NEET MDS General Pathology and Microbiology

Sample Paper – 7

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. Hypoxia and ischaemia are both oxygen deprivation states, yet tissue subjected to ischaemia is injured faster and more severely than tissue subjected to hypoxia alone. The reason is:

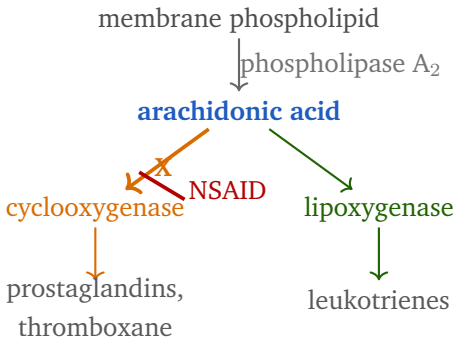
(A) Ischaemia cuts off the delivery of glucose substrate and the washing away of metabolites as well as the delivery of oxygen, so even anaerobic glycolysis cannot be sustained

(B) Hypoxia halts anaerobic glycolysis whereas ischaemia leaves glycolysis fully intact

(C) Ischaemia injures cells only by generating free radicals, a mechanism unavailable to hypoxia

(D) Hypoxia is by definition always irreversible whereas ischaemia is always reversible

Q2. The scheme below shows arachidonic acid metabolism. Aspirin and the other classical NSAIDs act by blocking the enzyme marked **X**. The consequence of that block is:



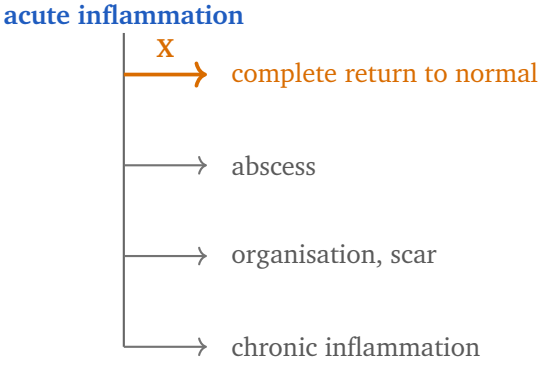
(A) Phospholipase A_2 is inhibited, so arachidonic acid is never released at all

(B) Lipoxygenase is inhibited, so leukotriene synthesis stops while prostaglandins continue

(C) Both arms are inhibited equally, so every arachidonic acid derived mediator disappears

(D) Cyclooxygenase is inhibited, so prostaglandin and thromboxane synthesis stops while the lipoxygenase arm continues to make leukotrienes

Q3. The tree below shows the possible outcomes of acute inflammation. The outcome marked **X**, which follows when the injurious agent is cleared quickly, the tissue is made of labile or stable cells and the stromal framework has survived intact, is:



(A) Complete resolution, with restoration of normal structure and function

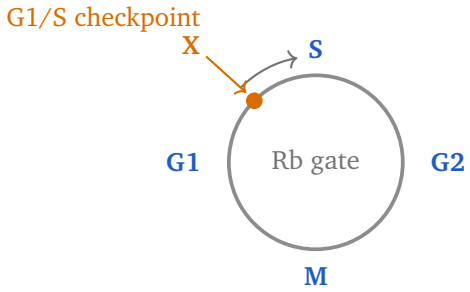
(B) Organisation, in which the exudate is replaced by fibrous scar tissue

(C) Abscess formation, with a walled-off collection of pus

(D) Progression to chronic inflammation

Neoplasia

Q4. The ring below is the cell cycle. The checkpoint marked **X** is guarded by the retinoblastoma (Rb) protein. The way Rb guards it is:



(A) Phosphorylated Rb binds E2F and arrests the cell, while dephosphorylation releases the cell into S phase

(B) Rb acts at the G2/M checkpoint, where it binds p53 and prevents mitosis

(C) Rb is an oncogene, and its activation drives the cell forward into S phase

(D) Hypophosphorylated Rb sequesters E2F and holds the cell in G1; phosphorylation by cyclin D–CDK4/6 releases E2F, which transcribes the genes needed for S phase entry

Q5. A solid tumour cannot enlarge beyond about 1 to 2 mm until it recruits its own blood supply. The principal pro-angiogenic factor secreted by tumour cells, whose transcription is driven by HIF-1α under hypoxic conditions, is:

(A) Tumour necrosis factor alpha

(B) Thrombospondin-1

(C) Vascular endothelial growth factor (VEGF)

(D) Angiostatin

Q6. A carcinoma of the lateral border of the tongue is being staged by the TNM system before treatment is planned. What T, N and M each record is:

(A) T records the size and local extent (including depth of invasion) of the primary tumour, N records the regional lymph node involvement, and M records the presence or absence of distant metastasis

(B) T records the tissue type, N the nuclear pleomorphism and M the mitotic count

(C) T records the time since onset, N the number of separate tumours and M the surgical margin status

(D) T records the tumour thickness alone, N perineural invasion and M the degree of differentiation

Haemodynamic Disorders and Wound Healing

Q7. A patient with Gram-negative septicaemia develops widespread microthrombi and, at the same time, oozing from every venepuncture site and from the gingivae. This apparent paradox of disseminated intravascular coagulation is explained by:

(A) Uncontrolled intravascular fibrin formation consumes platelets and clotting factors faster than they can be replaced, and the secondary fibrinolysis it triggers digests the fibrin already laid down, so the patient bleeds

(B) Two unrelated diseases happening to occur together in a septic patient

(C) A sudden failure of the bone marrow to release any platelets at all

(D) Dietary vitamin K deficiency arising over the course of the illness

Q8. From about day 3 of a healing extraction socket onwards, one cell dominates the wound. It clears necrotic debris and fibrin by phagocytosis and

then orchestrates repair by secreting PDGF, TGF-β, VEGF and FGF. That cell is the:

(A) Neutrophil

(B) Mast cell

(C) Macrophage

(D) Platelet

General Microbiology and Sterilisation

Q9. Prions survive a standard sterilisation cycle and are unaffected by routine chemical disinfectants. The reason for this extreme resistance is:

(A) They form an unusually thick spore coat that moist heat cannot penetrate

(B) They contain no nucleic acid and are nothing but a misfolded, tightly aggregated protein, so agents that target nucleic acid are useless and ordinary protein-denaturing conditions do not unfold the aggregate; inactivation needs 134°C for 18 minutes together with 1N sodium hydroxide

(C) They are readily killed by routine glutaraldehyde immersion, and only heat fails against them

(D) They replicate faster than the cycle can kill them, so survivors always remain

Q10. A surgical scrub before a third molar surgery differs from the routine hand wash performed between two routine check-ups in that:

(A) Both use plain soap and water, and only the duration is different

(B) The scrub uses an antiseptic such as 4 percent chlorhexidine gluconate or povidone iodine for 2 to 5 minutes over hands and forearms to the elbow, removing transient flora and also reducing resident flora with a persistent residual effect, whereas the routine wash uses plain soap for 15 to 20 seconds over the hands and removes transient flora only

(C) The routine hand wash sterilises the skin, while the scrub only disinfects it

(D) The scrub is performed only after the procedure, while the routine wash is performed only before it

Q11. A dentist is about to perform ultrasonic scaling on a patient with heavy calculus. The correct barrier protection for this procedure is:

(A) Examination gloves alone, since the patient has no known bloodborne infection

(B) Gloves and a surgical mask, with eye protection left to personal preference

(C) Gloves, a surgical mask, protective eyewear with side shields (or a face shield) and a protective gown, because the ultrasonic scaler generates a contaminated aerosol and spatter that can reach the conjunctiva, the nasal and oral mucosa and clothing

(D) The same pair of gloves reused between patients provided they are washed with soap and an alcohol rub

Systemic Bacteriology and Virology

Q12. *Treponema pallidum* produces a different oral lesion at each stage of syphilis. The correct sequence of primary, secondary and tertiary oral lesions is:

(A) A gumma first, then a chancre, then mucous patches

(B) Mucous patches first, then a painful ragged ulcer, then Hutchinson's incisors

(C) A painful ulcer with a yellow slough first, then interstitial keratitis, then a chancre

(D) A painless indurated chancre on the lip or tongue in the primary stage, mucous patches and snail track ulcers in the secondary stage, and a gumma of the palate, sometimes perforating it, in the tertiary stage

Q13. A 4-year-old has fever with crops of small vesicles and shallow ulcers confined to the soft palate, the anterior pillars of the fauces and the uvula, together with vesicles on the palms and soles. The gingivae are entirely normal and there is no gingival bleeding or perioral vesicle. The causative agent is:

(A) Herpes simplex virus type 1

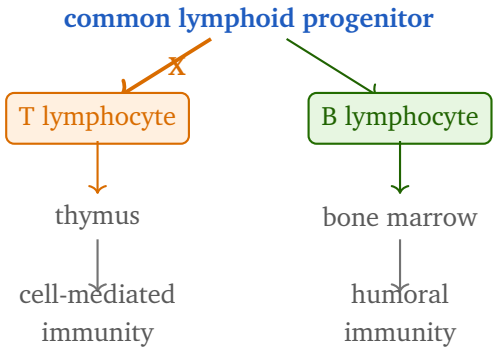
(B) Coxsackie virus, group A

(C) Varicella zoster virus

(D) Epstein–Barr virus

Immunology and Hypersensitivity

Q14. The scheme below shows the two lymphocyte lineages arising from a common lymphoid progenitor. The cell marked **X** matures at the site shown. The correct statement about the two lineages is:



(A) Both lineages mature in the thymus, and the difference lies only in the surface markers they carry

(B) T lymphocytes mature in the thymus and mediate cell-mediated immunity, whereas B lymphocytes mature in the bone marrow and mediate humoral immunity through antibody-secreting plasma cells

(C) T lymphocytes mature in the bone marrow and secrete antibody, whereas B lymphocytes mature in the thymus and kill infected cells directly

(D) B lymphocytes mediate cell-mediated immunity against intracellular organisms, while T lymphocytes handle all extracellular organisms by secreting immunoglobulin

Detailed Solutions

Q1.

Solution

Concept – hypoxia takes away one thing, ischaemia takes away three: Both insults starve the cell of oxygen, but ischaemia is a loss of blood *flow*, and flow carries more than oxygen.

Step 1 – define hypoxia: Hypoxia is a deficiency of **oxygen** reaching the tissue. The blood is still flowing. It occurs in anaemia, in carbon monoxide poisoning, at altitude and in cardiorespiratory failure.

Step 2 – define ischaemia: Ischaemia is a reduction or loss of **blood flow** to the tissue, usually from arterial obstruction or venous outflow obstruction. Nothing arrives and nothing leaves.

Step 3 – work out what the hypoxic cell can still do: In pure hypoxia, oxidative phosphorylation stops, so the cell switches to **anaerobic glycolysis**. Flowing blood keeps delivering **glucose**, so glycolysis can keep making a small amount of ATP, and the flowing blood also keeps washing away the lactic acid produced.

Step 4 – work out what the ischaemic cell cannot do: In ischaemia the glucose supply is cut off too, so the glycolytic substrate runs out and glycolysis grinds to a halt within minutes. There is now **no** route to ATP at all.

Step 5 – add the metabolite accumulation: Because nothing is washed away either, lactic acid and inorganic phosphate build up in the ischaemic tissue. The intracellular pH falls, which itself inhibits the glycolytic enzymes and denatures cellular proteins, deepening the injury.

Step 6 – conclude: Ischaemia removes oxygen, removes substrate and traps waste. Hypoxia removes only oxygen. That is exactly why ischaemic injury is faster and more severe, and why option A is correct.

Why the other options are wrong:

- B, hypoxia halting glycolysis while ischaemia leaves it intact: is the statement reversed. Glycolysis is preserved in hypoxia and fails in ischaemia.
- C, ischaemia acting only through free radicals: is wrong on two counts. Free radicals are generated mainly on **reperfusion**, not during ischaemia itself, and hypoxia is perfectly capable of free radical injury too.
- D, hypoxia always irreversible and ischaemia always reversible: is false in both halves. Either insult is reversible early and irreversible late, and the distinction has nothing to do with the label.

Final Answer: Ischaemia also cuts off glucose and metabolite clearance $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q1

Q2.

Solution

Concept – one substrate, two enzymes, two families of mediators: Arachidonic acid sits at a fork. Which arm a drug blocks decides which mediators disappear and which survive.

Step 1 – release the substrate: Arachidonic acid is a 20-carbon fatty acid esterified in **membrane phospholipid**. Cell injury and inflammatory stimuli activate **phospholipase A_2**, which liberates free arachidonic acid into the cytosol.

Step 2 – follow the first arm: Cyclooxygenase (COX-1 and COX-2) converts arachidonic acid into the cyclic endoperoxides, and from these come the **prostaglandins** (PGE_2, PGD_2, PGI_2) and **thromboxane A_2**. These mediate vasodilation, increased vascular permeability, pain sensitisation and fever, while thromboxane drives platelet aggregation.

Step 3 – follow the second arm: 5-lipoxygenase converts arachidonic acid into 5-HPETE and then into the **leukotrienes**. LTB_4 is a powerful chemoattractant for neutrophils, and LTC_4, LTD_4 and LTE_4 cause bronchoconstriction and increased permeability.

Step 4 – place the drug: Aspirin and the classical NSAIDs inhibit **cyclooxygenase** only. Aspirin does it irreversibly by acetylating the enzyme; ibuprofen and diclofenac do it reversibly. The figure marks this block as **X**.

Step 5 – read off the consequence: With COX blocked, prostaglandin and thromboxane synthesis stops, which is where the analgesic, antipyretic, anti-inflammatory and antiplatelet effects come from. The **lipoxygenase arm is untouched**, so leukotrienes continue to be made.

Step 6 – note why that matters clinically: Substrate no longer consumed by COX is available to the lipoxygenase arm, so leukotriene production can actually rise. This is the accepted explanation for aspirin-induced asthma, and it is why option D and no other describes the block correctly.

Why the other options are wrong:

- A, phospholipase A_2 inhibition: is the action of **corticosteroids**, which is why steroids suppress both arms. NSAIDs act one step further downstream.
- B, lipoxygenase inhibition: describes **zileuton**, not aspirin. It also reverses which family survives.
- C, both arms inhibited equally: is again the steroid effect, achieved by block-

ing the pathway before the fork. No classical NSAID does this.

Final Answer: COX blocked, prostaglandins and thromboxane lost, leukotrienes preserved $\Rightarrow$ D

Answer: (D) Go Back to Q2

Q3.

Solution

Concept – acute inflammation has exactly four exits: Which exit is taken depends on how much tissue was destroyed and whether the agent was cleared.

Step 1 – list the four outcomes: Complete **resolution**; healing by **organisation** and fibrosis; **abscess** formation; and progression to **chronic** inflammation.

Step 2 – read the three conditions the question gives: The agent is cleared quickly, the parenchymal cells are **labile or stable** so they can divide and replace losses, and the **stromal framework** is intact so the new cells have a scaffold to grow back on.

Step 3 – match those conditions: Those three conditions together are the precise definition of the setting in which **complete resolution** occurs.

Step 4 – describe what resolution involves: The neutrophils die by apoptosis and are phagocytosed; the fluid exudate is drained by lymphatics; the fibrin is digested by fibrinolysis; the vascular changes reverse; and the parenchymal cells regenerate. Structure and function return to normal, and no scar is left.

Step 5 – read the figure: Branch X is the one labelled "complete return to normal", which is resolution. The other three branches are the alternative exits, and they are taken only when one of the three conditions above fails.

Why the other options are wrong:

- B, organisation and scarring: is what happens when tissue destruction is **substantial**, when the framework is destroyed, or when the parenchymal cells are **permanent** and cannot regenerate. The question specifies the opposite of all three.
- C, abscess formation: requires a **pyogenic** organism producing pus that becomes walled off. The question describes no suppuration and says the agent was cleared.
- D, progression to chronic inflammation: occurs when the agent **persists** and cannot be eliminated. The question explicitly says it was cleared quickly.

Final Answer: Complete resolution $\Rightarrow$ A

Answer: (A) Go Back to Q3

Q4.

Solution

Concept – the G1/S restriction point is the cell's point of commitment: Once the cell crosses it, it will complete the cycle regardless of growth factors. Rb is the brake on that gate.

Step 1 – lay out the cycle: G1 (growth and preparation), S (DNA synthesis), G2 (checking of replicated DNA) and M (mitosis). Quiescent cells rest in G0 and re-enter at G1.

Step 2 – name the two main checkpoints: The **G1/S** checkpoint asks whether the DNA is fit to be replicated, and the **G2/M** checkpoint asks whether the DNA was replicated correctly and completely.

Step 3 – describe Rb in its active state: In the **hypophosphorylated** (active) form, Rb binds and sequesters the transcription factor **E2F**. With E2F held prisoner, the genes for S phase entry are not transcribed, and the cell is held in G1. This is the marked point X in the ring.

Step 4 – describe how the brake is released: Growth factor signalling induces **cyclin D**, which activates **CDK4** and **CDK6**. This complex phosphorylates Rb. **Hyperphosphorylated** Rb changes shape, lets go of E2F, and the freed E2F transcribes the S phase genes. The cell crosses into S phase.

Step 5 – note the key sense of the logic: Active Rb is the **unphosphorylated** form. Phosphorylation **inactivates** the brake. Candidates routinely reverse this, and that is what the question is testing.

Step 6 – link it to disease: Loss of Rb function, whether by mutation of *RB1*, by cyclin D or CDK4 amplification, by loss of the inhibitor p16(INK4a) or by the E7 protein of high-risk HPV binding Rb, leaves E2F permanently free and the G1/S gate permanently open. This is a common route into oral and cervical carcinogenesis.

Why the other options are wrong:

- A, phosphorylated Rb binding E2F: reverses the phosphorylation logic exactly. Phosphorylated Rb **releases** E2F; it does not bind it.
- B, Rb at G2/M with p53: is wrong on both the checkpoint and the partner. Rb guards **G1/S**, and p53 acts through p21 rather than binding Rb.
- C, Rb as an oncogene: is wrong in category. Rb is the prototype **tumour suppressor**, and it **restrains** rather than drives S phase entry.

Final Answer: Hypophosphorylated Rb sequesters E2F; cyclin D–CDK4/6 phosphorylation releases it $\Rightarrow$ D

Answer: (D) Go Back to Q4

Q5.

Solution

Concept – a tumour must build its own plumbing: Diffusion alone can feed only a couple of millimetres of tissue, so angiogenesis is a rate-limiting step in tumour growth.

Step 1 – explain the 1 to 2 mm limit: Oxygen and nutrients diffuse only about 1 to 2 mm from the nearest capillary. Beyond that, the tumour centre becomes hypoxic and cells begin to die, so growth stalls unless new vessels arrive.

Step 2 – follow the hypoxia signal: Hypoxia stabilises **HIF-1**α, the hypoxia inducible factor. Under normal oxygen it is hydroxylated and degraded; under hypoxia it escapes degradation, accumulates, and enters the nucleus.

Step 3 – read the gene it switches on: HIF-1α transcriptionally upregulates **VEGF**, vascular endothelial growth factor. This is the single most important pro-angiogenic factor in tumours.

Step 4 – describe what VEGF does: VEGF binds VEGFR-2 on endothelial cells and makes them proliferate, migrate towards the tumour and form new tubes. It also markedly raises vascular permeability, which is why tumour vessels are leaky, irregular and prone to haemorrhage.

Step 5 – name the concept being tested: The tumour shifts the **angiogenic balance** in favour of promoters (VEGF, basic FGF) and away from inhibitors (thrombospondin-1, angiostatin, endostatin). This shift is called the angiogenic switch.

Step 6 – note the therapeutic proof: Bevacizumab, an anti-VEGF monoclonal antibody, is used clinically precisely because blocking VEGF starves the tumour. That the drug works confirms VEGF as the principal factor.

Why the other options are wrong:

- A, TNF-α: is a major mediator of inflammation, cachexia and septic shock. It has some indirect angiogenic influence, but it is not the principal tumour angiogenic factor and it is not the HIF-1α target being described.
- B, thrombospondin-1: is an angiogenesis **inhibitor**, and it is a p53 target. It points the opposite way.
- D, angiostatin: is likewise an angiogenesis **inhibitor**, a plasminogen frag-

ment. It is part of the brake, not the accelerator.

Final Answer: Vascular endothelial growth factor $\Rightarrow$ C

Answer: (C) Go Back to Q5

Q6.

Solution

Concept – TNM records how far the disease has spread, in three separate readings: Each letter answers one anatomical question, and each is scored independently before they are combined.

Step 1 – read T: T is the **primary Tumour**. In oral cancer it records the greatest surface dimension and, since the eighth edition, the **depth of invasion**. T1 is 2 cm or less with depth of invasion 5 mm or less; T2 and T3 rise with size and depth; T4 means invasion of adjacent structures such as cortical bone, the maxillary sinus or the skin of the face.

Step 2 – read N: N is the **regional lymph Nodes**. It records whether the cervical nodes are involved, and if so their number, size, laterality (ipsilateral, contralateral or bilateral) and, in the current edition, extranodal extension.

Step 3 – read M: M is **distant Metastasis**. It is binary: M0 means none, M1 means distant deposits present, most often in the lungs, then bone and liver.

Step 4 – combine them: The three readings are grouped into stage I to IV. Any M1 makes the disease stage IVC no matter how small the primary is, which shows how much weight distant spread carries.

Step 5 – note why it matters at the chair: Stage decides treatment and prognosis. Node status is the strongest single prognostic factor in oral cancer, roughly halving survival when positive, which is why the N reading is taken so carefully.

Step 6 – match to the option: Option A states size and local extent for T, regional node involvement for N and distant metastasis for M. That is exactly correct.

Why the other options are wrong:

- B, tissue type, nuclear pleomorphism and mitotic count: are **histological** features. Pleomorphism and mitoses belong to grading, which is a different exercise from staging, and none of them is what T, N or M records.
- C, time, number of tumours and margin status: is invented. Duration of symptoms plays no part in TNM, and margin status is a separate pathological report finding.
- D, thickness alone, perineural invasion and differentiation: is a mixture.

Depth of invasion is only part of T and never the whole of it, perineural invasion is a separate adverse feature rather than N, and differentiation is grade rather than M.

Final Answer: T = primary tumour size and extent, N = regional nodes, M = distant metastasis $\Rightarrow$ A

Answer: (A) Go Back to Q6

Q7.

Solution

Concept – DIC is a consumptive coagulopathy: The clotting and the bleeding are not two problems. The bleeding is the direct consequence of the clotting.

Step 1 – name the trigger: DIC is always **secondary** to something else. Gram-negative septicaemia, obstetric emergencies, massive trauma, burns and disseminated malignancy all release **tissue factor** or damage endothelium widely.

Step 2 – follow the first half of the paradox: Widespread tissue factor exposure fires the coagulation cascade throughout the microcirculation. Fibrin microthrombi form in the small vessels of the kidneys, brain, lungs and skin, causing ischaemic organ damage. Red cells forced through those fibrin strands are sheared, giving the **schistocytes** of microangiopathic haemolytic anaemia.

Step 3 – follow the consumption: Making all that fibrin uses up the raw material. **Platelets, fibrinogen and clotting factors** V and VIII are consumed far faster than the liver and marrow can replace them. The patient is now, in effect, profoundly factor-deficient.

Step 4 – follow the fibrinolysis: Fibrin deposition activates **plasmin**, which digests the fibrin already laid down. The **fibrin degradation products** released are themselves anticoagulant, because they inhibit platelet aggregation and interfere with fibrin polymerisation. This is the second blow.

Step 5 – read the result at the bedside: Consumption plus fibrinolysis leaves the patient unable to clot anywhere it is needed. Hence the oozing from venepuncture sites and gingivae described in the stem, happening at the same time as microthrombi.

Step 6 – confirm with the laboratory picture: Prolonged PT and aPTT, low platelets, low fibrinogen and raised D-dimer together are diagnostic, and every one of those values is explained by the consumption-plus-lysis mechanism in option A.

Why the other options are wrong:

- B, two unrelated diseases: misses the point of the question. The whole teaching value of DIC is that the two opposite phenomena share one mechanism.
- C, sudden marrow failure: is wrong. The platelet count in DIC falls because the platelets are **consumed** in the microthrombi, not because the marrow stops making them; the marrow in fact responds.
- D, vitamin K deficiency: produces a different picture. It causes an isolated deficiency of factors II, VII, IX and X with a **normal** platelet count, normal fibrinogen and no microthrombi or raised D-dimer.

Final Answer: Consumption of platelets and factors plus secondary fibrinolysis $\Rightarrow$ A

Answer: (A) Go Back to Q7

Q8.

Solution

Concept – the macrophage is the conductor of the repair orchestra: It both cleans the site and issues the instructions that build the replacement tissue.

Step 1 – place it on the timeline: Neutrophils dominate the first 24 to 48 hours. **Macrophages** take over from about day 3 and remain the principal cell through the whole proliferative phase, exactly the window the question specifies.

Step 2 – describe the cleaning role: Macrophages phagocytose necrotic debris, spent neutrophils, fibrin and any foreign material. Until the site is cleared, granulation tissue cannot be laid down, so this step is a prerequisite for repair.

Step 3 – read the growth factors named: **PDGF** recruits fibroblasts and smooth muscle cells and makes them proliferate. **TGF-β** is the strongest fibrogenic agent known, driving collagen synthesis while suppressing collagen breakdown. **VEGF** and **FGF** drive angiogenesis, the new capillary ingrowth of granulation tissue.

Step 4 – assemble granulation tissue from those signals: New capillaries (VEGF, FGF) plus proliferating fibroblasts (PDGF) plus the collagen they deposit (TGF-β) is precisely the definition of granulation tissue. Every component is being commanded by the macrophage.

Step 5 – note the two phenotypes: The **M1** macrophage is the classically activated, microbicidal, pro-inflammatory cell. The **M2** macrophage is alternatively activated and is the pro-repair, fibrogenic, anti-inflammatory cell. Orderly healing depends on a timely switch from M1 to M2, and failure of that switch is one reason wounds turn chronic.

Step 6 – conclude: Only one cell does the phagocytic clearing and secretes that

entire quartet of growth factors across that time window, and it is the macrophage.

Why the other options are wrong:

- A, the neutrophil: has largely died off by day 3. It is a killing and debriding cell with a very short life and it does not orchestrate fibroblast proliferation or angiogenesis.
- B, the mast cell: releases histamine and heparin in the early vascular response and contributes to allergy. It has a minor supporting role in repair and never dominates the wound.
- D, the platelet: is the tempting distractor, because platelet α granules genuinely do release PDGF and TGF-β. But platelets act in the **first hours** at haemostasis and are gone long before day 3, so they cannot be the cell that dominates from day 3 onwards.

Final Answer: Macrophage $\Rightarrow$ C

Answer: (C) Go Back to Q8

Q9.

Solution

Concept – prions break the rules because they are not really organisms: Every routine sterilisation method was designed against organisms with nucleic acid and folded, denaturable protein. A prion has neither vulnerability.

Step 1 – define the agent: A prion is a **proteinaceous infectious particle**. It is the normal cellular protein PrP^{C} refolded into the abnormal isoform PrP^{Sc}. It contains **no DNA and no RNA**.

Step 2 – explain why the absence of nucleic acid matters: Ultraviolet and ionising radiation, formaldehyde, ethylene oxide and most disinfectants work substantially by damaging nucleic acid. With no nucleic acid to damage, these agents have no target and the prion is untouched.

Step 3 – explain why heat is not enough: Moist heat sterilises by **coagulating and denaturing** protein. PrP^{Sc} is already misfolded into a β-pleated sheet rich, tightly packed, protease-resistant aggregate. There is no native fold left to destroy, and the aggregate survives a standard 121°C, 15 psi, 15-minute cycle intact.

Step 4 – state what actually works: The accepted protocol is **134°C for 18 minutes** in a porous load autoclave, ideally preceded by immersion in **1N sodium hydroxide** or sodium hypochlorite (20,000 ppm available chlorine) for one hour. Incineration is the only wholly certain method.

Step 5 – state the practical dental rule: Because reprocessing is so unreliable, instruments used on a patient with known or suspected Creutzfeldt–Jakob disease, particularly on neural or lymphoid tissue, should be **single use and incinerated**. The pulp and the tonsil are the relevant high-infectivity tissues in dentistry.

Step 6 – match to the option: Option B gives the correct reason (no nucleic acid, already-misfolded aggregate) and the correct protocol (134°C for 18 minutes with 1N NaOH), so it is the answer.

Why the other options are wrong:

- A, a thick spore coat: is false. Prions are not bacteria and do not sporulate. Bacterial spores, unlike prions, are in fact killed by a proper autoclave cycle.
- C, killed by routine glutaraldehyde: is the opposite of the truth. Glutaraldehyde is not merely ineffective, it **fixes** the prion protein onto the instrument and makes it harder still to remove.
- D, replicating faster than the cycle kills: is a misconception. Prions propagate by **templating**, converting normal PrP^{C} into PrP^{Sc}, and they do not replicate at all inside an autoclave chamber.

Final Answer: No nucleic acid, already-misfolded resistant protein aggregate $\Rightarrow$ B

Answer: (B) Go Back to Q9

Q10.

Solution

Concept – the scrub and the routine wash have different targets: The routine wash aims at the transient flora only. The scrub aims at the transient flora *and* the resident flora, and it must keep working under the glove.

Step 1 – separate the two floras: **Transient** flora sit loosely on the skin surface, are picked up from patients and surfaces, and are removed mechanically by soap and water. **Resident** flora, mainly coagulase-negative staphylococci and diphtheroids, live in the deeper layers, the follicles and the ducts, and cannot be washed off.

Step 2 – describe the routine hand wash: Plain soap and water for **15 to 20 seconds**, covering the hands and wrists, performed before and after every patient contact and after glove removal. Its purpose is to remove transient flora, dirt and organic matter. It has no residual action.

Step 3 – describe the surgical scrub: An **antiseptic** agent, typically **4 percent**

chlorhexidine gluconate or **povidone iodine**, applied for **2 to 5 minutes** over the hands and forearms **up to the elbows**, with hands held above elbow level so water runs from clean to less clean, followed by drying with a sterile towel.

Step 4 – state the extra purpose the scrub serves: It removes transient flora, **substantially reduces** the resident flora, and leaves a **persistent residual** antimicrobial film on the skin. That residue matters because gloves develop microperforations during surgery, and the residual antiseptic suppresses regrowth of resident flora inside the warm, moist glove.

Step 5 – note why chlorhexidine is preferred: Chlorhexidine binds to the stratum corneum and keeps acting for about 6 hours. Povidone iodine has a broader immediate spectrum but a shorter residual effect and is inactivated by organic matter.

Step 6 – state the limit honestly: Neither procedure sterilises skin. Resident flora in the follicles always survive. That is precisely why sterile gloves are worn on top of the scrub, and it is why option B is the only complete answer.

Why the other options are wrong:

- A, both plain soap with only duration differing: misses the central difference. The **agent** changes, from plain soap to an antiseptic, and it is the antiseptic that provides the residual action a scrub exists for.
- C, the routine wash sterilising the skin: reverses the two and is false in any case. No hand procedure sterilises skin, because the resident flora deep in the follicles always persist.
- D, the scrub only after and the wash only before: is invented. The scrub is performed **before** donning sterile gloves for a surgical procedure, and the routine wash is done both before and after patient contact.

Final Answer: Antiseptic agent, 2 to 5 minutes to the elbows, resident flora reduced with residual action $\Rightarrow$ B

Answer: (B) Go Back to Q10

Q11.

Solution

Concept – barrier protection is chosen by the procedure, not by the patient's label: Standard precautions assume every patient is potentially infectious, so the aerosol decides the barrier.

Step 1 – read what the procedure generates: Ultrasonic scaling is one of the

highest aerosol-generating procedures in dentistry, alongside the air rotor and the air-water syringe. The aerosol carries saliva, blood, plaque bacteria and calculus fragments, and it stays suspended in the operatory air.

Step 2 – list the exposed portals: The **conjunctiva**, the **nasal and oral mucosa**, non-intact skin and contaminated clothing are all routes by which that aerosol reaches the operator.

Step 3 – assign a barrier to each portal: Gloves protect the hands. A **surgical mask** protects the nasal and oral mucosa and filters spatter. **Protective eyewear with side shields**, or a face shield, protects the conjunctiva, which is a genuine route of transmission for herpes simplex and for blood-borne viruses. A **gown** protects clothing and skin.

Step 4 – explain why eyewear is not optional: Ocular injury and infection from dental spatter is well documented, and the eye has no other barrier. Calculus fragments launched by an ultrasonic tip can also cause corneal abrasion. Eye protection during an aerosol-generating procedure is mandatory, not a preference.

Step 5 – invoke standard precautions: Standard precautions apply to **every** patient regardless of known infection status, because most carriers of HBV, HCV and HIV are undiagnosed. The absence of a known infection therefore changes nothing.

Step 6 – add the supporting measures: A pre-procedural mouth rinse and high-volume evacuation reduce aerosol load substantially, but they supplement rather than replace the personal barriers. Option C names the full set correctly.

Why the other options are wrong:

- A, gloves alone because no known infection: fails twice. It ignores standard precautions, and it leaves the eyes and respiratory mucosa completely unprotected against a heavy aerosol.
- B, eye protection optional: is unacceptable during an aerosol-generating procedure. The conjunctiva is a proven portal of entry, so eyewear is a requirement.
- D, reusing washed gloves: is never permitted. Gloves are single use; washing damages the glove material, causes wicking of fluid through microperforations, and does not decontaminate them.

Final Answer: Gloves, mask, protective eyewear with side shields and a gown $\Rightarrow$ C

Answer: (C) Go Back to Q11

Q12.

Solution

Concept – syphilis is staged, and each stage has its own oral lesion: *Treponema pallidum* is a spirochaete, and the oral picture changes completely as the disease progresses.

Step 1 – the primary stage: About 3 weeks after exposure a **chancre** appears at the site of inoculation. Extragenital chancres occur on the **lip** and **tongue**. It is a solitary, round, **painless**, **indurated** ulcer with a clean base, and it teems with treponemes, so it is highly infectious. It heals spontaneously in 3 to 8 weeks even without treatment, which is exactly why patients present late.

Step 2 – the secondary stage: About 6 weeks to 6 months later comes systemic dissemination, with fever, generalised lymphadenopathy and a rash that characteristically involves the **palms and soles**. In the mouth this stage produces **mucous patches**, which are painless greyish-white plaques on an erythematous base, and **snail track ulcers** where adjacent patches coalesce. Both are intensely infectious.

Step 3 – the latent stage: Serology stays positive but there are no lesions. The patient is not infectious by contact at this point.

Step 4 – the tertiary stage: Years later comes the **gumma**, a granulomatous, painless, destructive lesion. In the mouth it favours the **hard palate** and the tongue. Palatal gummata can **perforate**, producing an oro-nasal communication. Interstitial glossitis with atrophy of the papillae also occurs, and this leukoplakic tongue carries a raised risk of carcinoma.

Step 5 – separate congenital syphilis: Hutchinson's incisors, **mulberry molars**, interstitial keratitis and eighth nerve deafness are the features of **congenital** syphilis, not of acquired tertiary disease. Examiners deliberately mix these in.

Step 6 – match to the option: Chancre, then mucous patches and snail track ulcers, then a gumma. Option D gives that sequence exactly.

Why the other options are wrong:

- A, gumma first then chancre then mucous patches: has the whole sequence scrambled. The gumma is the **last** lesion, appearing years later, not the first.
- B, mucous patches first, then a painful ragged ulcer, then Hutchinson's incisors: is wrong at every step. The primary lesion is the chancre, not a mucous patch, and Hutchinson's incisors belong to **congenital** syphilis.
- C, a painful sloughing ulcer first, then interstitial keratitis, then a chancre: is wrong because the chancre is **painless** and comes **first**, and interstitial keratitis is again a **congenital** feature.

Final Answer: Chancre, then mucous patches and snail track ulcers, then gumma

$\Rightarrow$ D

Answer: (D) Go Back to Q12

Q13.

Solution

Concept – location in the mouth tells the two viruses apart: Coxsackie disease sits at the **back** of the mouth. Herpetic disease sits at the **front** and always attacks the gingiva.

Step 1 – read the distribution given: The lesions are on the **soft palate, anterior faucial pillars and uvula**. That is the posterior, non-keratinised mouth.

Step 2 – name that pattern: Fever with vesicles and shallow ulcers confined to the posterior oropharynx is **herpangina**, caused by **Coxsackie virus group A**, most often serotypes A1 to A6, A8, A10 and A22. It is an enterovirus, spread faeco-orally and by droplet, and it peaks in summer in young children.

Step 3 – use the hands and feet: The stem adds vesicles on the **palms and soles**. Oral ulcers plus vesicles on hands and feet is **hand, foot and mouth disease**, caused mainly by **Coxsackie A16** and by enterovirus 71. Both syndromes in the same child point squarely to the Coxsackie group.

Step 4 – use the negative findings: The stem says the **gingivae are entirely normal**. This is the decisive clue. Primary herpetic gingivostomatitis **always** produces an acute marginal gingivitis with fiery red, swollen, bleeding gingivae. No gingivitis effectively excludes herpes.

Step 5 – complete the herpes contrast: Herpetic lesions favour the **anterior** mouth and the **keratinised** attached gingiva and hard palate, are accompanied by perioral vesicles and marked systemic upset, and recur later as herpes labialis because the virus is latent in the trigeminal ganglion. Herpangina is posterior, spares the gingiva, has no perioral lesion, does not recur and settles in about a week.

Step 6 – conclude: Posterior distribution, plus hands and feet, plus normal gingiva, gives Coxsackie group A.

Why the other options are wrong:

- A, herpes simplex type 1: is excluded by the normal gingiva, by the strictly posterior distribution, by the absence of perioral vesicles and by the lesions on the palms and soles, which herpes does not cause.
- C, varicella zoster: gives a generalised centripetal rash on the trunk and face in crops at different stages, with only sparse oral vesicles. It does not confine

itself to the fauces, and it does not produce a palm and sole distribution.

- D, Epstein–Barr virus: causes infectious mononucleosis, with an exudative tonsillitis, palatal **petechiae** rather than vesicles, marked cervical lymphadenopathy and atypical lymphocytes. It causes no vesicles on the hands and feet.

Final Answer: Coxsackie virus, group A $\Rightarrow$ B

Answer: (B) Go Back to Q13

Q14.

Solution

Concept – one progenitor, two schools, two kinds of immunity: T and B lymphocytes look identical under the light microscope, so they are told apart by where they are educated and by what they do.

Step 1 – start at the common origin: Both arise from the **common lymphoid progenitor** in the bone marrow. The lineage split happens at the point marked X in the figure, and everything downstream follows from where each cell then goes.

Step 2 – follow the T cell: The precursor destined to be a T cell **leaves** the marrow and travels to the **thymus** to mature. There it rearranges its T cell receptor and passes positive and negative selection, which is how self-tolerance is established. The "T" stands for thymus.

Step 3 – follow the B cell: The B cell precursor **stays and matures in the bone marrow** itself in humans, rearranging its immunoglobulin genes there. The "B" comes from the bursa of Fabricius, the organ in birds where this was first shown, and the bone marrow is its mammalian equivalent.

Step 4 – assign the function of the T cell: T cells mediate **cell-mediated immunity**. CD4 helper T cells recognise antigen on MHC class II and coordinate the whole response by cytokine secretion. CD8 cytotoxic T cells recognise antigen on MHC class I and kill virus-infected and tumour cells directly. T cells handle intracellular organisms, viruses, fungi, graft rejection and delayed hypersensitivity.

Step 5 – assign the function of the B cell: B cells mediate **humoral immunity**. On activation, usually with T helper cell assistance, they differentiate into **plasma cells** that secrete antibody, and into memory B cells. Antibody handles extracellular organisms and toxins by neutralisation, opsonisation and complement activation.

Step 6 – note their locations and proportions: Circulating lymphocytes are roughly **60 to 70 percent T** and **10 to 20 percent B**. In lymph nodes, B cells

populate the **follicles and germinal centres** of the cortex, while T cells populate the **paracortex**. Option B alone states both maturation sites and both functions correctly.

Why the other options are wrong:

- A, both maturing in the thymus: is false. Only the T cell is educated in the thymus. The B cell completes its maturation in the bone marrow and never enters the thymus.
- C, T cells maturing in marrow and secreting antibody while B cells mature in the thymus and kill: reverses both the sites and the functions. Only the plasma cell, a B cell derivative, secretes antibody.
- D, B cells handling intracellular organisms and T cells secreting immunoglobulin: reverses the functions. Antibody cannot reach an intracellular organism, which is exactly why cell-mediated immunity exists, and T cells secrete cytokines rather than immunoglobulin.

Final Answer: T cells mature in the thymus (cell-mediated), B cells in the bone marrow (humoral) $\Rightarrow$ $\boxed{\text{B}}$

Answer: (B) Go Back to Q14

Answer Key

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