NEET MDS General Pathology and Microbiology

Sample Paper – 3

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. A tuberculous cervical lymph node is excised. On section the centre is soft, dry, white and cheesy, and under the microscope the tissue architecture is completely obliterated with no cell outlines preserved. This type of necrosis is:

(A) Coagulative necrosis, the pattern seen in the infarcted heart and kidney

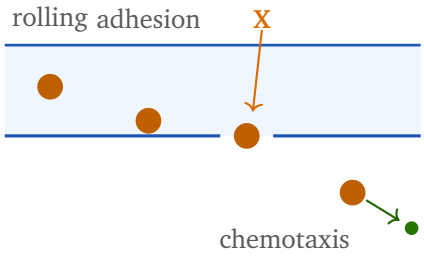
(B) Caseous necrosis

(C) Liquefactive necrosis, the pattern seen in a brain infarct

(D) Fat necrosis, the pattern seen around an inflamed pancreas

Q2. The figure shows a neutrophil leaving a venule at a site of acute inflammation. The full sequence runs margination, then rolling, then firm

adhesion, then the step marked **X**, and finally chemotaxis through the interstitium. Step **X** is:



(A) Transmigration, also called diapedesis, between two endothelial cells

(B) Opsonisation of the offending agent

(C) Phagocytosis of the offending agent

(D) Formation of the phagolysosome

Q3. Fluid aspirated from a swollen, tender, warm submandibular space is turbid, contains 4.6 g/dL of protein, has a specific gravity of 1.024 and is loaded with neutrophils. This fluid, and the mechanism that produced it, are:

(A) A transudate, produced by raised hydrostatic pressure in cardiac failure

(B) A transudate, produced by a fall in plasma oncotic pressure in hypoalbuminaemia

(C) An exudate, produced by increased vascular permeability in inflammation

(D) An exudate, produced entirely by lymphatic obstruction, with normal vessel permeability

Neoplasia

Q4. A biopsy from the hard palate of a reverse smoker shows the normal epithelium replaced by a fully formed, well ordered keratinised stratified squamous epithelium, with orderly maturation and no nuclear atypia. This reversible replacement of one differentiated cell type by another is:

(A) Dysplasia

(B) Hyperplasia

(C) Metaplasia

(D) Neoplasia

Q5. A report on an oral squamous cell carcinoma gives both a grade and a stage. The correct statement about the two is:

(A) Grading records the anatomical extent of the tumour, while staging records how well differentiated the tumour cells are

(B) Grading is based on the degree of differentiation of the tumour cells, while staging records the anatomical extent, and the TNM system records tumour size, regional node involvement and distant metastasis

(C) The TNM system records the degree of differentiation, the mitotic count and the amount of tumour necrosis

(D) Grading and staging are interchangeable terms, and neither carries prognostic value

Q6. Leukoplakia is defined as a white patch that cannot be scraped off and cannot be given any other clinical or pathological diagnosis. It is classed as a potentially malignant disorder because, taken over all reported series, it undergoes malignant transformation in about:

(A) Less than 0.1 percent of cases, which is the same as normal mucosa

(B) 3 to 6 percent of cases

(C) 30 to 40 percent of cases

(D) 100 percent of cases, since every leukoplakia becomes a carcinoma if followed long enough

Haemodynamic Disorders and Wound Healing

Q7. Hyperaemia and congestion both mean an increased volume of blood in a tissue, but they are not the same process. The correct distinction is:

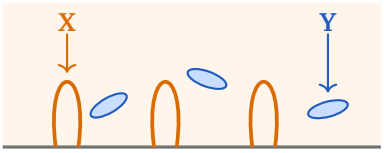
(A) Hyperaemia is active, caused by arteriolar dilatation bringing in oxygenated arterial blood, and the tissue looks bright red; congestion is passive, caused by impaired venous outflow, and the tissue looks blue-red because deoxygenated blood accumulates

(B) Hyperaemia is passive, caused by impaired venous outflow, while congestion is active, caused by arteriolar dilatation

(C) Both are active processes driven by arteriolar dilatation, and they differ only in how long they last

(D) Both are passive processes driven by venous obstruction, and they differ only in the calibre of the vessel involved

Q8. The figure shows the tissue filling the base of a healing extraction socket at day 5. It is soft, pink and studded with fine granules that bleed easily. The two components marked **X** and **Y**, which define this tissue and account for its red granular look, are:



socket base

(A) New capillaries formed by angiogenesis, and proliferating fibroblasts

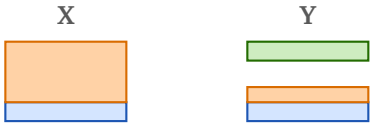
(B) Neutrophils and a mesh of fibrin

(C) Epithelioid cells and Langhans giant cells

(D) Dense mature collagen only, with no vessels

General Microbiology and Sterilisation

Q9. The figure shows two bacterial cell walls in section, marked **X** and **Y**. The correct statement about them is:



wall X wall Y

blue = plasma membrane, orange = peptidoglycan, green = extra outer layer

(A) X is the gram negative wall and Y is the gram positive wall, since gram negative organisms have the thicker peptidoglycan

(B) Both X and Y carry an outer membrane, and they differ only in the thickness of the peptidoglycan

(C) The lipopolysaccharide sits in wall X, while wall Y owes its staining behaviour to teichoic acid

(D) X is the gram positive wall, with a thick multilayered peptidoglycan and no outer membrane, while Y is the gram negative wall, with a thin peptidoglycan plus an outer membrane carrying lipopolysaccharide

Q10. Sterilisation, disinfection and antisepsis are distinct terms. The set of definitions that is correct is:

(A) Sterilisation destroys all microbial life including bacterial spores; disinfection destroys most pathogenic organisms on an inanimate object but need not kill spores; antisepsis is the application of a chemical agent to living tissue to reduce its microbial load

(B) Sterilisation destroys only vegetative organisms; disinfection destroys spores as well; antisepsis is a synonym for sterilisation of instruments

(C) Sterilisation and disinfection are identical, and antisepsis is the term used when either is done in an operating theatre

(D) Disinfection destroys all microbial life including spores; sterilisation is used only on living tissue; antisepsis applies only to floors and work surfaces

Q11. Pre-packed disposable syringes, needles and suture material are sterilised industrially inside their final sealed packaging, without any rise in temperature. The method used is:

(A) Autoclaving at 121°C for 15 minutes

(B) Ultraviolet irradiation

(C) Filtration through a 0.22 micrometre membrane filter

(D) Gamma radiation from a cobalt-60 source

Systemic Bacteriology and Virology

Q12. In a Ziehl-Neelsen stained sputum smear, *Mycobacterium tuberculosis* retains carbol fuchsin and appears as bright red bacilli against a blue counterstained background, even after treatment with 20 percent sulphuric acid and alcohol. This acid-fastness is due to:

(A) A thick multilayered peptidoglycan with abundant teichoic acid

(B) A large polysaccharide capsule surrounding the organism

(C) Lipopolysaccharide in an outer membrane, as in gram negative bacilli

(D) The high mycolic acid content of its waxy cell wall, which locks the dye in and resists decolourisation

Q13. An HIV positive man attends with white curdy plaques over the buccal mucosa and palate that wipe away with gauze to leave an erythematous base. This is the commonest oral lesion in HIV and an early clinical marker of immunosuppression. It typically appears once the CD4 count has fallen below about:

(A) 1200 cells per microlitre

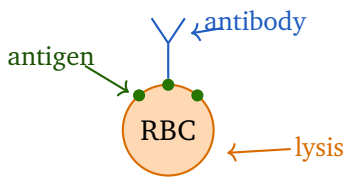
(B) 200 cells per microlitre

(C) 800 cells per microlitre

(D) The CD4 count is irrelevant, since the lesion occurs at any count

Immunology and Hypersensitivity

Q14. A group O patient is transfused with group A blood by mistake and develops fever, loin pain, haemoglobinuria and hypotension. The mechanism, shown below, is **IgM or IgG** binding an antigen fixed on the red cell surface, followed by complement activation and lysis of the cell. This reaction is:



(A) Type I hypersensitivity

(B) Type III hypersensitivity

(C) Type II hypersensitivity

(D) Type IV hypersensitivity

Detailed Solutions

Q1.

Solution

Concept – each pattern of necrosis belongs to a particular organ or disease: The morphological patterns are told apart by two things, the appearance of the dead tissue and the disease that produced it.

Step 1 – read the disease given: The node is **tuberculous**. That single word points to one pattern only.

Step 2 – read the gross appearance: Soft, dry, white and **cheesy**. The Latin *caseus* means cheese, and this is where the name caseous necrosis comes from.

Step 3 – read the microscopic appearance: The architecture is **completely obliterated** and no cell outlines survive. This is the decisive point, because it separates caseation from coagulative necrosis, in which the ghost outlines of the dead cells persist for days.

Step 4 – place it in context: Caseous necrosis sits at the centre of the tuberculous granuloma, surrounded by epithelioid cells and lymphocytes. It is a combination of coagulative and liquefactive change, and the lipid of the mycobacterial cell wall contributes to its cheesy consistency.

Why the other options are wrong:

- A, coagulative necrosis: is the pattern of **ischaemic infarction** in the heart, kidney and spleen. Its defining feature is that the tissue **outline is preserved**, so the question rules it out explicitly.
- C, liquefactive necrosis: is the pattern of **brain infarcts** and of **bacterial abscesses**, where hydrolytic enzymes digest the tissue into a liquid or pus. The material here is dry and cheesy, not liquid.
- D, fat necrosis: occurs in **acute pancreatitis** and after trauma to fatty tissue, where released lipase splits fat and the fatty acids bind calcium to form chalky white saponified deposits. It has no place in a lymph node.

Final Answer: Caseous necrosis $\Rightarrow$ B

Answer: (B) Go Back to Q1

Q2.

Solution

Concept – leucocyte extravasation is a fixed five-step journey: The neutrophil has to get out of the vessel and travel to the offender, and it does so in the same order every time.

Step 1 – list the sequence in full: Margination, then **rolling**, then **firm adhesion**, then **transmigration** (diapedesis), then **chemotaxis**.

Step 2 – identify what the question has already given: The stem names margination, rolling, adhesion and chemotaxis. Only one member of the sequence is missing, and it must sit between adhesion and chemotaxis.

Step 3 – name the missing step: That step is **transmigration**, also called diapedesis. The adherent leucocyte flattens, inserts a pseudopod into the junction between two endothelial cells, squeezes through and pierces the basement membrane.

Step 4 – confirm from the figure: The cell marked X straddles the vessel wall, half in the lumen and half in the tissue, with the endothelial line broken at that point. That is transmigration drawn.

Step 5 – name the molecules, since the examiner often asks: Rolling is mediated by **selectins** (E-selectin and P-selectin on endothelium, L-selectin on the leucocyte). Firm adhesion is mediated by **integrins** binding ICAM-1 and VCAM-1. Transmigration is mediated mainly by **PECAM-1** (CD31) at the interendothelial junction.

Why the other options are wrong:

- B, opsonisation: is the coating of a microbe with IgG or C3b to make it palatable. It happens to the **target**, not to the leucocyte, and it is not a step in extravasation at all.
- C, phagocytosis: happens only **after** the cell has completed chemotaxis and reached the offender, so it comes after the last step named in the stem, not before it.
- D, formation of the phagolysosome: is a stage **within** phagocytosis, when the phagosome fuses with a lysosome. It is even later in the story than option C.

Final Answer: Transmigration or diapedesis $\Rightarrow$ A

Answer: (A) Go Back to Q2

Q3.

Solution

Concept – protein content separates exudate from transudate: Both are extravascular fluid collections, but they are produced by different mechanisms and the laboratory numbers tell them apart.

Step 1 – apply the protein rule: An **exudate** has protein **above 3 g/dL**. A transudate has protein below 3 g/dL. The fluid here has **4.6 g/dL**, so it is an exudate.

Step 2 – apply the specific gravity rule: An exudate has specific gravity **above 1.020**; a transudate is below 1.012. The value here is **1.024**, which confirms an exudate.

Step 3 – apply the cellularity rule: An exudate is turbid and **cell rich**; a transudate is clear and almost acellular. The fluid is turbid and loaded with neutrophils, which confirms it a third time.

Step 4 – state the mechanism: An exudate forms because chemical mediators make the venule endothelium **leaky**. Interendothelial gaps open, and protein-rich plasma and cells escape. The high protein is a direct consequence of that leak.

Step 5 – confirm with the clinical picture: Swelling, tenderness and warmth of the submandibular space are the signs of acute inflammation, so an inflammatory exudate is exactly what should be expected.

Why the other options are wrong:

- A, a transudate from raised hydrostatic pressure: describes cardiac failure, where the vessel wall stays **intact**, so protein is pushed out only in trace amounts. It cannot give 4.6 g/dL.
- B, a transudate from low oncotic pressure: describes nephrotic syndrome or liver failure, where albumin is lost from the plasma. The escaping fluid would be protein **poor**, the opposite of the finding.
- D, an exudate from lymphatic obstruction with normal permeability: is self-contradictory. Lymphatic blockage alone raises interstitial fluid but does not open the vessel wall, so the fluid would not be a neutrophil-rich exudate.

Final Answer: An exudate from increased vascular permeability $\Rightarrow$ C

Answer: (C) Go Back to Q3

Q4.

Solution

Concept – metaplasia is a change of type, dysplasia is a change of quality: Both are adaptive epithelial changes, and the examiner separates them on one word, atypia.

Step 1 – define metaplasia: A **reversible** replacement of one fully differentiated cell type by **another fully differentiated** cell type, better suited to withstand the stress. The new epithelium is normal in every respect except that it is in the wrong place.

Step 2 – define dysplasia: Disordered growth, in which the epithelium loses its orderly maturation and shows **nuclear atypia**: hyperchromatism, pleomorphism, increased nuclear to cytoplasmic ratio and mitoses above the basal layer.

Step 3 – read the biopsy against those two definitions: The description says the new epithelium is **well ordered**, matures normally and shows **no nuclear atypia**. Every marker of dysplasia has been explicitly excluded.

Step 4 – name the change: A fully differentiated epithelium has replaced another fully differentiated epithelium, reversibly. That is **metaplasia**.

Step 5 – note the clinical link: Metaplasia is itself harmless, but the stimulus that drove it, here the heat and smoke of reverse smoking, may persist and go on to induce dysplasia and then carcinoma. That is why metaplastic sites are watched.

Why the other options are wrong:

- A, dysplasia: requires **atypia and disordered maturation**, both of which the biopsy report specifically denies.
- B, hyperplasia: is an increase in the **number** of cells of the *same* type. Here the cell type itself has changed, which hyperplasia does not do.
- D, neoplasia: is autonomous, **irreversible** new growth that continues after the stimulus is withdrawn. The change described is reversible and stimulus dependent.

Final Answer: Metaplasia $\Rightarrow$ C

Answer: (C) Go Back to Q4

Q5.

Solution

Concept – grade looks down the microscope, stage looks at the patient: The two words answer two different questions, and mixing them up is the classic error.

Step 1 – define grading: Grading asks **how bad the cells look**. It is a histological judgement based on the degree of **differentiation**, together with pleomorphism and mitotic activity. Broders graded oral carcinoma from Grade I, well differentiated, to Grade IV, anaplastic.

Step 2 – define staging: Staging asks **how far the tumour has gone**. It is a clinical and pathological judgement of **anatomical extent**, not of appearance.

Step 3 – unpack TNM: T records the size and local extent of the primary tumour. **N** records regional lymph node involvement. **M** records distant metastasis. All three are measures of extent, so TNM is a staging system, never a grading system.

Step 4 – match to the option: Option B states grading as differentiation, staging as extent, and TNM as size, nodes and metastasis. All three parts are correct.

Step 5 – note which one matters more: For most solid tumours, including oral squamous cell carcinoma, **stage carries more prognostic weight than grade**, and nodal status is the single strongest factor. Stage also drives the treatment plan.

Why the other options are wrong:

- A: has the two definitions **swapped**. Extent belongs to staging and differentiation belongs to grading, so every clause is reversed.
- C: attaches differentiation, mitotic count and necrosis to **TNM**. Those are grading criteria; TNM records nothing about how the cells look.
- D: claims the terms are interchangeable and carry no prognostic value. Both halves are false, since the terms are distinct and stage is the strongest prognostic indicator available.

Final Answer: Grading is differentiation, staging is extent, TNM records size, nodes and metastasis $\Rightarrow$ B

Answer: (B) Go Back to Q5

Q6.

Solution

Concept – potentially malignant means a raised risk, not a certainty: The whole point of the term is that the risk is real but modest, which is why these patients are kept under review rather than treated as cancer.

Step 1 – recall the accepted figure: Across pooled series, oral leukoplakia transforms to squamous cell carcinoma in roughly **3 to 6 percent** of cases. Individual studies scatter widely, from under 1 percent to about 17 percent, but the commonly quoted overall figure sits in this band.

Step 2 – put the figure in perspective: Normal oral mucosa carries a background risk far below 1 percent. A 3 to 6 percent risk is therefore many times raised, which justifies the label, yet it also means the large majority of leukoplakias never become malignant.

Step 3 – list what pushes an individual lesion higher: Speckled or nodular leukoplakia rather than homogeneous, a site on the **floor of the mouth or ventral tongue**, a lesion in a **non-smoker** (idiopathic), a large lesion, a female patient, and above all the presence and severity of **epithelial dysplasia** on biopsy.

Step 4 – draw the practical conclusion: Because the rate is low but real and cannot be predicted clinically, management is biopsy to grade dysplasia, removal of the habit, and long term review.

Why the other options are wrong:

- A, under 0.1 percent: is the background risk of **normal mucosa**. If leukoplakia carried this risk, the term potentially malignant would be meaningless.
- C, 30 to 40 percent: is far too high for leukoplakia as a whole. Figures of this order belong to **severe dysplasia** or to **erythroplakia**, which is a different and much more dangerous lesion.
- D, 100 percent: is false. Most leukoplakias persist unchanged for years, and some regress completely once the tobacco habit stops.

Final Answer: About 3 to 6 percent $\Rightarrow$ B

Answer: (B) Go Back to Q6

Q7.

Solution

Concept – ask which vessel is at fault and whether the tissue is doing it on purpose: Two questions settle the difference: is the process active or passive, and is the extra blood arterial or venous.

Step 1 – define hyperaemia: An **active** process. Neurogenic and mediator-driven **arteriolar dilatation** increases inflow, so more **oxygenated arterial** blood enters the tissue.

Step 2 – state how hyperaemia looks: Because the blood is oxygenated and haemoglobin is saturated, the tissue is **bright red** (erythema) and it feels **warm**. Inflamed gingiva, exercising muscle and a blushing face are the everyday examples.

Step 3 – define congestion: A **passive** process. Outflow is impaired, so blood dams back in the **venules and capillaries**. The tissue plays no active part, which is why it is called passive.

Step 4 – state how congestion looks: The stagnant blood gives up its oxygen, so deoxygenated haemoglobin accumulates and the tissue turns **blue-red**, that is cyanotic. Long standing congestion adds oedema and, in the lung and liver, the chronic changes of heart failure cells and nutmeg liver.

Step 5 – match to the option: Option A pairs active with arteriolar and red, and passive with venous and blue-red. Every clause matches.

Why the other options are wrong:

- B: is the correct pair of ideas with the **labels swapped**. Hyperaemia is the active arterial one, not the passive venous one.
- C: makes both active. Congestion is by definition **passive**, since the tissue is not dilating anything, and duration has nothing to do with the distinction.
- D: makes both passive. Hyperaemia is **active** arteriolar dilatation, so this is wrong on the same axis, and vessel calibre is not the dividing line.

Final Answer: Hyperaemia is active and arterial and red; congestion is passive and venous and blue-red $\Rightarrow$ A

Answer: (A) Go Back to Q7

Q8.

Solution

Concept – granulation tissue is two things and nothing else: The name misleads students, because it has nothing to do with granulomas. It describes a granular surface, and that surface has a precise cause.

Step 1 – identify the tissue: Soft, pink, granular tissue filling a socket at day 5 that bleeds easily is **granulation tissue**, the hallmark of healing by second intention.

Step 2 – name the first defining component: New capillaries, formed by **angiogenesis** under the drive of VEGF. In the figure these are the looped orange vessels rising from the socket base, marked X.

Step 3 – name the second defining component: Proliferating fibroblasts, recruited and stimulated by PDGF, FGF-2 and TGF-beta. In the figure these are the blue spindle-shaped cells scattered between the loops, marked Y. They lay down the extracellular matrix that will become the scar.

Step 4 – explain the red colour: The tissue is red because it is packed with **thin walled new capillaries** carrying blood, not because of any pigment. Those same fragile, leaky vessels are why it bleeds at the lightest touch.

Step 5 – explain the granular texture: The capillaries grow upward as **loops**. Each loop reaching the surface makes a tiny bead, and the massed beads give the surface its granular, velvety feel. That is where the name comes from.

Step 6 – state what happens next: Fibroblasts deposit collagen, the vessels regress, the tissue pales, and granulation tissue matures into a fibrous scar.

Why the other options are wrong:

- B, neutrophils and fibrin: describes the **acute inflammatory** phase or an early clot, which occupies the socket in the first day or two, before granulation tissue appears.
- C, epithelioid cells and Langhans giant cells: are the components of a **granuloma**, an entirely different lesion. This is the trap set by the similar name.
- D, dense mature collagen only, with no vessels: describes the **finished scar**, weeks later. Its defining feature is the absence of vessels, which is exactly why a scar is pale rather than red.

Final Answer: New capillaries and proliferating fibroblasts $\Rightarrow$ A

Answer: (A) Go Back to Q8

Q9.

Solution

Concept – the gram stain works because the two walls are built differently: Read the figure as architecture, and the staining behaviour follows.

Step 1 – read wall X: A plasma membrane with a single, very **thick** layer above it and nothing outside that layer. Thick multilayered peptidoglycan, up to 20 to 80 nm, with **no outer membrane**, is the **gram positive** wall.

Step 2 – read wall Y: A plasma membrane, a **thin** peptidoglycan layer of only 2 to 7 nm, and then a separate **outer membrane** above it, with a periplasmic space between. That is the **gram negative** wall.

Step 3 – name what sits in the gram negative outer membrane: Lipopolysaccharide (LPS), whose lipid A component is **endotoxin**. Endotoxin is released on cell lysis and drives fever and septic shock. LPS is found only in gram negative organisms.

Step 4 – name what sits in the gram positive wall: Teichoic and lipoteichoic acids, which run through the thick peptidoglycan. These are exclusive to gram positive organisms.

Step 5 – explain the stain itself: Crystal violet and iodine form a large complex inside the cell. In the gram positive organism the thick peptidoglycan is dehydrated by alcohol, its pores close, and the complex is trapped, so the cell stays **purple**. In the gram negative organism the alcohol dissolves the lipid-rich outer membrane, the thin peptidoglycan cannot hold the complex, it washes out, and the safranin counterstain leaves the cell **pink**.

Why the other options are wrong:

- A: is the correct architecture with the two labels **reversed**, and the added claim that gram negative organisms have the thicker peptidoglycan is the exact opposite of the truth.
- B: says both walls carry an outer membrane. The gram positive wall has **no outer membrane at all**, which the figure shows and which is the single most important structural difference.
- C: puts lipopolysaccharide in wall X. LPS belongs to the gram negative outer membrane, so it belongs in Y, and teichoic acid belongs in X, so both halves are placed in the wrong wall.

Final Answer: X gram positive with thick peptidoglycan and no outer membrane, Y gram negative with thin peptidoglycan plus an LPS outer membrane $\Rightarrow$ $\boxed{\text{D}}$

Answer: (D) Go Back to Q9

Q10.

Solution

Concept – the three terms differ on what is killed and on what surface: Two variables decide the answer: whether spores die, and whether the surface is living or inanimate.

Step 1 – define sterilisation: The process that destroys **all forms of microbial life**, including **bacterial spores**, viruses, fungi and prions to the extent achievable. It is the most demanding of the three, and it is an absolute term. Nothing is partly sterile.

Step 2 – define disinfection: The removal or killing of **most pathogenic organisms** from an **inanimate** object. It is not required to kill spores, and high-level disinfection kills everything except large numbers of spores. It is a relative term, graded from low to high level.

Step 3 – define antisepsis: The application of a chemical agent to **living tissue**, such as skin, a mucous membrane or a wound, to reduce the microbial load. Chlorhexidine, povidone iodine and alcohol handrub are the everyday dental examples.

Step 4 – fix the distinction that examiners test: The same chemical can be both. An agent used on an instrument is a **disinfectant**; used on the patient it is an **antiseptic**. The surface, living or not, decides the word.

Step 5 – match to the option: Option A gives all three definitions correctly and in the right relation to one another, including the two dividing lines, spores and living tissue.

Why the other options are wrong:

- B: says sterilisation destroys only vegetative organisms and disinfection kills spores. This inverts the hierarchy completely, since killing spores is the very definition of sterilisation.
- C: calls sterilisation and disinfection identical. They are not, because disinfection need not be sporicidal, and the setting has no bearing on which word applies.
- D: makes disinfection the sporicidal process and confines sterilisation to living tissue. No living tissue can be sterilised, which alone makes the option impossible.

Final Answer: Sterilisation kills all life including spores, disinfection kills most pathogens on objects, antisepsis is chemical use on living tissue $\Rightarrow$ A

Answer: (A) Go Back to Q10

Q11.

Solution

Concept – read the two constraints in the stem: The item is already sealed in its final packet, and the temperature must not rise. Only one method satisfies both.

Step 1 – use the temperature constraint: No rise in temperature rules out every heat method at once, so the autoclave and the oven are gone before anything else is considered. Disposable plastics would deform anyway.

Step 2 – use the packaging constraint: The item must be sterilised **inside its sealed pack**, so the agent must penetrate paper, plastic film and cardboard. Only a penetrating radiation can do this.

Step 3 – name the method: Gamma radiation from a **cobalt-60** source, delivered at a standard dose of **25 kiloGray**. It is called **cold sterilisation** precisely because no significant heat is generated.

Step 4 – explain how it kills: Gamma rays are **ionising**. They break DNA strands directly and also generate free radicals from water that damage nucleic acid further. It is **sporicidal**, so it achieves true sterilisation rather than disinfection.

Step 5 – note where it is used and where it is not: It needs a large shielded installation, so it is an **industrial** method, used for disposable syringes, needles, catheters, sutures, gloves and bone grafts. It is never available in a dental clinic, which is why the question says industrially.

Why the other options are wrong:

- A, autoclaving at 121°C: raises the temperature, which the stem forbids, and moist heat would melt or distort the plastic syringe barrel and its packaging.
- B, ultraviolet irradiation: is **non-ionising** and has very poor penetration. It is stopped by glass, plastic, paper and even a film of dust, so it cannot reach an item inside a sealed pack. It is used only for surface and air disinfection.
- C, membrane filtration: removes organisms from a **liquid** passed through it. It cannot be applied to a solid object at all, and it does not reliably remove viruses.

Final Answer: Gamma radiation from a cobalt-60 source $\Rightarrow$ $\boxed{\text{D}}$

Answer: (D) Go Back to Q11

Q12.

Solution

Concept – acid-fastness is a property of the cell wall lipid: The stain is really a test of one chemical, so name the chemical and the question is answered.

Step 1 – follow the stain step by step: Carbol fuchsin is applied and heated, so the dye penetrates the waxy wall. **20 percent sulphuric acid with 3 percent alcohol** is then applied to decolourise. **Methylene blue** is applied as a counterstain.

Step 2 – state the result: Acid-fast bacilli hold the carbol fuchsin and stand out **bright red** against the **blue** background of everything that let the dye go.

Step 3 – name the responsible molecule: Mycolic acid, a long chain beta-hydroxy fatty acid, makes up a large part of the mycobacterial wall and forms a thick waxy layer.

Step 4 – explain the mechanism: Heat melts that wax and lets the dye in. The wax then resolidifies on cooling and traps the carbol fuchsin inside. Because the layer is lipid and impermeable, the acid and alcohol cannot get back in to strip the dye out, and the organism stays red.

Step 5 – note the practical points: The wall also explains why mycobacteria are slow growing, why they resist drying and many disinfectants, and why they are hard to stain by gram at all. Auramine-rhodamine fluorescence stains the same mycolic acid and is more sensitive on screening.

Why the other options are wrong:

- A, thick peptidoglycan with teichoic acid: describes the **gram positive** wall. It explains why crystal violet is retained in the gram stain, not why carbol fuchsin resists acid.
- B, a polysaccharide capsule: is an antiphagocytic layer, as in *Streptococcus pneumoniae* or *Klebsiella*. It is demonstrated by negative staining with India ink, and it has no role in acid-fastness.
- C, lipopolysaccharide in an outer membrane: is the **gram negative** arrangement. Mycobacteria are not gram negative bacilli, and LPS does not retain carbol fuchsin.

Final Answer: The high mycolic acid content of the waxy cell wall ⇒ D

Answer: (D) Go Back to Q12

Q13.

Solution

Concept – the CD4 count is the yardstick of immunosuppression, and each lesion has its threshold: HIV destroys CD4 T lymphocytes, and the mouth records the loss earlier than almost anywhere else.

Step 1 – identify the lesion: White curdy plaques that **wipe off** to leave an erythematous base are **pseudomembranous candidiasis**, or thrush, caused by *Candida albicans*. Wiping off is the diagnostic manoeuvre, and it is what separates candidiasis from leukoplakia.

Step 2 – recall the normal CD4 count: Roughly **500 to 1500 cells per microlitre** in a healthy adult.

Step 3 – state the threshold: Oral candidiasis becomes common once the count falls below about **200 cells per microlitre**. It may begin to appear in the 200 to 500 band, but below 200 it is frequent, and it becomes progressively harder to clear.

Step 4 – place it among the other markers: A CD4 count below 200 also defines **AIDS** in an HIV positive adult, and it is the threshold at which *Pneumocystis jirovecii* prophylaxis is started. Oesophageal candidiasis, in contrast to the oral form, is itself an AIDS-defining illness.

Step 5 – state the dental relevance: Oral thrush in a young adult with no obvious cause, no denture, no steroid inhaler and no antibiotic course, should prompt the dentist to consider undiagnosed HIV. This is the sentinel role the question is testing.

Why the other options are wrong:

- A, 1200 cells per microlitre: lies **within the normal range**. An immunocompetent count of this order would not permit candidal overgrowth, so the option describes no immunosuppression at all.
- C, 800 cells per microlitre: is also **normal**, and it is not the lower limit of normal, which is nearer 500. Candidiasis is not expected at this count.
- D, the count is irrelevant: is false. The whole clinical value of the lesion lies in the fact that it **tracks** the CD4 count, which is why it is used as an early marker.

Final Answer: About 200 cells per microlitre $\Rightarrow$ B

Answer: (B) Go Back to Q13

Q14.

Solution

Concept – name the antibody and name the target, and the Gell and Coombs type follows: Type II is defined by antibody directed against an antigen **fixed on a cell surface**.

Step 1 – read the mediator given: IgM or IgG, not IgE, and not a T cell. That already excludes Types I and IV.

Step 2 – read the target given: The antigen is **fixed on the red cell surface**, not free in the circulation. That excludes Type III, which needs soluble antigen.

Step 3 – name the type: Antibody against a cell-bound antigen is **Type II**, antibody-dependent cytotoxic hypersensitivity.

Step 4 – follow the mechanism through: A group O recipient already carries **naturally occurring anti-A and anti-B IgM**. Transfused group A cells are bound at once. The bound antibody **activates complement** down the classical pathway, the membrane attack complex punches the membrane, and the cells lyse inside the vessels. Opsonisation by C3b also delivers surviving cells to splenic macrophages.

Step 5 – match the clinical picture: Fever, loin pain, haemoglobinuria and hypotension are the features of **acute intravascular haemolysis**. Free haemoglobin spills into the plasma, is filtered and turns the urine red, and it can precipitate acute renal failure. Released mediators cause the hypotension.

Step 6 – note the other Type II examples worth remembering: Autoimmune haemolytic anaemia, haemolytic disease of the newborn from Rh incompatibility, Goodpasture syndrome, myasthenia gravis and pemphigus vulgaris, the last being the one a dentist meets in the mouth.

Why the other options are wrong:

- A, Type I: is mediated by **IgE** bound to mast cells and needs prior sensitisation. The question names IgM or IgG, so the antibody class is wrong.
- B, Type III: is **immune complex** disease, in which antibody meets **soluble** antigen in the circulation and the complexes then deposit in vessel walls, joints and glomeruli. Here the antigen is anchored to the cell, so no complex forms.
- D, Type IV: is **cell mediated** and driven by T cells and macrophages, delayed by 24 to 72 hours, and involves **no antibody at all**. The reaction described is antibody driven and immediate.

Final Answer: Type II hypersensitivity $\Rightarrow$ C

Answer: (C) Go Back to Q14

Answer Key

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