^-$
MECHANISMS OF PRODUCTION	Incomplete reduction of O_2 during oxidative phosphorylation; by phagocyte oxidase in leukocytes	Generated by SOD from O_2^- and by oxidases in peroxisomes	Generated from H_2O by hydrolysis, e.g., by radiation; from H_2O_2 by Fenton reaction; from $O_2^{\bullet -}$	Produced by interaction of $O_2^{\bullet -}$ and NO generated by NO synthase in many cell types (endothelial cells, leukocytes, neurons, others)
MECHANISMS OF INACTIVATION	Conversion to H_2O_2 and O_2 by SOD	Conversion to H_2O and O_2 by catalase (peroxisomes), glutathione peroxidase (cytosol, mitochondria)	Conversion to H_2O by glutathione peroxidase	Conversion to HNO_2 by peroxiredoxins (cytosol, mitochondria)
PATHOLOGIC EFFECTS	Stimulates production of degradative enzymes in leukocytes and other cells; may directly damage lipids, proteins, DNA; acts close to site of production	Can be converted to •OH and OCl^-, which destroy microbes and cells; can act distant from site of production	Most reactive oxygen-derived free radical; principal ROS responsible for damaging lipids, proteins, and DNA	Damages lipids, proteins, DNA

1. Most reactive free radical is: *(AIIMS May 16)*

a. Peroxide
b. Carboxyl
c. Hydroxyl
d. Superoxide

Ans. c. Hydroxyl

Features of Reversible Cell Injury

Cellular swelling is the first manifestation of almost all forms of injury to cells. On microscopic examination, small clear vacuoles may be seen within the cytoplasm; these represent distended and pinched-off segments of the ER. This pattern of nonlethal injury is sometimes called hydropic change or vacuolar degeneration. Swelling of cells is reversible. Cells may also show increased eosinophilic staining, which becomes much more pronounced with progression to necrosis.

The ultrastructural changes of reversible cell injury include:

1. Plasma membrane alterations, such as blebbing, blunting, and loss of microvilli
2. Mitochondrial changes, including swelling and the appearance of small amorphous densities
3. Dilation of the ER, with detachment of polysomes; intracytoplasmic myelin figures may be present
4. Nuclear alterations, with disaggregation of granular and fibrillar elements.

2. All of the following are seen in reversible injury *except*: *(AIIMS May 19)*

a. Loss of microvilli
b. Myelin figures
c. Plasma membrane blebs
d. Cell swelling

Ans. b. Myelin figures

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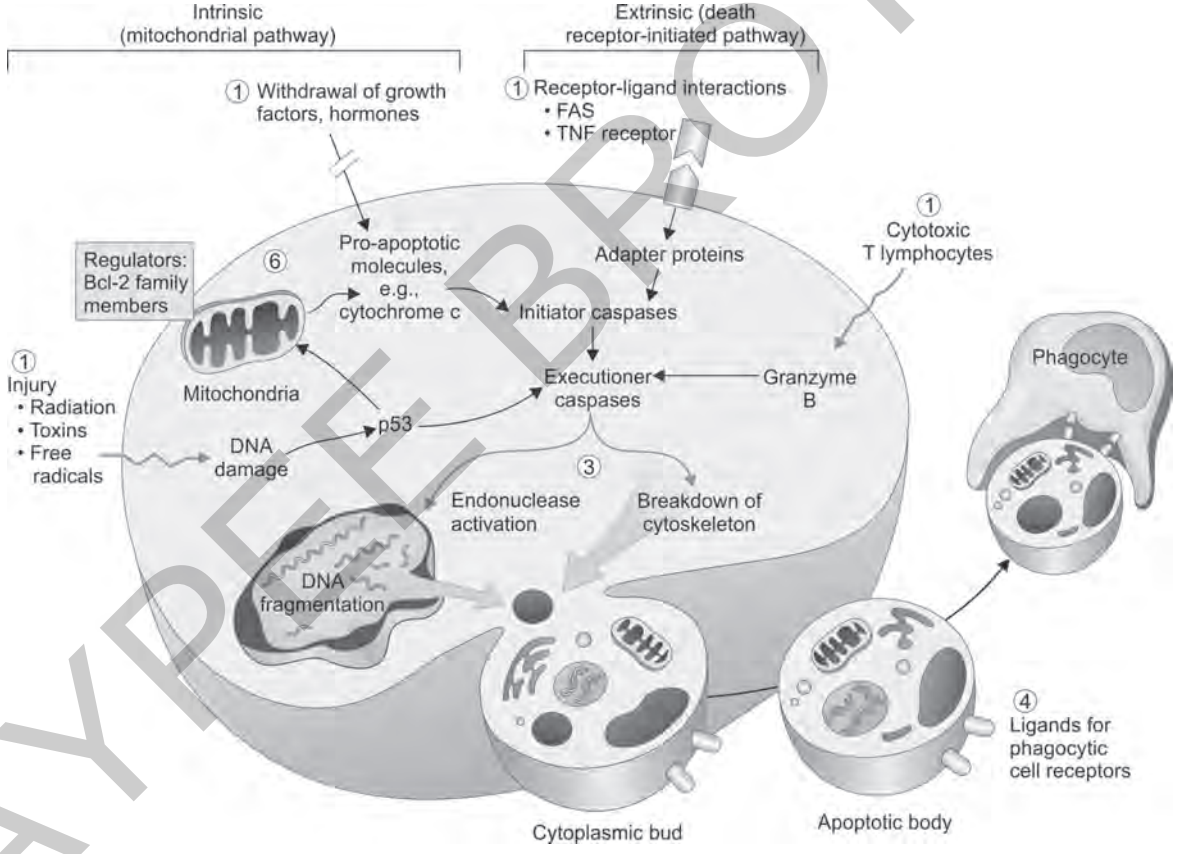
Irreversible Cell Death

Feature	Necrosis	Apoptosis
Cell size	Enlarged (swelling)	Reduced (shrinkage)
Nucleus	Pyknosis → karyorrhexis → karyolysis	Fragmentation into nucleosome-size fragments
Plasma membrane	Disrupted	Intact; altered structure, especially orientation of lipids
Cellular contents	Enzymatic digestion; may leak out of cell	Intact; may be released in apoptotic bodies
Adjacent inflammation	Frequent	No
Physiologic or pathologic role	Invariably pathologic (culmination of irreversible cell injury)	Often physiologic, means of eliminating unwanted cells; may be pathologic after some forms of cell injury, especially DNA damage
Agarose gel electrophoresis of DNA	Diffuse smearing of DNA	Ladder pattern of DNA fragments

Apoptosis

The 2 pathways of apoptosis are summarized below:



Apoptosome is a complex formed in the intrinsic pathway by the combination of cytochrome C with Apaf-1. This then binds to caspase-9, which is one of the initiator caspases, leading to apoptosis.

Execution Phase of Apoptosis

The two initiating pathways converge to a cascade of caspase activation, which mediates the final phase of apoptosis. The mitochondrial pathway leads to activation of the initiator caspase-9, and the death receptor pathway to the initiators caspase-8 and -10. After an initiator caspase is cleaved to generate its active form, the enzymatic death program is set in motion by rapid and sequential activation of the executioner caspases. Executioner caspases, such as caspase-3 and -6, act on many cellular components. For instance, these caspases, once activated, cleave an inhibitor of a cytoplasmic

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DNase and thus make the DNase enzymatically active; this enzyme induces the characteristic cleavage of DNA into nucleosomesized pieces, described earlier. Caspases also degrade structural components of the nuclear matrix, and thus promote fragmentation of nuclei. Some of the steps in apoptosis are not fully defined. For instance, we do not know how the structure of the plasma membrane is changed in apoptotic cells, or how membrane blebs and apoptotic bodies are formed.

3. Apoptosome binds to which of the following molecules? *(INI-CET June 20)*

a. Procaspase 8
b. Procaspase 9
c. Procaspase 4
d. Procaspase 5

Ans. b. Procaspase 9

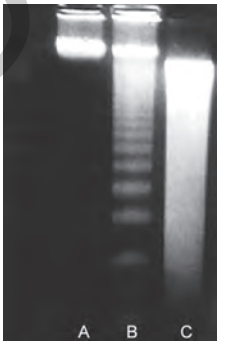
4. Which of the following can be activated by intrinsic or extrinsic pathway? *(AIIMS Nov 19)*

a. Necroptosis
b. Necrosis
c. Apoptosis
d. Ferroptosis

Ans. c. Apoptosis

5. Agarose gel electrophoresis from DNA of a population of cells as seen under ultraviolet light is shown below. What is the correct explanation for the finding as seen in the bank labeled as "C"? *(AIIMS May 16)*

a. Apoptotic cells
b. A population of viable cells
c. Mixed population of normal and apoptotic cells
d. Predominantly necrotic cells



Ans. d. Predominantly necrotic cells

6. Which is false about apoptosis? *(AIIMS Nov 18)*

a. No inflammation
b. Plasma membrane intact
c. Organelle swelling
d. Affected by dedicated genes

Ans. c. Organelle swelling

7. Acute inflammation seen in: *(AIIMS May 19)*

a. Necrosis
b. Apoptosis
c. Pyroptosis
d. Necroptosis

Ans. a. Necrosis

8. The correct description of Hayflick Limit is: *(INI-CET June 20)*

a. Total number of times a cell can divide during its lifetime
b. The maximum temperature up to which a cell can be heated before its death
c. The maximum distance up to which signals can travel through neurons
d. The minimum temperature at which a mammalian cell can survive

Ans. a. Total number of times a cell can divide during its lifetime

The Hayflick Limit is a concept that helps to explain the mechanisms behind cellular aging. The concept states that a normal human cell can only replicate and divide forty to sixty times before it cannot divide anymore, and will break down by programmed cell death or apoptosis. The concept of the Hayflick Limit revised Alexis Carrel's earlier theory, which stated that cells can replicate themselves infinitely.

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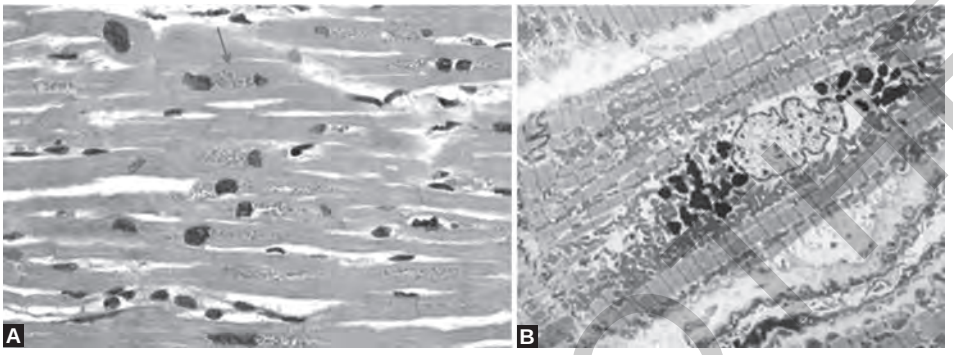
Lipofuscin

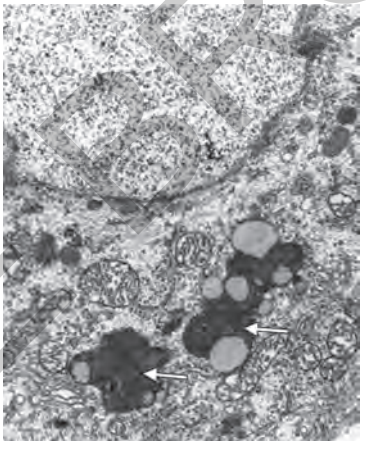
Lipofuscin is an insoluble pigment, also known as lipochrome or wear-and-tear pigment. Lipofuscin is composed of polymers of lipids and phospholipids in complex with protein, suggesting that it is derived through lipid peroxidation of polyunsaturated lipids of subcellular membranes. Lipofuscin is not injurious to the cell or its functions. Its importance lies in its being a telltale sign of free radical injury and lipid peroxidation. The term is derived from the Latin (fuscus, brown), referring to brown lipid. In tissue sections it appears as a yellow-brown, finely granular cytoplasmic, often perinuclear, pigment. It is seen in cells undergoing slow, regressive changes and is particularly prominent in the liver and heart of aging patients or patients with severe malnutrition and cancer cachexia.

The following figure shows lipofuscin granules in a cardiac myocyte shown by (A) light microscopy (deposits indicated by arrows), and (B) electron microscopy (note the perinuclear, intralysosomal location).

(For color version see Fig. 1, Plate 5)



9. Identify the marked structure: *(AIIMS Nov 18)*



a. Primary lysosome
b. Secondary lysosome
c. Phagolysosome
d. Lipofuscin granule

Ans. d. Lipofuscin granule

Dystrophic Calcification

Dystrophic calcification is seen any type of necrosis—coagulative, caseous, liquefactive type, and necrosis of fat. Calcification is frequently seen in atheromas of advanced atherosclerosis. It also commonly develops in aging or damaged heart valves, further hampering their function. The calcium salts appear macroscopically as fine, white granules or clumps, often felt as gritty deposits. Sometimes a tuberculous lymph node is virtually converted to stone.

Metastatic Calcification

Metastatic calcification occurs in normal tissues in the setting of hypercalcemia. Hypercalcemia also accentuates dystrophic calcification. The major causes are:

1. Increased secretion of parathyroid hormone (PTH) with subsequent bone resorption, as in hyperparathyroidism due to parathyroid tumors, and ectopic secretion of PTH-related protein by malignant tumors
2. Destruction of bone tissue—secondary to primary tumors of bone marrow (e.g., multiple myeloma, leukemia) or diffuse skeletal metastasis (e.g., breast cancer), accelerated bone turnover (e.g., Paget disease), or immobilization

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3. Vitamin D-related disorders, including vitamin D intoxication, sarcoidosis (in which macrophages activate a vitamin D precursor), and idiopathic hypercalcemia of infancy (Williams syndrome), characterized by abnormal sensitivity to vitamin D
4. Renal failure—causes retention of phosphate, leading to secondary hyperparathyroidism.

10. Dystrophic calcification is seen in: *(AIIMS May 19)*

a. Paget disease
b. Sarcoidosis
c. Myositis ossificans
d. Immobilization after fracture

Ans. c. Myositis ossificans

Stem Cells

Stem cells are characterized by their self-renewal properties and by their capacity to generate differentiated cell lineages. To give rise to these lineages, stem cells need to be maintained during the life of the organism. Such maintenance is achieved by two mechanisms: (a) *obligatory asymmetric replication*, in which with each stem cell division, one of the daughter cells retains its self-renewing capacity while the other enters a differentiation pathway, and (b) *stochastic differentiation*, in which a stem cell population is maintained by the balance between stem cell divisions that generate either two self-renewing stem cells or two cells that will differentiate. In early stages of embryonic development, stem cells, known as *embryonic stem cells* or *ES cells*, are *pluripotent*, that is, they can generate all tissues of the body. Pluripotent stem cells give rise to multipotent stem cells, which have more restricted developmental potential, and eventually produce differentiated cells from the three embryonic layers. The term *transdifferentiation* indicates a change in the lineage commitment of a stem cell. In adults, stem cells (often referred to as adult stem cells or somatic stem cells) with a more restricted capacity to generate different cell types have been identified in many tissues. They have been studied in detail in the skin, the lining of the gut, the cornea, and particularly in the hematopoietic tissue. Somatic stem cells for the most part reside in special microenvironments called niches, composed of mesenchymal, endothelial, and other cell types. It is believed that niche cells generate or transmit stimuli that regulate stem cell self-renewal and the generation of progeny cells. Although stem cells have regenerative capacity, they do so only when activated by a stimulus.

11. Which of the following is a wrong statement about stem cells? *(INI-CET June 20)*

a. They can differentiate into other cells
b. They can replicate
c. They can regenerate damaged tissue
d. They can do division without any regulatory control

Ans. d. They can do division without any regulatory control

12. Hematopoietic stem cells are capable of all of the following *except*: *(INI-CET June 20)*

a. Replication
b. Differentiation
c. Replication without differentiation
d. Trans differentiation

Ans. c. Replication without differentiation

Inflammation

Acute Inflammation

Acute inflammation has three major components:
1. Alterations in vascular caliber that lead to an increase in blood flow.
2. Structural changes in the microvasculature that permit plasma proteins and leukocytes to leave the circulation.
3. Emigration of the leukocytes from the microcirculation, their accumulation in the focus of injury, and their activation to eliminate the offending agent.

Vasodilation is one of the earliest manifestations of acute inflammation; sometimes it follows a transient constriction of arterioles, lasting a few seconds. Vasodilation first involves the arterioles and then leads to opening of new capillary beds in the area. The result is increased blood flow, which is the cause of heat and redness (erythema) at the site of inflammation. Vasodilation is induced by the action of several mediators, notably histamine and nitric oxide (NO), on vascular smooth muscle.

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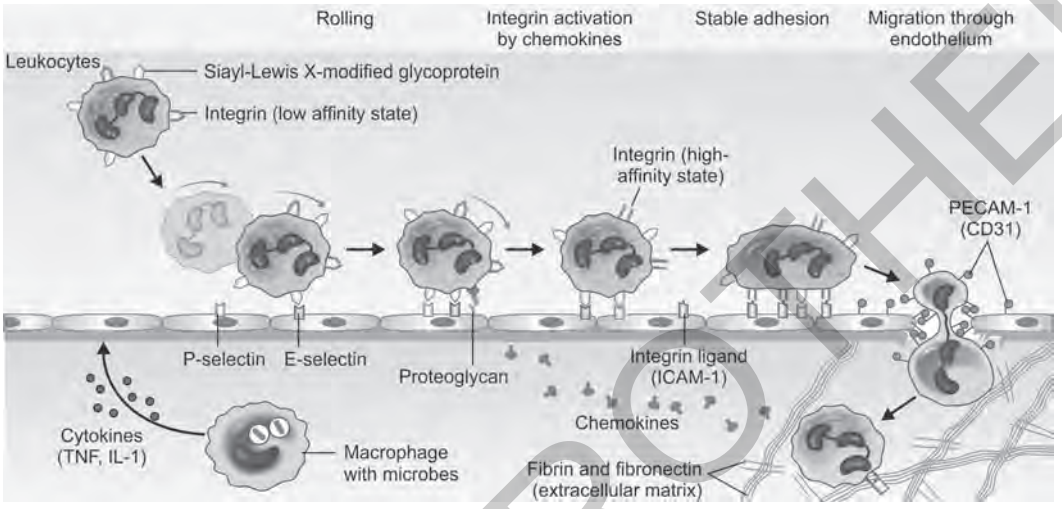
Vasodilation is quickly followed by increased permeability of the microvasculature, with the outpouring of protein-rich fluid into the extravascular tissues.

The journey of leukocytes from the vessel lumen to the interstitial tissue, called extravasation, can be divided into the following steps:

1. In the lumen: margination, rolling, and adhesion to endothelium. Vascular endothelium in its normal, unactivated state does not bind circulating cells or impede their passage. In inflammation, the endothelium is activated and can bind leukocytes, as a prelude to their exit from the blood vessels.
2. Migration across the endothelium and vessel wall.
3. Migration in the tissues toward a chemotactic stimulus.



13. Which of the following is the correct sequence of cellular events of acute inflammation?

(AIIMS May 18, INI-CET June 21)

a. Rolling—stable adhesion—activation of integrins—migration via endothelium
b. Rolling—activation of integrins—stable adhesion—migration via endothelium
c. Stable adhesion—rolling—activation of integrins—migration via endothelium
d. Activation of integrins—migration via endothelium—stable adhesion—rolling

Ans. b. Rolling—activation of integrins—stable adhesion—migration via endothelium

14. Integrin binds to: *(AIIMS Nov 18)*

a. Vitronectin
b. Laminin
c. Fibronectin
d. Collagen

Ans. c. Fibronectin

15. Vasodilation is mediated by: *(AIIMS Nov 19)*

a. TXA2
b. PGE2
c. Histamine
d. Serotonin

Ans. c. Histamine

Mechanisms of Increased Vascular Permeability

Several mechanisms increase vascular permeability, some of which are physiologic and some of which are pathologic. Endothelial contraction and retraction are physiologic mechanisms and are due to mediators. Direct endothelial injury is a pathologic mechanism due to damaging agents not under the body's control.

❖ **Endothelial contraction** (referred to as immediate-transient response)
- *Mediators*: Histamine, bradykinin, and leukotrienes.
- *Vessels affected*: Postcapillary venules.
- *Time course*: Immediate; short lived (up to 30 minutes).

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- **Endothelial cell retraction**
  - *Mediators*: Tumor necrosis factor (TNF) and interleukins (e.g., IL-1).
  - *Mechanism*: Structural rearrangement of cytoskeleton.
  - *Time course*: 4–6 hours (referred to as delayed response); long lived.
- **Direct endothelial injury**
  - *Mediators*: Bacterial enzymes.
  - *Vessels affected*: All.
  - *Mechanism*: Endothelial cell necrosis.
  - *Time course*: Immediate (referred to as immediate-sustained response).
- **Delayed prolonged response**
  - Due to ultraviolet light, X-ray, and mild thermal injury.
  - Uncertain mechanism.
- **Leukocyte-mediated damage**

16. Match the following types of endothelial dysfunction with their causes: *(AIIMS May 19)*

1. Sunburn	A. Immediate sustained
2. Bee sting	B. Delayed prolonged
3. Bacterial enzymes	C. Endothelial cell retraction
4. Tumor necrosis factor	D. Immediate transient

Ans. 1-B, 2-D, 3-A, 4-C

Acute Phase Reactants

Proteins that change their serum concentration by >25% in response to inflammatory cytokines (IL-1, IL-6, TNFα) are acute phase reactants

Mostly synthesized by liver.

The acute-phase response is considered part of the innate immune system, and APPs play a role in mediating such systemic effects as fever, leukocytosis, increased cortisol, decreased thyroxine, decreased serum iron, and many others.

APPs can be categorized as positive (increasing serum concentration) or negative (decreasing serum concentration).

Increased production of positive acute phase proteins is a sensitive indicator of inflammation.

Positive APPs	Negative APPs
C-reactive protein (CRP)	Albumin
Serum amyloid A (SAA)	Transferrin
Haptoglobin (Hp)	Transthyretin
Ceruloplasmin	Retinol-binding protein
α2-Macroglobulin	
A1-Acid glycoprotein (AGPr)	
Fibrinogen	
Complement (C3, C4)	

17. Which of the following is a negative acute phase reactant? *(AIIMS May 17)*

a. Ferritin
b. Albumin
c. Haptoglobin
d. C-reactive protein

Ans. b. Albumin

Emperipolesis

It is defined "The active penetration of one cell by another which remains intact." It differs from phagocytosis in that an engulfed cell exists temporarily within another cell and with an intact normal structure while in phagocytosis, the engulfed cell is destroyed by the protective action of lysosomal enzymes. The cells involved in Emperipolesis are mainly histiocytes and megakaryocytes but can also be seen in association with tumor cells and Reed Sternberg cells. In addition, plasma cells, myeloid cells, erythroblasts and neutrophils can also be engulfed in histiocytes or megakaryocytes cell cytoplasm.

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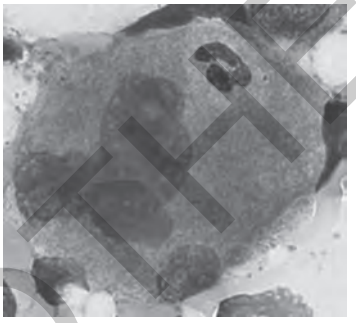
Emperipolesis is a hallmark feature of Rosai – Dorfman disease (RDD) also called Sinus Histiocytosis with Massive Lymphadenopathy (SHML) but can also be part of other malignant conditions such as hematolymphoid disorders (Hodgkin's disease, leukemia, acute and chronic myeloid leukemia, Non-Hodgkin's lymphoma, myeloproliferative disorders, myelodysplastic syndrome) and Non-hematological malignancies (multiple myeloma, neuroblastoma, rhabdomyosarcoma).

The histiocytes in SHML are characterized by round to oval vesicular or hyperchromatic nuclei with distinct nucleoli and abundant amphophilic to eosinophilic granular or clear cytoplasm. These histiocytes show features of antigen presenting cell or Langerhans cell (S100 positive) and phagocytic histiocytes (α1-antitrypsin, lysozymes, CD 68, MAC-378 positivity and CD 1a negativity).

18. Macrophage engulfs different cells as shown in the image, the process is called: *(AIIMS Nov 18)*

a. Phagocytosis
b. Emperipolesis
c. Killing
d. Cytotoxicity

Ans. b. Emperipolesis



(For color version see Fig. 2, Plate 5)

Entosis

Entosis refers to the invasion of one living cell into another of the same type with involvement of adhesion molecules, actin cytoskeleton and expenditure of energy. Entosis is derived from the Greek word that means "inside" or "within", based on the observation that one viable cell invades the other viable cell. Entosis is characteristic for epithelial cells and epithelial cancers, and is triggered by detachment of cells from the basement membrane. As a result of entosis, characteristic cell-in-cell (CIC) structures are formed. Once engulfed, entotic cells can be eliminated through regulated cell death within the entotic vacuole (entosome), via a specific autophagy-related process, commonly known as LC3-associated phagocytosis (LAP). This process is independent of the apoptotic pathway. However, various fates of entosis are possible. The inner entotic cell can undergo division within the host cell or can escape from the host cell, without any signs of degeneration.

19. Cell in cell appearance seen in: *(INI-CET May 22)*

a. Necrosis
b. Apoptosis
c. Necroptosis
d. Entosis

Ans. d. Entosis

Amyloidosis

Approximately 95% of the amyloid material consists of fibril proteins, the remaining 5% being the P component and other glycoproteins. Three most common types are: (1) AL (amyloid light chain) is derived from Ig light chains produced in plasma cells; (2) AA (amyloid-associated) is derived from a unique non-Ig protein synthesized by the liver; and (3) Aβ amyloid is produced from β amyloid precursor protein and is found in the cerebral lesions of Alzheimer disease.

- The AL protein is made up of complete immunoglobulin light chains, the amino-terminal fragments of light chains, or both. Most of the AL proteins analyzed are composed of λ light chains or their fragments, but in some cases κ chains have been identified. The amyloid fibril protein of the AL type is produced from free Ig light chains secreted by a monoclonal population of plasma cells, and its deposition is associated with certain forms of plasma cell tumors.
- The second major class of amyloid fibril protein (AA) does not have structural homology to immunoglobulins. AA fibrils are derived by proteolysis from a larger precursor in the serum called SAA (serum amyloid–associated) protein that is synthesized in the liver and circulates in association with high density lipoproteins. The production of SAA protein is increased in inflammatory states as part of the "acute phase response"; therefore, this form of amyloidosis is associated with chronic inflammation, and is often called secondary amyloidosis.

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- ❖ β-amyloid protein (Aβ) constitutes the core of cerebral plaques found in Alzheimer disease as well as the amyloid deposited in walls of cerebral blood vessels in individuals with this disease. The Aβ protein is derived by proteolysis from a much larger transmembrane glycoprotein, called amyloid precursor protein.
- ❖ Transthyretin (TTR) is a normal serum protein that binds and transports thyroxine and retinol. A mutant form of TTR (and its fragments) is deposited in a group of genetically determined disorders referred to as familial amyloid polyneuropathies. TTR is also deposited in the heart of aged individuals (senile systemic amyloidosis), but in such cases the amino acid sequence of the TTR molecule is normal.
- ❖ β_2-microglobulin, a component of MHC class I molecules and a normal serum protein, has been identified as the amyloid fibril subunit ($A\beta_2m$) in amyloidosis that complicates the course of patients on long-term hemodialysis.

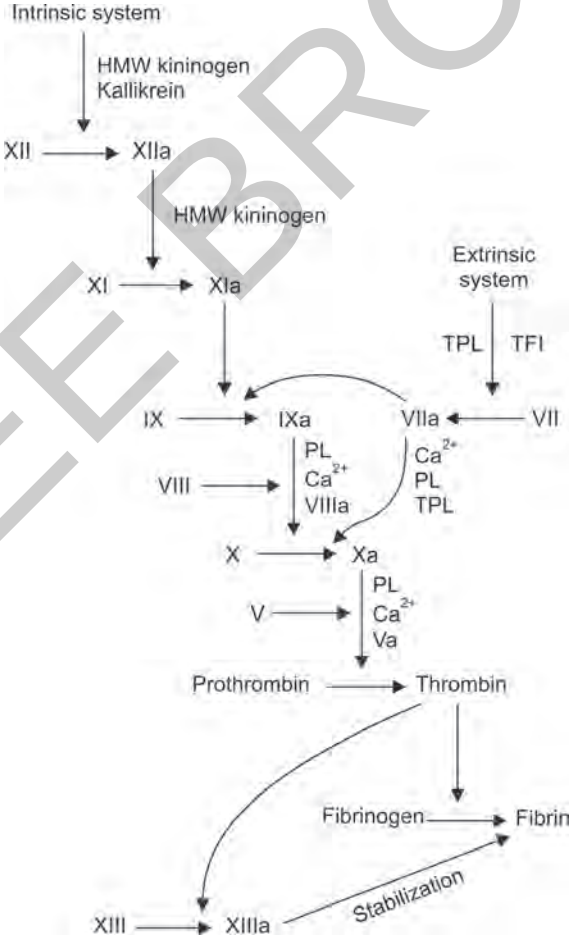
20. True/false about amyloidosis: *(AIIMS May 19)*

a. β_2 macroglobulin forms amyloid in systemic senile amyloidosis
b. In AL type, amyloid is most commonly formed from kappa chain
c. Apple green birefringence is seen when Congo Red stain is used
d. SAA forms amyloid in multiple myeloma
e. Most common cause of amyloidosis is secondary

Ans. a-F, b-F, c-T, d-F, e-F

Hemodynamics

Coagulation Cascade



Clotting involves aggregation of platelets followed by formation of fibrin. Fibrin is derived from fibrinogen with the help of thrombin. Thrombin is formed by activation of prothrombin by factor X and V. Factor X can be activated by either of two systems, known as intrinsic and extrinsic. The initial reaction in the intrinsic system is conversion of inactive factor XII to active factor XII (XIIa). This activation, which is catalyzed by high-molecular-weight kininogen

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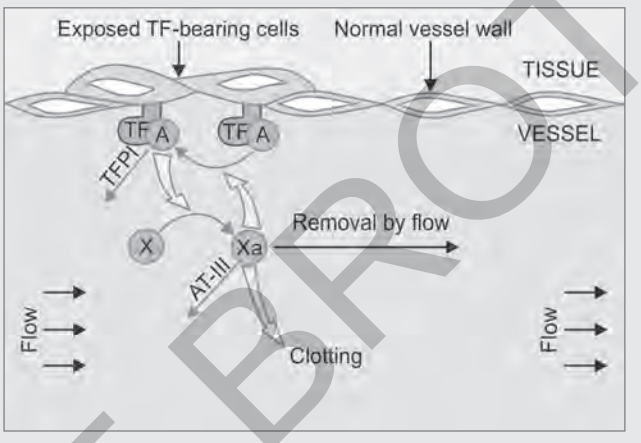
and kallikrein, can be brought about *in vitro* by exposing the blood to glass, or *in vivo* by collagen fibers underlying the endothelium. Active factor XII then activates factor XI, and active factor XI activates factor IX. Activated factor IX forms a complex with active factor VIII, which is activated when it is separated from von Willebrand factor. The complex of IXa and VIIIa activate factor X. Phospholipids from aggregated platelets (PL) and Ca^{2+} are necessary for full activation of factor X. The extrinsic system is triggered by the release of tissue thromboplastin (TPL), a protein–phospholipid mixture that activates factor VII. Tissue thromboplastin and factor VII activate factors IX and X. In the presence of PL, Ca^{2+}, and factor V, activated factor X catalyzes the conversion of prothrombin to thrombin.

21. Identify the sequence in which clotting factors are activated in intrinsic coagulation pathway: *(AIIMS May 19)*

a. XII
b. XI
c. X
d. IX

Ans. a-XII, b-XI, d-IX, c-X

22. The tissue factor that is released during endothelial injury combines with a factor that is labelled as A in the given diagram to initiate the coagulation cascade. A depicts which of the following factors? *(INI-CET June 21)*



a. Factor VIII
b. Factor VII
c. vWF
d. Factor XII

Ans. b. Factor VII

23. Deficiency of which of the following clotting factors do not affect clotting in-vivo? *(INI-CET Nov 20)*

a. V
b. VII
c. VIII
d. XII

Ans. d. XII

Isolated deficiency of factor XII do not cause excessive bleeding as other factors compensate for it.

Coagulation Assays

Based on assays carried out in clinical laboratories, the coagulation cascade has traditionally been divided into the extrinsic and intrinsic pathways.

The prothrombin time (**PT**) assay assesses the function of the proteins in the **extrinsic** pathway (factors VII, X, V, II, and fibrinogen). In brief, **tissue factor**, phospholipids, and **calcium** are added to plasma and the time for a fibrin clot to form is recorded.

The partial thromboplastin time (PTT) assay screens the function of the proteins in the intrinsic pathway (factors XII, XI, IX, VIII, X, V, II, and fibrinogen). In this assay, clotting of plasma is initiated by addition of negative charged particles (e.g., ground glass) that activate factor XII (Hageman factor) together with phospholipids and calcium, and the time to fibrin clot formation is recorded.

Thromboelastography

Thromboelastography is a rapid, point-of-care assay of coagulation function with both platelet and soluble coagulation factors present simultaneously. By not separating the coagulation components and by following clot formation and

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dissolution over time, thromboelastography may provide additional information on coagulation and fibrinolysis. The principle of measurement is based on changes in viscoelastic rotational forces between a wire or pin suspended in a cup of serum and platelets. Clot forms in the specimen cup after addition of calcium and a coagulation activator. The resulting thrombus causes adhesion and resistance to rotation which is measured electromechanically or optically in the continuously rotating system.

24. Rapid assessment of entire coagulation cascade can be performed using which of the following tests? *(INI-CET Nov 20)*

a. PT
b. aPTT
c. Clotting time
d. Thromboelastography

Ans. d. Thromboelastography

25. Which of the following lab test is used to assess the intrinsic pathway of coagulation? *(AIIMS May 19)*

a. aPTT
b. PT
c. Bleeding time
d. Clotting time

Ans. a. aPTT

26. In PT Test, the addition of Ca^{2+} and tissue thromboplastin activates which pathway? *(AIIMS Nov 19)*

a. Extrinsic
b. Intrinsic
c. Fibrinolytic
d. Common

Ans. a. Extrinsic

Passive Venous Congestion of Liver

- Hepatic manifestations of systemic circulatory compromise-passive congestion and centrilobular necrosis can be seen in both left and right-sided heart failure.

Pathology

- Right-sided cardiac decompensation leads to passive congestion of the liver. The liver is slightly enlarged, tense, and cyanotic, with rounded edges. Microscopically there is congestion of centrilobular sinusoids. With time, centrilobular hepatocytes become atrophic, resulting in markedly attenuated liver cell plates.
- Left-sided cardiac failure or shock may lead to hepatic hypoperfusion and hypoxia, causing ischemic coagulative necrosis of hepatocytes in the central region of the lobule (centrilobular necrosis).
- Combination of hypoperfusion and retrograde congestion acts synergistically to cause centrilobular hemorrhagic necrosis. The liver takes on a variegated mottled appearance, reflecting hemorrhage and necrosis in the centrilobular regions. This finding is known as nutmeg liver due to its resemblance to the cut surface of a nutmeg.

27. A liver biopsy reveals following findings. What is true about this condition? *(AIIMS Nov 15, INI-CET Nov 21)*



(For color version see Fig. 3, Plate 6)

a. Nutmeg liver with dark areas of perivenular dead hepatocytes and gray areas of periportal viable hepatocytes
b. Nodular regenerative hyperplasia of liver induced due to OCPs
c. Nutmeg liver with pale areas of necrosis and dark congested areas of perivenular viable hepatocytes
d. Cirrhotic liver with fibrotic nodules.

Ans. a. Nutmeg liver with dark areas of perivenular dead hepatocytes and gray areas of periportal viable hepatocytes

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Genetics

Chromosomal Translocations

These are DNA rearrangements in which a segment of DNA breaks away from one chromosome and attaches to another. The rearranged chromosomes are called derivative (der) chromosomes. There are two types—reciprocal and Robertsonian translocations.

Reciprocal Translocations

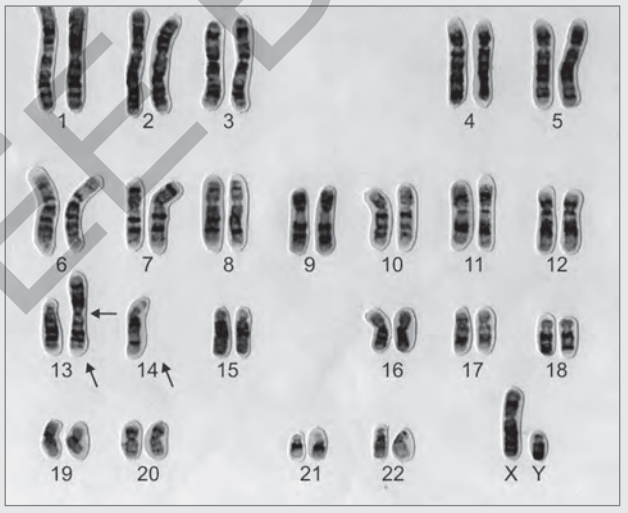
A double-segment or reciprocal translocation begins when breaks occur in two different chromosomes. The broken fragments are then exchanged, so that each affected chromosome contains a fragment of the other. If no chromosomal material is gained or lost in this process, the translocation is considered balanced. Although the balanced translocation carrier is usually normal phenotypically, repositioning of specific genes within chromosomal segments can cause abnormalities.

Balanced translocation carriers are at risk to produce unbalanced gametes, resulting in abnormal offspring. If an oocyte or sperm contains a translocated chromosome, fertilization results in an unbalanced translocation—monosomy for part of one affected chromosome and trisomy for part of the other.

Robertsonian Translocations

These involve only *acrocentric* chromosomes, which are chromosomes 13, 14, 15, 21, and 22. The acrocentric chromosomes have extremely short p arms. In a Robertsonian translocation, the q arms of two acrocentric chromosomes fuse at one centromere to form a derivative chromosome. The other centromere and both sets of p arms are lost. Fortunately, the p arms of the acrocentric chromosomes—the satellite regions—contain redundant copies of genes that code for ribosomal RNA. As these are present in multiple copies on other acrocentric chromosomes, their loss does not affect the translocation carrier, who is usually phenotypically normal. Robertsonian translocations are found in 1 in 1000 individuals. The most common Robertsonian translocation is der(13;14)(q10;q10), which accounts for up to 20% of cases of Patau syndrome (Trisomy 18—Edwards Syndrome).

28. The mutation shown in the given karyotype occurs in which of the following types of chromosomes? *(INI-CET Nov 21)*



a. Acrocentric
b. Metacentric
c. Submetacentric
d. Telocentric

Ans. a. Acrocentric

The karyotype shows an example of Robertsonian translocation.

Chromosome Banding

Chromosome banding techniques produce a series of consistent landmarks along the length of metaphase chromosomes that allow for both recognition of individual chromosomes within a genome and identification of specific segments of individual chromosomes.

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The Next Level of Preparation
REVIEW OF AIIMS (2015-2019) and
INI-CET (2020-2022)



Apurv Mehra MBBS (MAMC) MS ORTHO (UCMS) DNB ORTHO DIP SICOT (Belgium) Fellowship Joint Replacements (Germany, Australia, Malaysia) is the Leading Faculty of Orthopedics and is recognized Nationally and Internationally as an outstanding teacher, world class scholar and dedicated mentor. He is best known for his stupendous efforts in teaching, motivating and inspiring budding doctors. The quality that sets him apart is his unique style of teaching with fun and passion. Dr Apurv Mehra is the Founder of e-Conceptual – No. 1 Online Platform for Residency and Super Specialty Training Programs, Conceptual Orthopedics and Conceptual Surgery. He is also the Co-Founder of Cerebellum Academy, An Institute formed for the students by the teachers, where the Guiding Principle is "Students First" and motto of Cerebellum is to provide Simple Complete, Relevant, Structured and Revisable content for students preparing for NEET PG/NEXT INI-CET & FMGE Exams. Dr Apurv Mehra is a youth icon, guide, mentor and role model for aspiring doctors. He is known for his highly inspirational and motivational Speeches. Inspired from his lectures, students not only "learn" but actually follow Dr Apurv Mehra and also implement his advice and guidance in their day to day lives.



Thameem Saif MD (Medicine) is the Leading Faculty of Medicine and a renowned teacher head in very high regard by medicos both in India and the US. He is an intellect of high order with brilliant teaching skills. His input have been found to be very valuable by his students across the country and he has helped lots of students achieve their dreams of clearing the tough Indian Medical PG Entrance Exam and the USMLE. Most of the toppers in the country today thank him in their heart for playing an important role in their success. He is well-known for solving MCQs in an organized approach to reach towards the answer. His Orientation about how to approach MCQs with latest patterns proposed for National Eligibility Entrance Test is an important aspect of his session.



Vishnu Prasad MBBS MD DNB MNAMS did his MD in Radiodiagnosis from the prestigious All India Institute of Medical Sciences, New Delhi, India. With special interests in teaching and coaching, he is currently pursuing a superspecialty degree in Interventional Radiology at Kovai Medical Center, Coimbatore, Tamil Nadu, India. He cleared the AIIMS UG and PG and NEET-SS examinations in his first attempt with single digit ranks. He has used his expertise in identifying the hot topics asked in entrance examinations to compile all must know topics into the book– INI-CET – the precise version.



Akhil Monga MD (Radiodiagnosis) (AIIMS, New Delhi) Fellow Neuro and Vascular Interventional Radiology cleared his AIIMS PG MEE in November 2015. It was the exam that saw the introduction of the image-based questions. He proved then, that as long as you have strong concepts and strong hold on the commonly asked topics, you shall not be surprised by any new element in the exam. Ever since, he has been actively following the exam pattern and developing intuitive study material for the online platform "MedMiracle". Now, with the introduction of the 7-pattern of questions in the AIIMS entrance exam, he along with Dr Vishnu Prasad has come up with this book to emphasize that though practicing new MCQs and understanding the scoring pattern of these questions is important, the subject matter needed to do a focused revision is still similar. The goal is to reduce the "last-month anxiety" of revising 19 subjects before the exam.

Printed in India

Available at all medical bookstores
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JAYPEE BROTHERS
Medical Publishers (P) Ltd.
EMCA House, 23/23-B, Ansari Road,
Daryaganj, New Delhi - 110 002, INDIA
www.jaypeebrothers.com

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ISBN 978-93-5465-765-8

9 789354 657658