NEET MDS General Pathology and Microbiology

Sample Paper – 10

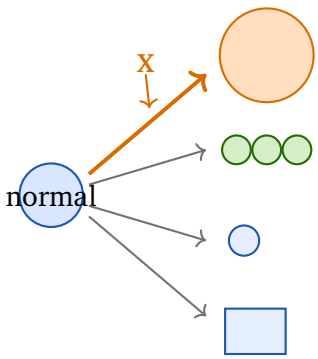
Duration: 11 Minutes **Maximum Marks: 56**

Instructions

- This paper contains **14** Multiple Choice Questions (Single Best Response), modelled on the General Pathology and Microbiology content of **NEET MDS** conducted by NBEMS.
- The **14-question** length matches the number of Pathology and Microbiology items you actually meet in the real 240-question paper.
- Each correct answer carries **+4 marks**. There is **negative marking of 1 mark** for every incorrect answer; unattempted questions carry no penalty.
- Every question has **exactly one best answer**. Choose the single most appropriate option.
- Attempt this practice paper in one timed sitting of about **11 minutes**, the pace the real computer-based test demands.

Cell Injury and Inflammation

Q1. The figure shows a normal cell responding to a persistent stimulus in four different ways. A patient with long-standing nocturnal bruxism has bilaterally enlarged masseter muscles, and biopsy shows fibres of increased individual size with no increase in fibre number. The response marked **X** that this patient shows is:



(A) Hyperplasia

(B) Metaplasia

(C) Hypertrophy

(D) Atrophy

Q2. A patient reports with facial swelling and the laboratory returns a C-reactive protein of 96 mg/L against a reference of under 5 mg/L. The correct interpretation of this raised CRP is that it indicates:

(A) An acute phase response, in which interleukin-6 has driven the liver to raise CRP; it is a sensitive but non-specific marker of inflammation, infection or tissue injury, and it names no cause by itself

(B) A specifically bacterial infection, so that a viral illness or a non-infective inflammatory cause is excluded

(C) A direct measure of the liver's synthetic capacity, comparable to serum albumin

(D) An antibody made by plasma cells against the C polysaccharide of the pneumococcus, and therefore part of adaptive immunity

Q3. Chronic granulomatous disease and Chediak-Higashi syndrome are both defects of neutrophil function, and both present in childhood with severe early periodontal destruction, recurrent oral ulceration and premature loss of teeth. The step that fails in each of them is, respectively:

(A) Failure of the NADPH oxidase respiratory burst, and failure of phagosome-lysosome fusion because of giant defective granules

(B) Failure of rolling adhesion to endothelium, and failure of the respiratory burst

(C) Failure of complement-mediated opsonisation, and failure of chemotaxis alone

(D) Failure of neutrophil production in the marrow, and failure of integrin-mediated adhesion

Neoplasia

Q4. A 55-year-old tobacco chewer has a 3 cm indurated ulcer with rolled everted margins on the lateral border of the tongue, and cervical nodes are palpable. The correct biopsy for this suspected malignancy is:

(A) An excisional biopsy removing the whole lesion with a wide margin, because it diagnoses and treats in a single sitting

(B) An incisional biopsy taking a wedge from the most suspicious part of the margin, including the transition into adjacent normal tissue, because the definitive resection and neck management must be planned on a confirmed histological diagnosis and grade

(C) An incisional biopsy taken from the centre of the ulcer, where the disease process is most advanced

(D) No biopsy at this stage, since exfoliative cytology of the ulcer surface is sufficient to confirm or exclude carcinoma

Q5. A patient two weeks into a 60 Gy course of radiotherapy for an oral squamous cell carcinoma has widespread painful erythema and ulceration of the buccal mucosa, and complains of a persistently dry mouth. The correct account of these findings is:

(A) The mucosal ulceration is early osteoradionecrosis, which characteristically appears in the second week of treatment

(B) The ulceration is radiation mucositis, caused by killing of the dividing basal keratinocytes so that the surface is not replaced as it is shed; it is acute and self-limiting once treatment ends. The dry mouth reflects damage to the radiosensitive serous acini, which is largely irreversible, and osteoradionecrosis of the irradiated mandible remains a late risk for years

(C) The dry mouth is a transient functional effect that always recovers fully within a week of the last fraction, and the ulceration is due to direct radiation damage to submucosal collagen

(D) Mucositis is exclusive to radiotherapy, and cytotoxic chemotherapy does not produce it because it acts systemically rather than on the mucosa

Q6. An odontogenic myxoma of the mandible is reported as histologically benign, yet it has no capsule, permeates the marrow spaces well beyond its radiographic outline and recurs repeatedly after curettage. The principle this illustrates is:

(A) Histological type and biological behaviour are separate axes; a tumour that is benign on histology can still behave in a locally aggressive, infiltrative and recurrent way, so it is treated by resection with a margin, yet it does not metastasise and is not a malignancy

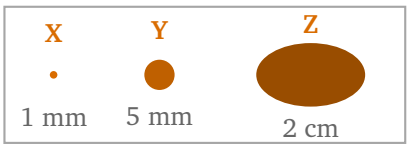
(B) The histological report must be in error, because any tumour that infiltrates is malignant by definition

(C) Repeated local recurrence is itself proof of metastatic potential, so the lesion should be staged as a sarcoma

(D) Behaviour is fully predicted by histology, so a benign report guarantees that curettage alone is adequate

Haemodynamic Disorders and Wound Healing

Q7. The figure compares three haemorrhages into skin and mucosa drawn to scale. Lesion **X** measures about 1 mm, lesion **Y** about 5 mm and lesion **Z** about 2 cm. They are named, in that order:



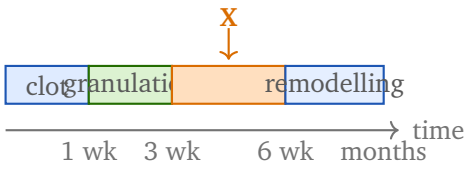
(A) Purpura, ecchymosis and petechia

(B) Ecchymosis, petechia and purpura

(C) Petechia, ecchymosis and purpura

(D) Petechia, purpura and ecchymosis

Q8. The bar below sets out the healing of an extraction socket over the weeks after the tooth is removed. The tissue that occupies the socket at the stage marked **X**, about 4 to 6 weeks after extraction, is:



(A) The organising blood clot, still unreplaced

(B) Granulation tissue alone, with no mineralised tissue yet formed

(C) Fully mature lamellar bone with an established trabecular pattern and a restored cortex

(D) Woven, immature bone laid down by osteoblasts and progressively filling the socket from the base and walls

General Microbiology and Sterilisation

Q9. An autoclave sterilises a load at 121°C in 15 minutes, whereas a hot air oven needs 160°C for 2 hours to sterilise. The reason moist heat achieves sterilisation at a much lower temperature and in far less time than dry heat is that:

(A) Dry heat kills by coagulating cell proteins while moist heat kills by oxidative destruction, and coagulation is the slower of the two processes

(B) Moist heat kills by coagulating and denaturing cell proteins and nucleic acids, which needs far less energy than the oxidative destruction and charring that dry heat depends on; in addition, steam gives up its large latent heat the instant it condenses on the cooler load and it penetrates fabric wrapping, so lethal heat is delivered to every surface quickly and evenly

(C) The raised pressure inside the chamber ruptures the bacterial spores mechanically, and the temperature is incidental to the kill

(D) The autoclave is simply the hotter of the two machines, so its greater temperature accounts for the shorter cycle

Q10. While recapping a needle a dental surgeon sustains a deep percutaneous injury from a hollow bore needle used on a patient whose viral status is unknown. The correct management is to:

(A) Stop the procedure, remove gloves, wash the wound with soap under running water without scrubbing it and without squeezing or sucking it, report and document the exposure at once, obtain consent to test the source patient and the exposed worker, and if the source proves positive or high risk start HIV post-exposure prophylaxis as early as possible, ideally within 2 hours and not beyond 72 hours, while confirming the worker's hepatitis B antibody titre and giving immunoglobulin or a booster as indicated

(B) Squeeze the wound hard to express as much blood as possible, then apply undiluted sodium hypochlorite to the puncture and continue the appointment

(C) Take no action until the source patient's serology has returned, since prophylaxis started before a confirmed result is never justified

(D) Take no further action if the worker has been vaccinated against hepatitis B, since that vaccination also protects against hepatitis C and HIV

Q11. Monitoring of a sterilisation cycle is grouped into physical, chemical and biological categories. What each of the three actually proves is:

(A) All three prove the same thing, that the organisms in the load have been killed, and they differ only in cost

(B) Physical monitoring proves killing, chemical monitoring proves the parameters were reached, and biological monitoring proves only that the pack entered the chamber

(C) Chemical monitoring by autoclave tape is the gold standard, because a colour change can only occur once every organism in the pack is dead

(D) Physical monitoring by gauges, timers and printouts shows that the machine recorded the correct time, temperature and pressure; chemical indicators show that the pack was exposed to those conditions and separate a processed load from an unprocessed one; only the biological indicator, carrying *Geobacillus stearothermophilus* spores, proves that killing was actually achieved

Systemic Bacteriology and Virology

Q12. A child with diphtheria has a greyish adherent membrane across the tonsils and pharynx, and develops myocarditis in the second week. The correct account of *Corynebacterium diphtheriae*, its membrane and its toxin is:

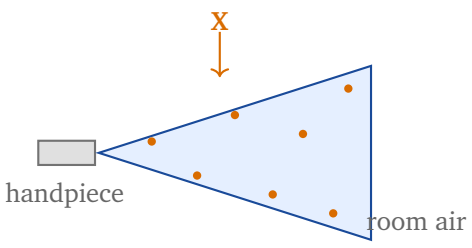
(A) The organism invades the bloodstream and seeds the myocardium directly, and the membrane is simply a collection of pus over the tonsil

(B) The organism remains confined to the mucosal surface and does not invade; its phage-encoded exotoxin blocks protein synthesis by ADP-ribosylating elongation factor 2, and the resulting local necrosis produces a pseudomembrane of fibrin, dead epithelium, leucocytes, red cells and organisms that is firmly adherent and bleeds when peeled off. Toxin absorbed into the blood then reaches the heart and peripheral nerves

(C) The membrane is pure fibrin that wipes off cleanly leaving intact mucosa, and the toxin acts by lysing red cells

(D) The damage is caused by endotoxin released from the lipopolysaccharide of this gram negative bacillus when it dies

Q13. The figure shows the plume marked **X** generated at the tip of a high speed air rotor during a routine cavity preparation. Aerosol generating procedures are a specific hazard in dentistry because:



(A) The plume is made only of large droplets over 100 micrometres, which fall to the floor within a metre, so the risk is confined to the operator's own face

(B) The plume is coolant water from the handpiece line and is therefore sterile, so it carries no organisms at all

(C) Along with the visible spatter, the plume contains droplet nuclei under 5 micrometres that evaporate, remain suspended in the operatory air for long periods and drift well beyond the chair, so respiratory viruses shed by the patient, such as influenza and SARS-CoV-2, can be inhaled by the team and by the next patient long after the procedure has ended

(D) The only organisms of concern in the plume are the blood-borne viruses, and hepatitis B in particular is chiefly acquired by inhaling aerosol

Immunology and Hypersensitivity

Q14. Innate and adaptive immunity are separated on three counts, namely speed, specificity and memory. The statement that sets them apart correctly on all three is:

(A) Innate immunity is slow to start, is specific for individual epitopes and leaves lasting memory, while adaptive immunity is immediate and broadly non-specific

(B) Innate immunity takes days to develop and recognises single epitopes, while adaptive immunity acts within minutes and recognises broad microbial patterns

(C) Innate immunity acts within minutes to hours, recognises broad conserved microbial patterns through a fixed set of germline-encoded receptors, and leaves no memory, so a second exposure is met in exactly the same way as the first; adaptive immunity takes days on first exposure, is specific for individual epitopes through receptors generated by gene rearrangement, and leaves memory, so a second exposure is met faster and more strongly

(D) Both arms leave memory and both are equally specific; they differ only in how quickly they act

Detailed Solutions

Q1.

Solution

Concept – the four cellular adaptations are told apart by what changes: A cell under persistent stress does not simply die. It resets to a new steady state, and there are only four ways it can do so. The trick is to read the scenario for *what* changed: cell size, cell number, or cell type.

Step 1 – list the four adaptations: Hypertrophy is an increase in cell **size**. **Hyperplasia** is an increase in cell **number**. **Atrophy** is a decrease in cell size and number. **Metaplasia** is the replacement of one differentiated cell **type** by another.

Step 2 – read the two facts the biopsy gives: The fibres are of **increased individual size**, and there is **no increase in fibre number**. Both facts are given deliberately, and together they permit only one answer.

Step 3 – apply the first fact: Increased cell size is hypertrophy. That eliminates atrophy at once, since atrophy means the opposite.

Step 4 – apply the second fact: No increase in number rules out hyperplasia. This is the fact that separates the two enlargement adaptations, and it is why the examiner supplied it.

Step 5 – confirm with the tissue involved: Skeletal muscle is a **permanent** tissue. Its cells left the cell cycle after birth and cannot divide, so muscle physically **cannot** undergo hyperplasia. Any enlargement of a muscle must be hypertrophy. Bruxism is a chronic increased workload, which is the classic stimulus for hypertrophy.

Step 6 – read the figure: Arrow **X** points to a single cell that has become larger, with no extra cells drawn beside it. That is hypertrophy, and it matches the biopsy exactly.

Why the other options are wrong:

- A, hyperplasia: is an increase in cell **number**, drawn in the figure as the row of several small cells. The biopsy explicitly states the fibre number is unchanged, and skeletal muscle cannot divide in any case.
- B, metaplasia: is a change of cell **type**, drawn as the square cell. The masseter fibres are still muscle fibres, so no cell type has been replaced.
- D, atrophy: is a **reduction** in cell size, drawn as the shrunken cell. It is the exact reverse of what the biopsy shows, and it follows disuse, denervation or loss of blood supply, not increased load.

Final Answer: Hypertrophy $\Rightarrow$ $\boxed{\text{C}}$

Answer: (C) Go Back to Q1

Q2.

Solution

Concept – CRP is an acute phase reactant, not a diagnosis: The acute phase reactants are plasma proteins whose concentration changes sharply when inflammation is present anywhere in the body. Reading them correctly means knowing what they can and cannot tell you.

Step 1 – define the acute phase response: Inflammatory cells release the cytokines **IL-6**, IL-1 and TNF. These travel to the liver, where IL-6 in particular reprograms hepatocyte protein synthesis.

Step 2 – name the proteins that rise: The **positive** acute phase reactants rise. They include **CRP**, serum amyloid A, fibrinogen, haptoglobin, ferritin and complement components. CRP is the one used clinically because it rises fastest and falls fastest.

Step 3 – name the proteins that fall: The **negative** acute phase reactants fall, chiefly **albumin** and transferrin, because the liver diverts its synthetic effort. This is why albumin drops in any prolonged illness.

Step 4 – state what CRP actually does: CRP binds phosphocholine on microbial surfaces and on dying cells, and having bound it acts as an **opsonin** and fixes complement by the classical pathway. It is an innate molecule made in the liver, produced within hours and needing no prior exposure.

Step 5 – interpret the value given: 96 mg/L against a reference under 5 mg/L confirms that a significant inflammatory process is present. It says nothing about **where** that process is or **what** is causing it. It is sensitive, not specific.

Step 6 – state the clinical use: Because CRP has a short half-life of about 19 hours, it tracks the disease in near real time. A CRP that falls day by day after drainage of a fascial space infection tells you the treatment is working, which is exactly how it earns its place.

Why the other options are wrong:

- B, specific for bacterial infection: is wrong. Bacterial sepsis does raise CRP most, but trauma, surgery, burns, myocardial infarction, malignancy and rheumatoid arthritis all raise it too. A raised CRP can never exclude a viral or non-infective cause.
- C, a measure of liver synthetic function: is wrong and inverted. Albumin, not CRP, is used that way, and albumin is a **negative** acute phase reactant

that *falls* in inflammation.

- D, an antibody against pneumococcal C polysaccharide: is the deliberate trap in the name. CRP was named because it precipitates the **C** polysaccharide of the pneumococcus, but it is a pentraxin made by hepatocytes, not an immunoglobulin made by plasma cells. It has no antigen specificity and belongs to **innate**, not adaptive, immunity.

Final Answer: A non-specific IL-6 driven acute phase response ⇒ A

Answer: (A) Go Back to Q2

Q3.

Solution

Concept – neutrophil defects are classified by which step of the killing sequence fails: A neutrophil must adhere, emigrate, chemotax, phagocytose, fuse its granules with the phagosome, and finally kill. Each named disorder blocks one specific step, and the question asks you to place two of them.

Step 1 – place chronic granulomatous disease: CGD is a defect of the **NADPH oxidase** complex. The neutrophil adheres, migrates and engulfs the organism perfectly well, but it cannot mount the **respiratory burst**.

Step 2 – follow the consequence in CGD: No NADPH oxidase means no superoxide, therefore no hydrogen peroxide, therefore no hypochlorite from myeloperoxidase. The organism sits alive inside the phagosome. Because the neutrophil cannot finish the job, macrophages wall the organism off, and **granulomas** form. That is where the name comes from.

Step 3 – note the CGD test and organisms: The nitroblue tetrazolium test, or the modern dihydrorhodamine flow assay, fails to show a burst. Infections are with **catalase-positive** organisms such as *Staphylococcus aureus*, *Aspergillus*, *Serratia* and *Nocardia*, because catalase-negative organisms obligingly supply the peroxide the neutrophil lacks.

Step 4 – place Chediak-Higashi syndrome: Chediak-Higashi is a defect of the LYST gene affecting lysosomal trafficking. Granules fuse abnormally with each other into **giant granules** instead of fusing with the phagosome, so the killing enzymes never reach the engulfed organism. Chemotaxis and degranulation are impaired as a consequence.

Step 5 – recognise the other Chediak-Higashi features: The same trafficking defect affects melanosomes and platelet dense granules, giving **oculocutaneous albinism** and a bleeding tendency, with peripheral neuropathy. The blood film showing giant cytoplasmic granules in neutrophils is diagnostic.

Step 6 – explain the shared oral picture: The periodontium depends on a continuous, competent neutrophil traffic through the junctional epithelium into the sulcus. Take that away and plaque organisms are not controlled, so both disorders present with aggressive prepubertal periodontitis, deep pockets, oral ulceration and early exfoliation of teeth. The dentist is often the first to see the disease.

Why the other options are wrong:

- B, adhesion then respiratory burst: names **leucocyte adhesion deficiency** first. LAD is a real disorder, caused by absent CD18 integrins, and it also gives severe periodontitis and delayed cord separation, but it is not CGD. The second half is wrong too, since the burst defect is CGD and not Chediak-Higashi.
- C, opsonisation then chemotaxis alone: describes complement deficiency, which is a humoral rather than a neutrophil function defect. Chemotaxis is impaired in Chediak-Higashi, but the defining lesion is the failure of granule fusion, so "alone" makes the option wrong.
- D, marrow production then adhesion: describes **neutropenia** and LAD. Both CGD and Chediak-Higashi have normal or near-normal neutrophil **counts**. The neutrophils are present but cannot do their work, which is precisely why these are called function defects rather than number defects.

Final Answer: NADPH oxidase failure, and phagosome-lysosome fusion failure $\Rightarrow$ A

Answer: (A) Go Back to Q3

Q4.

Solution

Concept – the choice of biopsy is decided by the size of the lesion and the suspicion of malignancy: Incisional means taking a representative piece. Excisional means removing the whole lesion. Choosing between them is a standard NEET MDS item.

Step 1 – state the rule for excisional biopsy: Excisional biopsy suits a **small** lesion, usually under about 1 cm, that is **clinically benign**, and where removing it entirely does no harm. The fibroma and the mucocele are typical examples.

Step 2 – state the rule for incisional biopsy: Incisional biopsy suits a **large** lesion, a lesion in a hazardous site, or a lesion **suspected of malignancy**, where the definitive treatment must be planned on histology before anything is removed.

Step 3 – read this lesion: It is 3 cm, indurated, has rolled everted margins, sits

on the lateral tongue in a tobacco chewer, and there are palpable nodes. Every one of those features says carcinoma, so the second rule applies.

Step 4 – explain why excision is wrong here: An "excision" of a 3 cm carcinoma done as a biopsy would be an inadequate cancer operation. It would breach the tumour, disturb the field, and leave the surgeon planning a resection and a neck dissection on a site where the anatomy has already been altered and the true extent obscured. Definitive surgery must follow the diagnosis, not precede it.

Step 5 – explain where to take the incisional specimen: From the **margin** of the ulcer, taking a wedge that includes both abnormal tissue and the transition into adjacent normal mucosa. The pathologist needs that interface to judge invasion across the basement membrane and to grade the tumour.

Step 6 – explain why not the centre: The centre of a malignant ulcer is necrotic slough and fibrin. A specimen from there frequently returns as "necrotic debris and inflammation" and forces a second biopsy, delaying treatment in a disease where weeks matter.

Why the other options are wrong:

- A, excisional biopsy with a wide margin: confuses biopsy with treatment. It is correct only for small clinically benign lesions, and here it would compromise the definitive resection.
- C, incisional biopsy from the centre: chooses the right technique but the wrong site. The centre yields necrotic tissue and no assessable invasive front.
- D, exfoliative cytology alone: is unacceptable for a suspected carcinoma. Cytology samples only surface cells, cannot demonstrate **invasion** through the basement membrane, and a negative result would never be allowed to overrule a clinical picture this strong. Cytology is a screening adjunct; a histological biopsy is the definitive investigation.

Final Answer: Incisional biopsy from the margin including adjacent normal tissue $\Rightarrow$ B

Answer: (B) Go Back to Q4

Q5.

Solution

Concept – radiation injures the tissues that divide fastest first, and the tissues that repair slowest last: The oral complications of cancer therapy divide neatly into acute effects on rapidly dividing cells and late effects on the vasculature and stroma.

Step 1 – explain the mucositis: Oral epithelium turns over roughly every 10 to 14 days, driven by the dividing **basal keratinocytes**. Radiation kills dividing cells, so replacement stops while surface loss continues. Around the end of the second week the epithelium thins, then reddens, then ulcerates. The timing in the question fits exactly.

Step 2 – describe the course of mucositis: It peaks toward the end of the course, is intensely painful, limits eating and may force a treatment break. Once irradiation stops, the surviving basal cells repopulate and the mucosa heals over roughly 2 to 4 weeks. It is therefore an **acute and self-limiting** reaction.

Step 3 – explain the xerostomia: The **serous** acini of the salivary glands are unexpectedly radiosensitive despite being slowly dividing, so the parotid suffers most. Doses above about 30 Gy cause acinar loss and fibrosis. Unlike the mucosa, the glands do not repopulate, so the dry mouth is **largely permanent**.

Step 4 – follow the consequences of xerostomia: Loss of saliva removes buffering, clearance and antimicrobial proteins. The result is rampant radiation caries at the cervical margins, candidiasis, loss of taste and difficulty wearing dentures.

Step 5 – explain osteoradionecrosis: Irradiated bone becomes **hypocellular, hypovascular and hypoxic**. It cannot mount a healing response, so an extraction or an ulcer can expose bone that never heals. The mandible is affected far more than the maxilla because its blood supply is poorer. This is a **late** complication, appearing months to years later, which is why extractions are done **before** radiotherapy begins.

Step 6 – add chemotherapy: Cytotoxics, especially methotrexate and 5-fluorouracil, are antimitotic and hit the same dividing basal layer, so chemotherapy causes mucositis in its own right. Chemotherapy also causes myelosuppression, so the ulcerated mucosa becomes a portal for sepsis in a neutropenic patient.

Why the other options are wrong:

- A, calling the ulceration osteoradionecrosis: mistakes an acute mucosal reaction for a late bony one. Osteoradionecrosis presents as exposed non-healing **bone** months to years afterwards, not as mucosal erythema in week two.
- C, transient xerostomia and collagen damage: is wrong on both halves. Serous acinar loss is largely irreversible rather than recovering in a week, and mucositis arises from killing of dividing basal keratinocytes, not from damage to submucosal collagen.
- D, mucositis exclusive to radiotherapy: is plainly wrong. Chemotherapy-induced mucositis is common and can be severe, precisely because cytotoxic drugs target dividing cells wherever they are.

Final Answer: Acute mucositis, largely irreversible xerostomia, late osteoradionecrosis risk ⇒ B

Answer: (B) Go Back to Q5

Q6.

Solution

Concept – what a tumour is and what a tumour does are two different questions: Histological **type** answers "what tissue is this made of and how differentiated is it". Biological **behaviour** answers "what will it do to the patient". They usually agree, and the lesions worth examining are the ones where they do not.

Step 1 – read the histology: The report says benign. The myxoma consists of bland stellate and spindle cells in an abundant loose myxoid matrix, with no pleomorphism, no atypical mitoses and no anaplasia. On the microscope, nothing is malignant.

Step 2 – read the behaviour: It has no capsule, it permeates the marrow spaces beyond its radiographic outline, and it recurs after curettage. Clinically it is behaving aggressively.

Step 3 – explain how both can be true: Malignancy is defined by **invasion plus the capacity to metastasise**. The myxoma invades locally, but it has no capacity to metastasise. It therefore occupies the intermediate category recognised in surgical pathology: **locally aggressive, benign but non-encapsulated**.

Step 4 – explain why the myxoid matrix matters: The gelatinous matrix has almost no tensile strength, so the tumour flows into cancellous bone rather than pushing it aside. Curettage removes the bulk but leaves tumour cells scattered in the marrow, which is exactly why recurrence rates after curettage alone are high.

Step 5 – state the treatment that follows: Because the behaviour, not the histology, dictates surgery, the myxoma is managed by **resection with a margin of normal bone**, the same principle used for a malignancy, even though the lesion is benign and no neck dissection is required.

Step 6 – generalise the principle: Several oral lesions sit in this intermediate band, including the odontogenic myxoma, the aggressive central giant cell granuloma and the desmoplastic fibroma. In each, the histological label alone would badly under-treat the patient.

Why the other options are wrong:

- B, the report must be wrong: assumes invasion equals malignancy. It does not. Malignancy requires invasion **and** metastatic capacity, and several be-

nign lesions infiltrate without ever metastasising.

- C, recurrence proves metastatic potential: confuses two different things. Local recurrence reflects incomplete removal of an infiltrative lesion; metastasis means colonising a distant site. Neither implies the other, and staging a myxoma as a sarcoma would expose the patient to needless treatment.
- D, behaviour fully predicted by histology: is the exact error the question was written to expose. Trusting the benign label and curetting is what produces the repeated recurrences described in the stem.

Final Answer: Type and behaviour are separate; benign histology can still be locally aggressive $\Rightarrow$ A

Answer: (A) Go Back to Q6

Q7.

Solution

Concept – haemorrhages into skin and mucosa are named purely by size: There is no histological difference between the three. They are the same event, blood leaking out of vessels into tissue, and the name changes only with the diameter. The size then hints at the underlying cause.

Step 1 – define the petechia: 1 to 2 mm, pinpoint. Lesion X at 1 mm is a petechia.

Step 2 – define purpura: Roughly **3 mm to 1 cm**. Lesion Y at 5 mm falls squarely in this band and is purpura.

Step 3 – define the ecchymosis: Larger than about **1 to 2 cm**, the everyday bruise. Lesion Z at 2 cm is an ecchymosis.

Step 4 – read the order the question demands: X then Y then Z, that is smallest to largest, giving petechia, purpura, ecchymosis. Only one option runs in that sequence.

Step 5 – link petechiae to their causes: Petechiae need only the smallest capillaries to leak, so they point to a **platelet** problem or a vessel wall problem: thrombocytopenia, defective platelet function, or raised intravascular pressure. Palatal petechiae are a recognised early sign in a patient with thrombocytopenia, and the dentist may see them first.

Step 6 – link ecchymoses to their causes: Larger bleeds need a larger vessel to leak, so ecchymoses point to trauma or to a **coagulation factor** defect such as haemophilia or warfarin excess. Spontaneous ecchymoses without trauma always demand a coagulation screen before any extraction.

Step 7 – note the colour sequence: An ecchymosis changes colour as haemoglobin is degraded: red-blue, then blue-green as biliverdin forms, then golden-brown as bilirubin and haemosiderin appear. That sequence lets you date a bruise.

Why the other options are wrong:

- A, purpura, ecchymosis, petechia: gives the 1 mm lesion the middle-sized name and the 2 cm lesion is placed second, so the sizes do not run in order.
- B, ecchymosis, petechia, purpura: calls the 1 mm lesion an ecchymosis, which is off by more than a factor of ten.
- C, petechia, ecchymosis, purpura: starts correctly but then swaps the two larger lesions, calling the 5 mm lesion an ecchymosis and the 2 cm lesion purpura. This is the intended trap for a candidate who reads only the first word.

Final Answer: Petechia, purpura, ecchymosis $\Rightarrow$ D

Answer: (D) Go Back to Q7

Q8.

Solution

Concept – an extraction socket heals to a fixed bony timetable: The socket is a bony defect, and the tissues that occupy it change in a set order. Knowing which tissue sits in the socket at which week is standard examinable material.

Step 1 – week 0 to 1, the clot: Torn vessels fill the socket with blood, which clots. The clot is scaffold, not filler: fibrin gives migrating cells a surface to crawl on, and the platelets in it release PDGF and TGF-beta that call in the next cells.

Step 2 – week 1, granulation tissue: Capillaries sprout in and fibroblasts follow, so the clot is replaced by **granulation tissue**. Epithelium migrates across the surface at the same time, closing the orifice.

Step 3 – weeks 2 to 3, connective tissue: The granulation tissue matures into provisional **connective tissue** with collagen laid down. Osteoprogenitor cells derived from the socket walls and the periodontal ligament remnants begin to differentiate into osteoblasts, and the first osteoid appears at the base and walls.

Step 4 – weeks 4 to 6, woven bone: Osteoblasts deposit **woven bone**, laid down quickly with randomly arranged collagen. It progressively fills the socket from the base and walls toward the crest. This is the stage the figure marks as X, so the tissue in the socket at 4 to 6 weeks is immature woven bone.

Step 5 – weeks 6 to 8 onward, remodelling: Osteoclasts resorb the woven bone and osteoblasts replace it with organised **lamellar bone**. Radiographically the socket outline, the lamina dura, gradually disappears.

Step 6 – months 4 to 6, maturity: A full trabecular pattern indistinguishable from the surrounding bone, with a restored cortex across the crest, takes about 4 to 6 months. Alongside this the ridge resorbs in width and height, which is why the timing of implant placement depends on the timetable above.

Step 7 – eliminate on timing: The clot is gone by week 1 and granulation tissue by roughly week 3, so at 4 to 6 weeks neither is present; and lamellar maturity is months away. Only woven bone fits the window.

Why the other options are wrong:

- A, the unreplaced clot: is 4 to 5 weeks too early. The clot is fully replaced within the first week. A clot still present at 6 weeks would be pathological.
- B, granulation tissue with no mineralised tissue: describes about week 1 to 2. By 4 to 6 weeks osteoid has been mineralised and woven bone is well established, so it is too early.
- C, mature lamellar bone with a full trabecular pattern: is far too late. Remodelling from woven to lamellar bone takes months, and a complete trabecular pattern with a restored cortex is a 4 to 6 **month** finding, not a 4 to 6 **week** one. This is the intended trap.

Final Answer: Woven, immature bone filling the socket $\Rightarrow$ $\boxed{\text{D}}$

Answer: (D) Go Back to Q8

Q9.

Solution

Concept – moist heat and dry heat kill by two different mechanisms: The whole comparison between the autoclave and the hot air oven rests on this one difference. Once you know the mechanism, every number follows from it.

Step 1 – state how moist heat kills: Saturated steam kills by **coagulation and denaturation** of structural proteins, enzymes and nucleic acids. Water molecules destabilise the hydrogen bonds holding a protein in shape, so the protein unfolds and precipitates at a comparatively low temperature.

Step 2 – state how dry heat kills: Dry hot air has no water to assist, so it must kill by **oxidation** of cell constituents, essentially a slow charring. Oxidative destruction demands far more thermal energy, which is why the oven must run at

160°C for 2 hours.

Step 3 – add the latent heat argument: When steam at 121°C touches the cooler instrument it **condenses**, and in condensing it releases its large latent heat of vaporisation directly into the surface. That is a very large energy transfer in an instant, which hot air cannot match.

Step 4 – add the volume argument: As steam condenses its volume collapses, drawing more steam onto the load. The load therefore keeps pulling fresh steam onto itself until it reaches chamber temperature.

Step 5 – add the penetration argument: Steam penetrates fabric and paper wrapping and reaches lumens, so every surface of a wrapped pack comes to temperature quickly. Hot air is a poor conductor and heats unevenly, which is another reason the oven needs its long cycle.

Step 6 – explain the role of pressure correctly: Pressure is not itself lethal. Water boils at 100°C at atmospheric pressure, and 100°C does not kill spores. Raising the pressure to 15 psi raises the boiling point so that steam can exist at **121**°C. Pressure is only the means of getting hotter steam.

Step 7 – draw the practical conclusion: The autoclave is the first choice for anything that tolerates moisture, and the hot air oven is reserved for what steam cannot handle or would spoil: glassware, oils, powders and sharp cutting instruments that would corrode.

Why the other options are wrong:

- A, the mechanisms swapped: reverses the two. Coagulation belongs to moist heat and oxidation to dry heat, not the other way round.
- C, pressure ruptures spores mechanically: is false. Spores are not destroyed by 15 psi, which is only about one extra atmosphere. Pressure serves solely to raise the boiling point so that steam can reach 121°C.
- D, the autoclave is simply hotter: is factually backwards. At 121°C the autoclave is **cooler** than the 160°C oven, yet it sterilises in one eighth of the time. That contradiction is the entire point of the question.

Final Answer: Moist heat coagulates protein and steam delivers latent heat and penetrates $\Rightarrow$ B

Answer: (B) Go Back to Q9

Q10.

Solution

Concept – needlestick management is a fixed protocol, and its most time-critical element is prophylaxis: The steps run: first aid, then report, then assess the source, then prophylaxis, then follow up.

Step 1 – first aid, and what not to do: Wash the wound with soap and **running water**. Do **not** scrub, do **not** squeeze, and never suck the wound. Squeezing traumatises the tissue, causes local hyperaemia and may draw virus deeper rather than expelling it, and no evidence supports it. Caustics and neat bleach on the wound are not recommended either. If mucosa or eyes are splashed, irrigate copiously with water or saline.

Step 2 – report and document: Report the exposure immediately to the designated officer. Record the device, the depth, whether the needle was hollow bore and visibly bloodstained, and whether gloves were worn. A deep injury from a blood-filled hollow bore needle is the highest risk category, and this stem describes one.

Step 3 – assess the source: With informed consent, test the source patient for **HBsAg, anti-HCV and HIV**. Test the exposed worker at baseline as well. Note that a rapid HIV result is usually available within the hour, which the timing of prophylaxis depends on.

Step 4 – time HIV prophylaxis correctly: This is the step the question turns on. Antiretroviral PEP should start **as soon as possible, ideally within 2 hours**, and there is no benefit beyond **72 hours**. If the source status is unknown but the risk is significant, PEP is started and then stopped if the source proves negative. Waiting for full serology can waste the window.

Step 5 – manage the hepatitis B exposure: Check the worker's **anti-HBs** titre. A titre of 10 mIU/mL or more means protection and nothing further is needed. If unvaccinated or a non-responder, give hepatitis B immunoglobulin within 24 to 72 hours together with the vaccine series.

Step 6 – manage the hepatitis C exposure: There is no vaccine and no prophylaxis for hepatitis C. Management is surveillance: HCV RNA at 4 to 6 weeks and antibody at 3 to 6 months, with early antiviral treatment if seroconversion occurs.

Step 7 – follow up: Serology is repeated at 6 weeks, 3 months and 6 months. Until cleared the worker practises safe sex and does not donate blood.

Why the other options are wrong:

- B, squeeze and apply hypochlorite: is actively harmful and contrary to guidance on both counts, and continuing the appointment abandons the report-

ing and prophylaxis pathway entirely.

- C, wait for source serology before acting: is the most dangerous option because it wastes the narrow window in which HIV PEP works. Prophylaxis is started on risk assessment and stopped later if the source is negative.
- D, hepatitis B vaccination covers everything: is wrong. The vaccine protects only against HBV. There is no vaccine for HIV or hepatitis C, so a vaccinated worker still needs source testing, HIV risk assessment and HCV surveillance.

Final Answer: Wash without squeezing, report, test the source, PEP within 2 to 72 hours $\Rightarrow$ A

Answer: (A) Go Back to Q10

Q11.

Solution

Concept – the three categories of monitoring answer three different questions: They are not three ways of doing the same job. Each one proves something the other two cannot, which is why a proper sterilisation policy uses all three.

Step 1 – physical monitoring, and what it proves: The gauges, the timer, the thermocouple and the printed cycle record. These prove that the **machine** registered the correct temperature, pressure and holding time. They are read at every cycle and are the first thing checked when a cycle fails.

Step 2 – state the limitation of physical monitoring: A chamber gauge reports conditions **at the sensor**, not inside the pack. A correct printout with air trapped in the load is entirely possible, and trapped air stops steam contacting the instruments.

Step 3 – chemical monitoring, and what it proves: Heat- and steam-sensitive inks that change colour on exposure. **External** indicators such as autoclave tape separate a processed pack from an unprocessed one on the shelf. **Internal** indicators placed inside the pack confirm that steam actually reached the contents.

Step 4 – state the limitation of chemical monitoring: A colour change proves **exposure to conditions**, not death of organisms. Autoclave tape in particular reacts to temperature and may turn even where steam penetration failed, so it can never stand as proof of sterility.

Step 5 – biological monitoring, and what it proves: A vial of *Geobacillus stearothermophilus* spores is run through the cycle and then incubated. If the spores do not grow, the cycle killed the most heat-resistant organism available, so everything less resistant is dead too. This is the only category that proves **killing** and therefore the only direct evidence of sterilisation.

Step 6 – state the limitation of biological monitoring: The result takes time to incubate, so the load may already have been used before the answer is known. That is exactly why the fast physical and chemical checks are run on every cycle and the biological check is run periodically, typically weekly, and always after installation, repair or a change in the load.

Step 7 – pair each indicator with its process: *G. stearothermophilus* for moist heat. *Bacillus atrophaeus* for dry heat and for ethylene oxide. The pairing follows from which spore best resists which process.

Why the other options are wrong:

- A, all three prove killing: is wrong. Only the biological indicator demonstrates killing; the other two demonstrate parameters and exposure respectively.
- B, the roles shuffled: assigns killing to physical monitoring, which a pressure gauge cannot possibly show, and reduces the biological indicator to proving the pack entered the chamber, which is the job of external chemical tape.
- C, autoclave tape as gold standard: inverts the hierarchy. Tape changes colour on exposure to heat, whether or not steam penetrated and whether or not organisms died, so it is the weakest of the three, not the strongest. The biological indicator is the gold standard.

Final Answer: Physical shows parameters, chemical shows exposure, biological proves killing $\Rightarrow$ D

Answer: (D) Go Back to Q11

Q12.

Solution

Concept – diphtheria is a toxin disease, not an invasive one: The organism never leaves the mucosal surface. Everything that happens, locally and systemically, is done by a single exotoxin.

Step 1 – identify the organism: *Corynebacterium diphtheriae* is a **gram positive**, non-motile, non-sporing, club-shaped bacillus. It arranges in Chinese letter or cuneiform patterns and shows metachromatic Babes-Ernst granules on Albert stain. It grows on Loeffler's serum slope and on potassium tellurite medium, where colonies turn black.

Step 2 – explain where the toxin comes from: Only strains lysogenised by the **beta phage** carry the *tox* gene and produce toxin. A non-lysogenic strain colonises harmlessly. This is a classic example of phage conversion, and the Elek gel precip-

itation test demonstrates toxigenicity.

Step 3 – explain how the toxin acts: The toxin has two fragments. Fragment B binds the cell and delivers fragment A inside. Fragment A **ADP-ribosylates elongation factor 2**, which halts translocation on the ribosome, so protein synthesis stops and the cell dies. A single molecule of fragment A can kill a cell.

Step 4 – build the pseudomembrane: Local cell death produces necrosis and an inflammatory exudate. Fibrin, necrotic epithelium, leucocytes, red cells and the organisms themselves mat together into a tough greyish-white **pseudomembrane** over the tonsils, pharynx and sometimes the larynx.

Step 5 – explain why it bleeds: The membrane is not sitting on the surface. It is knitted into the underlying necrotic tissue, so peeling it leaves a raw **bleeding** base. That single sign separates it from candidal plaques, which wipe off leaving intact erythematous mucosa. The membrane can also detach and obstruct the airway, which is the immediate danger.

Step 6 – explain the systemic disease: The organism stays put but the toxin is **absorbed into the blood**. It reaches the myocardium, giving myocarditis with arrhythmias in the second week, and the peripheral nerves, giving palatal palsy with nasal regurgitation, then ocular palsy, then a later polyneuropathy. The palatal palsy is often noticed first in the dental chair.

Step 7 – state the treatment principle: Antitoxin must be given **immediately** and on clinical suspicion, because it neutralises only **circulating** toxin and cannot detach toxin already bound to cells. Antibiotics stop further toxin production and clear carriage but do nothing about toxin already released. Prevention is by toxoid in DPT.

Why the other options are wrong:

- A, bloodstream invasion seeding the myocardium: is wrong on the central point. *C. diphtheriae* is non-invasive and remains on the mucosa; it is the **toxin** that circulates. Calling the membrane pus also misses that it is fibrinous and adherent.
- C, membrane wipes off cleanly and the toxin is haemolytic: has both halves wrong. A pseudomembrane that wipes off leaving intact mucosa describes candidiasis, and the diphtheria toxin inhibits protein synthesis rather than lysing red cells.
- D, endotoxin from a gram negative bacillus: fails at the first step. *C. diphtheriae* is **gram positive**, so it has no lipopolysaccharide and no endotoxin. Its damage comes from a secreted **exotoxin**, which is also why a toxoid vaccine works and why an antitoxin can neutralise it.

Final Answer: Non-invasive organism, exotoxin ADP-ribosylating EF-2, adherent bleeding pseudomembrane ⇒ B

Answer: (B) Go Back to Q12

Q13.

Solution

Concept – the hazard of an aerosol is that it does not settle: Dentistry is unusual among clinical specialties in deliberately generating aerosol, at close range, from the airway of a conscious patient. Understanding why that matters means separating spatter from true aerosol.

Step 1 – separate spatter from aerosol by size: Spatter is droplets larger than about 50 to 100 micrometres. They are heavy, travel ballistically and settle within about a metre in seconds. **Aerosol** is particles under about 50 micrometres, and the fraction under **5 micrometres** is what matters.

Step 2 – explain droplet nuclei: A small droplet evaporates in flight, leaving a **droplet nucleus**: the organism plus a shell of dried residue. Nuclei are so light that they follow the room's air currents rather than falling, and they stay suspended for tens of minutes to hours.

Step 3 – explain what the plume contains: Coolant water is only the carrier. The plume marked X mixes coolant with the patient's **saliva, blood, gingival crevicular fluid, plaque, tooth and restorative debris**, and whatever is being shed from the patient's respiratory tract at the time.

Step 4 – explain why size decides where it lands: Particles under 5 micrometres bypass the upper airway filters and deposit in the **terminal bronchioles and alveoli**. That is precisely the tissue tropism of the respiratory viruses, so the aerosol delivers the organism to the site where it establishes best.

Step 5 – name the agents: Influenza, SARS-CoV-2, respiratory syncytial virus, rhinovirus, measles and *Mycobacterium tuberculosis* all transmit by this route. The critical point is that the patient does not need to be symptomatic to be shedding.

Step 6 – explain the specific dental hazard: The operator works about 30 cm from the source for a prolonged period, in a room in which the aerosol accumulates. Because the nuclei remain airborne after the appointment ends, the exposure extends to the assistant, to the reception area if air is shared, and to the **next patient** seated before the air has turned over.

Step 7 – state the controls that follow: The controls follow directly from the physics: high volume evacuation at the source, rubber dam to isolate the field, a pre-procedural mouthrinse to lower the salivary load, an FFP2 or N95 respirator

rather than a surgical mask because a surgical mask does not seal against nuclei, adequate air changes, and a fallow period between aerosol generating patients.

Why the other options are wrong:

- A, only large droplets settling within a metre: describes **spatter** and ignores the aerosol fraction entirely. If this were true, a visor alone would solve the problem and no fallow period would ever be needed.
- B, sterile coolant water: is wrong twice over. The plume is dominated by the patient's own saliva and blood, and dental unit waterlines themselves harbour biofilm, so the coolant is not sterile either.
- D, only blood-borne viruses, hepatitis B by inhalation: misstates the transmission route. Hepatitis B is transmitted **percutaneously and by mucosal contact**, not by inhaling aerosol, and confining the concern to blood-borne viruses ignores the respiratory pathogens that are the actual aerosol hazard.

Final Answer: Sub-5 micrometre droplet nuclei stay suspended and carry respiratory viruses $\Rightarrow$ C

Answer: (C) Go Back to Q13

Q14.

Solution

Concept – innate and adaptive immunity trade speed against precision: The immune system runs two arms because no single design can be both instant and exquisitely specific. Compare them on the three axes the question names and the contrast is clean.

Step 1 – compare on speed: Innate immunity acts within **minutes to hours**, because its effectors are already present and need no manufacture: the epithelial barrier, lysozyme in saliva, complement, neutrophils, macrophages, natural killer cells and the acute phase proteins. Adaptive immunity takes **4 to 7 days** on first exposure, because the rare lymphocyte carrying the right receptor must first find its antigen, then divide repeatedly.

Step 2 – compare on specificity: Innate receptors are **germline-encoded** and fixed, a limited set of a few hundred pattern recognition receptors such as the Toll-like receptors. They read **PAMPs**, conserved structures shared by whole classes of microbes: lipopolysaccharide, peptidoglycan, flagellin, double stranded RNA. One receptor therefore covers many organisms but distinguishes none of them individually.

Step 3 – explain how adaptive specificity is generated: Adaptive receptors

are built by **somatic gene rearrangement** of V, D and J segments, generating a repertoire of the order of 10^{11} distinct specificities. Each lymphocyte carries one specificity, aimed at a single **epitope**. That is why an antibody to one serotype may fail against another.

Step 4 – compare on memory: Innate immunity leaves **no memory** in the classical sense. The thousandth encounter with the same organism is met exactly as the first: same cells, same speed, same magnitude. Adaptive immunity leaves **memory** B and T cells that persist for years.

Step 5 – describe the secondary response: On re-exposure the memory pool is already expanded and pre-selected, so the response is faster with a shorter lag, larger in magnitude, of higher affinity through affinity maturation, and class-switched from IgM to IgG. This is the entire basis of vaccination, and it is something innate immunity cannot offer.

Step 6 – explain how the two arms connect: They are not independent. The innate system decides whether an adaptive response happens at all: a dendritic cell must first recognise a PAMP, mature, migrate to the node and present peptide on MHC with costimulation before a naive T cell will respond. This is why vaccines need adjuvants, which work by supplying the innate danger signal.

Step 7 – apply it clinically: A periapical abscess is contained first by neutrophils within hours, which is innate. The antibody and T cell response that follows over the next week is adaptive. Both arms are working on the same lesion, at different times, on different principles.

Why the other options are wrong:

- A, innate slow, specific and with memory: describes the adaptive arm and attaches the label to the wrong one on all three counts.
- B, the two arms swapped: gives innate the days-long, epitope-specific, memory-forming profile and gives adaptive the immediate non-specific one, which is the reverse of the truth.
- D, both have memory and both are equally specific: is wrong on two of the three axes. Innate immunity leaves no classical memory and reads broad patterns rather than epitopes. If it did have memory, vaccination would work without lymphocytes, and it does not.

Final Answer: Innate is fast, pattern-based and memoryless; adaptive is slow, epitope-specific and remembers $\Rightarrow$ C

Answer: (C) Go Back to Q14

Answer Key

Q	Ans	Q	Ans	Q	Ans	Q	Ans	Q	Ans
1	C	2	A	3	A	4	B	5	B
6	A	7	D	8	D	9	B	10	A
11	D	12	B	13	C	14	C		