NEET MDS General Pathology and Microbiology

Sample Paper – 5

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 chronically inflamed, partly necrotic dental pulp shows radio-opaque calcium salt deposits on radiograph. The patient's serum calcium and phosphate are entirely normal. This calcification is best termed:

(A) Metastatic calcification, because calcium is being laid down away from bone

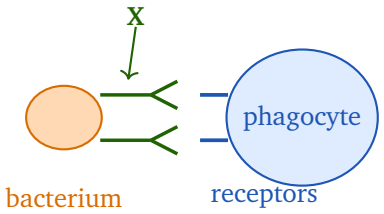
(B) Physiological calcification of an otherwise normal tissue

(C) Metastatic calcification, which by definition occurs in previously damaged tissue

(D) Dystrophic calcification

Q2. In the figure below a bacterium has been coated by the molecules marked

X, whose tails are engaged by matching receptors on the phagocyte. The molecules **X** are:



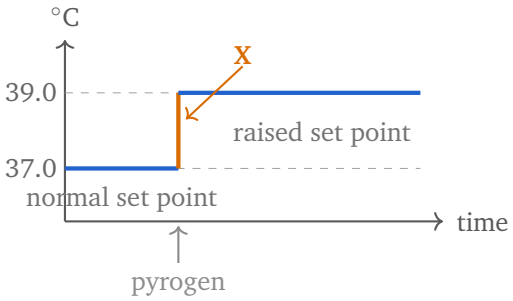
(A) Histamine and bradykinin

(B) C3b and the Fc portion of IgG

(C) Interferon gamma and interleukin 2

(D) Selectins and integrins

Q3. The trace below shows core temperature after release of the endogenous pyrogens interleukin 1, tumour necrosis factor and interleukin 6. The step marked **X** is produced because these cytokines:



(A) Act directly on peripheral thermoreceptors in the skin, causing cutaneous vasodilation

(B) Induce cyclo-oxygenase in the hypothalamic vascular endothelium, so that prostaglandin E2 resets the thermoregulatory set point upwards

(C) Uncouple oxidative phosphorylation in brown fat, raising temperature independently of the hypothalamus

(D) Destroy the anterior hypothalamic neurons, abolishing thermoregulation altogether

Neoplasia

Q4. A follicular lymphoma carries the t(14;18) translocation, which places the *BCL2* gene under the immunoglobulin heavy chain promoter and drives massive overexpression of the Bcl-2 protein. The tumour cells accumulate because Bcl-2:

(A) Accelerates entry into mitosis by activating cyclin D1 and CDK4

(B) Abolishes contact inhibition, so the cells pile up in culture

(C) Guards the mitochondrial outer membrane, preventing its permeabilisation and the release of cytochrome c, so the intrinsic apoptotic pathway cannot fire

(D) Directly activates caspase 9, which then cleaves the executioner caspases

Q5. A squamous cell carcinoma of the lateral tongue shows sheets of cells that closely resemble normal squamous epithelium, with abundant keratin pearls, obvious intercellular bridges, minimal pleomorphism and few mitoses. Well over three quarters of the tumour cells are keratinising. On the Broders system of grading by degree of differentiation, this tumour is:

(A) Grade I, well differentiated

(B) Grade II, moderately differentiated

(C) Grade III, poorly differentiated

(D) Grade IV, anaplastic

Q6. A 38-year-old man who has never used tobacco or alcohol develops a squamous cell carcinoma of the tonsil. The tumour is strongly p16 positive. The class of carcinogen responsible, and the agent, are:

(A) A chemical carcinogen, namely the polycyclic aromatic hydrocarbon benzo(a)pyrene

(B) A viral (biological) carcinogen, namely human papillomavirus types 16 and 18

(C) A physical carcinogen, namely ultraviolet radiation

(D) A chemical carcinogen, namely the tobacco-specific nitrosamines

Haemodynamic Disorders and Wound Healing

Q7. A patient with a spreading Ludwig's angina becomes hypotensive. The peripheries are **warm** and flushed, the pulse pressure is wide, systemic vascular resistance is low and cardiac output is high. This haemodynamic pattern is that of:

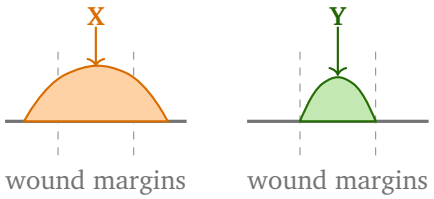
(A) Septic shock, a form of distributive shock

(B) Hypovolaemic shock from acute haemorrhage

(C) Cardiogenic shock following myocardial infarction

(D) Hypovolaemic shock from extensive burns

Q8. The two scars below followed identical skin incisions. The dashed lines mark the **original wound margins**. Lesion **X**, whose collagen has grown well beyond those margins onto adjacent normal skin, is a:



(A) Hypertrophic scar, whereas Y is a keloid

(B) Mass of granulation tissue

(C) Normal mature scar

(D) Keloid, whereas Y is a hypertrophic scar

General Microbiology and Sterilisation

Q9. Culture media are classified as enriched, selective or differential according to what has been added to them and why. Which one of the following pairings of a medium with its type is correct?

(A) Blood agar is a selective medium, because blood suppresses unwanted organisms

(B) Nutrient agar is an enriched medium, because it supports fastidious organisms

(C) Lowenstein-Jensen medium is a purely differential medium, because colonies differ in colour

(D) MacConkey agar is both a selective and a differential medium

Q10. An ultraviolet lamp is switched on overnight in a closed dental operatory to reduce the microbial load. The correct statement about ultraviolet radiation used this way is that it is:

(A) An ionising radiation that will sterilise wrapped instrument packs to their full depth

(B) A non-ionising radiation that acts only on directly exposed surfaces and on air, since it penetrates poorly and does nothing in shadowed areas or under debris

(C) An ionising radiation of the same class as gamma rays, used industrially for bulk sterilisation of disposables

(D) Fully effective through the glass of the cabinet and through plastic instrument wrapping

Q11. Water sampled from the handpiece outlet of a dental chair repeatedly grows heterotrophic bacterial counts far above the potable standard, even though the mains supply feeding the surgery is of drinking quality. The principal explanation is:

(A) A biofilm lining the narrow-bore waterline tubing, encouraged by very low flow rates and long periods of overnight and weekend stagnation, which continually sheds organisms into the output water

(B) Gross contamination of the municipal water at its source

(C) A reaction between residual chlorine in the mains and the tubing polymer, which generates bacteria

(D) Multiplication of bacteria confined to the independent reservoir bottle, the tubing itself playing no part

Systemic Bacteriology and Virology

Q12. A 66-year-old man who wears a full upper denture day and night, uses a long-term inhaled corticosteroid for asthma and complains of a dry mouth, has white curdy plaques on the palate that wipe off with gauze, leaving an erythematous base. The best account of this lesion is:

(A) *Candida glabrata*, an organism that characteristically forms true hyphae in the tissues

(B) *Candida albicans*, but the plaques of this lesion are by definition non-scrapable

(C) *Candida albicans*, an oral commensal turned pathogen by the topical steroid, continuous denture wear and reduced salivary flow

(D) *Candida albicans*, which is never a commensal, so the lesion must represent exogenous infection

Q13. A 62-year-old woman reports two days of burning pain over the right forehead, followed by a crop of grouped vesicles on the right forehead, upper eyelid and the tip of the nose. The eruption stops abruptly at the midline. The virus and the mechanism are:

(A) Herpes simplex virus type 1, reactivating from latency in the trigeminal ganglion

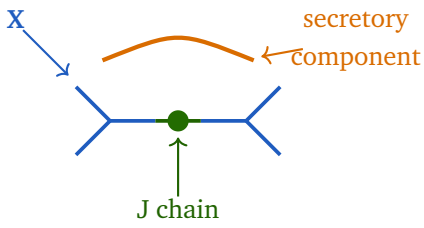
(B) Varicella zoster virus, reactivating from latency in the submandibular salivary gland

(C) Varicella zoster virus, reactivating from latency in the trigeminal ganglion and tracking down the ophthalmic division

(D) Coxsackie A virus, reactivating from latency in the dorsal root ganglia

Immunology and Hypersensitivity

Q14. Of the five immunoglobulin classes, one predominates in saliva and in other mucosal secretions, and is shown below as the molecule marked **X**. Molecule **X** is:



(A) Secretory IgA, a dimer of two monomers joined by a J chain and carrying a secretory component

(B) IgG, which is the most abundant immunoglobulin in serum

(C) IgM, a pentamer joined by a J chain

(D) Monomeric IgA, structurally identical to the serum form

Detailed Solutions

Q1.

Solution

Concept – two kinds of pathological calcification, told apart by two questions: Ask what the tissue was like, and ask what the serum calcium is. Those two answers decide the label every time.

Step 1 – define dystrophic calcification: Calcium salts are deposited in tissue that is **already dead, dying or damaged**. The serum calcium is **normal**, and calcium metabolism is entirely undisturbed.

Step 2 – explain why damaged tissue attracts calcium: Injured and dying cells lose the ability to pump calcium out. Calcium enters, accumulates in mitochondria and in membrane debris, and nucleates crystals of calcium phosphate on the phospholipids left behind. The abnormality is local, in the tissue.

Step 3 – define metastatic calcification: Calcium salts are deposited in **normal, undamaged** tissue, and this happens only because the serum calcium is **raised**. The abnormality is systemic, in the blood.

Step 4 – recall the causes of hypercalcaemia that drive it: Hyperparathyroidism, destruction of bone by metastases or myeloma, vitamin D excess and renal failure. Deposits favour tissues that lose acid and so run an alkaline internal pH, such as gastric mucosa, kidney, lung and systemic arteries.

Step 5 – read the case against the two definitions: The pulp is chronically inflamed and partly **necrotic**, so the tissue is damaged. The serum calcium and phosphate are **normal**, so there is no systemic driver.

Step 6 – conclude: Damaged tissue with normal serum calcium is the exact definition of **dystrophic** calcification. Pulp stones and denticles in an irritated pulp, and calcification in an atheromatous plaque or in a damaged heart valve, are the familiar clinical examples.

Why the other options are wrong:

- A, metastatic calcification because calcium lies away from bone: is wrong reasoning. Metastatic calcification is not defined by being away from bone. It is defined by **hypercalcaemia**, which this patient does not have.
- B, physiological calcification: is wrong because the tissue is neither normal nor a site where calcification is expected. A necrotic pulp calcifying is pathology, not physiology.
- C, metastatic calcification occurring in previously damaged tissue: reverses the two definitions. Damaged tissue is the setting for **dystrophic**, not

metastatic, calcification.

Final Answer: Dystrophic calcification $\Rightarrow$ D

Answer: (D) Go Back to Q1

Q2.

Solution

Concept – opsonisation is the step that makes a microbe edible: A phagocyte struggles to grip a bare bacterium. Coating it with host molecules the phagocyte already has receptors for solves the problem.

Step 1 – read what the figure shows: The molecules X coat the bacterium on one side and present their tails to matching receptors on the phagocyte. That is the definition of an opsonin: a molecule that binds the microbe with one end and a phagocyte receptor with the other.

Step 2 – name the two major opsonins: They are **C3b**, the large fragment of complement C3, and **IgG**, which binds antigen through its Fab arms and the phagocyte through its **Fc** portion. Mannose-binding lectin and other collectins are minor additions to this list.

Step 3 – name the receptors they engage: C3b is read by **complement receptor 1** (CR1, CD35). The Fc portion of IgG is read by **Fc gamma receptor I** (CD64) and its relatives. Both receptor families are constitutively expressed on neutrophils and macrophages.

Step 4 – explain why the coating matters so much: Binding through these receptors does far more than stick the microbe on. It triggers actin polymerisation, so pseudopods zip around the particle, and it triggers the respiratory burst. Opsonised organisms are engulfed several hundred times faster than unopsonised ones.

Step 5 – link the two opsonins to the two arms of immunity: C3b is generated by the alternative pathway within minutes and needs no prior exposure, so it opsonises innately. IgG appears only after an adaptive response, which is precisely why a second encounter with an organism clears it so much faster.

Step 6 – close the loop clinically: Loss of either arm shows the point. Asplenic patients handle encapsulated organisms poorly because splenic macrophages are the main readers of opsonised bacteria, and this is why they are vaccinated against the pneumococcus.

Why the other options are wrong:

- A, histamine and bradykinin: are **vasoactive mediators**. They raise vascular permeability and cause pain and vasodilation. Neither coats a bacterium and neither has a phagocyte receptor.
- C, interferon gamma and interleukin 2: are **cytokines**. Interferon gamma activates macrophages, making them better killers, but it acts on the phagocyte, not on the bacterial surface, so it is not an opsonin.
- D, selectins and integrins: are **adhesion molecules** of the leucocyte extravasation sequence. They stick the leucocyte to endothelium; they do not coat microbes.

Final Answer: C3b and the Fc portion of IgG $\Rightarrow$ B

Answer: (B) Go Back to Q2

Q3.

Solution

Concept – fever is not a broken thermostat, it is a thermostat set higher: The body still regulates temperature perfectly in fever. It simply defends a new, higher target.

Step 1 – name the endogenous pyrogens: Interleukin 1, **tumour necrosis factor** and **interleukin 6**, released by macrophages when they meet an exogenous pyrogen such as bacterial lipopolysaccharide.

Step 2 – follow them to the brain: These cytokines are too large to cross the blood-brain barrier freely. They act instead on the **vascular endothelium** of the hypothalamus, and particularly at the organum vasculosum of the lamina terminalis, where the barrier is leaky.

Step 3 – state the enzymatic step: There they induce **cyclo-oxygenase 2**, which converts arachidonic acid to **prostaglandin E2**.

Step 4 – state what PGE2 does: PGE2 acts on neurons of the **preoptic anterior hypothalamus** and raises the thermoregulatory **set point**, in the figure from 37.0 to 39.0 degrees. That upward step is exactly what X marks.

Step 5 – explain the symptoms from the set point: The moment the target moves up, the actual temperature of 37 reads as too cold. The body therefore behaves as a cold person does: cutaneous vasoconstriction, shivering and the patient reaching for a blanket. Those **rigors** are heat generation, not heat sickness.

Step 6 – explain defervescence the same way: When the pyrogen clears, the set point drops back. Now 39 reads as too hot, so vasodilation and **sweating** follow until temperature falls. This is why sweating marks the breaking of a fever.

Step 7 – confirm with the drugs: Aspirin, paracetamol and the other NSAIDs are antipyretic precisely because they **inhibit cyclo-oxygenase**. Removing PGE2 lowers the set point back to normal. Nothing else in the pathway is drug-targeted, and this is the strongest proof that the prostaglandin step is the true mechanism.

Why the other options are wrong:

- A, direct action on skin thermoreceptors causing vasodilation: is wrong on both counts. The cytokines act centrally, not on skin receptors, and the rising limb of fever is characterised by **vasoconstriction**, not vasodilation.
- C, uncoupling oxidative phosphorylation in brown fat: describes non-shivering thermogenesis, which is a mechanism the hypothalamus can **recruit** in a neonate once the set point has risen. It is downstream, and it is not how the pyrogens produce fever. Saying it happens independently of the hypothalamus makes the option plainly false.
- D, destruction of anterior hypothalamic neurons: would abolish regulation and give **hyperthermia**, in which temperature drifts with the environment. Fever is regulated, so the neurons must be intact and working.

Final Answer: COX induction, PGE2, and an upward reset of the hypothalamic set point $\Rightarrow$ B

Answer: (B) Go Back to Q3

Q4.

Solution

Concept – a tumour can grow by dividing faster or by dying less: Evasion of apoptosis is one of the recognised hallmarks of cancer, and follicular lymphoma is its purest example.

Step 1 – read the translocation: t(14;18) juxtaposes *BCL2* on chromosome 18 with the **immunoglobulin heavy chain** locus on chromosome 14. That locus is fiercely transcribed in a B cell, so *BCL2* is dragged along and hugely overexpressed.

Step 2 – recall what Bcl-2 normally does: Bcl-2 is **anti-apoptotic**. It sits on the outer mitochondrial membrane and holds the pro-apoptotic proteins **Bax and Bak** in check.

Step 3 – recall the intrinsic pathway it guards: When a cell is stressed, Bax and Bak oligomerise and punch pores in the outer mitochondrial membrane. **Cytochrome c** escapes into the cytosol, binds Apaf-1 to form the apoptosome, and that activates **caspase 9**, which activates caspases 3 and 7 and dismantles the cell.

Step 4 – see what excess Bcl-2 does to that pathway: Excess Bcl-2 keeps Bax and Bak inactive, so the pores never form, cytochrome c never leaves, no apoptosome assembles and caspase 9 is never activated. The mitochondrial gate simply never opens.

Step 5 – work out the consequence for the tumour: The lymphoma cells do not divide unusually fast. They **fail to die**. A normal germinal centre B cell that fails to make a useful antibody is deleted by apoptosis; these cells survive that quality check and accumulate instead.

Step 6 – match to the option: Option C states exactly this, that Bcl-2 prevents mitochondrial outer membrane permeabilisation and cytochrome c release, so the intrinsic pathway cannot fire.

Step 7 – note why it matters therapeutically: Because the cells are alive rather than proliferating, they resist drugs aimed at dividing cells, which is part of why follicular lymphoma is indolent yet very hard to cure. Venetoclax, a direct Bcl-2 inhibitor, was designed to reopen that mitochondrial gate.

Why the other options are wrong:

- A, accelerating mitosis via cyclin D1 and CDK4: describes a **proliferation** driver, and cyclin D1 belongs to mantle cell lymphoma with t(11;14). Bcl-2 does not touch the cell cycle.
- B, abolishing contact inhibition: is a hallmark of transformation in adherent cells in culture. It is irrelevant to a circulating B cell, and Bcl-2 has no role in it.
- D, Bcl-2 directly activating caspase 9: is the exact **opposite** of the truth. Bcl-2 prevents caspase 9 activation. If it activated caspase 9 the cells would die faster, not accumulate.

Final Answer: Bcl-2 blocks mitochondrial permeabilisation, so apoptosis cannot fire $\Rightarrow$ $\boxed{\text{C}}$

Answer: (C) Go Back to Q4

Q5.

Solution

Concept – Broders graded squamous carcinoma by how much of it still behaves like squamous epithelium: The grade is a statement about **differentiation**, and nothing else.

Step 1 – recall the Broders scheme: Broders divided squamous cell carcinoma

into four grades on the proportion of tumour cells that remain differentiated and keratinising:

- Grade I: more than **75 percent** differentiated, well differentiated.
- Grade II: **50 to 75 percent**, moderately differentiated.
- Grade III: **25 to 50 percent**, poorly differentiated.
- Grade IV: **less than 25 percent**, anaplastic.

Step 2 – read the proportion given: The question states that well over three quarters of the cells are keratinising, which is more than 75 percent. On the numbers alone this is Grade I.

Step 3 – confirm with the keratin pearls: Keratin pearls are concentric whorls of keratinised cells laid down in the middle of tumour nests. They can only form if the tumour cells still remember how to keratinise, so abundant pearls are the histological signature of good differentiation.

Step 4 – confirm with the intercellular bridges: Obvious intercellular bridges are desmosomes, the junctions of normal squamous epithelium. Retaining them is further proof that the tumour has kept its squamous identity.

Step 5 – confirm with pleomorphism and mitoses: Minimal pleomorphism means the nuclei are uniform, and few mitoses means the growth fraction is low. Both fit a well differentiated tumour. A poorly differentiated tumour would show marked nuclear variation, hyperchromasia and frequent abnormal mitotic figures.

Step 6 – state what the grade means and does not mean: Grade describes the appearance of the cells down the microscope, so a Grade I tumour tends to behave less aggressively. Grade is an **intrinsic** property of the tumour cells only; how far the tumour has physically spread is a separate matter entirely and is not what Broders measured.

Why the other options are wrong:

- B, Grade II: requires 50 to 75 percent differentiated cells, with less pearl formation and more pleomorphism than described. The stated figure is above 75 percent, so it falls outside this band.
- C, Grade III: requires only 25 to 50 percent differentiated cells, with pearls scanty or absent and mitoses frequent. That contradicts the abundant pearls described.
- D, Grade IV, anaplastic: requires fewer than 25 percent differentiated cells. Such tumours are so primitive that immunohistochemistry may be needed to prove they are squamous at all. Nothing in the description fits.

Final Answer: Grade I, well differentiated $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q5

Q6.

Solution

Concept – carcinogens come in three classes: Chemical, physical (radiant) and viral or biological. Naming the class first, then the agent, keeps these questions simple.

Step 1 – list the classes with their typical agents:

- **Chemical**: polycyclic aromatic hydrocarbons, aromatic amines, nitrosamines, alkylating agents, aflatoxin B1, arecoline in areca nut.
- **Physical**: ultraviolet radiation and ionising radiation.
- **Viral or biological**: HPV, EBV, hepatitis B and C, HTLV-1, and by convention *Helicobacter pylori*.

Step 2 – use the negative history: The man has never used tobacco or alcohol, so the two chemical carcinogens that dominate oral cancer in India are excluded at once.

Step 3 – use the site: The **tonsil** is oropharynx, not oral cavity proper. HPV-driven head and neck cancer has a strong predilection for the tonsil and the base of tongue, where the reticulated crypt epithelium gives the virus easy access to basal cells.

Step 4 – use the p16 result, which is the decisive clue: p16 overexpression is the accepted surrogate marker of transcriptionally active high-risk HPV. It looks paradoxical, because p16 is a tumour suppressor, so the reasoning must be followed carefully.

Step 5 – explain why p16 is high: HPV **E7** binds and degrades the retinoblastoma protein, Rb. Free E2F then drives the cell cycle unchecked. The cell responds to that unchecked signal by pouring out p16 in an attempt to brake it, but p16 works by inhibiting CDK4/6 upstream of Rb, and Rb is already gone. So p16 accumulates uselessly and stains strongly.

Step 6 – add the second oncoprotein: HPV **E6** binds p53 and directs it to proteasomal degradation. Losing p53 removes the apoptotic and repair response, so E6 and E7 together disable both major tumour suppressor pathways.

Step 7 – name the types and match the option: The high-risk types responsible are **HPV 16 and 18**, with 16 accounting for the great majority of oropharyngeal cases. This is a virus, so the class is viral. Option B names both correctly.

Step 8 – note the clinical payoff: HPV-positive oropharyngeal carcinoma re-

sponds far better to treatment and carries a distinctly better prognosis than the tobacco-driven, p53-mutated tumours, which is exactly why p16 is now tested routinely.

Why the other options are wrong:

- A, benzo(a)pyrene: is a genuine chemical carcinogen of tobacco smoke, acting through its epoxide metabolite on *TP53*. The patient has never smoked, and such tumours are p16 **negative**.
- C, ultraviolet radiation: is a true physical carcinogen, but it causes lip vermilion and skin cancer on sun-exposed surfaces. UV cannot reach the tonsil.
- D, tobacco-specific nitrosamines: such as NNK and NNN, are again chemical carcinogens of tobacco. The history of no tobacco excludes them, and they do not produce a p16 positive tumour.

Final Answer: Viral carcinogen, HPV 16 and 18 $\Rightarrow$ B

Answer: (B) Go Back to Q6

Q7.

Solution

Concept – three types of shock, and the peripheries tell you which: Shock is inadequate tissue perfusion, but the three types get there by different routes and give opposite physical signs.

Step 1 – define hypovolaemic shock: Loss of circulating volume, from haemorrhage, burns, vomiting or diarrhoea. Preload falls, so cardiac output falls. The body compensates with intense sympathetic **vasoconstriction**, giving **cold, clammy, pale** peripheries and a **narrow** pulse pressure. Systemic vascular resistance is **high**.

Step 2 – define cardiogenic shock: The pump fails, from infarction, arrhythmia or tamponade. Cardiac output falls with a normal or raised blood volume, so blood dams back and the patient has raised jugular venous pressure and pulmonary oedema. Again the body vasoconstricts, so the peripheries are **cold** and resistance is **high**.

Step 3 – define distributive shock: Volume and pump are both adequate, but the vessels dilate, so the circulating volume is maldistributed into a container that has suddenly grown too large. Septic, anaphylactic and neurogenic shock all belong here.

Step 4 – work out the numbers distributive shock must give: Widespread va-

sodilation drops systemic vascular resistance, which drops diastolic pressure and so **widens** the pulse pressure. The heart, unloaded, ejects easily, so cardiac output is **high**. Dilated skin vessels make the peripheries **warm and flushed**.

Step 5 – read the case against those numbers: Warm peripheries, wide pulse pressure, low resistance and high output. Every one matches distributive shock, and every one is the opposite of the hypovolaemic and cardiogenic patterns.

Step 6 – confirm from the source: Ludwig's angina is a severe spreading odontogenic infection of the submandibular spaces. Bacterial products, above all endotoxin, drive macrophage release of TNF and IL-1, which induce **inducible nitric oxide synthase**. The nitric oxide produced relaxes vascular smooth muscle throughout the body, which is the direct cause of the low resistance.

Step 7 – name it and note the trap: This is **septic shock**, a distributive shock, and the early picture is called **warm shock**. If untreated, capillary leak and myocardial depression supervene and it turns into cold shock, which is why a warm septic patient must never be mistaken for a stable one.

Why the other options are wrong:

- B, hypovolaemic shock from haemorrhage: would give **cold** peripheries, a **narrow** pulse pressure, **high** resistance and **low** output. All four findings are the reverse of those given, and there is no bleeding source anyway.
- C, cardiogenic shock: would give cold peripheries, high resistance and low output, with raised JVP. It also has no relation to a spreading dental infection.
- D, hypovolaemic shock from burns: is hypovolaemia by another route, from plasma loss across the burnt surface. The haemodynamic pattern is identical to B and equally opposite to the case, and there is no burn.

Final Answer: Septic (distributive) shock $\Rightarrow$ A

Answer: (A) Go Back to Q7

Q8.

Solution

Concept – one line separates keloid from hypertrophic scar: Both are excessive collagen. Only one crosses the boundary of the original wound.

Step 1 – state the discriminator: A **keloid** grows **beyond** the original wound margins and invades adjacent normal skin. A **hypertrophic scar** stays **within** the original wound margins, however raised it becomes.

Step 2 – read the figure: Lesion X spills well outside both dashed margins onto normal skin. Lesion Y is raised but is contained neatly between its dashed margins.

Step 3 – apply the rule: X crosses the margins, so X is the **keloid**. Y respects them, so Y is the hypertrophic scar. Option D says exactly this.

Step 4 – add the natural history, which supports the same answer: A hypertrophic scar appears early, within weeks, and then usually **regresses** over a year or two. A keloid often appears later, after months, and **does not regress**; it may keep enlarging for years.

Step 5 – add the histology: A hypertrophic scar contains fine collagen bundles arranged parallel to the skin surface, along with abundant myofibroblasts. A keloid contains thick, glassy, haphazardly arranged **hyalinised keloidal collagen** bundles and few myofibroblasts.

Step 6 – add the mechanism: Both reflect an imbalance between collagen synthesis and its breakdown by matrix metalloproteinases, driven by excess TGF-beta. In keloid the fibroblasts are also relatively resistant to apoptosis, which is why the process never switches itself off.

Step 7 – add the clinical predictors: Keloids favour darker skin, run in families, and cluster at the sternum, shoulder, upper back and ear lobe, the ear lobe being the classic site after piercing.

Step 8 – note why the difference matters: Simple excision of a keloid usually produces a larger keloid, since the excision is itself a fresh wound. Adjuvant treatment such as intralesional steroid, pressure or radiotherapy is required. A hypertrophic scar, by contrast, may be safely revised and often needs no more than observation.

Why the other options are wrong:

- A, X hypertrophic and Y keloid: reverses the two lesions. Y is the one contained within its margins, so Y cannot be the keloid.
- B, granulation tissue: is the transient red, soft, granular repair tissue of an **open** wound, made of new capillaries, fibroblasts and inflammatory cells. It is not a healed raised scar.
- C, normal mature scar: is flat or slightly depressed and pale. Neither X nor Y is normal, and a normal scar could never extend past the wound margins.

Final Answer: X is a keloid, Y is a hypertrophic scar $\Rightarrow$ $\boxed{\text{D}}$

Answer: (D) Go Back to Q8

Q9.

Solution

Concept – classify a medium by asking what was added and why: Added nutrients make it enriched. Added inhibitors make it selective. Added indicators make it differential. A medium may hold more than one label at once.

Step 1 – define enriched: Extra nutrients such as blood, serum or egg are added so that **fastidious** organisms will grow. Nothing is inhibited. **Blood agar** and **chocolate agar** are the standard examples.

Step 2 – define selective: An **inhibitor** is added so that unwanted organisms are suppressed while the wanted one grows. Examples are Lowenstein-Jensen with malachite green for *Mycobacterium*, thiosulphate citrate bile sucrose for *Vibrio*, and Mitis Salivarius Bacitracin for *Streptococcus mutans*.

Step 3 – define differential: An **indicator** such as a sugar plus a pH dye is added so that different organisms produce visibly different colonies on one plate. Nothing is inhibited; the plate simply tells them apart.

Step 4 – examine MacConkey agar ingredient by ingredient: It contains **bile salts and crystal violet**, which inhibit Gram positive organisms and let Gram negatives through. That makes it **selective**. It also contains **lactose and neutral red**. Lactose fermenters such as *E. coli* acidify the medium and turn the dye, giving **pink** colonies; non-fermenters such as *Salmonella*, *Shigella* and *Proteus* stay **pale**. That makes it **differential**.

Step 5 – conclude on MacConkey: Both criteria are met on the same plate, so MacConkey agar is correctly described as both selective and differential. Option D is right.

Step 6 – note that blood agar is a similar dual case, for contrast: Blood agar is enriched, and because haemolysis can be read on it as alpha, beta or gamma, it also serves as a **differential** medium. What it is not, under any reading, is selective.

Why the other options are wrong:

- A, blood agar as selective: is wrong. Blood is a **nutrient**, not an inhibitor, so blood agar is enriched. It suppresses nothing, which is exactly why it grows almost everything.
- B, nutrient agar as enriched: is wrong. Nutrient agar is the plain **basal** medium of peptone, beef extract, salt and agar. Adding nothing to it is the whole point, and fastidious organisms will not grow on it.
- C, Lowenstein-Jensen as purely differential: is wrong. Its malachite green is an **inhibitor** of contaminating flora, which makes it selective, and its egg

makes it enriched. Colonies are not distinguished by an indicator reaction, so calling it purely differential fails on every count.

Final Answer: MacConkey agar is both selective and differential $\Rightarrow$ D

Answer: (D) Go Back to Q9

Q10.

Solution

Concept – ultraviolet light is a surface agent, not a sterilant: It is genuinely germicidal, but only exactly where its photons land.

Step 1 – classify the radiation: Ultraviolet is **non-ionising**. Its photons carry too little energy to eject electrons and form ion pairs, which is precisely what separates it from gamma rays and X-rays.

Step 2 – state the effective wavelength: The germicidal peak is around **254 nm**, matching the absorption maximum of nucleic acid, which is why mercury vapour lamps are used.

Step 3 – state the mechanism: UV energy absorbed by DNA joins adjacent pyrimidines into **thymine dimers**. These distort the helix, so replication and transcription stall and the organism cannot divide.

Step 4 – draw the consequence of being non-ionising: Low photon energy means **very poor penetration**. UV is stopped by ordinary glass, by plastic wrap, by paper, by dust and by a film of blood or saliva. It cannot reach the inside of a wrapped pack, and it does nothing in any **shadowed** area.

Step 5 – state what UV is therefore fit for: Decontaminating **directly exposed surfaces** and **air**, for example in operating theatre air handling, in biological safety cabinets and in laboratory rooms overnight. It is a surface and air decontaminant, not a sterilant for instruments.

Step 6 – match to the option: Option B names the class, the mechanism, the poor penetration and the shadow limitation, all correctly.

Step 7 – note the safety point: UV causes keratoconjunctivitis and erythema of skin, so the lamp is run only in an empty, closed room. Some organisms can also repair thymine dimers by photoreactivation if exposed to visible light afterwards, another reason UV cannot be trusted as a sterilant.

Why the other options are wrong:

- A, ionising and able to sterilise a wrapped pack to full depth: is wrong twice. UV is non-ionising, and its poor penetration means it cannot get through the

wrapper at all.

- C, ionising and of the same class as gamma rays: confuses the two. **Gamma** radiation is ionising, penetrates deeply and is used industrially to cold-sterilise pre-packed disposables such as syringes and sutures. UV can do none of that.
- D, fully effective through glass and plastic wrapping: is the exact opposite of the truth. Ordinary glass **absorbs** UV at 254 nm, and this is a standard trap.

Final Answer: Non-ionising, surface and air only, poor penetration, useless in shadow $\Rightarrow$ B

Answer: (B) Go Back to Q10

Q11.

Solution

Concept – the dental unit waterline is an incubator by design: Clean water goes in. What comes out depends on what has been living in the tubing.

Step 1 – read the clue in the question: The mains supply is of drinking quality, but the handpiece output is not. The contamination must therefore be generated **between** the two points, which is the tubing.

Step 2 – explain why that tubing is so hospitable: The lines are of very **narrow bore**, so the surface area to volume ratio is enormous and almost every water molecule is near a wall. Flow is **laminar and slow**, and it is close to zero at the wall itself, so organisms that settle there are never scoured off.

Step 3 – add stagnation: The unit sits idle overnight, over weekends and over holidays. Long stagnation lets the sessile population multiply undisturbed.

Step 4 – describe biofilm formation: A conditioning film of organic molecules adsorbs to the polymer wall. Planktonic bacteria attach, then secrete an extracellular polysaccharide matrix and form a mature, layered **biofilm**.

Step 5 – explain how the biofilm reaches the patient: The biofilm continually **sheds** organisms into the flowing water, so the count at the outlet rises far above that at the inlet. Counts in neglected lines can reach hundreds of thousands of colony forming units per millilitre against a potable standard of 500 or fewer, and 100 or fewer for dental treatment water.

Step 6 – name what grows there: Mostly harmless environmental heterotrophs, but the flora includes *Pseudomonas aeruginosa*, non-tuberculous mycobacteria, *Legionella pneumophila* and free-living amoebae. Aerosol from the handpiece and syringe puts these directly into the breathing zone of patient and operator.

Step 7 – explain why simple flushing is not enough: The matrix protects the sessile organisms, so they are far more resistant to disinfectants than the same species free in water. Flushing clears the planktonic organisms in the lumen but leaves the biofilm intact to reseed within hours.

Step 8 – state the control measures: Flush lines at the start of the day and for 20 to 30 seconds between patients, fit **anti-retraction valves** to stop suck-back of oral fluid, use an independent reservoir with treated water, and above all run periodic **chemical shock treatment** plus a continuous residual biocide, since only chemistry removes an established biofilm. Use sterile saline for surgical irrigation, never line water.

Why the other options are wrong:

- B, contamination of the municipal water at source: is excluded by the question itself, which states the incoming supply is of drinking quality. If the source were at fault, the inlet count would already be high.
- C, chlorine reacting with the tubing to generate bacteria: is not possible. Bacteria are living organisms and must be seeded and multiply. A chemical reaction cannot create them, and residual chlorine in fact **suppresses** growth.
- D, growth confined to the reservoir bottle: is wrong on the key point. The bottle can certainly become contaminated, but the biofilm on the **tubing wall** is the dominant reservoir, which is why units fed with sterile water from a clean bottle still deliver contaminated output.

Final Answer: Biofilm on the waterline tubing, shedding into the output $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q11

Q12.

Solution

Concept – candidosis is a disease of the host, not of the organism: *Candida* is already in the mouth of roughly half of healthy people. Something must change for it to cause disease.

Step 1 – identify the lesion from the description: White **curdy** plaques that **wipe off** leaving an erythematous, sometimes bleeding base define **pseudomembranous candidosis**, or oral thrush. The plaque is a mat of hyphae, desquamated epithelium, fibrin and inflammatory cells sitting loosely on the surface.

Step 2 – name the organism: ***Candida albicans***, which causes the large majority of oral candidosis. It is a dimorphic yeast, and the switch from the yeast form to

the invasive **hyphal** form is what turns a commensal into a pathogen.

Step 3 – establish that it is a commensal: *C. albicans* is carried harmlessly in the mouth of a large proportion of healthy adults. This is the pivotal fact, because it means the question to ask is not where the organism came from but why the host stopped controlling it.

Step 4 – take the first predisposing factor, the inhaled steroid: An inhaled corticosteroid deposits drug on the oral mucosa at every dose. Locally it suppresses mucosal immunity and epithelial turnover, which is why patients are told to rinse the mouth after each inhalation and why a spacer is recommended.

Step 5 – take the second, continuous denture wear: The fitting surface of an acrylic denture is a porous, poorly cleansed niche. Worn day and night it covers the palatal mucosa, so saliva cannot reach it, oxygen tension falls and pH drops. *Candida* adheres avidly to acrylic and to the plaque on it.

Step 6 – take the third, the dry mouth: Saliva is a mechanical washer and carries **lysozyme, lactoferrin, histatins** and secretory IgA. Reduced flow removes both the flushing and the chemical defence, and histatins in particular are directly candidacidal.

Step 7 – put the three together: Each factor alone might be tolerated. Together they tip a commensal population into overgrowth and hyphal invasion. Option C names the organism and this exact triad, so it is the answer.

Step 8 – note the wider list and the clinical duty: Extremes of age, broad-spectrum antibiotics, systemic steroids, diabetes, iron and B12 deficiency, xerostomia after radiotherapy, and immunodeficiency all belong to the same list. Unexplained thrush in an otherwise healthy adult must prompt a search for an underlying cause rather than antifungal treatment alone.

Why the other options are wrong:

- A, *Candida glabrata* forming true hyphae: is wrong twice. *C. glabrata* is a less common cause, seen mainly in denture wearers and the immunosuppressed, and it notably does **not** form true hyphae or pseudohyphae, existing only as blastoconidia. *C. albicans* is the hyphal one.
- B, non-scrapable by definition: contradicts the case. Non-scrapable white patches define **chronic hyperplastic** candidosis (candidal leukoplakia), a different clinical variant that sits typically at the commissure. The plaques here wipe off, so the lesion is pseudomembranous.
- D, never a commensal, so infection must be exogenous: is factually false and reverses the whole logic of the disease. *C. albicans* is a normal oral commensal in a large share of healthy people, and virtually all oral candidosis is **endogenous** overgrowth.

Final Answer: *Candida albicans*, a commensal overgrowing because of steroid, denture and dry mouth ⇒ C

Answer: (C) Go Back to Q12

Q13.

Solution

Concept – a rash that stops at the midline is following a nerve: Dermatomal distribution is the single most useful sign in this case, and it points to a virus latent in a sensory ganglion.

Step 1 – read the distribution: Grouped vesicles, strictly **unilateral**, stopping **abruptly at the midline**. Blood-borne and allergic rashes do not respect the midline. Only a rash travelling down a single nerve does.

Step 2 – name the dermatome: Forehead, upper eyelid and the tip of the nose are supplied by the **ophthalmic division (V1)** of the trigeminal nerve.

Step 3 – read the pain that came first: Two days of burning pain before any rash is **prodromal neuralgia**. It means the virus is replicating in the ganglion and travelling down the axon before it reaches the skin. This sequence is characteristic of zoster.

Step 4 – name the virus and its life history: Varicella zoster virus, human herpesvirus 3. Primary infection is **chickenpox**, usually in childhood. The virus then travels up sensory axons and lies **latent** in the dorsal root and cranial sensory ganglia, including the **trigeminal ganglion**.

Step 5 – explain reactivation: Latency is held in check by VZV-specific cell-mediated immunity. When that wanes with age, illness, stress or immunosuppression, the virus reactivates, replicates in the ganglion and travels **down** the sensory axons to the skin of that one dermatome. The result is **shingles**, herpes zoster.

Step 6 – match to the option: Option C names the virus, the trigeminal ganglion as the site of latency, and the ophthalmic division as the route, so it is correct.

Step 7 – note Hutchinson's sign: Vesicles on the **tip of the nose** indicate involvement of the nasociliary branch of V1, which also supplies the globe. This predicts ocular involvement and makes urgent ophthalmological referral mandatory. This is **herpes zoster ophthalmicus**.

Step 8 – note the sequel and the dental relevance: Post-herpetic neuralgia, pain persisting after the rash has healed, is the major complication and rises steeply with age. When zoster affects V2 or V3 the vesicles appear intraorally, are

strictly unilateral to the midline, and are a well recognised cause of unexplained unilateral facial or dental pain.

Why the other options are wrong:

- A, herpes simplex type 1: is also latent in the trigeminal ganglion, so the site is right, but the virus is wrong. Recurrent HSV-1 gives **herpes labialis** at the vermilion border, is not dermatomal, and does not respect the midline in this way. Naming the correct herpesvirus is the whole point here.
- B, VZV latent in the submandibular salivary gland: names the right virus but the wrong site. Herpesviruses of this group establish latency in **neurons**, not in glandular tissue. A salivary gland could not produce a dermatomal rash.
- D, Coxsackie A: is an **enterovirus**, not a herpesvirus, and establishes no latency at all. It causes herpangina and hand, foot and mouth disease, both bilateral and neither dermatomal.

Final Answer: Varicella zoster reactivating from the trigeminal ganglion in the V1 dermatome $\Rightarrow$ C

Answer: (C) Go Back to Q13

Q14.

Solution

Concept – each immunoglobulin class has one place where it dominates: Learn the classes by location and by shape, and questions like this answer themselves.

Step 1 – list the five classes:

- **IgG**: monomer, most abundant in **serum** at about 75 to 80 percent, the only class that crosses the **placenta**.
- **IgA**: monomer in serum, **dimer** in secretions, the most abundant immunoglobulin in the **body as a whole** because of the volume secreted daily.
- **IgM**: **pentamer**, the **first** antibody made in a primary response, the best activator of complement.
- **IgD**: monomer, a surface receptor on naive B cells.
- **IgE**: monomer, binds mast cells and basophils, involved in parasite defence.

Step 2 – pick the class of the mucosal surfaces: The predominant immunoglobulin of **saliva**, tears, colostrum, and respiratory and gut secretions is **secretory IgA**, sometimes written sIgA. It is the answer to the question as posed.

Step 3 – read the first structural feature in the figure: Two monomers are

joined tail to tail through a small linking polypeptide. That is the **J chain** (joining chain), and it is made by the plasma cell in the mucosal lamina propria at the same time as the antibody.

Step 4 – read the second structural feature: The curved band draped over the dimer is the **secretory component**. Its origin is different, and worth following.

Step 5 – follow how the secretory component is acquired: The dimer with its J chain binds the **polymeric immunoglobulin receptor** on the basolateral surface of the epithelial cell. The complex is transcytosed across the cell. At the apical surface the receptor is cleaved, and the piece that stays attached to the antibody **is** the secretory component. So it is epithelial in origin, not made by the plasma cell.

Step 6 – explain what the secretory component is for: It wraps the hinge region and protects the molecule from the **proteolytic enzymes** of saliva and gut. Without it the antibody would be digested within minutes in the very environment it must work in. It also helps anchor sIgA in the mucus layer.

Step 7 – explain how sIgA protects: Chiefly by **immune exclusion**. Its four antigen-binding sites let it agglutinate microbes and block their adhesins, so the organism never attaches to the epithelium in the first place. It is a poor complement activator by the classical pathway and a poor opsonin, which is a virtue rather than a fault: it neutralises without provoking inflammation at a surface that meets antigen constantly.

Step 8 – state the dental relevance: Salivary sIgA against the adhesins and glucosyltransferase of *Streptococcus mutans* is a genuine part of caries resistance, which is why sIgA has been the target of anti-caries vaccine work. Option A names the class, the dimeric form, the J chain and the secretory component, all correctly.

Why the other options are wrong:

- B, IgG: is correctly described as the most abundant in **serum**, and it is indeed present in gingival crevicular fluid, where it does predominate. But saliva as a whole is dominated by sIgA, so the location is wrong, and IgG is a **monomer** with no J chain and no secretory component, which contradicts the figure.
- C, IgM: is a pentamer and does carry a J chain, which makes it the closest distractor. But the figure clearly shows **two** units, not five, and IgM carries **no secretory component**. IgM appears in secretions only as a minor backup, notably in selective IgA deficiency.
- D, monomeric IgA identical to the serum form: gets the class right but the form wrong. Serum IgA is a monomer with neither J chain nor secretory component. The secretory form is a **dimer** with both, and that difference is

the entire point of the question.

Final Answer: Secretory IgA, a J chain-linked dimer bearing the secretory component $\Rightarrow$ A

Answer: (A) Go Back to Q14

Answer Key

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