NEET MDS General Pathology and Microbiology

Sample Paper – 2

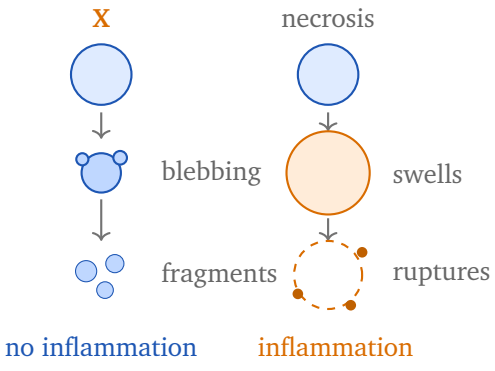
Duration: 11 Minutes **Maximum Marks: 56**

Instructions

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

Cell Injury and Inflammation

Q1. The figure below contrasts two forms of cell death. In the pathway marked **X** the cell shrinks, throws out surface blebs and breaks into membrane-bound fragments. The statement that correctly distinguishes **X** from necrosis is:



(A) X is a passive, energy independent process, whereas necrosis requires ATP throughout

(B) X is energy dependent and leaves the plasma membrane intact, so no inflammation follows, whereas necrosis is energy independent, ruptures the membrane and provokes inflammation

(C) X ruptures the plasma membrane and spills enzymes into the tissue, whereas necrosis keeps the membrane intact

(D) Both X and necrosis always excite a brisk acute inflammatory response in the surrounding tissue

Q2. Two chemical mediators of acute inflammation are compared. The first is preformed in mast cell granules and released within seconds of injury; the second is a nonapeptide generated in plasma from high molecular weight kininogen by kallikrein. The pairing of each mediator with its principal effect is:

(A) Histamine produces the pain of inflammation; bradykinin produces vasodilation

(B) Histamine and bradykinin both act chiefly as chemotactic agents that draw neutrophils to the site

(C) Histamine produces arteriolar vasodilation and increased venular permeability; bradykinin produces pain

(D) Histamine is the plasma-derived kinin and bradykinin is the granule-stored amine

Q3. Of the five cardinal signs of acute inflammation, *calor* and *rubor* are both produced by one and the same vascular event. That event is:

(A) Vasodilation of arterioles with a resulting increase in local blood flow

(B) Increased vascular permeability with escape of a protein-rich exudate

(C) Emigration of neutrophils across the venular wall into the tissue

(D) Action of bradykinin and prostaglandins on local nerve endings

Neoplasia

Q4. Anaplasia, literally a "backward formation", is the morphological hallmark of malignant transformation. The set of features that best defines it is:

(A) Uniform cells with regular nuclei, abundant cytoplasm and a low nucleocytoplasmic ratio

(B) Marked pleomorphism, hyperchromatic nuclei, a nucleocytoplasmic ratio approaching 1:1 and atypical mitotic figures

(C) An increase in the number of cells in an organ with the normal architecture fully retained

(D) Replacement of one differentiated adult cell type by another differentiated adult cell type

Q5. p53 is called the guardian of the genome. The statement that correctly places p53 within the two classes of cancer-related genes is:

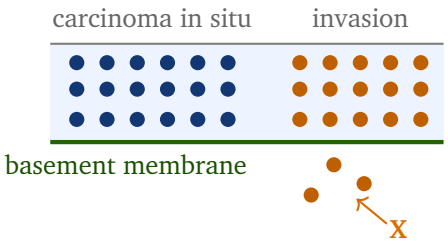
(A) It is a tumour suppressor gene, so both alleles must be lost before function is lost; its normal product arrests the cell in G1 to allow DNA repair, and drives the cell into apoptosis if repair fails

(B) It is a proto-oncogene, so a gain-of-function mutation in a single allele is enough to drive unrestrained proliferation

(C) It is a tumour suppressor gene whose normal function is to accelerate the cell through the G1/S checkpoint

(D) It is a DNA repair enzyme that directly excises damaged bases from the double helix

Q6. The figure below shows an epithelium in which full-thickness dysplastic change has occurred. On the left the tumour cells remain above the green line; on the right they have carried out the step marked **X**. Step **X**, which converts carcinoma in situ into invasive carcinoma, is:



(A) The appearance of mitotic figures above the basal layer of the epithelium

(B) Dysplasia extending through the full thickness of the epithelium

(C) Penetration of the basement membrane by the tumour cells

(D) Loss of surface keratinisation over the abnormal epithelium

Haemodynamic Disorders and Wound Healing

Q7. Two days after internal fixation of a fractured shaft of femur, a 24-year-old man becomes breathless and confused and develops a petechial rash over the chest, neck and axillae. The type of embolism responsible is:

(A) Fat embolism, from the marrow of the fractured long bone

(B) Air embolism, from entry of air into an open vein

(C) Amniotic fluid embolism

(D) Septic embolism from a vegetation of infective endocarditis

Q8. A poorly controlled diabetic on long-term systemic corticosteroids has an extraction socket that is still unhealed at three weeks. The socket is infected and a root fragment has been retained in it. Every factor listed delays healing, but the one that does so chiefly by **suppressing the inflammatory phase and inhibiting collagen synthesis** is:

(A) The long-term corticosteroid therapy

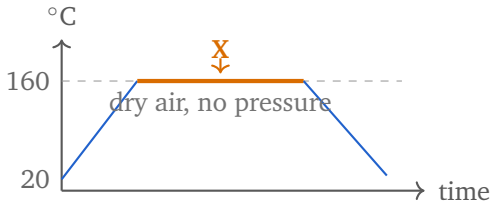
(B) The infection in the socket

(C) The retained root fragment acting as a foreign body

(D) The poorly controlled diabetes

General Microbiology and Sterilisation

Q9. The chart below plots a **dry heat** sterilisation cycle run in a hot air oven, used for glass Petri dishes, oils, powders and sharp cutting instruments. The holding phase is marked **X**. The parameters at **X** are:



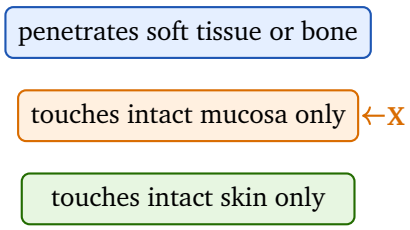
(A) 121°C for 15 minutes

(B) 134°C for 3 minutes

(C) 180°C for 2 hours

(D) 160°C for 2 hours

Q10. The Spaulding classification sorts instruments by the tissue they contact, as shown below. A dental mouth mirror and an impression tray fall into the tier marked **X**. That tier and the processing it demands are:



(A) Critical, requiring only intermediate-level disinfection

(B) Non-critical, requiring only cleaning with detergent and water

(C) Critical, requiring sterilisation without exception

(D) Semi-critical, requiring sterilisation or, where the item cannot tolerate heat, high-level disinfection

Q11. The chemical agent used in dentistry as a cold sterilant, working as a high-level disinfectant in about 20 minutes and as a sterilant only after roughly 10 hours of immersion, and used for items that cannot withstand heat, is:

(A) 70 per cent ethyl alcohol

(B) 2 per cent alkaline glutaraldehyde

(C) 0.2 per cent chlorhexidine gluconate

(D) 4 per cent formaldehyde as a surface wipe

Systemic Bacteriology and Virology

Q12. Pus from a facial abscess grows a Gram-positive coccus arranged in grape-like clusters, catalase positive, forming golden yellow colonies. The single test that separates the pathogenic species from the other members of this genus is:

(A) The catalase test

(B) Optochin sensitivity

(C) Bacitracin sensitivity

(D) The coagulase test

Q13. A 3-year-old child has fever, malaise, drooling and tender cervical lymphadenopathy. The gingiva is fiery red and oedematous throughout, and multiple small vesicles on the gingiva, palate and tongue have ruptured into shallow yellowish ulcers with red margins. The causative agent is:

(A) Coxsackievirus group A

(B) Herpes simplex virus type 1

(C) Varicella zoster virus

(D) Candida albicans

Immunology and Hypersensitivity

Q14. A dental surgeon develops an itchy, erythematous, scaly eczematous rash confined to the dorsum of both hands, appearing about 48 hours after a day of operating in latex gloves. Patch testing is positive at 72 hours. The same mechanism underlies the tuberculin test and nickel dermatitis. This reaction is:

(A) Type I hypersensitivity

(B) Type II hypersensitivity

(C) Type IV hypersensitivity

(D) Type III hypersensitivity

Detailed Solutions

Q1.

Solution

Concept – two ways a cell can die, and they behave oppositely: The figure describes apoptosis on the left and necrosis on the right. Every examinable difference between them follows from one fact, that apoptosis is a controlled programme the cell runs on itself while necrosis is an accident that happens to the cell.

Step 1 – identify X from the morphology given: Cell shrinkage, surface blebbing and fragmentation into membrane-bound bodies are the classic sequence of **apoptosis**. Necrosis does the reverse, the cell swells.

Step 2 – decide the energy requirement: Apoptosis is an active, **ATP dependent** programme. It needs energy to activate caspases, to cleave DNA at internucleosomal sites and to dismantle the cytoskeleton in an orderly way. Necrosis is **energy independent**, because it is precisely the failure of ATP production that causes it.

Step 3 – decide what happens to the membrane: In apoptosis the plasma membrane stays **intact** throughout. The cell parcels its contents into apoptotic bodies, each still wrapped in membrane. In necrosis the membrane **ruptures**, and lysosomal enzymes and cell contents spill into the tissue.

Step 4 – follow the membrane to the inflammation: Because apoptotic bodies keep their contents sealed in and are eaten by neighbouring cells and macrophages within minutes, nothing leaks out, so **no inflammation** results. Because necrotic cells spill their contents, those contents act as danger signals, so a brisk **acute inflammatory** reaction follows. This is why the two bottom labels in the figure differ.

Step 5 – match to the option: Option B states energy dependent, membrane intact and no inflammation for X, and energy independent, membrane ruptured and inflammation for necrosis. All three pairs are correct, so B is the answer.

Step 6 – note two further contrasts worth carrying into the exam: Apoptosis affects **single scattered cells** and may be entirely physiological, as in the shedding of deciduous tooth germ cells or in involution. Necrosis affects **sheets of contiguous cells** and is always pathological.

Why the other options are wrong:

- A: reverses the energy statement. Apoptosis is the **active** process and necrosis is the passive one, so calling X passive and necrosis ATP requiring is exactly backwards.
- C: reverses the membrane statement. Membrane rupture with spillage of en-

zymes is **necrosis**, not apoptosis, and it is the very reason necrosis inflames while apoptosis does not.
- D: is wrong because apoptosis provokes **no** inflammatory response at all. Only necrosis does.

Final Answer: X is ATP dependent with an intact membrane and no inflammation; necrosis is not $\Rightarrow$ B

Answer: (B) Go Back to Q1

Q2.

Solution

Concept – each mediator of inflammation owns a particular effect: The examiner tests whether you can attach the right function to the right molecule rather than list mediators as a block.

Step 1 – identify the first mediator from the clue: Preformed in **mast cell granules** and released within seconds identifies **histamine**. It is also stored in basophils and platelets, and it is the very first mediator to act in acute inflammation.

Step 2 – state what histamine does: Acting on H1 receptors, histamine causes **arteriolar vasodilation**, which is what produces the redness and the heat, and **increased permeability of venules** by making endothelial cells contract, which lets the exudate out and produces the swelling.

Step 3 – identify the second mediator from the clue: A **nonapeptide** generated in plasma from **high molecular weight kininogen** by **kallikrein** identifies **bradykinin**, the end product of the kinin system.

Step 4 – state what bradykinin does: Bradykinin also dilates vessels and raises permeability, but its distinguishing action, and the one it is asked about, is that it stimulates local nerve endings to produce **pain**. Prostaglandins do not cause pain by themselves, they sensitise those nerve endings so that bradykinin hurts far more.

Step 5 – assemble the pairing: Histamine gives vasodilation and permeability; bradykinin gives pain. That is option C.

Why the other options are wrong:

- A: swaps the two effects. Histamine is not the pain mediator and bradykinin is not primarily the vasodilator of the first seconds, so both halves are wrong.
- B: is wrong because neither molecule is a significant chemotaxin. Neutrophil chemotaxis is the job of **C5a**, **leukotriene B4**, **IL-8** and bacterial peptides.

- D: swaps the origins. Histamine is the **amine** stored in granules, while bradykinin is the **plasma-derived kinin**, so the option reverses the source of each.

Final Answer: Histamine causes vasodilation and permeability; bradykinin causes pain $\Rightarrow$ C

Answer: (C) Go Back to Q2

Q3.

Solution

Concept – the five cardinal signs, each with its own mechanism: Celsus described four and Virchow added the fifth. Each sign maps to a specific event, and the question asks which single event explains two of them at once.

Step 1 – list the five signs with their mechanisms: *Rubor* (redness) and *calor* (heat) come from **vasodilation and increased blood flow**. *Tumor* (swelling) comes from **increased permeability with exudate**. *Dolor* (pain) comes from **bradykinin and prostaglandins** acting on nerve endings, helped by the pressure of the exudate. *Functio laesa* (loss of function) results from the combination of pain and swelling.

Step 2 – take the sign asked about first, rubor: After a brief transient vasoconstriction, the arterioles dilate. More blood enters the capillary bed, the bed opens up, and the tissue turns **red**.

Step 3 – take calor: The blood arriving is at core temperature, about 37°C, whereas peripheral tissue such as skin or oral mucosa sits below that. Pouring more warm blood through the part makes it feel **hot**. So heat is simply the thermal consequence of the same hyperaemia that caused the redness.

Step 4 – confirm that one event covers both: Redness and heat therefore share a single cause, **vasodilation with increased blood flow**, which is option A.

Step 5 – note the clinical use: This is why an acute periapical abscess is red and warm from the outset, before any measurable swelling appears. The vascular calibre change happens within seconds, whereas exudation takes minutes.

Why the other options are wrong:

- B, increased permeability with exudate: is the mechanism of **tumor**, the swelling, not of redness or heat.
- C, neutrophil emigration: is the cellular phase of inflammation. It produces pus if it continues, but it accounts for none of the five cardinal signs directly.

- D, bradykinin and prostaglandins on nerve endings: is the mechanism of **dolor**, the pain, and has nothing to do with redness or heat.

Final Answer: Vasodilation of arterioles with increased blood flow $\Rightarrow$ A

Answer: (A) Go Back to Q3

Q4.

Solution

Concept – anaplasia is the loss of differentiation: The word means to form backward. An anaplastic tumour has lost both the structure and the function of the tissue it came from.

Step 1 – take pleomorphism: The cells and their nuclei vary widely in **size and shape**, in contrast to the monotonous uniformity of a benign tumour. Giant tumour cells with a single huge nucleus or several nuclei may appear.

Step 2 – take the nuclear changes: The nuclei are **hyperchromatic**, staining darkly because they hold an abnormally large quantity of DNA. Nucleoli are large and often multiple, reflecting heavy ribosomal RNA synthesis for a cell that is constantly dividing.

Step 3 – take the nucleocytoplasmic ratio: Normally the nucleus occupies about a quarter of the cell, giving a ratio near **1:4** or **1:6**. In anaplasia the nucleus enlarges while cytoplasm is not made, so the ratio approaches **1:1**.

Step 4 – take the mitoses: Mitoses are not merely numerous, they are **atypical**, with tripolar, quadripolar and multipolar spindles. Abnormal spindles are far more suggestive of malignancy than a raised mitotic count on its own, since a healing wound also shows many mitoses but all of them normal.

Step 5 – match to the option: Option B lists pleomorphism, hyperchromasia, a ratio near 1:1 and atypical mitoses together, which is the definition of anaplasia.

Step 6 – note the loss of polarity: Anaplastic cells also lose their orientation to one another and to the basement membrane, growing in disorganised sheets. Loss of polarity is the architectural half of anaplasia and completes the picture.

Why the other options are wrong:

- A: describes **normal or benign** cells. Uniform nuclei and a low nucleocytoplasmic ratio are the exact opposite of anaplasia.
- C: describes **hyperplasia**, an increase in cell number with normal architecture retained. It is an adaptive, potentially reversible change, not neoplasia.
- D: describes **metaplasia**, the replacement of one adult cell type by another,

as in the bronchial epithelium of a smoker. It is also adaptive and reversible.

Final Answer: Pleomorphism, hyperchromatic nuclei, a 1:1 ratio and atypical mitoses $\Rightarrow$ B

Answer: (B) Go Back to Q4

Q5.

Solution

Concept – oncogenes push, tumour suppressors brake: Cancer genes come in two families. Understanding which family a gene belongs to tells you how many alleles must be hit and in which direction the mutation acts.

Step 1 – define the oncogene side: A **proto-oncogene** normally promotes growth, and its mutated form, the **oncogene**, is a **gain-of-function** mutation. One mutated allele is enough, so it behaves as **dominant** at the cellular level. Examples are RAS, MYC and ERBB2. Think of a stuck accelerator.

Step 2 – define the tumour suppressor side: A **tumour suppressor** normally restrains growth, and its mutation is a **loss-of-function**. Both alleles must be inactivated, which is Knudson's two-hit hypothesis, so it behaves as **recessive** at the cellular level. Examples are RB, APC and p53. Think of failed brakes.

Step 3 – place p53: p53, on chromosome 17p, is a **tumour suppressor**. It is the single most commonly mutated gene in human cancer, altered in more than half of all tumours, including oral squamous cell carcinoma.

Step 4 – follow what normal p53 actually does: DNA damage stabilises p53. It then transcribes **p21**, which inhibits cyclin dependent kinases and **arrests the cell in G1**. That pause buys time for the repair machinery to work on the damage.

Step 5 – follow what happens if repair fails: If the damage cannot be repaired, p53 switches to transcribing **BAX** and other pro-apoptotic genes and pushes the cell into **apoptosis**. Arrest, repair, or kill, that triad is why it is called the guardian of the genome.

Step 6 – see the consequence of losing it: With both p53 alleles lost, damaged DNA is neither repaired nor eliminated. Mutations accumulate at every division, and the cell walks steadily toward malignancy. Option A states all of this correctly.

Why the other options are wrong:

- B: puts p53 in the wrong family. It is not a proto-oncogene and its cancer-causing mutations are **loss-of-function**, not gain-of-function. The dominant single-allele model belongs to RAS and MYC.

- C: reverses the function. p53 **halts** the cell at G1/S; it does not accelerate the cell through that checkpoint. That description would fit cyclin D or CDK4.
- D: confuses p53 with a repair enzyme. p53 is a **transcription factor**. It orders repair by switching genes on, but it excises no bases itself. Direct excision is the work of the nucleotide and base excision repair enzymes.

Final Answer: A tumour suppressor needing two hits, causing G1 arrest for repair or apoptosis $\Rightarrow$ A

Answer: (A) Go Back to Q5

Q6.

Solution

Concept – the basement membrane is the line that defines invasion: The whole difference between carcinoma in situ and invasive carcinoma is which side of one thin layer the tumour cells sit on.

Step 1 – define carcinoma in situ: The epithelium shows **full-thickness** dysplasia, so cytologically the cells are already malignant. But the basement membrane is **intact** and every tumour cell lies above it. That is the left half of the figure.

Step 2 – define invasive carcinoma: The same malignant cells **breach** the basement membrane and enter the underlying connective tissue. That is the right half of the figure, and it is step X.

Step 3 – explain how the cells get through: They loosen from one another by losing **E-cadherin**, attach to laminin in the membrane, then secrete **matrix metalloproteinases** such as type IV collagenase that digest a path through it, and finally migrate into the stroma.

Step 4 – explain why this one step matters so much: The connective tissue below the membrane carries **lymphatics and blood vessels**; the epithelium above it has none. Crossing the membrane is therefore the moment the tumour gains access to the routes of **metastasis**. Carcinoma in situ, by definition, cannot metastasise.

Step 5 – see the clinical consequence: Carcinoma in situ of the oral mucosa is cured by complete local excision. Once the membrane is breached, node status must be assessed and neck treatment becomes part of the plan. One thin line changes the whole management.

Why the other options are wrong:

- A, mitoses above the basal layer: is a feature of **dysplasia** and is already

present in carcinoma in situ, so it cannot be the step that converts one into the other.

- B, full-thickness dysplasia: is the **definition** of carcinoma in situ itself. The question already tells you it has occurred, so it is the starting point, not the change.
- D, loss of surface keratinisation: is another minor dysplastic feature seen in the epithelium. It has no bearing on whether the basement membrane has been crossed.

Final Answer: Penetration of the basement membrane $\Rightarrow$ C

Answer: (C) Go Back to Q6

Q7.

Solution

Concept – name the embolus from the clinical setting: An embolus is any detached intravascular mass carried by the blood to a site distant from its origin. The setting almost always names the type.

Step 1 – read the setting: A fractured **shaft of femur**, a long bone with a large marrow cavity rich in fat, plus operative instrumentation of that marrow.

Step 2 – read the timing: Symptoms at **24 to 72 hours** after the injury. The fat embolism syndrome characteristically has this latent interval, which is a strong pointer, since air and amniotic fluid embolism strike within seconds to minutes.

Step 3 – read the triad: Breathlessness, **confusion** and a **petechial rash** over the chest, neck and axillae are the classic triad of fat embolism syndrome. The rash is the most specific of the three, arising from occlusion of dermal capillaries by fat globules.

Step 4 – explain the mechanism: Marrow fat globules enter torn venous sinusoids in the fracture site, travel to the lungs and lodge in the pulmonary microvasculature, causing hypoxia. Some globules pass on to the brain and skin. Free fatty acids released from those globules then injure endothelium directly, which is why the syndrome worsens over the first day or two rather than beginning at full force.

Step 5 – confirm the answer: Setting, timing, triad and mechanism all point to **fat embolism**, which is option A.

Why the other options are wrong:

- B, air embolism: needs air to enter an open vein, as in neck surgery, a sitting-position procedure or a mishandled central line. It causes **immediate** col-

lapse, not a syndrome appearing two days later, and it produces no petechial rash.

- C, amniotic fluid embolism: occurs only during **labour or immediately postpartum** in an obstetric patient. It is impossible in a 24-year-old man with a femoral fracture.
- D, septic embolism: requires an infective focus such as an endocardial vegetation. Nothing here suggests endocarditis, and the picture would be of fever and infected infarcts, not a petechial rash with confusion at 48 hours.

Final Answer: Fat embolism from the fractured long bone $\Rightarrow$ A

Answer: (A) Go Back to Q7

Q8.

Solution

Concept – every option delays healing, so the mechanism decides the answer: The question deliberately gives four genuine causes of delayed healing and then asks which one works by a named mechanism. Read the mechanism, not the list.

Step 1 – read the mechanism demanded: Suppression of the **inflammatory phase** and inhibition of **collagen synthesis**.

Step 2 – test corticosteroids against it: Glucocorticoids are potent anti-inflammatories. They inhibit phospholipase A2, so both prostaglandins and leukotrienes fall, and they blunt neutrophil and macrophage recruitment. Since macrophages are the cells that release the growth factors driving repair, the whole inflammatory phase is damped down.

Step 3 – take the second half of the mechanism: Corticosteroids also directly **inhibit fibroblast proliferation and collagen synthesis**, and increase collagen breakdown. Less collagen means less tensile strength, so the wound heals slowly and weakly. Both halves of the stated mechanism therefore belong to corticosteroids, and A is the answer.

Step 4 – see why the delay is prolonged here: The therapy is **long term** and systemic, so the effect is continuous. This is also why such patients need care with elective extractions.

Step 5 – fix the classification in memory: **Local** factors delaying healing are infection, foreign body, poor blood supply, movement of the wound and ionising radiation. **Systemic** factors are diabetes, corticosteroids, malnutrition especially protein and vitamin C deficiency, old age and zinc deficiency.

Why the other options are wrong:

- B, infection: is the commonest cause of delayed healing, but its mechanism is the **opposite** of the one described. It **prolongs and intensifies** the inflammatory phase rather than suppressing it, and bacterial enzymes destroy tissue directly.
- C, the retained root fragment: is a **foreign body**, and it too **sustains** inflammation rather than suppressing it, keeping the wound in a chronic inflammatory state until it is removed. It does not inhibit collagen synthesis.
- D, the diabetes: delays healing through **microangiopathy** causing poor perfusion, impaired neutrophil chemotaxis and killing, and non-enzymatic glycation of collagen. It impairs the inflammatory response qualitatively, but it does not suppress the phase in the manner described, and it is not a direct inhibitor of collagen synthesis. This is the closest distractor.

Final Answer: Long-term corticosteroid therapy $\Rightarrow$ A

Answer: (A) Go Back to Q8

Q9.

Solution

Concept – dry heat sterilisation needs a much higher temperature and a much longer time: The hot air oven and the autoclave both use heat, but because dry air is a poor conductor and carries no latent heat, the oven must run hotter and far longer.

Step 1 – recall the standard hot air oven cycle: 160°C for 2 hours (120 minutes) is the accepted standard cycle, and the figure confirms it with dry air at atmospheric pressure and no pressure limb.

Step 2 – explain the killing mechanism: Dry heat kills by **oxidative destruction** of cell constituents, denaturing proteins and charring organic matter. Moist heat instead kills by coagulating proteins, which happens at a much lower temperature.

Step 3 – explain why the numbers must be so high: Steam carries a large **latent heat of vaporisation** which it dumps onto the load the moment it condenses, and it penetrates rapidly. Dry air has no latent heat and conducts poorly, so the temperature must rise to 160°C and be held for 2 full hours to kill spores.

Step 4 – note the accepted equivalents: 170°C for 1 hour and 180°C for 30 minutes are the recognised alternative dry heat cycles. As the temperature climbs the time falls, but 180°C is never paired with 2 hours.

Step 5 – name what the oven is for: Items that steam would spoil or cannot reach, namely **glassware**, **oils**, **greases**, **powders** and **sharp cutting instruments**, since dry heat does not blunt the cutting edge and does not corrode. In

dentistry these are the burs, endodontic files, glass slabs and hand cutting instruments.

Step 6 – note the holding time point: As with the autoclave, the 2 hours is the **holding** time at 160°C marked X, measured after the oven reaches temperature. It excludes the long heat-up and the slow cool-down.

Why the other options are wrong:

- A, 121°C for 15 minutes: is the **autoclave** cycle, which is moist heat under pressure. The question specifies dry heat in a hot air oven, so this is the intended trap.
- B, 134°C for 3 minutes: is the fast **autoclave** cycle at 30 psi used in vacuum autoclaves. Again moist heat, not dry.
- C, 180°C for 2 hours: mixes a real dry heat temperature with the wrong time. At 180°C the correct holding time is **30 minutes**; 2 hours there would needlessly damage the load.

Final Answer: 160°C for 2 hours ⇒ D

Answer: (D) Go Back to Q9

Q10.

Solution

Concept – Spaulding sorts instruments by the risk of the tissue they touch: The rule is simple. The more sterile the tissue an instrument enters, the more rigorous its processing must be.

Step 1 – define the critical tier: Instruments that **penetrate soft tissue or bone** or enter the bloodstream. They must be **sterilised**, with no alternative permitted. In dentistry these are the surgical burs, elevators, forceps, scalers, scalpel blades and endodontic files.

Step 2 – define the semi-critical tier: Instruments that **contact intact mucous membrane or non-intact skin but do not penetrate**. They should be **sterilised**; if the item cannot survive heat, then **high-level disinfection** is the accepted minimum.

Step 3 – define the non-critical tier: Items that touch only **intact skin**, such as the chair, the light handle and the blood pressure cuff. Intact skin is an effective barrier, so cleaning with detergent and **low or intermediate-level disinfection** is enough.

Step 4 – place the instruments named in the question: A **mouth mirror** and

an **impression tray** touch the mucosa but do not cut into it or reach bone. They are therefore **semi-critical**, which the figure marks as X.

Step 5 – state the processing X requires: Sterilise them if they will take it, and a metal mirror handle and a metal tray will. Where the item is heat sensitive, such as some plastic trays, high-level disinfection with 2 per cent glutaraldehyde is acceptable. Option D states exactly this.

Step 6 – note the practical caveat: Because a mirror can contact blood if the mucosa is broken, most guidelines simply advise heat sterilising every semi-critical item that can tolerate it, and using single-use plastic trays otherwise.

Why the other options are wrong:

- A: is wrong twice. A mirror is not critical, and intermediate-level disinfection would in any case never be enough for a critical item.
- B: places the mirror in the wrong tier. Non-critical applies only to items touching **intact skin**, and the mouth is mucosa, not skin.
- C: names the right processing but the wrong tier. Critical is reserved for instruments that **penetrate** tissue or bone, and a mirror penetrates nothing. The option also removes the recognised high-level disinfection fallback for heat-sensitive items.

Final Answer: Semi-critical, needing sterilisation or high-level disinfection $\Rightarrow$ D

Answer: (D) Go Back to Q10

Q11.

Solution

Concept – the cold sterilant: One chemical is sporicidal at room temperature given long enough contact, and that property is what earns it the name cold sterilant.

Step 1 – read the two contact times given: About **20 minutes** for high-level disinfection and about **10 hours** for sterilisation. That pair of numbers belongs to **2 per cent alkaline glutaraldehyde** and to nothing else in the list.

Step 2 – explain how it works: Glutaraldehyde is a dialdehyde that **alkylates** the amino, sulphydryl and hydroxyl groups of microbial proteins and nucleic acids, cross-linking them so the organism cannot function.

Step 3 – explain the difference between the two times: At 20 minutes it kills vegetative bacteria, fungi, mycobacteria and viruses, which is the definition of high-level disinfection. Only prolonged immersion of about 10 hours kills **spores**,

and only then can the process be called sterilisation.

Step 4 – explain the alkaline part: Glutaraldehyde is supplied acidic and stable but is not sporicidal in that state. Adding the activator raises the pH to about 7.5 to 8.5 and switches on the sporicidal activity, at the cost of shelf life. Once activated the solution lasts roughly 14 days.

Step 5 – explain what it is used for: Heat-sensitive items that cannot be autoclaved, in dentistry and medicine, including endoscopes and some plastic and rubber components. It is non-corrosive to metals and does not blunt cutting edges.

Step 6 – note the drawbacks: It irritates skin, eyes and the respiratory tract, so it needs a closed container, gloves and ventilation, and the item must be rinsed in sterile water before use.

Why the other options are wrong:

- A, 70 per cent ethyl alcohol: is only an **intermediate-level** disinfectant. It is **not sporicidal** at any contact time, and it evaporates too quickly to maintain contact, so it can never sterilise.
- C, 0.2 per cent chlorhexidine: is an **antiseptic** for skin and mucosa, used as a mouthwash. It is a low-level agent, not sporicidal, and is not an instrument sterilant at all.
- D, formaldehyde as a surface wipe: is sporicidal in the vapour form used for fumigating rooms, but as a wipe it is a surface disinfectant. It is more irritant and more carcinogenic than glutaraldehyde and has been displaced by it for instrument immersion, and the times quoted do not fit it.

Final Answer: 2 per cent alkaline glutaraldehyde $\Rightarrow$ B

Answer: (B) Go Back to Q11

Q12.

Solution

Concept – identify the genus first, then the species: Laboratory identification is a staircase. Each test narrows the field by one step, and the question asks for the step that separates the species within the genus.

Step 1 – use the morphology: Gram-positive cocci in **grape-like clusters** identify the genus **Staphylococcus**. Chains would have meant Streptococcus.

Step 2 – confirm with catalase: Catalase positive separates **Staphylococcus** from **Streptococcus**, which is catalase negative. But the question already tells you the organism is a cluster-forming coccus, so catalase adds nothing new here. It is a

genus-level test.

Step 3 – separate within the genus: The **coagulase test** splits the staphylococci into **Staphylococcus aureus**, which is coagulase positive, and the coagulase-negative staphylococci such as *S. epidermidis* and *S. saprophyticus*. This is the species-level test the question wants.

Step 4 – support with the colony colour: Golden yellow colonies come from the carotenoid pigment staphyloxanthin, which gives *S. aureus* its name. *S. epidermidis* forms white colonies. The colour supports the answer but coagulase confirms it.

Step 5 – explain what coagulase does: It converts fibrinogen to fibrin, walling the organism in a fibrin coat that shields it from phagocytosis, and it helps localise the infection into an **abscess**. It is therefore both a marker of the species and a genuine virulence factor, which fits the facial abscess described.

Step 6 – note the two forms tested: The slide test detects **bound** coagulase or clumping factor and gives an immediate result, while the tube test detects **free** coagulase and is the confirmatory test, read after incubation.

Why the other options are wrong:

- A, catalase: separates **Staphylococcus from Streptococcus**, a genus-level distinction. Every staphylococcus is catalase positive, so it cannot separate *S. aureus* from the others. This is the main trap.
- B, optochin sensitivity: separates **Streptococcus pneumoniae**, which is sensitive, from the other alpha-haemolytic viridans streptococci. It is irrelevant to staphylococci.
- C, bacitracin sensitivity: separates **Streptococcus pyogenes**, group A, which is sensitive, from other beta-haemolytic streptococci. Again a streptococcal test, not a staphylococcal one.

Final Answer: The coagulase test $\Rightarrow$ D

Answer: (D) Go Back to Q12

Q13.

Solution

Concept – primary herpetic gingivostomatitis: This is the commonest symptomatic primary viral infection of the mouth in a young child, and its picture is highly stereotyped.

Step 1 – use the age: 6 months to 5 years is the classic age band. Maternal

antibody has waned and the child meets the virus for the first time.

Step 2 – use the systemic picture: Fever, malaise, irritability, drooling because swallowing hurts, and **tender cervical lymphadenopathy**. A primary infection is systemic; a recurrence is not.

Step 3 – use the gingival sign, which is the decisive one: The gingiva is **fiery red and oedematous throughout**, involving the whole marginal and attached gingiva. This acute marginal gingivitis is the single most characteristic feature and is what separates herpetic gingivostomatitis from every viral exanthem with oral ulcers.

Step 4 – use the lesion morphology and distribution: Small **vesicles** that rupture into shallow yellowish ulcers with red haloes, spread across gingiva, palate and tongue. Note that both **keratinised and non-keratinised** mucosa are involved, which is the pattern of primary infection.

Step 5 – name the agent: Herpes simplex virus type 1, an enveloped double-stranded DNA virus of the Herpesviridae. HSV-2 is chiefly genital, though it may cause oral disease too.

Step 6 – follow the latency: After the primary illness settles in 10 to 14 days, the virus travels up the sensory nerve and lies **latent in the trigeminal ganglion**. Sunlight, fever, trauma or stress reactivate it, producing **herpes labialis**, the cold sore at the vermilion border. That link between the primary and the recurrent disease is frequently examined.

Why the other options are wrong:

- A, Coxsackievirus group A: causes **herpangina** and **hand, foot and mouth disease**. Herpangina spares the gingiva entirely and its ulcers sit on the soft palate and fauces. The absence of generalised gingivitis rules it out, and hand, foot and mouth disease would show the skin lesions.
- C, varicella zoster virus: causes chickenpox, whose oral lesions are few and accompany a widespread **vesicular skin rash** in crops. No skin rash is described here, and diffuse gingivitis is not its pattern.
- D, Candida albicans: causes **pseudomembranous candidiasis**, which forms creamy white plaques that wipe off to leave a red base. It produces no vesicles and no fiery gingivitis with cervical lymphadenopathy.

Final Answer: Herpes simplex virus type 1 $\Rightarrow$ B

Answer: (B) Go Back to Q13

Q14.

Solution

Concept – timing and mediator together name the hypersensitivity type: Of the four Gell and Coombs types, only one is mediated by cells rather than antibody, and only one takes days rather than minutes or hours.

Step 1 – use the timing: The rash appears at **48 hours** and the patch test is read at **72 hours**. A delay of 24 to 72 hours is the signature of **Type IV, delayed hypersensitivity**. No antibody-mediated reaction is that slow.

Step 2 – identify the antigen: Chemical accelerators in latex, and nickel in jewellery, are **haptens**. They are too small to be immunogenic alone, so they penetrate the skin and bind host proteins to become complete antigens.

Step 3 – follow the sensitisation phase: Langerhans cells take up the hapten-protein complex, migrate to the regional lymph node and present it on MHC class II to **CD4 T cells**. Those cells expand into a memory population. This first exposure produces no rash at all.

Step 4 – follow the elicitation phase: On re-exposure the memory CD4 cells are activated in the skin and release **interferon gamma**, TNF and IL-2, which recruit and activate **macrophages**. The macrophages do the actual tissue damage, and the lag while all this happens is exactly why the reaction takes 24 to 72 hours.

Step 5 – confirm that no antibody is involved: Type IV cannot be transferred by serum. It transfers only with **T cells**, which is the classic experimental proof that it is cell mediated, and it means no IgE test will detect it.

Step 6 – note the other examples that share the mechanism: The **tuberculin test**, granuloma formation in tuberculosis and leprosy, graft rejection, and contact dermatitis to nickel, chromium or eugenol. All are Type IV.

Step 7 – note the clinical contrast that matters in dentistry: Latex causes two entirely different reactions. This eczematous, hand-limited, 48-hour picture is **Type IV** allergy to the rubber chemicals. Latex *protein* allergy is instead Type I, appearing as urticaria, wheeze or anaphylaxis within minutes. Distinguishing them decides whether the risk is a rash or a resuscitation.

Why the other options are wrong:

- A, Type I: is IgE-mediated and **immediate**, acting within minutes. It would present with urticaria, angioedema or anaphylaxis, not an eczematous rash first noticed two days later.
- B, Type II: is **IgG or IgM** binding antigen fixed on a cell surface, leading to complement-mediated or antibody-dependent cell lysis, as in a mismatched transfusion or autoimmune haemolytic anaemia. It causes no contact der-

matitis.

- D, Type III: is **immune complex** deposition with complement activation and neutrophil influx, as in serum sickness or the Arthus reaction. It develops in hours, not days, and the patch test would not be its investigation.

Final Answer: Type IV, delayed, T cell mediated hypersensitivity ⇒ C

Answer: (C) Go Back to Q14

Answer Key

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