NEET MDS General Pathology and Microbiology

Sample Paper – 4

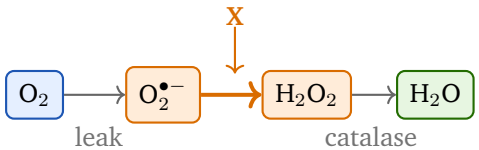
Duration: 11 Minutes **Maximum Marks: 56**

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 scheme below traces the handling of reactive oxygen species inside a cell. The enzyme at the step marked **X**, which converts superoxide anion into hydrogen peroxide, is:



(A) Catalase

(B) Superoxide dismutase

(C) Glutathione peroxidase

(D) Myeloperoxidase

Q2. A patient with long standing hypertension has a thickened left ventricle in which the myocytes are larger but no more numerous than normal. The reason the heart meets an increased workload by hypertrophy alone, whereas the endometrium or the liver can also mount hyperplasia, is:

(A) Cardiac myocytes are permanent cells that have left the cell cycle and cannot divide, so the only adaptation available is an increase in the size of the existing cells

(B) Cardiac myocytes are labile cells that divide continuously, and division is simply suppressed by pressure

(C) Hyperplasia occurs only in tissues that lack a blood supply of their own

(D) Hypertrophy of the heart reflects oedema within the myocytes rather than any increase in structural protein

Q3. A mandibular fracture is managed with intermaxillary fixation for 6 weeks. When the fixation is removed the masseter is visibly reduced in bulk, yet biopsy shows intact viable fibres of reduced diameter with no inflammation. The process responsible is:

(A) Hypoplasia, a failure of the muscle to develop to its normal size

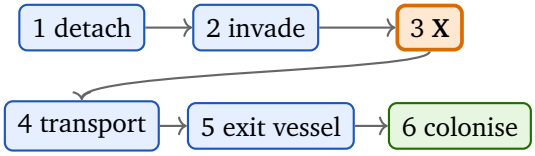
(B) Coagulative necrosis of the muscle fibres

(C) Atrophy, a reduction in cell size and number driven by reduced protein synthesis and increased degradation through the ubiquitin-proteasome pathway and autophagy

(D) Metaplasia of skeletal muscle into fibrous tissue

Neoplasia

Q4. The chain below sets out the metastatic cascade in its correct sequence. The step marked **X**, which immediately follows invasion through the basement membrane and stroma, is:



(A) Colonisation and growth of a deposit at the distant site

(B) Intravasation, the entry of tumour cells into the lumen of a vessel

(C) Detachment of tumour cells from one another through loss of E-cadherin

(D) Extravasation, the exit of tumour cells from the vessel into the distant tissue

Q5. A 62 year old man with squamous cell carcinoma of the floor of the mouth has a serum calcium of 3.2 mmol/L. A skeletal survey and bone scan show no metastatic deposits, and serum parathyroid hormone is suppressed. The most likely mechanism of his hypercalcaemia is:

(A) Tumour secretion of parathyroid hormone-related peptide, a paraneoplastic syndrome

(B) Ectopic secretion of antidiuretic hormone by the tumour

(C) Ectopic secretion of adrenocorticotrophic hormone by the tumour

(D) Osteolytic destruction of bone by metastatic deposits

Q6. A 34 year old woman has a soft, painless, pedunculated exophytic growth on the soft palate, about 6 mm across, made up of numerous white finger-like fronds giving a cauliflower appearance. Histology shows fronds of proliferating stratified squamous epithelium on fibrovascular cores, with koilocytes; HPV types 6 and 11 are identified. The lesion is a:

(A) Irritation fibroma

(B) Verrucous carcinoma

(C) Pyogenic granuloma

(D) Squamous papilloma

Haemodynamic Disorders and Wound Healing

Q7. The figure contrasts two infarcts. The wedge in the lung is haemorrhagic (red); the wedge in the spleen is anaemic (white). The feature that best explains why the lung infarct is red is:

lung: red infarct spleen: white infarct

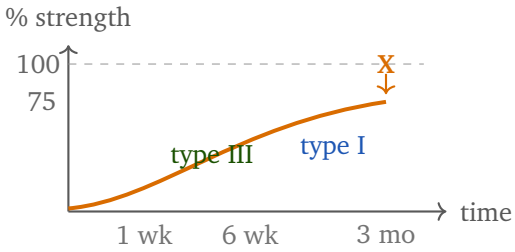
(A) The lung is a solid organ supplied by a single end artery, so blood cannot enter the dead zone

(B) The lung has a dual blood supply and a loose spongy texture, so blood from the bronchial circulation seeps into the necrotic zone through the porous tissue

(C) Infarction of the lung is always caused by venous rather than arterial occlusion

(D) The lung infarct is simply older, and every infarct turns red as it organises

Q8. The curve shows the tensile strength of a healing skin wound as a percentage of unwounded skin. At the point marked **X**, about 3 months after wounding, the strength and the collagen are best described as:



(A) About 70 to 80 percent of unwounded skin, with the type III collagen of early granulation tissue largely replaced by type I

(B) A full 100 percent of unwounded skin, with type I collagen replaced by type III

(C) About 10 percent of unwounded skin, with only type III collagen present and no remodelling yet

(D) About 30 percent of unwounded skin, with the collagen composition unchanged from day 1

General Microbiology and Sterilisation

Q9. The Bowie-Dick test is run each morning in an empty vacuum autoclave chamber, using a chemical indicator sheet inside a test pack. The two sheets below show a pass and a fail. The test is designed to detect:

pass: uniform fail: pale centre

(A) Survival of *Geobacillus stearothermophilus* spores after the cycle

(B) Failure of air removal from the chamber, such as an air leak or a vacuum pump fault, which leaves an air pocket that steam cannot penetrate

(C) Residual endotoxin on the surface of the processed instruments

(D) Adequacy of the drying phase at the end of the cycle, and nothing else

Q10. An unwrapped extraction forceps is needed urgently in a busy clinic and must be processed in the shortest cycle that still sterilises. The rapid (flash) autoclave cycle used for this purpose runs at:

(A) 134°C at about 2.2 bar (roughly 30 psi) for a 3 minute holding time

(B) 121°C at 15 psi for a 15 minute holding time

(C) 160°C at atmospheric pressure for a 60 minute holding time

(D) 134°C at about 2.2 bar for a 30 minute holding time

Q11. An alcohol-based hand rub containing 70 percent isopropanol is used between patients. The statement that correctly describes both its mechanism and its limitation is:

(A) It alkylates microbial nucleic acids, and it is fully sporicidal, so soap and water are never required

(B) It is most effective at a concentration of 100 percent, because the absence of water speeds up killing

(C) It denatures and coagulates microbial proteins, and it is not sporicidal, so hands must be washed with soap and water after contact with a patient carrying *Clostridioides difficile* or other spores

(D) It works purely by mechanically removing organisms, and it removes spores more effectively than soap and water do

Systemic Bacteriology and Virology

Q12. A 28 year old woman had rheumatic fever in childhood following a *Streptococcus pyogenes* pharyngitis, and now has a prosthetic mitral valve. She is listed for a surgical extraction that will involve manipulation of gingival tissue. She has no drug allergy. The correct infective endocarditis prophylaxis is:

(A) None, because her childhood penicillin prophylaxis against rheumatic fever also protects the valve

(B) Amoxicillin 500 mg three times daily for 5 days, started after the extraction

(C) Amoxicillin 2 g orally as a single dose, 30 to 60 minutes before the procedure

(D) None, because current guidelines have withdrawn prophylaxis for every category of patient

Q13. A dental surgeon sustains a needlestick from a patient known to be hepatitis C RNA positive. The statement that correctly describes hepatitis C is:

(A) Fewer than 5 percent of infections become chronic, and an effective vaccine is available and is given to all health workers

(B) It spreads mainly by the faeco-oral route, so a needlestick carries no meaningful risk

(C) It is a defective virus that can only infect a person already carrying HBsAg

(D) Roughly 70 to 85 percent of acute infections become chronic, there is no vaccine, and post-exposure management is RNA testing with early direct acting antiviral treatment, which cures over 95 percent

Immunology and Hypersensitivity

Q14. A patient given equine antivenom is well at the time of injection, but 10 days later develops fever, an urticarial rash, arthralgia, lymphadenopathy and proteinuria. Serum C3 and C4 are low. Circulating antigen-antibody complexes have deposited in vessel walls, glomeruli and joints and have activated complement. This serum sickness reaction is:

(A) Type I hypersensitivity

(B) Type II hypersensitivity

(C) Type IV hypersensitivity

(D) Type III hypersensitivity

Detailed Solutions

Q1.

Solution

Concept – free radical injury is a chain, and each link has its own antioxidant: Reactive oxygen species are produced constantly, and the cell survives only because a specific enzyme sits at each step of the chain.

Step 1 – read the step the figure marks: The figure marks the conversion of **superoxide anion** ($O_2^{\bullet -}$) into **hydrogen peroxide** (H_2O_2).

Step 2 – name the enzyme that performs exactly that step: Superoxide dismutase dismutates two molecules of superoxide into one molecule of hydrogen peroxide and one of oxygen. It exists as a manganese-containing form in mitochondria and a copper-zinc form in the cytosol.

Step 3 – see why superoxide must be removed: Superoxide is generated by leak from the mitochondrial electron transport chain and by NADPH oxidase in neutrophils. It is short lived but it inactivates iron-sulphur enzymes and, worse, it feeds the Fenton and Haber-Weiss reactions that generate the hydroxyl radical.

Step 4 – follow what happens after the marked step: The hydrogen peroxide produced is itself toxic, so **catalase** in peroxisomes splits it into water and oxygen, and **glutathione peroxidase** reduces it to water using reduced glutathione. The figure shows catalase on the later limb, which is a further clue that the marked step must be an earlier enzyme.

Step 5 – recall the damage the pathway prevents: If the chain is not intercepted, radicals attack lipids (lipid peroxidation of membranes), proteins (oxidation of side chains and fragmentation) and DNA (single strand breaks and thymine lesions). These three targets are the standard examination answer for how reactive oxygen species injure a cell.

Why the other options are wrong:

- A, catalase: acts one step **later**, on hydrogen peroxide, and the figure already labels it on that limb. It does not act on superoxide.
- C, glutathione peroxidase: also acts on hydrogen peroxide, and on lipid peroxides, converting them to water using glutathione. Again it is the wrong substrate.
- D, myeloperoxidase: is not an antioxidant at all. It is the neutrophil enzyme that **makes** hypochlorite from hydrogen peroxide and chloride to kill microbes, so it increases oxidant load rather than removing it.

Final Answer: Superoxide dismutase $\Rightarrow$ $\boxed{\text{B}}$

Answer: (B) Go Back to Q1

Q2.

Solution

Concept – which adaptation a tissue can use is set by its proliferative capacity: Hypertrophy is more cell substance; hyperplasia is more cells. Only a tissue that can divide is allowed hyperplasia.

Step 1 – classify the tissues by proliferative capacity: Labile cells divide continuously, such as surface epithelium and bone marrow. **Stable** cells are quiescent but can be recalled into the cycle, such as hepatocytes and renal tubular cells. **Permanent** cells have left the cycle for good, such as cardiac myocytes, skeletal muscle and neurons.

Step 2 – place the heart: Cardiac myocytes are **permanent** cells. Once terminally differentiated they cannot re-enter mitosis in any useful number.

Step 3 – draw the consequence: Because division is unavailable, the only response left to an increased pressure load is to make each existing myocyte bigger. That is **pure hypertrophy**, and it is why a hypertensive left ventricle is thick but contains no extra myocytes.

Step 4 – explain what hypertrophy actually is at cell level: It is a genuine increase in **structural protein and organelles**. Mechanical stretch and agonists such as angiotensin II switch on transcription factors, more sarcomeres are assembled, and foetal genes such as atrial natriuretic factor are re-expressed. It is not swelling and not fluid.

Step 5 – contrast with the tissues named in the stem: The endometrium is hormone responsive and **labile**, so oestrogen drives hyperplasia. The liver is **stable**, so after partial resection hepatocytes re-enter the cycle and the organ regenerates by hyperplasia. Both have the option the heart does not.

Why the other options are wrong:

- B, myocytes are labile: is factually reversed. Labile cells are those dividing continuously, such as gut and skin epithelium, and cardiac muscle is the classic **permanent** tissue.
- C, hyperplasia only in tissues without their own blood supply: is invented. Blood supply has nothing to do with the choice; proliferative capacity does. The endometrium is richly vascular and is the standard example of hyperplasia.
- D, hypertrophy as myocyte oedema: confuses adaptation with **reversible cell injury**. Cellular swelling is hydropic change from pump failure and it is

an injury, not an adaptive gain of contractile protein.

Final Answer: Cardiac myocytes are permanent cells, so only hypertrophy is available ⇒ A

Answer: (A) Go Back to Q2

Q3.

Solution

Concept – atrophy is shrinkage of a tissue that once reached normal size: The word applies only when the organ developed properly and then lost substance.

Step 1 – read the clinical setting: Six weeks of intermaxillary fixation means six weeks of **disuse**. The masseter has not been loaded at all.

Step 2 – read the biopsy: The fibres are **intact and viable**, only reduced in diameter, and there is no inflammation. So the muscle has not died and has not been replaced; each fibre has simply shrunk.

Step 3 – name the process: That is **atrophy**, defined as a reduction in the size of an organ or tissue by loss of cell substance, and often loss of cell number too. It is an adaptive response to reduced demand, and it is reversible once loading resumes.

Step 4 – give the mechanism: Protein **synthesis** falls because the anabolic stimulus of load is gone, and protein **degradation** rises. Degradation runs mainly through the **ubiquitin-proteasome** pathway, in which proteins are tagged with ubiquitin and fed into the proteasome, with **autophagy** handling whole organelles. The net negative balance shrinks the fibre.

Step 5 – list the standard causes so the pattern is complete: Disuse (as here, or a limb in plaster), denervation, loss of blood supply, inadequate nutrition, loss of endocrine stimulation, ageing, and pressure. Alveolar bone resorption after tooth loss is the everyday dental example of disuse atrophy.

Why the other options are wrong:

- A, hypoplasia: means the tissue **never developed** to full size, a developmental failure. This masseter was normal for 28 years and shrank afterwards, so the word does not apply.
- B, coagulative necrosis: would show dead eosinophilic fibres with loss of nuclei and an inflammatory response. The biopsy explicitly shows viable fibres and no inflammation.
- D, metaplasia: is replacement of one differentiated cell type by **another dif-**

ferentiated cell type, such as ciliated to squamous epithelium in a smoker. Skeletal muscle turning into fibrous tissue is not metaplasia, and no such change is described.

Final Answer: Disuse atrophy $\Rightarrow$ C

Answer: (C) Go Back to Q3

Q4.

Solution

Concept – the metastatic cascade is a fixed sequence, and the examiner tests the order: Every step is separately necessary, and a tumour cell that fails any one of them never forms a metastasis.

Step 1 – write the sequence out in full: (1) **Detachment** of cells from one another, (2) **invasion** of basement membrane and stroma, (3) **intravasation** into a vessel lumen, (4) **transport** in the circulation, (5) **extravasation** into the distant tissue, (6) **colonisation** and growth of the new deposit.

Step 2 – locate the marked step: The stem says X **immediately follows invasion**. Invasion is step 2, so X is step 3, which the figure also shows in position 3.

Step 3 – name step 3: Step 3 is **intravasation**, the penetration of a capillary or lymphatic wall so the cell enters the lumen.

Step 4 – explain each earlier step so the order is secure: Detachment depends on loss of **E-cadherin**, which normally glues epithelial cells together. Invasion needs degradation of type IV collagen of the basement membrane by **matrix metalloproteinases**, followed by attachment to laminin and fibronectin and locomotion through the loosened stroma.

Step 5 – explain the later steps: In transport most cells are destroyed by shear stress and by natural killer cells, so they survive by forming platelet-coated emboli. Extravasation is adhesion to endothelium at the distant site and passage back out. Colonisation is the hardest step, since the cell must find a permissive microenvironment and recruit a blood supply, which is why most circulating tumour cells never produce a metastasis.

Why the other options are wrong:

- A, colonisation: is the **last** step (6), not the third. It cannot precede transport and exit from the vessel.
- C, detachment through loss of E-cadherin: is the **first** step (1) and comes before invasion, whereas X comes after it.

- D, extravasation: is step 5. It is the mirror image of intravasation but happens at the far end, after transport, so it is out of place here. Confusing intravasation with extravasation is the trap in this question.

Final Answer: Intravasation into a vessel lumen $\Rightarrow$ B

Answer: (B) Go Back to Q4

Q5.

Solution

Concept – a paraneoplastic syndrome is a distant effect of a tumour that is not caused by the tumour mass or its metastases: The tumour acts as an endocrine gland it has no right to be.

Step 1 – use the negative findings first: The bone scan and skeletal survey are **clear**, so there are no lytic deposits eating bone. That removes the mechanical explanation for hypercalcaemia at once.

Step 2 – use the suppressed parathyroid hormone: Low PTH proves the parathyroid glands are behaving correctly and switching themselves off in the face of a high calcium. So primary hyperparathyroidism is excluded, and the calcium must be driven by something PTH-like that the assay does not measure.

Step 3 – name the mediator: Parathyroid hormone-related peptide (PTHrP). Squamous cell carcinomas, of the head and neck, oesophagus and lung, are the classic secretors.

Step 4 – explain why PTHrP fits every finding: PTHrP shares its N-terminal sequence with PTH, so it binds the same PTH1 receptor. It therefore raises osteoclastic bone resorption and increases renal calcium reabsorption, pushing calcium up. Because it is a different molecule, the standard intact-PTH assay reads **low**, exactly as reported. This picture is called humoral hypercalcaemia of malignancy.

Step 5 – fix the wider list of paraneoplastic syndromes: SIADH with hyponatraemia and Cushing syndrome from ectopic ACTH both go with **small cell** lung carcinoma; hypercalcaemia from PTHrP goes with **squamous** carcinoma; and hypertrophic osteoarthropathy, myasthenic Lambert-Eaton syndrome, acanthosis nigricans and migratory thrombophlebitis (Trousseau sign) complete the standard set. These syndromes matter because they may be the first sign of an occult tumour.

Why the other options are wrong:

- B, ectopic ADH: would cause **hyponatraemia** with concentrated urine, not

hypercalcaemia, and it belongs with small cell carcinoma.

- C, ectopic ACTH: would produce Cushing syndrome with hypertension, hypokalaemic alkalosis and hyperglycaemia. It does not raise calcium, and again it is a small cell association.
- D, osteolytic metastases: is directly **excluded by the imaging**. It is also not a paraneoplastic mechanism at all, since it is the physical presence of tumour in bone.

Final Answer: Tumour secretion of PTHrP $\Rightarrow$ A

Answer: (A) Go Back to Q5

Q6.

Solution

Concept – benign oral tumours are recognised from architecture plus aetiology: The shape of the lesion and the virus named together settle the diagnosis.

Step 1 – read the architecture: Numerous white **finger-like fronds** giving a cauliflower surface, each frond built of stratified squamous epithelium over a **fibrovascular core**. That papillary architecture is the definition of a papilloma.

Step 2 – read the aetiology: HPV 6 and 11, the low-risk types, with **koilocytes** (perinuclear haloes) as the cytological footprint of HPV. Squamous papilloma is the HPV 6/11 lesion of the mouth.

Step 3 – read the behaviour: Soft, painless, pedunculated, only 6 mm, and on the **soft palate**, which with the uvula and the tongue is the favoured site. Squamous papilloma is benign, does not invade, and is cured by conservative excision including the base of the stalk.

Step 4 – contrast with the other benign lesion of the mouth you must know: The **irritation fibroma** is the commonest benign oral growth, but it is a reactive fibrous hyperplasia rather than a true neoplasm. It is a smooth, firm, dome-shaped, normally coloured nodule on the buccal mucosa along the occlusal line, or on the lip or tongue, caused by chronic trauma such as cheek biting. Histology shows dense collagen covered by intact epithelium, with **no fronds and no koilocytes**.

Step 5 – confirm the diagnosis: Fronds with fibrovascular cores plus HPV 6/11 plus koilocytes plus a benign pedunculated growth equals **squamous papilloma**.

Why the other options are wrong:

- A, irritation fibroma: is smooth and **not papillary**. It is a collagenous reactive nodule from trauma, with no viral aetiology, so both the surface and the

HPV finding are against it.

- B, verrucous carcinoma: is a **malignant** low-grade variant of squamous cell carcinoma. It is a broad, sessile, warty white plaque in an older tobacco user, and histology shows pushing invasion of the underlying tissue with bulbous rete pegs. A 6 mm pedunculated frondy lesion with no invasion is not verrucous carcinoma.
- C, pyogenic granuloma: is a lobular capillary haemangioma. It is a soft **red**, bleeding, often ulcerated mass, typically on the gingiva and often in pregnancy, and its histology is exuberant granulation tissue with numerous capillaries, not squamous fronds.

Final Answer: Squamous papilloma $\Rightarrow$ $\boxed{\text{D}}$

Answer: (D) Go Back to Q6

Q7.

Solution

Concept – an infarct is red or white depending on whether blood can get back into the dead tissue: The necrosis is the same; only the amount of haemorrhage into it differs.

Step 1 – define infarction: An area of **ischaemic necrosis** caused by occlusion of the arterial supply or the venous drainage of a tissue. Almost all infarcts are wedge shaped with the occluded vessel at the apex, which is why the figure draws wedges.

Step 2 – state the rule for red infarcts: Infarcts are **red (haemorrhagic)** when the organ has a **dual or anastomotic blood supply**, when the tissue is **loose and spongy** so extravasated blood can collect, when the tissue was previously **congested**, and when flow is **re-established** into a zone of already necrotic vessels (reperfusion).

Step 3 – apply the rule to the lung: The lung is supplied by both the **pulmonary** and the **bronchial** arteries. When a pulmonary artery branch is embolised, bronchial arterial blood still enters the dead segment through the anastomoses, and because lung is loose spongy tissue that blood leaks freely into the alveoli. The infarct therefore fills with blood and is red.

Step 4 – state the rule for white infarcts and apply it to the spleen: Infarcts are **white (anaemic)** in **solid** organs with a **single end-arterial** supply, because once the artery is blocked no second source can deliver blood and the dense tissue limits seepage. Spleen, kidney and heart behave this way, and the spleen wedge is therefore pale.

Step 5 – add the other red-infarct examples: Small bowel (rich mesenteric anastomoses and loose tissue), liver (hepatic artery plus portal vein), testis and ovary after torsion (venous outflow blocked while arterial inflow continues), and any tissue reperfused after thrombolysis.

Why the other options are wrong:

- A, single end-arterial supply: describes the **spleen**, not the lung, and it is the recipe for a **white** infarct. The option states the opposite of the truth.
- C, always venous occlusion: is false. Venous occlusion does give red infarcts, as in testicular torsion, but the standard pulmonary infarct follows an **arterial** thromboembolus lodging in a pulmonary artery branch. The word "always" also breaks the statement on its own.
- D, age of the infarct: reverses the natural history. Infarcts do not become red with time; a red infarct **loses** its blood as red cells are lysed and phagocytosed, and both red and white infarcts end as a pale fibrous scar (except in the brain, which liquefies).

Final Answer: Dual blood supply and loose spongy tissue $\Rightarrow$ B

Answer: (B) Go Back to Q7

Q8.

Solution

Concept – wound strength is a collagen story with a fixed timetable: Strength comes first from the type of collagen laid down, then from how it is cross-linked and rearranged.

Step 1 – read the point marked on the curve: X sits at **3 months** and the curve there is at roughly three quarters of the dashed 100 percent line, which is the plateau.

Step 2 – give the numbers along the whole timeline: At **1 week** the wound has only about **10 percent** of the strength of unwounded skin, which is why sutures are still doing the work and why removing them early causes dehiscence. Strength then rises quickly over the next 4 weeks. By about **3 months** it plateaus at approximately **70 to 80 percent**, and it never returns to 100 percent. A scar is permanently weaker than the skin it replaced.

Step 3 – give the collagen switch: Early granulation tissue lays down thin **type III** collagen, which is quick to produce but weak. During remodelling it is progressively degraded by matrix metalloproteinases and replaced by thick **type I** collagen, which is the strong collagen of normal dermis. The figure labels type III

on the early limb and type I on the later limb for this reason.

Step 4 – explain why the plateau is where it is: Strength depends on the amount of collagen, on the **cross-linking** between fibres, and on their **realignment** along lines of tension. Net collagen accumulation stops by about month 2 to 3, so any further gain in strength after that comes from cross-linking and reorientation, and it is small. The disorganised weave of a scar can never match the basketweave of normal dermis.

Step 5 – note the clinical use: This is why an extraction or surgical site is fragile in the first week, why sutures are removed around day 7 in the mouth, and why patients are told a scar keeps maturing and fading for months.

Why the other options are wrong:

- B, 100 percent with type I replaced by type III: is wrong twice. A scar never reaches 100 percent, and the replacement runs **type III to type I**, not the reverse.
- C, 10 percent with only type III: gives the figure for the **first week**, not for 3 months, and by 3 months type I dominates. The curve at X is clearly near its plateau, not at its origin.
- D, 30 percent with unchanged collagen: understates the plateau, and "collagen composition unchanged" denies remodelling, which is the entire point of the third phase of healing.

Final Answer: About 70 to 80 percent, with type III largely replaced by type I $\Rightarrow$ A

Answer: (A) Go Back to Q8

Q9.

Solution

Concept – steam sterilises only where steam actually reaches: A vacuum autoclave depends on removing all the air first, and the Bowie-Dick test is the daily check that it did.

Step 1 – state the physical problem: Air is heavier than steam and does not mix with it. Any air left in the chamber, in a pack, or in a lumen forms a **cold air pocket**. Steam cannot penetrate it, so the instruments inside that pocket never reach 134°C and are not sterilised, even though the chamber gauge reads correctly.

Step 2 – explain what the test is: A standard porous test pack with a **chemical indicator sheet** at its centre is run in an **empty** chamber on a full cycle, first thing

each morning before any load is processed.

Step 3 – read the two sheets in the figure: A **pass** is a **uniform** colour change across the whole sheet, meaning steam reached every part of the pack at the same moment. A **fail** is a pale or patchy centre, which is the footprint of an air pocket that steam could not displace, and it points to an air leak, a failing vacuum pump, or non-condensable gases in the steam supply.

Step 4 – keep chemical and biological indicators separate: A **chemical** indicator is a dye that changes colour when exposed to defined conditions. Class 1 is process indicator tape, which only says the pack went through a machine. Class 6 emulating indicators respond to time, temperature and steam together. The Bowie-Dick sheet is a chemical indicator used for a specific purpose, namely **air removal**. A **biological** indicator instead uses live spores and proves killing.

Step 5 – state what the test does not do: It is not a sterility test and it says nothing about any load, because the chamber is empty. It is a daily performance check of steam penetration, and a failed Bowie-Dick means the autoclave is taken out of service until the fault is found.

Why the other options are wrong:

- A, spore survival: describes a **biological** indicator, which is a different test with a different purpose. The Bowie-Dick pack contains a dye sheet, not spores.
- C, residual endotoxin: is detected by the limulus amoebocyte lysate assay and matters for pyrogen testing of injectables. Endotoxin is heat stable and no autoclave indicator addresses it.
- D, the drying phase only: is far too narrow and misses the point. A wet pack is a separate fault. Bowie-Dick tests **air removal and steam penetration**, which is a problem of the early vacuum phase, not the end of the cycle.

Final Answer: Failure of air removal from the chamber $\Rightarrow$ B

Answer: (B) Go Back to Q9

Q10.

Solution

Concept – higher temperature buys shorter time: Sterilisation is a trade between temperature and holding time, and the flash cycle sits at the hot, fast end of that trade.

Step 1 – recall the flash cycle numbers: **134°C**, at about **2.2 bar** above at-

mospheric (roughly 30 psi), for a **3 minute** holding time. This is the rapid or immediate-use cycle.

Step 2 – explain why 3 minutes is enough: Killing follows first-order kinetics, and every rise in temperature shortens the decimal reduction time steeply. Raising steam from 121°C to 134°C cuts the holding time needed for the same sporicidal effect from 15 minutes to 3.

Step 3 – explain the pressure: Water boils at 100°C at atmospheric pressure, and steam cannot be hotter than its boiling point at that pressure. To hold saturated steam at 134°C the chamber must be pressurised to about 2.2 bar. The pressure is not what kills; it is only what allows the temperature.

Step 4 – state when the cycle is used: For **unwrapped** instruments needed immediately, as in the stem. The whole cycle including heat-up and cool-down takes only a few minutes, which is why it suits a busy chairside.

Step 5 – state its limits: Because the item is unwrapped it has **no barrier** once the chamber opens, so it must be used at once and cannot be stored. There is no drying phase, so the instrument comes out wet, and recontamination during transfer is a real risk. For that reason the flash cycle is for genuine urgency, not for routine reprocessing, which should use a wrapped cycle.

Why the other options are wrong:

- B, 121°C for 15 minutes: is the correct **standard** autoclave cycle, but it is the slow one. The stem asks specifically for the shortest cycle that still sterilises, so 15 minutes of holding time fails the requirement.
- C, 160°C for 60 minutes at atmospheric pressure: is a **hot air oven** cycle, which is dry heat. It is far slower, it is not an autoclave at all, and 160°C would blunt or damage the instrument.
- D, 134°C for 30 minutes: has the right temperature but ten times the necessary holding time, so it defeats the purpose of a rapid cycle. The 30 minute figure at 134°C belongs to the special prion-inactivation cycle, not to routine flash sterilisation.

Final Answer: 134°C at about 2.2 bar for 3 minutes ⇒ A

Answer: (A) Go Back to Q10

Q11.

Solution

Concept – alcohol is fast and broad but it has one hard boundary: It kills almost everything on the hands, and it kills no spores at all.

Step 1 – state the mechanism: Alcohol **denatures and coagulates proteins**. It unfolds enzymes and structural proteins, and it also disrupts lipid membranes, so the organism loses both its envelope and its metabolism within seconds.

Step 2 – explain why 70 percent and not 100 percent: Water is required for protein denaturation. Absolute alcohol dehydrates the cell and merely fixes the surface protein, forming a coagulated shell that shields the interior. A 60 to 80 percent solution carries enough water to let the alcohol penetrate and denature protein throughout, so it is far more effective than 100 percent.

Step 3 – state the spectrum: Alcohol rubs are rapidly bactericidal, including against MRSA, and are fungicidal and virucidal against **enveloped** viruses such as hepatitis B, hepatitis C, HIV and influenza, because the envelope is lipid.

Step 4 – state the limits: Alcohol is **not sporicidal**. Spores have a keratin-like coat and a dehydrated cortex, so there is little free water and no membrane for the alcohol to attack. It is also unreliable against some **non-enveloped** viruses such as norovirus, and it works poorly on **visibly soiled** hands, since organic matter absorbs the alcohol before it reaches the organism.

Step 5 – draw the practical rule: Use the alcohol rub for routine hand hygiene between patients when hands look clean. Switch to **soap and water** when hands are soiled and after contact with a patient carrying *Clostridioides difficile* or other spore formers, because there the killing does not happen and the spores must be physically washed away instead.

Why the other options are wrong:

- A, alkylation and sporicidal: describes **ethylene oxide** and **glutaraldehyde**, which alkylate nucleic acids and proteins and are genuinely sporicidal. Alcohol does neither, and calling it sporicidal is the central error.
- B, 100 percent is best: reverses the pharmacology, as Step 2 shows. Absolute alcohol is less effective because it lacks the water needed for denaturation.
- D, purely mechanical removal: describes what **soap and water** do. Alcohol rubs are not rinsed off and remove nothing mechanically; they kill in situ. The claim that they clear spores better than washing is the exact reverse of practice.

Final Answer: Protein denaturation, and not sporicidal $\Rightarrow$ $\boxed{\text{C}}$

Answer: (C) Go Back to Q11

Q12.

Solution

Concept – prophylaxis is now restricted to the highest risk cardiac lesions and the highest risk procedures: Both halves of that sentence must be satisfied before an antibiotic is given.

Step 1 – trace how this patient reached her risk: *Streptococcus pyogenes* (group A, beta-haemolytic, bacitracin sensitive) caused her pharyngitis. Antibodies to the M protein cross-reacted with cardiac myosin and valve glycoprotein, an example of **molecular mimicry**, giving rheumatic fever 2 to 4 weeks later and scarring the mitral valve. That valve has since been replaced.

Step 2 – check the cardiac indication: A **prosthetic valve** is one of the highest risk categories, alongside previous infective endocarditis, certain unrepaired or recently repaired congenital heart disease, and cardiac transplant valvulopathy. She qualifies.

Step 3 – check the procedural indication: Prophylaxis applies to dental procedures involving **manipulation of gingival tissue**, the periapical region, or perforation of the oral mucosa. A surgical extraction is exactly that, so both halves are satisfied.

Step 4 – give the regimen: Amoxicillin 2 g orally as a single dose, 30 to 60 minutes before the procedure, in an adult with no allergy. The single preoperative dose is timed so that a peak blood level is present during the bacteraemia the procedure creates. Children receive 50 mg/kg. If she could not swallow, ampicillin or cefazolin 2 g intravenously would be used; with penicillin allergy, clindamycin 600 mg or azithromycin 500 mg.

Step 5 – name the organism the prophylaxis actually targets: Not *S. pyogenes*. The bacteraemia of a dental procedure is dominated by the **viridans streptococci** of plaque, and amoxicillin is chosen because it covers them. Rheumatic fever is the reason she has the prosthetic valve; viridans streptococci are the reason she needs the antibiotic today. Separating those two ideas is the whole point of the question.

Why the other options are wrong:

- A, childhood rheumatic prophylaxis is enough: confuses two different regimens. Secondary prophylaxis with long-acting penicillin prevents **recurrent group A streptococcal pharyngitis**. It does not cover the viridans streptococcal bacteraemia of a dental procedure, and low-dose continuous peni-

cillin in fact selects for resistant oral streptococci, which is why an alternative class is preferred if she is on it.

- B, a 5 day post-operative course: is wrong on timing and duration. The antibiotic must already be circulating **before** the bacteraemia occurs, since the bacteraemia is over within minutes. Starting afterwards is useless, and prolonged courses only add resistance and adverse effects.
- D, prophylaxis withdrawn for everyone: overstates one guideline. NICE in the United Kingdom did move to advising against routine prophylaxis, but the AHA, ESC and Indian practice all retain it for the **highest risk** group, which includes prosthetic valves. "Every category of patient" makes the option false.

Final Answer: Amoxicillin 2 g orally, single dose, 30 to 60 minutes before $\Rightarrow$ C

Answer: (C) Go Back to Q12

Q13.

Solution

Concept – hepatitis C is the chronic one with no vaccine but a cure: Its profile is almost the opposite of hepatitis B on both counts, which is why the two are examined together.

Step 1 – classify the virus: An enveloped single-stranded RNA flavivirus, transmitted **parenterally** by blood. So a needlestick is a genuine route of exposure, though the risk from a single injury is only about 3 percent, well below that of hepatitis B.

Step 2 – state the chronicity: Roughly **70 to 85 percent** of acute infections fail to clear and become **chronic**. Acute infection is usually silent, which is why most carriers are found incidentally. Of those chronic carriers, about 20 to 30 percent progress to cirrhosis over 20 to 30 years, and cirrhosis carries an ongoing risk of hepatocellular carcinoma.

Step 3 – explain why the chronicity is so high: HCV RNA polymerase has no proofreading, so the virus mutates continually and circulates as a swarm of quasispecies. The hypervariable region of the E2 envelope protein changes faster than neutralising antibody can be raised, so the immune response is always chasing a virus that has already moved.

Step 4 – draw the vaccine conclusion from the same fact: That same antigenic variability, across six major genotypes, is precisely why **no vaccine exists**. There is no stable target to immunise against, and unlike hepatitis B there is also no useful post-exposure immunoglobulin, since antibody is not protective.

Step 5 – state what is actually done after the needlestick: Baseline testing, then HCV **RNA** by PCR at about 3 to 6 weeks (RNA appears well before antibody), with antibody at 4 to 6 months. There is no prophylaxis to give. If infection is confirmed, **direct acting antivirals** such as sofosbuvir with velpatasvir cure over **95 percent** in 8 to 12 weeks. So hepatitis C is the virus you cannot prevent by vaccine but can cure by treatment, exactly the reverse of hepatitis B.

Why the other options are wrong:

- A, under 5 percent chronicity with a vaccine: inverts both key facts. Chronicity is high, not low, and no hepatitis C vaccine exists in any country. The figure of under 5 percent chronicity belongs to adult-acquired **hepatitis B**.
- B, faeco-oral: describes hepatitis **A** and **E**. Hepatitis C is blood-borne, and dismissing the needlestick risk on this basis would be a serious clinical error.
- C, defective virus needing HBsAg: describes hepatitis **D**, which cannot assemble without the hepatitis B surface antigen coat. Hepatitis C replicates entirely on its own.

Final Answer: High chronicity, no vaccine, curable with direct acting antivirals $\Rightarrow$ D

Answer: (D) Go Back to Q13

Q14.

Solution

Concept – Type III damage is done by the immune complex, not by the antibody sitting on a target: Where the complex lodges decides which organ is hurt, which is why the illness is multisystem.

Step 1 – read the mediator the stem gives: Circulating antigen-antibody complexes that deposit in vessel walls, glomeruli and joints. Soluble complexes forming in the circulation and depositing at a distance from where the antigen entered is the definition of **Type III** hypersensitivity.

Step 2 – use the timing: The reaction appears at **10 days**, not in minutes. That interval is the time the patient needs to raise IgG against the foreign horse protein. Once antibody appears in the presence of persisting antigen, complexes form. Serum sickness classically begins 7 to 14 days after the first exposure.

Step 3 – use the complement: Low C3 and C4 are the fingerprint of Type III. Deposited complexes fix complement through the classical pathway, consuming it, so serum levels fall. The fragments released, C3a and C5a, are anaphylatoxins and chemotaxins: they raise vascular permeability and pull neutrophils in. The

neutrophils cannot phagocytose a complex stuck to a wall, so they discharge lysosomal enzymes and reactive oxygen species outside the cell and destroy the vessel, giving a **fibrinoid necrotising vasculitis**. This frustrated phagocytosis is how the damage is done.

Step 4 – map each symptom onto a deposition site: Skin vessels give the **urticarial rash**; joints give the **arthralgia**; the glomerulus gives the **proteinuria**; systemic complement activation gives the **fever** and the lymphadenopathy. One mechanism, many organs.

Step 5 – name the local counterpart: The **Arthus reaction** is the localised form. Antigen injected into the skin of an individual who already has high circulating antibody forms complexes at the injection site within 4 to 12 hours, producing local oedema, haemorrhage and necrosis. Systemic version equals serum sickness, local version equals Arthus, and both are Type III.

Why the other options are wrong:

- A, Type I: is IgE bound to mast cells, degranulating within **minutes** of exposure. The 10 day delay excludes it, and Type I consumes no complement, so the low C3 and C4 would be unexplained.
- B, Type II: requires antibody binding an antigen **fixed on a cell surface or in tissue**, as in a mismatched transfusion or Goodpasture syndrome. Here the complexes form in the circulation first and then deposit, which is the defining difference from Type II.
- C, Type IV: is **cell mediated** and driven by T cells and macrophages, as in contact dermatitis or the tuberculin test. It involves **no antibody and no complement**, so it cannot explain either the immune complexes or the consumed C3 and C4, and its usual delay is 24 to 72 hours.

Final Answer: Type III immune complex hypersensitivity $\Rightarrow$ $\boxed{\text{D}}$

Answer: (D) Go Back to Q14

Answer Key

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