NEET MDS General Pathology and Microbiology

Sample Paper – 8

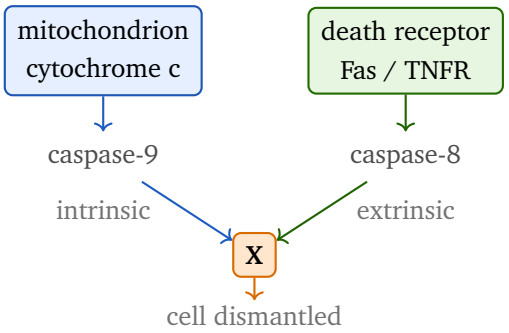
Duration: 11 Minutes **Maximum Marks: 56**

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 diagram below shows the two pathways of apoptosis converging on a common enzyme marked **X**. The enzyme at **X** is:



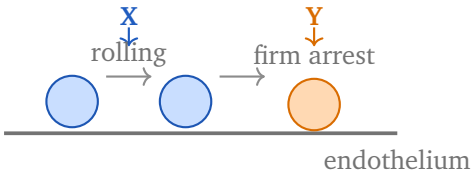
(A) Caspase-1, which cleaves pro-interleukin-1 beta

(B) Bcl-2, which holds the mitochondrial membrane closed

(C) Caspase-8, which only the death receptor limb can activate

(D) Caspase-3, the executioner caspase on which both pathways converge

Q2. In the figure a leucocyte first rolls slowly along the endothelium at step **X**, then stops and adheres firmly at step **Y**. The adhesion molecules responsible for **X** and **Y** respectively are:



(A) Integrins at **X**, and selectins at **Y**

(B) PECAM-1 (CD31) at **X**, and selectins at **Y**

(C) Selectins at **X**, and PECAM-1 (CD31) at **Y**

(D) Selectins binding sialyl-Lewis X at **X**, and integrins (LFA-1, VLA-4) binding ICAM-1 and VCAM-1 at **Y**

Q3. Chronic inflammation is defined by its mononuclear cell infiltrate. Which one of the following pairs a cell of chronic inflammation correctly with what it contributes?

(A) Plasma cell – a terminally differentiated B cell that secretes antibody against the persistent antigen

(B) Neutrophil – the mononuclear cell of chronic inflammation, which sustains it by secreting interferon gamma

(C) Eosinophil – the dominant cell of all chronic inflammation, recruited to every site by interleukin-8

(D) Mast cell – an agranular lymphoid cell whose role is to present antigen to CD4 T cells

Neoplasia

Q4. A man who chewed tobacco for 30 years has been treated successfully for a small squamous cell carcinoma of the lateral tongue, with clear margins. Three years later a fresh, genetically unrelated carcinoma appears on the opposite buccal mucosa. The concept that explains this high risk of a second primary tumour is:

(A) Local recurrence from tumour cells left behind at the original margin

(B) Field cancerisation, in which the whole carcinogen-exposed mucosa is already genetically altered, so several independent clones can transform separately

(C) Lymphatic micrometastasis that seeded the opposite side at the time of the first surgery

(D) Intratumoural heterogeneity within the single original clone

Q5. A 35-year-old man has a painless, slowly enlarging, multilocular radiolucent swelling of the posterior mandible, and biopsy confirms ameloblastoma. It is classified as a benign odontogenic tumour and it does not metastasise, yet it is treated by resection with a bony margin of about 1 to 1.5 cm rather than by simple enucleation. The reason is:

(A) It is locally invasive, infiltrating through the cancellous marrow spaces well beyond the radiographic outline, so enucleation leaves tumour behind and recurrence is high

(B) It seeds the cervical lymph nodes early, so the neck field must be cleared with the specimen

(C) It is in truth a low grade sarcoma of the mandible, and its benign histological appearance is an artefact

(D) It undergoes malignant transformation into osteosarcoma within a few months in every untreated case

Q6. TP53 is the gene most frequently mutated in oral squamous cell carcinoma, and by the time an invasive tumour is established both alleles are usually lost. The direct consequence of losing p53, the guardian of the genome, is:

(A) The cell can no longer enter S phase, so growth arrests permanently

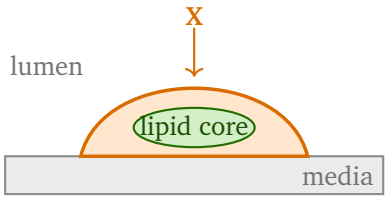
(B) A cell with damaged DNA is neither held in G1 for repair nor pushed into apoptosis, so it divides and passes its mutations to every daughter cell

(C) Telomerase is permanently switched off, so the tumour cells become mortal

(D) p53 begins to act as a growth factor receptor, and mutation raises the number of receptors on the cell surface

Haemodynamic Disorders and Wound Healing

Q7. The figure shows an atheromatous plaque in cross section. Structure **X**, whose thinning and rupture precipitates acute thrombosis, is:



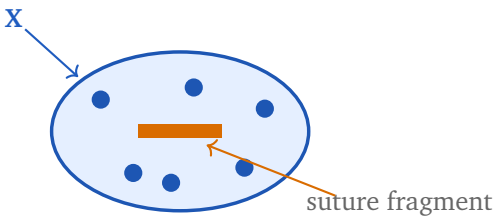
(A) The lipid-rich necrotic core, packed with cholesterol clefts and foam cells

(B) The tunica adventitia with its vasa vasorum

(C) The fibrous cap of smooth muscle cells and dense collagen lying over the core

(D) The internal elastic lamina

Q8. Three weeks after wound closure a firm nodule is excised. Histology shows the cell marked **X** in the figure: a large multinucleate cell with nuclei scattered haphazardly through its cytoplasm, wrapped around a retained refractile suture fragment. The cell at **X** is:



(A) A Langhans giant cell, with its nuclei in a peripheral horseshoe, indicating tuberculosis

(B) An osteoclast recruited from the marrow to resorb the material

(C) A Touton giant cell, as seen in a xanthoma

(D) A foreign body giant cell, formed by the fusion of macrophages that cannot ingest the fragment singly

General Microbiology and Sterilisation

Q9. Chlorhexidine gluconate 0.2 percent is called the gold standard antiplaque mouthwash. The statement that correctly gives both its mechanism and the property responsible for that status is:

(A) It is a dicationic bisbiguanide that binds the anionic bacterial cell wall and causes leakage and precipitation of the cytoplasm, and it is adsorbed onto oral surfaces and released slowly over 8 to 12 hours, a property called substantivity

(B) It is an anionic detergent that emulsifies the biofilm, and its low surface tension is what keeps it working all day

(C) It is a phenolic that alkylates bacterial DNA, and its activity ends the moment the rinse is spat out

(D) It is a quaternary ammonium compound whose whole effect is the mechanical flushing of the rinse, with no binding to oral surfaces at all

Q10. Sodium hypochlorite is used both as an operatory surface disinfectant and as the principal endodontic irrigant. Its action inside the root canal depends on:

(A) Release of hypochlorous acid, which oxidises and chlorinates microbial proteins and also dissolves necrotic pulp tissue and the organic part of the smear layer

(B) Chelation of calcium from the canal wall, which softens dentine and opens the tubules

(C) Dissolution of the inorganic component of the smear layer only, with no effect on organic debris

(D) Formation of a persistent antibacterial film that is left behind deliberately at obturation

Q11. Extracted instruments caked with dried blood must be cleaned before they go into the autoclave. The statement that correctly gives the reason and the role of the ultrasonic bath is:

(A) Cleaning is only cosmetic, since steam sterilises straight through any debris, and the ultrasonic bath merely lubricates the hinges

(B) The ultrasonic bath itself sterilises the instruments, so the autoclave is needed only for wrapped items

(C) Dried blood and debris shield organisms from contact with steam, so the cycle cannot be relied on; the ultrasonic bath removes that debris by cavitation, more thoroughly and far more safely than hand scrubbing

(D) Debris chemically neutralises the steam, and the ultrasonic bath works by holding the solution at 80°C, which kills the organisms outright

Systemic Bacteriology and Virology

Q12. Pus aspirated from an acute dentoalveolar abscess is cultured anaerobically. After 5 days the blood agar carries black-pigmented colonies, and the isolate is a non-motile, asaccharolytic, gram negative anaerobic rod. The organism is:

(A) Prevotella intermedia

(B) Porphyromonas gingivalis

(C) Fusobacterium nucleatum

(D) Streptococcus anginosus (of the milleri group)

Q13. An HIV positive man with a CD4 count of 180 has a white, corrugated, vertically ridged plaque along the lateral border of the tongue. It cannot

be rubbed off and it does not respond to a course of antifungals. This is oral hairy leukoplakia, and the virus responsible is:

(A) Human papillomavirus type 16

(B) Epstein-Barr virus (human herpesvirus 4)

(C) Herpes simplex virus type 1

(D) Human herpesvirus 8

Immunology and Hypersensitivity

Q14. A 52-year-old woman has a dry mouth with rampant cervical caries and gritty, dry eyes. A labial minor salivary gland biopsy shows focal lymphocytic sialadenitis. The autoantibodies most characteristic of this autoimmune disease are:

(A) Anti-double stranded DNA antibody

(B) Anti-desmoglein 3 antibody

(C) Anti-Ro (SS-A) and anti-La (SS-B) antibodies

(D) Anti-mitochondrial antibody

Detailed Solutions

Q1.

Solution

Concept – two pathways, one final executioner: Apoptosis can be triggered from inside the cell or from outside it, but both triggers end in the same enzyme.

Step 1 – read the intrinsic limb of the figure: The mitochondrial pathway begins when Bcl-2 family proteins such as Bax and Bak open the outer mitochondrial membrane. **Cytochrome c** leaks into the cytosol.

Step 2 – follow it to its initiator caspase: Cytochrome c binds Apaf-1 to form the apoptosome, and the apoptosome activates the initiator caspase of the intrinsic limb, which is **caspase-9**. That is the left arm of the figure.

Step 3 – read the extrinsic limb of the figure: The death receptor pathway begins when Fas ligand engages Fas (CD95), or TNF engages TNFR1. The adaptor FADD is recruited, and the initiator caspase of this limb, **caspase-8**, is activated. That is the right arm.

Step 4 – find where the two arms meet: Both caspase-9 and caspase-8 cleave and activate the **executioner caspases**, chiefly **caspase-3**, along with caspase-6 and caspase-7. The convergence point marked X is therefore caspase-3.

Step 5 – state what the executioner does: Caspase-3 cleaves the cytoskeleton, activates the endonuclease that cuts DNA between nucleosomes, and drives the cell to break into apoptotic bodies, which neighbouring cells phagocytose without any inflammation.

Why the other options are wrong:

- A, caspase-1: is an **inflammatory** caspase of the inflammasome. It processes pro-interleukin-1 beta and drives pyroptosis, which is a different, inflammatory form of death and not the convergence point of apoptosis.
- B, Bcl-2: is not an enzyme in this chain at all. It is an **anti-apoptotic** protein that keeps the mitochondrial membrane shut, so it lies upstream of the left arm and it blocks apoptosis rather than executing it.
- C, caspase-8: is correct for the **extrinsic** limb alone, and the figure already names it on the right arm. It cannot be X, because X must be common to both arms.

Final Answer: Caspase-3, the executioner caspase $\Rightarrow$ D

Answer: (D) Go Back to Q1

Q2.

Solution

Concept – each adhesion molecule family does one job: Weak, reversible binding gives rolling. Strong, sustained binding gives arrest. Two different families supply the two strengths.

Step 1 – identify what X requires: Rolling needs bonds that form and break repeatedly, so the cell tumbles along the wall instead of sticking or floating away.

Step 2 – name the molecules that do that: The **selectins**. E-selectin and P-selectin appear on cytokine-activated endothelium, and L-selectin sits on the leucocyte. They bind sialylated oligosaccharides, chiefly **sialyl-Lewis X**, with low affinity, which is exactly why the bond breaks and reforms and the cell rolls.

Step 3 – identify what Y requires: Firm arrest needs a high affinity bond strong enough to hold the cell still against the shear of flowing blood.

Step 4 – name the molecules that do that: The **integrins**, chiefly LFA-1 and Mac-1 on the leucocyte binding **ICAM-1**, and VLA-4 binding **VCAM-1**, on the endothelium.

Step 5 – explain the switch between X and Y: Integrins sit on the resting leucocyte in a low affinity state. Chemokines displayed on the endothelial surface, such as IL-8, signal through the rolling cell and change the integrin to its high affinity conformation. Rolling therefore converts to arrest, and the cell then crawls to a junction and transmigrates.

Why the other options are wrong:

- A, integrins then selectins: reverses the two families. Integrin bonds are far too strong to permit rolling, and selectin bonds are far too weak to hold a cell against shear.
- B, PECAM-1 at X: is wrong twice over. PECAM-1 (CD31) works at neither X nor Y; it mediates the later step of **transmigration** through the endothelial junction.
- C, selectins then PECAM-1: gets X right but Y wrong. PECAM-1 acts after arrest, at diapedesis, not at the moment of firm adhesion.

Final Answer: Selectins for rolling, integrins for firm adhesion $\Rightarrow$ $\boxed{\text{D}}$

Answer: (D) Go Back to Q2

Q3.

Solution

Concept – the cast of chronic inflammation: Four cells matter, and each has one job you are expected to name: the macrophage, the lymphocyte, the plasma cell and the eosinophil.

Step 1 – test option A against the plasma cell: The plasma cell is a B lymphocyte that has been driven to terminal differentiation, usually with T cell help. Its entire function is to secrete antibody, and in chronic inflammation that antibody is directed against the persistent antigen or against altered self components. Option A states exactly this, so it is correct.

Step 2 – confirm the plasma cell fits the mononuclear picture: It has a single eccentric nucleus with a clock-face chromatin pattern and a perinuclear hof, which is the expanded Golgi it needs for antibody export. It is a mononuclear cell, so it belongs in this infiltrate.

Step 3 – place the other true members for contrast: The **macrophage** is the chief effector, presenting antigen and secreting the cytokines and enzymes that both sustain the reaction and destroy tissue. The **lymphocyte** supplies T cell help, with CD4 T cells and macrophages driving each other in a loop. The **eosinophil** is a genuine member, but only in parasitic infection and in IgE-mediated reactions, where eotaxin recruits it.

Step 4 – confirm by elimination: Only option A pairs a cell correctly with what it actually does, so it is the single best answer.

Why the other options are wrong:

- B, the neutrophil: is wrong on both halves. It is a **polymorphonuclear** cell, not a mononuclear one, and it is the cell of **acute** inflammation. Interferon gamma comes from **T cells and NK cells**, not from neutrophils. Neutrophils do persist in some chronic conditions such as chronic osteomyelitis, but they neither define chronic inflammation nor secrete IFN-gamma.
- C, the eosinophil: is wrong because it is not the dominant cell of all chronic inflammation, only of parasitic and allergic reactions. Its chemoattractant is **eotaxin**, not interleukin-8, which recruits neutrophils.
- D, the mast cell: is wrong on both halves. The mast cell is heavily **granular**, and it is a myeloid cell, not lymphoid. Its role is to bind IgE and release histamine, not to present antigen to CD4 T cells.

Final Answer: Plasma cell, a B cell secreting antibody $\Rightarrow$ A

Answer: (A) Go Back to Q3

Q4.

Solution

Concept – the carcinogen treats the whole mouth, not one spot: Tobacco bathes the entire oral mucosa, so the entire mucosa mutates, not only the patch that eventually becomes a lump.

Step 1 – define field cancerisation: Slaughter's concept, described for the oral cavity in 1953: prolonged exposure to a carcinogen leaves a wide **field** of mucosa that already carries genetic damage, even though it looks and feels normal.

Step 2 – draw the consequence: Within that field, several independent clones can accumulate further hits at their own pace. Removing the one clone that reached invasion first does nothing to the rest of the field.

Step 3 – match to the details in the stem: The new tumour is on the **opposite** buccal mucosa, well away from the tongue, and it is described as **genetically unrelated**. Both facts say it arose from a separate clone in the damaged field, which is a second primary and not a spread of the first.

Step 4 – note the clinical weight: Second primaries develop in roughly 3 to 5 percent of treated oral cancer patients per year, and the risk keeps accruing. This is why the whole mouth must be inspected at every follow-up visit, and why tobacco cessation is treatment, not advice.

Why the other options are wrong:

- A, local recurrence: is excluded by the stem, which states the margins were clear. Recurrence also arises **at or near the original site** and is genetically identical to the first tumour, whereas this lesion is at a distant site and genetically unrelated.
- C, lymphatic micrometastasis: would produce a deposit in a **lymph node**, not a fresh mucosal carcinoma on the opposite cheek. A metastasis is also a clone of the original tumour, so it could not be genetically unrelated.
- D, intratumoural heterogeneity: describes subclonal variation **inside one tumour**. It explains why parts of a tumour respond differently to treatment, and it says nothing about a new tumour appearing at a separate site.

Final Answer: Field cancerisation ⇒ B

Answer: (B) Go Back to Q4

Q5.

Solution

Concept – benign and harmless are not the same word: Ameloblastoma is the classic example of a tumour that is benign by definition yet destructive in behaviour.

Step 1 – confirm why it is benign: It never metastasises in its ordinary form. Metastasis is the one criterion no benign tumour meets, so ameloblastoma stays on the benign side of the line. Its cells are also cytologically bland, with no anaplasia and few mitoses.

Step 2 – state the behaviour that spoils the picture: It is **locally invasive**. Tumour islands push out into the cancellous marrow spaces of the mandible, and they run ahead of the edge you can see on the radiograph, typically by several millimetres.

Step 3 – draw the surgical conclusion: If you enucleate or curette to the visible edge, you leave those islands behind. Recurrence after enucleation runs at roughly 50 to 90 percent for the solid multicystic type, while resection with a 1 to 1.5 cm margin brings it down to about 5 to 15 percent.

Step 4 – put the paradox in one line: Ameloblastoma is benign because it does not metastasise, but it is treated aggressively because it invades locally. The margin is dictated by **invasion**, not by malignancy.

Step 5 – note the rare exception, so it does not confuse you: Metastasising ameloblastoma and ameloblastic carcinoma exist, but they are separate rare entities. They do not change the behaviour of the ordinary tumour described here.

Why the other options are wrong:

- B, early nodal seeding: contradicts the stem, which states the tumour does not metastasise. Neck dissection plays no part in the management of a conventional ameloblastoma.
- C, a low grade sarcoma: is wrong on origin and on classification. Ameloblastoma is an **epithelial** odontogenic tumour derived from the dental lamina and enamel organ remnants, and its benign histology is real, not an artefact.
- D, transformation into osteosarcoma: does not happen. Ameloblastoma is epithelial and osteosarcoma is mesenchymal, so one cannot become the other, and there is no such routine sequence.

Final Answer: Locally invasive beyond the radiographic margin $\Rightarrow$ A

Answer: (A) Go Back to Q5

Q6.

Solution

Concept – what the guardian actually guards: p53 is the checkpoint that stands between a damaged cell and the next cell division. Remove it and damaged cells keep dividing.

Step 1 – recall what normal p53 does when DNA is damaged: p53 protein levels rise, because the damage signal stops MDM2 from degrading it. The accumulated p53 acts as a transcription factor.

Step 2 – list its three outputs: It transcribes **p21**, which inhibits the cyclin-CDK complexes and arrests the cell in **G1**, buying time for repair. It upregulates repair genes such as GADD45. If the damage is beyond repair, it transcribes **Bax** and PUMA and drives the cell into apoptosis.

Step 3 – delete p53 and follow through: With both alleles lost, no p21 is induced, so there is no G1 arrest. No Bax or PUMA is induced, so there is no apoptotic exit. The damaged cell simply proceeds into S phase and replicates its damaged template.

Step 4 – state the consequence for the tumour: Every daughter inherits those mutations and adds its own. Genomic instability builds up, and this is the engine that carries a dysplastic oral clone forward to invasive carcinoma.

Step 5 – fix the gene class in your mind: TP53 is a **tumour suppressor**, so it obeys the two-hit rule and is lost, not gained. That is why option B is phrased as a loss of braking and not as a gain of drive.

Why the other options are wrong:

- A, permanent arrest before S phase: is the exact opposite of the truth. Arrest is what **intact** p53 imposes; losing it removes the arrest and lets the cell into S phase.
- C, telomerase switched off: is backwards. Oral carcinoma cells **reactivate** telomerase and become immortal. p53 loss removes a barrier to immortalisation, and it certainly does not restore mortality.
- D, p53 acting as a receptor: misstates the protein. p53 is a nuclear **transcription factor**, not a surface receptor, and it is a suppressor, not an oncogene, so mutation causes loss of function rather than an amplified signal.

Final Answer: No G1 arrest and no apoptosis, so mutations are passed on $\Rightarrow$ B

Answer: (B) Go Back to Q6

Q7.

Solution

Concept – a plaque is a soft core under a hard lid: Read the atheroma as two parts, and the clinical behaviour follows from which part fails.

Step 1 – name the two components: A **fibrous cap** on the luminal side, made of smooth muscle cells that migrated from the media plus the dense collagen and matrix they secreted, and beneath it a **necrotic lipid core** of cholesterol, cholesterol clefts, foam cells and cell debris.

Step 2 – read the figure: X is marked at the luminal surface of the plaque, on the layer lying over the labelled lipid core. That layer is the fibrous cap.

Step 3 – explain why the cap is what matters clinically: The core is intensely **thrombogenic**, because it is loaded with tissue factor. The cap is the only thing keeping that core away from flowing blood.

Step 4 – explain how the cap thins: Macrophage foam cells in the shoulder region secrete matrix metalloproteinases that digest the collagen, while interferon gamma from T cells stops smooth muscle cells replacing it. Collagen is lost faster than it is made and the cap thins.

Step 5 – complete the sequence: A thin cap ruptures. Blood meets the core, tissue factor fires the coagulation cascade, and an occlusive thrombus forms in minutes. This, and not slow luminal narrowing, is what causes myocardial infarction, so a plaque with a thin cap is called vulnerable regardless of how little it narrows the lumen.

Why the other options are wrong:

- A, the lipid core: is already labelled in the figure as the structure **beneath** X, so it cannot be X. It is also the passive victim in this story: it does not rupture, it is exposed.
- B, the adventitia: is the outermost coat, well away from the lumen and far outside the plaque. It plays no part in cap rupture.
- D, the internal elastic lamina: lies between intima and media, deep to the plaque. Atheroma is an **intimal** disease, so the lamina sits under the lesion rather than over it.

Final Answer: The fibrous cap $\Rightarrow$ C

Answer: (C) Go Back to Q7

Q8.

Solution

Concept – giant cells are told apart by where their nuclei sit: All giant cells are fused macrophages. The arrangement of the nuclei, read together with the clinical setting, names the type.

Step 1 – read the nuclear pattern in the figure: The nuclei are **scattered haphazardly** throughout the cytoplasm, with no ring and no ordered arrangement. That pattern defines the **foreign body giant cell**.

Step 2 – read the clinical setting: A retained, refractile, non-absorbable **suture fragment** in a healing wound. This is the textbook foreign body stimulus.

Step 3 – explain why fusion happens at all: A macrophage can ingest a particle a few micrometres across. The suture fragment is far larger than any single cell, so phagocytosis fails. Frustrated macrophages fuse into one syncytium and attempt to surround and wall off what they cannot swallow.

Step 4 – explain what the giant cell then does: It seals itself against the fragment and releases lysosomal enzymes and reactive oxygen species into the closed space between them, a process called frustrated phagocytosis. Fibroblasts then lay collagen around the whole assembly, producing the firm nodule the stem describes.

Step 5 – note the practical point: The reaction persists as long as the fragment does. Removing the suture removes the stimulus and the nodule settles, which is why excision is both the diagnosis and the cure.

Why the other options are wrong:

- A, the Langhans giant cell: has its nuclei arranged **peripherally in a horseshoe or ring**, which is the opposite of the scattered pattern shown, and it belongs to immune granulomas such as tuberculosis. There is no mycobacterial setting here, only a suture.
- B, the osteoclast: is a multinucleate cell of the same monocyte-macrophage lineage, but it lives on **bone** surfaces in resorption lacunae and resorbs mineralised matrix. It has no role against a soft tissue suture fragment.
- C, the Touton giant cell: has a **ring of nuclei** enclosing a central zone, surrounded by foamy lipid-laden cytoplasm, and it is found in xanthomas and other lipid-rich lesions. Neither the nuclear ring nor the lipid is present here.

Final Answer: Foreign body giant cell $\Rightarrow$ D

Answer: (D) Go Back to Q8

Q9.

Solution

Concept – chlorhexidine wins on staying power, not on raw killing: Plenty of agents kill plaque bacteria in the beaker. Chlorhexidine is the gold standard because it keeps working long after the rinse is gone.

Step 1 – name the molecule correctly: Chlorhexidine is a **bisbiguanide**. It carries two positive charges at oral pH, which is why it is called dicationic.

Step 2 – work out the mechanism from that charge: The bacterial cell surface, with its teichoic acids and lipopolysaccharide, is **negatively charged**. The dicationic molecule binds it electrostatically.

Step 3 – follow the dose-dependent effect: At low concentration it disturbs the membrane and low molecular weight substances such as potassium leak out, which is **bacteriostatic**. At the concentration used in a rinse it precipitates the cytoplasmic contents outright, which is **bactericidal**.

Step 4 – define substantivity: The same charge lets chlorhexidine adsorb onto the anionic surfaces of the mouth, which are the pellicle on enamel, the mucosa and the salivary glycoproteins. It is then released slowly, in active form, over roughly **8 to 12 hours**. That sustained release is substantivity.

Step 5 – connect substantivity to the clinical claim: Because one rinse keeps an effective concentration on the tooth surface for most of the day, twice daily use suppresses plaque regrowth continuously. That is precisely why it outperforms agents that are washed away in minutes, and it is why it is prescribed after periodontal surgery and when a patient cannot brush.

Step 6 – remember the trade-off: Extrinsic brown staining, taste disturbance and, with long use, calculus formation. This is why it is a short-term prescription and not a permanent one.

Why the other options are wrong:

- B, an anionic detergent: is chemically backwards. Chlorhexidine is **cationic**; an anionic detergent such as sodium lauryl sulphate in toothpaste actually **inactivates** chlorhexidine, which is why the two must be spaced apart. Low surface tension also has nothing to do with substantivity.
- C, a phenolic that alkylates DNA: is wrong on class and on mechanism, and its second half denies substantivity, which is the very property being asked about. Alkylation of nucleic acids is how ethylene oxide works, not chlorhexidine.
- D, a quaternary ammonium compound with only a flushing action: names the wrong class, since cetylpyridinium chloride is the quaternary ammonium

rinse. It also denies any binding to oral surfaces, which contradicts the defining property of chlorhexidine.

Final Answer: A dicationic bisbiguanide with substantivity $\Rightarrow$ A

Answer: (A) Go Back to Q9

Q10.

Solution

Concept – hypochlorite is an oxidising agent and a tissue solvent at the same time: Its two useful actions come from the same chemistry, and no other irrigant offers both.

Step 1 – write what happens in solution: Sodium hypochlorite in water equilibrates to **hypochlorous acid** (HOCl) and the hypochlorite ion (OCl^-). Hypochlorous acid is the small uncharged species, so it crosses the bacterial membrane and it is the active killer.

Step 2 – state the antimicrobial action: It **oxidises** sulphydryl groups on essential bacterial enzymes and **chlorinates** amino groups to form chloramines. Metabolism stops and the organism dies. At the concentrations used it also kills spores, fungi and viruses, and it disrupts the biofilm holding the canal flora together.

Step 3 – state the tissue-dissolving action: Hypochlorite breaks peptide bonds and turns proteins into soluble fragments. This dissolves **necrotic pulp remnants**, including tissue in fins, isthmuses and lateral canals that no file can reach, and it dissolves the **organic** part of the smear layer.

Step 4 – state the limit that defines the answer: It does **not** dissolve the inorganic part of the smear layer, which is dentine chips. That is why EDTA is used alongside it: EDTA chelates calcium and clears the inorganic component, and hypochlorite clears the organic one. Together they open the tubules.

Step 5 – note the safety point: It is cytotoxic to periapical tissues. Extrusion beyond the apex causes a hypochlorite accident, with immediate severe pain and rapid swelling, so it must be delivered passively with a side-vented needle and never wedged and pressed.

Why the other options are wrong:

- B, chelation of calcium: describes **EDTA**, not hypochlorite. Hypochlorite has no chelating action at all.
- C, dissolving the inorganic part only: inverts the truth. Hypochlorite dis-

solves the **organic** component and leaves the inorganic component untouched, which is exactly why EDTA is needed as well.

- D, a persistent film left at obturation: is wrong twice. Hypochlorite is unstable and leaves no lasting residue, and any residue must be flushed out with a final rinse before obturation because it interferes with the seal. A substantive residue is the property of chlorhexidine, not of hypochlorite.

Final Answer: Release of hypochlorous acid, which oxidises protein and dissolves organic tissue $\Rightarrow$ A

Answer: (A) Go Back to Q10

Q11.

Solution

Concept – no steriliser can kill what steam cannot touch: Every sterilisation method assumes the agent reaches the organism. Debris breaks that assumption before the cycle even starts.

Step 1 – state why cleaning comes first: Blood, saliva and dentine debris dry into a hard crust. Organisms sheltering under that crust are physically shielded from **contact** with steam, and steam sterilises only what it touches.

Step 2 – add the chemical half of the reason: Dried protein also insulates, so it slows heat transfer, and a heavy bioburden raises the load the cycle must overcome. A cycle validated for clean instruments cannot be assumed to work on dirty ones.

Step 3 – explain how the ultrasonic bath works: High frequency waves, around 25 to 40 kHz, create alternating high and low pressure zones in the detergent solution. In the low pressure phase microscopic bubbles form and then implode, which is **cavitation**. Each implosion is a tiny jet of energy that lifts debris off the surface.

Step 4 – explain why it beats hand scrubbing: Cavitation reaches hinges, serrations, box joints and burs, which a brush cannot enter. It is also far safer, because the operator's hands never touch contaminated sharps, so the risk of a percutaneous injury during the dirtiest step of the day is removed.

Step 5 – put the sequence in order: Clean, then rinse and dry, then inspect, then wrap, then sterilise, then store. Cleaning is a prerequisite for sterilisation, never a substitute for it.

Why the other options are wrong:

- A, cleaning is cosmetic: is the dangerous misconception the question is aimed at. Steam does **not** penetrate dried debris, and the bath's job is cavitation cleaning, not lubrication. Lubrication is a separate step for handpieces.
- B, the ultrasonic bath sterilises: is false. The bath **cleans**; it does not kill. Instruments leaving it are contaminated but clean, and every one of them still needs a sterilisation cycle, wrapped or not.
- D, debris neutralises steam and the bath kills at 80°C: is wrong on both halves. Steam is not chemically neutralised, it is simply blocked, and the ultrasonic bath works by mechanical cavitation at low temperature, not by heat. Holding at 80°C would not kill spores in any case.

Final Answer: Debris shields organisms from steam; the bath removes it by cavitation $\Rightarrow$ C

Answer: (C) Go Back to Q11

Q12.

Solution

Concept – read every word of a microbiology stem: Four clues are given, and the fourth one separates the only two organisms that survive the first three.

Step 1 – use the anaerobic culture: An acute dentoalveolar abscess is a **polymicrobial, predominantly anaerobic** infection. Strict anaerobes outnumber facultative organisms in pus from a periapical abscess, so an anaerobic isolate is entirely expected.

Step 2 – use the black pigment: Black-pigmented colonies on blood agar narrow the field to the **black-pigmented anaerobes**, which are *Porphyromonas* and *Prevotella*. The pigment is protohaem accumulated from the blood in the medium, and it takes several days to appear, which is why the stem says 5 days.

Step 3 – use the gram stain and morphology: A gram negative, non-motile rod fits both of those genera and eliminates any gram positive organism at once.

Step 4 – use the decisive clue, asaccharolytic: *Porphyromonas gingivalis* is **asaccharolytic**: it cannot ferment carbohydrate and instead derives its energy from peptides and amino acids. *Prevotella* species are **saccharolytic**. This single word settles the answer in favour of *P. gingivalis*.

Step 5 – confirm the organism makes sense clinically: *P. gingivalis* is a red complex periodontal pathogen. Its gingipain proteases, capsule, fimbriae and lipopolysaccharide let it destroy tissue, evade phagocytosis and thrive in the protein-rich anaerobic environment of an abscess.

Why the other options are wrong:

- A, Prevotella intermedia: is the intended trap. It is a genuine black-pigmented gram negative anaerobe of oral infections, but it is **saccharolytic**, so it is excluded by the one word the stem supplies to separate them.
- C, Fusobacterium nucleatum: is a gram negative anaerobe common in abscesses and a key bridging organism in plaque, but it is a long, thin **spindle-shaped** rod and it does **not** produce black pigment.
- D, Streptococcus anginosus: is a real abscess-forming organism of the milleri group, but it is a **gram positive coccus**, not a gram negative rod, and it forms no pigment.

Final Answer: Porphyromonas gingivalis $\Rightarrow$ B

Answer: (B) Go Back to Q12

Q13.

Solution

Concept – one lesion, one virus: Oral hairy leukoplakia has a single cause, and the association is absolute enough to be an examination certainty.

Step 1 – use the site and appearance: A white, corrugated, vertically ridged plaque on the **lateral border of the tongue**, usually bilateral, is the defining description of oral hairy leukoplakia.

Step 2 – use the negative clues: It does **not rub off**, which separates it from pseudomembranous candidiasis, and it does **not respond to antifungals**, which confirms that separation after a therapeutic trial.

Step 3 – name the virus: Epstein-Barr virus, human herpesvirus 4. The lesion is EBV replicating productively in the epithelium of the tongue, producing the hyperkeratosis and the balloon cells with nuclear beading seen on histology.

Step 4 – explain why this patient: EBV infects most adults and then persists latently in B lymphocytes. Cell-mediated immunity keeps it silent. With a CD4 count of 180 that control has failed, so the virus replicates freely in the epithelium. The lesion is therefore a marker of immunosuppression, and its appearance predicts progression of HIV disease.

Step 5 – note the management point: It is benign and it has **no malignant potential**, despite the word leukoplakia in its name. It regresses on antiretroviral therapy, so treating the immune deficiency treats the lesion, and no excision is needed.

Why the other options are wrong:

- A, HPV 16: is the high risk type associated with **oropharyngeal squamous cell carcinoma**, and low risk types 6 and 11 cause papillomas and condylomas. HPV causes no hairy leukoplakia.
- C, herpes simplex virus type 1: causes primary herpetic gingivostomatitis and recurrent herpes labialis, both **vesicular and ulcerative** lesions. HSV-1 is a herpesvirus like EBV, but it produces vesicles that break down into ulcers, not a fixed white plaque.
- D, human herpesvirus 8: is the cause of **Kaposi sarcoma**, which is also an HIV-associated oral lesion but presents as a purple or red macule or nodule, most often on the palate, and not as a white plaque on the tongue.

Final Answer: Epstein-Barr virus $\Rightarrow$ B

Answer: (B) Go Back to Q13

Q14.

Solution

Concept – autoimmunity is a loss of self tolerance, and each disease has its signature antibody: Name the disease from the clinical triad, then attach its antibodies.

Step 1 – read the clinical picture: Dry mouth (**xerostomia**) plus dry gritty eyes (**keratoconjunctivitis sicca**) in a middle-aged woman is the sicca complex of **Sjogren's syndrome**.

Step 2 – confirm with the biopsy: Focal lymphocytic sialadenitis in a labial minor gland is the histological criterion. Autoreactive CD4 T cells and B cells infiltrate and destroy the exocrine acini and replace them, so secretion fails. A focus score of at least 1 per 4 mm^2 counts as positive.

Step 3 – name the antibodies: Anti-Ro (SS-A) and **anti-La (SS-B)**, directed against ribonucleoprotein antigens. Anti-Ro is present in roughly 60 to 70 percent of cases and anti-La in about 40 percent. Rheumatoid factor and ANA are also often positive but are far less specific.

Step 4 – explain why this matters to a dentist: Loss of saliva removes buffering, clearance and antimicrobial protection, so the patient gets rampant **cervical and root surface caries**, candidiasis, angular cheilitis and difficulty wearing dentures. Management is preventive: high fluoride, saliva substitutes, pilocarpine if residual gland function remains, and short recall intervals.

Step 5 – note the long-term risk: Sjogren's carries a substantially raised lifetime

risk of **MALT lymphoma** in the salivary glands, so any persistent unilateral gland enlargement must be investigated rather than watched.

Why the other options are wrong:

- A, anti-double stranded DNA: is highly specific for **systemic lupus erythematosus** and tracks its renal activity. Sjogren's can occur secondary to lupus, but anti-dsDNA is not its signature antibody and it does not explain the sicca picture.
- B, anti-desmoglein 3: is the antibody of **pemphigus vulgaris**. It attacks the desmosome, causing acantholysis, so the oral picture is flaccid bullae and painful erosions with a positive Nikolsky sign, not dryness.
- D, anti-mitochondrial antibody: is the marker of **primary biliary cholangitis**. That disease may coexist with Sjogren's, but the antibody itself has no role in salivary or lacrimal destruction.

Final Answer: Anti-Ro (SS-A) and anti-La (SS-B) $\Rightarrow$ C

Answer: (C) Go Back to Q14

Answer Key

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