

BDS SECOND PROFESSIONAL EXAMINATION 2007
GENERAL PATHOLOGY AND MICROBIOLOGY
Model Paper (SEQs)

Total No. of SEQs: 15

Total Marks: 45

Time 2 hours 15 min.

Note: 3 Marks for each question.

Note: THE FINAL PAPER WILL BE SIMILAR TO THE MODEL PAPER BUT WILL FOLLOW "TOS" EXACTLY.

Q.1 Write down various steps of healing in a clean uninfected surgically incised wound with reference to changes at different time periods. (3)

Topic Specification: Healing and Repair.

Key of Q.1:

Such a process of healing is also called as Healing by Primary Intention-The incision causes death of a limited number of epithelial and connective tissue cells as well as disruption of epithelial basement membrane continuity-The narrow Incisional space immediately gets filled with clotted blood cells. Dehydration of surface clot forms the well known scab that covers the wound.

-Changes within 24 hours:

Neutrophils start moving towards fibrin clot.

-Changes within 24-48 hours:

Spurs of epithelial cells move from wound margin.

-Changes by day 3:

Neutrophils replaced by macrophages and granulation tissue starts developing.

-Changes by day 5:

Granulation tissue fills the incised gap, neovascularisation is maximum, collagen is abundant, epidermis recovers its normal thickness.

-Changes by 2nd week:

Continued collagen accumulation, prolif of fibroblasts-leucocyte infiltr, oedema and increased vascularity disappears.

-Changes by end of 1st month:

Scar made up of cellular infiltrate, CT devoid of inflam. Infiltrate and is covered by intact epidermis.

Dermal appendages of the area-permanently last.

Reference: Robbins Pathology, 7th Edition, Healing and Repair, Pages 112-113

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- Q.2 A. What are nuclear changes seen in a necrotic cell. (1)**
B. Define steatosis, give morphologic appearance of liver in this condition. (2)

Topic Specification: Cell Injury, Death and Cellular Adaptation.

Key of Q.2:

Key A: (1)
Nuclear changes seen in a necrotic cell are all due to non-specific breakdown of DNA and they are:

Karyolysis:

Basophilia of chromatin fades away presumably due to DNA-ase activity.

Pyknosis:

Nuclear shrinkage and increased basophilia

DNA condenses into solid shrunken basophilic mass.

Karyorrhexis:

Pyknotic or partially pyknotic nucleus undergoes fragmentation and disappears in a day or two.

Key B: (2)
Steatosis or fatty change is abnormal accumulation of triglycerides within parenchymal cells.

Liver:

Gross-There may be no change in mild/ early cases. But with progressive accumulation liver enlarges and becomes increasingly yellow soft and greasy with stretched capsule and starts weighing 3-6 Kg.

Microscopically: fatty changes begins as small vacuoles in the cytoplasm with central nucleus but in progressive accumulation the vacuoles coalesce, become larger and push the nucleus to one side-occasionally rupture of these lipocytes may form lipocysts.

Reference: Robbins Pathology, 7th Edition, Cell Injury, Death and Cellular Adaptation, Part A Pages 21/ Part B Pages 35, 36.

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Q.3 Define Acute inflammation and write down its:

A: three major components. (1)

B: write down process of increased vascular permeability in response to acute inflammation. (2)

Topic Specification: Acute and Chronic Inflammation.

Key of Q.3:

Key A: (1)

Acute inflammation is rapid or immediate response of body to injurious agent, that serve to deliver mediators of host defence, leucocytes and plasma proteins. Its three major components are:

- a. Alteration in vascular caliber leading to an increase in blood flow.
- b. Structural changes in the microvasculature that permit plasma proteins and leucocytes to leave the circulation.
- c. Emigration of leucocytes from micro circulation their accumulation in the focus of injury and their activation to eliminated offending agent.

Key B: (2)

Increased vascular permeability- a hall mark feature of acute inflammation- leading to escape of protein rich fluid (exudates) in extra vascular tissue.

-Loss of proteins from plasma → ↓ intravascular osmotic pressure and ↑ in osmotic pressure → in extra vascular interstitial fluid.

- ↑ Hydrostatic pressure due to increased blood flow in dilated blood vessels → leads to a marked outflow of fluid and its accumulation in interstitium → with net ↑ in extra vascular fluid → oedema.

- Endothelium becomes leaky because of:

- Formation of gaps in venular endothelium.
- Direct endothelial injury → endothelial cell necrosis and detachment.
- Delayed prolonged leakage.
- Leucocyte mediated endothelial injury.
- ↑ Transcytosis
- Leakage from new blood vessels.

Reference: Robbins Pathology, 7th Edition, Acute and Chronic Inflammation Pages Part A 49, Part B 50, 51.

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Q.4 What is a macrophage? Write down its role in chronic inflammation. (1+2=3)

Topic Specification: Acute and Chronic Inflammation.

Key of Q.4:

(1)

Macrophage is a dominant cell of chronic inflammation and is one of the components of mononuclear phagocyte system. This system consists of closely related cells of bone marrow origin blood monocytes and tissue macrophages, diffusely scattered in different organs. Mononuclear phagocytes arise from a common precursor in the bone marrow which gives rise to blood monocytes-from the blood monocytes migrate into various tissues and differentiate into macrophages.

(2)

In chronic inflammation macrophage accumulation persists and is mediated by different mechanisms.

- a. Recruitment of monocytes from the circulation.
- b. Local proliferation of macrophages.
- c. Immobilization of macrophages at the site of inflammation.

Reference: Robbins Pathology 7th Edition, Acute and Chronic Inflammation Pages 79, 80, 81.

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Q.5 Explain Virchow's Triad in thrombosis. (3)

Topic Specification: Haemodynamic Disorders (Thrombo-Embolic Diseases and Shock).

Key of Q.5:

Virchow's Triad reflects three primary influences in thrombus formation which are:

1. Endothelial Injury:

A dominant influence and can by itself lead to thrombosis, particularly in heart and arterial circulation where normally high flow states might otherwise hamper clotting by preventing platelet adhesion or dilating coagulation factors.

Physical loss of endothelial extracellular matrix (ECM) → platelet adhesion and release of tissue factors and local depletion of PGI₂ and P_{as}-however it is important that endothelium need not be denuded or physically disrupted to contribute to development of thrombosis, any perturbation in dynamic balance of the pro and anti thrombotic SH effects of endothelium can influence local clotting SH events.

2. Turbulence of Blood Flow or Stasis:

This factor contributes to arterial and cardiac thrombosis by causing endothelial injury / dysfunction as well as by forming counter currents and local packets of stasis.

Stasis is a major factor in the development of venous thrombi.-stasis or turbulence therefore:

- a. Disrupt laminar flow of blood.
- b. Prevent dilution of activated clotting factors by fresh flowing blood.
- c. Retard inflow of clotting factors inhibitors and permit thrombi formation.
- d. Premature endothelial cell activation.

3. Hypercoagulability:

Contributes less frequently to thrombotic states, but is an important component in the equation-any alteration in the coagulation pathway predisposes to coagulation.

Reference: Robbins Pathology, 7th Edition, Haemodynamic Disorders (Thrombo-Embolic Diseases and Shock).

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- Q.6 A. Define how autosomal dominant disorders are manifested? (1)**
B. Tabulate the system and the disorder caused by autosomal dominant disorders. (2)

Topic Specification: Genetic Disorders.

Key of Q.6:

Key A: (1)
 Autosomal dominant disorders are manifested in the heterozygous state, so at least one parent of an index case is usually affected. If both male and female are affected and both can transmit the condition- if an affected person marries an unaffected one, every child has one in two of having the disease.

Key B: (2)

Autosomal Dominant Disorders

Sr.#	System	Disorders
1.	Nervous	Huntington Disease, Neurofibromatosis, Myotonic Dystrophy, Tuberous Sclerosis.
2.	Urinary	Polycystic Kidney Disease.
3.	Gastrointestinal	Familial Polyposis Coli.
4.	Hematopoietic	Hereditary Spherocytosis, Van-Wille-Brand Disease.
5.	Skeletal	Marfan Syndrome, Ehlers-Danlos Syndrome, Osteogenesis Imperfecta, Achondroplasia.
6.	Metabolic	Familial Hypercholesterolemia, Acute Intermittent Porphyria.

Reference: Robbins Pathology, 7th Edition, Genetic Disorders, Part A Page 150, Part B Page 151.

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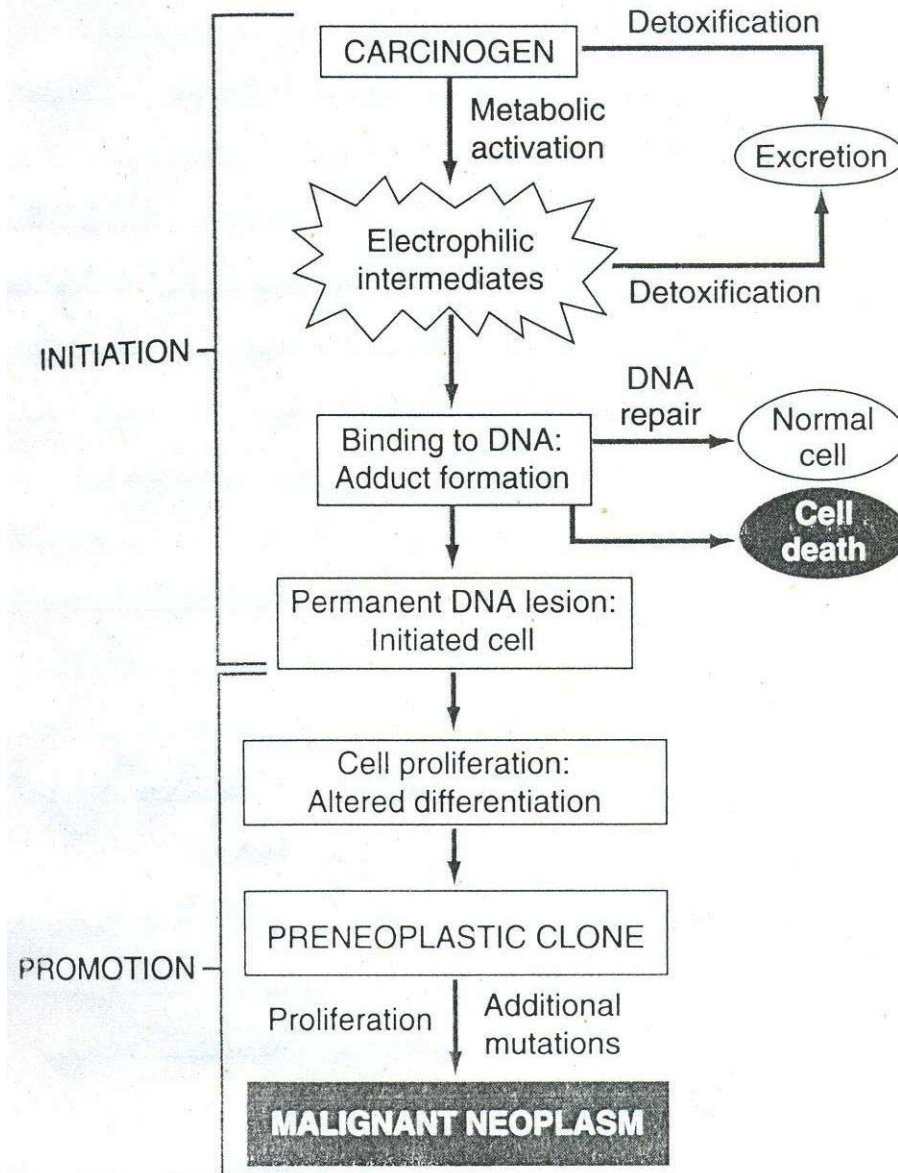
- Q.7 A. Make a flow chart showing general scheme of events in chemical carcinogenesis. (2)**
B: Enumerate any three of DNA oncogenic viruses with reference to malignancies they can cause. (1)

Topic Specification: Neoplasias.

Key of Q.7:

Key A:

(2)



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Key B:

(1)

1. Human Papilloma Virus: (Type 16,18 and less commonly 31, 33, 35, 51).
2. Epstein Barr Virus (EBV): Burkitt's Lymphoma, Nasopharyngeal Carcinoma.
3. Hepatitis B Virus (HBV): Hepatocellular Carcinoma.

Reference: Robbins Pathology, 7th Edition, Neoplasias, Part A Page 321, Part B Pages 324, 325, 326, 327.

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Q.8 Name any six tumor markers, also mentioning the tumors they are used for. (3)

Topic Specification: Neoplasias.

Key of Q.8: (0.5 each)

- 1.** Human Chorionic Gonadotrophin-HCG = Trophoblastic Tumours, Non Seminomatous Testicular Tumours.
- 2.** α -Fetoprotein = Hepatocellular Carcinoma, Non Seminomatous Testicular Tumours.
- 3.** Carcino-Embryonic Antigen-CEA = Carcinoma of Colon, Pancreas, Stomach etc.
- 4.** Prostatic Specific Antigen-PSA = Carcinoma Prostate.
- 5.** CA-125 = Ovarian Cancer.
- 6.** Calcitonin = Medullary Carcinoma Thyroid.

(or any other)

Reference: Robbins Pathology, 7th Edition, Neoplasias, Page 339.

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- Q.9 Compare Th1 and Th2 lymphocytes in terms of the following:**
- A. the cytokines they produce. (1+1)**
 - B. whether they promote cell mediated immunity or antibody production. (1)**

Topic Specification: Immunology.

Key of Q.9:

Key A:

Th1 Lymphocytes Produce Cytokines: (1)

1. Interleukin-2 (IL-2).
2. Interferon-gamma (IFN-gamma).
3. Lymphotoxin.
4. Tumor Necrosis Factor-beta (TNF-beta)

Th2 Lymphocytes Produce Cytokines: (1)

1. Interleukins-2.
2. Interleukis-4.
3. Interleukins-5.
4. Interleukins-10 & 13.

Key B: (1)

Th1-lymphocytes recognize antigens presented by macrophages and function primarily to activate and heighten cell mediated immunity.

Th2- lymphocytes recognize antigens presented by B-lymphocytes. They produce cytokines that primate antibody production.

**Reference: Microbiology and Immunology by Ernest Jawetz,
9th Edition Chapter 58-Page 404-05.**

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- Q.10 State a disease caused by each of the following protozoans and indicate how they are transmitted to humans: (3)**
- a. Entamoeba histolytica.**
 - b. Giardia lamblia.**
 - c. Trichomonas vaginalis.**
 - d. Toxoplasma gondii.**
 - e. Cryptosporidium.**
 - f. Plasmodium species.**

Topic Specification: Virology.

Key of Q.10:

- a.** Entamoeba Histolytica, causes amoebic dysentery and extra intestinal amebiasis (liver abscess). The organism produces protective cysts which pass out of the intestines of infected host and are ingested by the next host (faeco-oral route). (0.5)
- b.** Giardia Lamblia, can cause a gastro-intestinal infection-"giardiasis" – cysts pass out of the intestines of the infected host (fecal-oral route). (0.5)
- c.** Trichomonas vaginalis- infects vagina and male urinary tract- does not produce a cyst and is transmitted sexually. (0.5)
- d.** Toxoplasma Gandii-causes toxoplasmosis, contracted by inhaling or ingesting cysts from the feces of infected domestic cats or by ingesting raw meat of infected animal. Infects brain, heart or lungs of people of immunosuppressed, but is wild in those who have normal immune system-can also be transmitted congenitally and infect the nervous system of infected child. (0.5)
- e.** Cryptosporidium causes diarrhea and is transmitted by fecal oral route. (0.5)
- f.** Plasmodium Species- a haemoparasite and causes malaria of different kinds with different species and transmitted parantarilly by bite of Anopheles Mosquito. (0.5)

**Reference: Text Book of Medical Parasitology CKJ Pamikar, Chapter 5
Page 61-81.**

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Q.11 Describe how certain viruses may contribute to the development of tumours. (3)

Topic Specification: Parasitology.

Key of Q.11:

Virus Plays a Role in Cancer Development Both Directly and Indirectly.

Directly: (1.5)

1. By integrating into the host cell's chromosomes. Some viruses may alter the normal function of the proto-oncogenes and tumour suppressor genes, as is seen the HPV and HBV.
2. In case of cervical cancer, carcinogenic strains of HPV, produces and oncoprotein called E6. two variants of tumor suppressor gene known as P-53 produces a suppressor protein, that is much more susceptible to degradation by E6.

Indirectly: (1.5)

1. The viruses may induce immunosuppression so that cancer cells are removed by immune responses as in the case of HIV/AIDS.
2. They may cause long term damage to tissues resulting I na large scale cell regeneration which increase the chances of natural mutation in proto-oncogenes and tumor suppressor genes, as in the case of HBV and HCV.

Reference: Microbiology and Immunology, Ernest Jawetz, 9th Edition, Chapter 43, Page 306.

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Q.12 A. Define the following:

1. Pathogenicity. (1)

2. Virulence. (1)

B. Name four things an organism must be able to do, to cause an infectious disease. (1)

Topic Specification: Microbiology General.

Key of Q.12:

Key A:

Pathogenicity and virulence are terms that refer to an organism's ability to cause disease.

Pathogenicity is used with respect to differences between microbial species. (1)

Virulence denotes differences between strains of the same species- in practice they are often used interchangeably. (1)

Key B:

To cause disease an organisms must:

1. Maintain a reservoir before and after infection (humans, animals, environment etc.).
2. Leave the reservoir and gain access to the new host.
3. Colonize the body, and
4. Harm the body.

**Reference: Microbiology and Immunology, Ernst Jawetz, 9th Edition
Chapter 07.**

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- Q.13 A female infant was delivered by a midwife at home, within one day of birth she develops meningitis and dies next day.**
- a. Name 2 most common organisms which are most likely to cause this disease. (1)**
 - b. Name 3 risk factors which increase the chances of a new born acquiring this infection. (1)**
 - c. Name 3 laboratory tests which can be helpful in identifying the causative agent. (1)**

Topic Specification: Microbiology General.

Key of Q.13:-

- a:** (1)
- 1. Group B-beta hemolytic streptococci.
 - 2. E. coli.
- b:** (1)
- 1. Prematurity.
 - 2. Prolonged rupture of membranes.
 - 3. Group B-streptococcal carriage in birth canal.
- c:** (1)
- 1. Gm. stain of CSF.
 - 2. Culture of CSF.
 - 3. Blood culture.

Reference: Microbiology and Immunology Ernst Jawetz, 9th Edition, Chapter 15.

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- Q.14 A. Describe 2 characteristics which make mycobacteria leprae different from other mycobacteria. (1)**
B. Differentiate between tuberculoid and lepromatous leprosy. (1)
C. Name 2 causes of responsible for disfigurement in leprosy. (1)

Topic Specification: Microbiology Systemic (Special)

Key of Q.14:

Key A: (1)

It is a strict parasite that has not been grown in artificial media or tissue culture.

It is slowest growing of all the species.

Key B: (1)

Sr.#	Features	Tuberculoid Leprosy (TL)	Lepromatous Leprosy (LL)
1.	Type of lesion	One or few macular depigmented lesions with little tissue damage	Many erythematous and granulomatous skin lesions with masked tissue damage
2.	Number of acid fast bacilli	Few	Many
3.	Likelihood of transmitting leprosy	Low	High
4.	Cell mediated response to M. leprae	Present	Reduced or absent
5.	Lepramin Test	Positive	Negative

Key C: (1)

1. Loss of sensations results in burns and trauma.
2. Resorption of bones results in loss of nose and finger tips.
3. Infiltration of skin and nerves results in thickening and folding.

Reference: Microbiology and Immunology, Ernst Jawetz, 9th Edition, Chapter 21, Pages 167-168.

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- Q.15 Write briefly the mechanism of sterilization by:**
- A. Autoclaving. (1.5)**
- B. Pasteurization. (1.5)**

Topic Specification: Microbiology Systemic (Special).

Key of Q.15:

Key A:

Autoclaving: (1.5)

Most frequently used method of sterilization by moist heat because bacterial spores are resistant to boiling (100°C at sea level) they must be exposed to a higher temperature, which can only be achieved under increased pressure – for this purpose an autoclave chamber is used in which steam at a pressure of 15 lb/in² reaches a temperature of 121°C and is held for 15-20 min. this Clostridium botulinum.

Sterilization by dry heat on the other hand requires temperature in the range of 180°C for 2 hours.

Key B:

Pasteurization: (1.5)

Used primarily for milk, consists of heating the milk to 62°C for 30 min. followed by rapid cooling (Flash Pasteurization at 72°C for 15 seconds is often used). This is sufficient to kill the vegetative cells of the milk born pathogen i.e Mycobacterium Bovis, Salmonella, Streptococcus Listeria and Brucella, but not sterilize milk.

Reference: Medical Microbiology and Immunology, 7th Edition by Ernst Jawetz, Page 86.