NEET MDS General Pathology and Microbiology

Sample Paper – 6

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

Instructions

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

Cell Injury and Inflammation

Q1. A gingival biopsy from a patient with long-standing multiple myeloma shows homogeneous eosinophilic extracellular deposits. Stained with Congo red and viewed under polarised light, the deposits show **apple-green birefringence**. The material deposited is:

(A) Haemosiderin

(B) Amyloid

(C) Dystrophic calcium salt

(D) Lipofuscin

Q2. An endogenous pigment that is insoluble, brownish-yellow, granular, accumulates in the cytoplasm of ageing hearts, livers and neurons, and

represents the indigestible end product of membrane lipid peroxidation, is:

(A) Lipofuscin

(B) Melanin

(C) Haemosiderin

(D) Anthracotic carbon

Q3. A mucocele of the lower lip is aspirated and yields a thin, clear, watery fluid that is low in protein and contains very few cells. In terms of the morphological patterns of acute inflammation, this exudate represents:

(A) Fibrinous inflammation

(B) Suppurative inflammation

(C) Serous inflammation

(D) Ulcerative inflammation

Neoplasia

Q4. Normal somatic cells shorten their telomeres with every division and arrest permanently once the telomeres reach a critical length. Cancer cells acquire limitless replicative potential largely by re-expressing the enzyme:

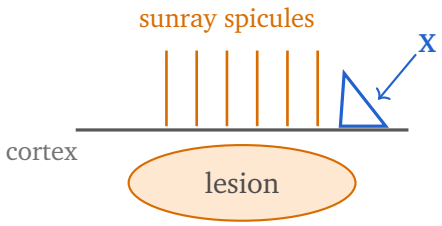
(A) Topoisomerase II

(B) Telomerase

(C) DNA ligase IV

(D) Poly ADP-ribose polymerase

Q5. A 24-year-old man has a rapidly enlarging, painful swelling of the body of the mandible. The radiograph, sketched below, shows spicules of new bone radiating perpendicular to the expanded cortex, together with a triangular cuff of reactive periosteal bone lifted at the margin of the lesion (marked **X**, Codman's triangle). The lesion is most likely:



(A) Osteosarcoma of the mandible

(B) Ossifying fibroma

(C) Compact osteoma

(D) Odontogenic keratocyst

Q6. Metastatic deposits in the jaw bones are uncommon, settle by preference in the posterior body and ramus of the mandible, and may present with numbness of the lower lip. The primary tumours that most often send such deposits to the jaws are those of the:

(A) Brain, retina and spinal cord

(B) Skin, lip and floor of mouth

(C) Stomach, pancreas and gall bladder

(D) Breast, lung, prostate, kidney and thyroid

Haemodynamic Disorders and Wound Healing

Q7. A child with nephrotic syndrome loses large amounts of albumin in the urine and develops generalised oedema with puffiness of the face and eyelids. Of the mechanisms of oedema, the one operating first and principally here is:

(A) Increased capillary hydrostatic pressure

(B) Reduced plasma colloid osmotic (oncotic) pressure

(C) Obstruction of lymphatic drainage

(D) Increased vascular permeability from inflammatory mediators

Q8. A clean surgical incision is closed with sutures and heals by first intention. The correct day-by-day sequence of events is:

(A) Macrophages fill the gap within 24 hours, neutrophils replace them on day 3, a clot forms on day 5, and granulation tissue appears only in week 3

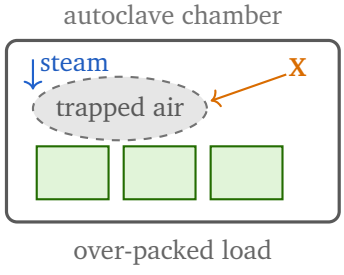
(B) A fibrin clot fills the narrow gap within 24 hours with neutrophils at the margins; by day 3 macrophages replace the neutrophils and granulation tissue begins to invade; by day 5 the space is filled with granulation tissue with maximal neovascularisation and the epidermis regains its normal thickness; through week 2 fibroblast proliferation and collagen accumulation continue while the vessels regress; by the end of month 1 an intact epidermis covers a pale, avascular scar

(C) Epithelial continuity is restored only after 3 weeks, and no clot or granulation tissue forms at any stage

(D) Myofibroblast-driven contraction shrinks the incision to a tenth of its original area by week 3, and this contraction is the main mechanism of closure

General Microbiology and Sterilisation

Q9. The sketch below shows a loaded autoclave chamber part-way through a cycle. The gauge reads the correct temperature, yet the packs at the centre of the load come out unsterile. Region **X** is a pocket of residual air lying over the packs. The commonest reason an autoclave cycle fails is:



(A) Use of distilled water instead of tap water in the reservoir

(B) A chamber pressure held marginally above 15 psi

(C) Failure to displace air from the chamber and from the packs, so that trapped air shields the load from direct steam contact, an error made worse by overloading

(D) Opening the door before the load has cooled

Q10. Under the Biomedical Waste Management Rules followed in Indian dental clinics, blood-soaked cotton and gauze from an extraction, together with the extracted tooth itself, must be discarded into the bin coloured:

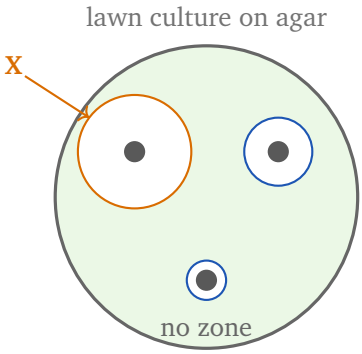
(A) Red

(B) White (translucent, puncture proof)

(C) Blue

(D) Yellow

Q11. A pus swab is plated as a lawn culture and tested by the Kirby-Bauer disc diffusion method, shown below. The zone of inhibition around disc **X** measures 22 mm, while the other two discs give a smaller zone and none at all. The 22 mm figure is correctly used by:



(A) Comparing it with the standard interpretive breakpoint table for that organism and that drug, and reporting the isolate as susceptible, intermediate or resistant

(B) Reading it directly as the minimum inhibitory concentration in micrograms per millilitre

(C) Declaring the drug with the widest zone to be the most potent drug in absolute terms

(D) Confirming that the drug is bactericidal rather than bacteriostatic

Systemic Bacteriology and Virology

Q12. Six weeks after a difficult third molar extraction, a man has a firm, indurated, board-like swelling at the angle of the mandible with several sinuses discharging pus. The pus contains hard yellow granules which, when crushed and stained, show a central tangle of Gram-positive branching filaments. The organism is:

(A) Actinomyces israelii

(B) Nocardia brasiliensis

(C) Blastomyces dermatitidis

(D) Mycobacterium tuberculosis

Q13. A 9-year-old unimmunised boy has fever, headache and painful bilateral parotid swelling that lifts the ear lobes. Stensen's duct is red but yields no pus on milking the gland, the serum amylase is raised, and two classmates have the same illness. The diagnosis is:

(A) Acute bacterial suppurative parotitis

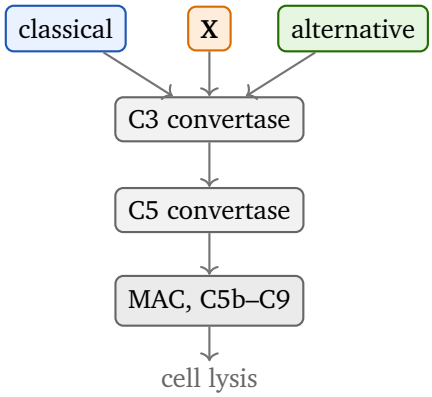
(B) Parotid sialolithiasis with obstruction

(C) Sjogren's syndrome

(D) Mumps (epidemic viral parotitis)

Immunology and Hypersensitivity

Q14. The scheme below shows the three complement pathways converging on C3 and then on a common terminal membrane attack complex. Pathway **X** is the one triggered when a plasma protein binds terminal mannose residues on a microbial surface, without any antibody being involved. Pathway **X** is the:



(A) Classical pathway

(B) Alternative pathway

(C) Lectin pathway

(D) Properdin-independent coagulation pathway

Detailed Solutions

Q1.

Solution

Concept – Congo red plus polarised light is the signature of one substance only: Amyloid is defined not by its chemistry, which varies, but by its physical form and by how it behaves with Congo red.

Step 1 – read the staining clue: The deposit takes up **Congo red**. Congo red is the standard screening stain for amyloid because the dye molecule is long and straight and slots between the stacked protein sheets.

Step 2 – read the polarised light clue: Under polarised light the Congo red stained deposit gives **apple-green birefringence**. This is the confirmatory test and it is considered specific for amyloid.

Step 3 – explain why the green colour appears: Amyloid, whatever protein it is made from, always folds into a **beta-pleated sheet** arranged in non-branching fibrils about 7.5 to 10 nm wide. That regular, ordered array is what aligns the Congo red molecules and splits polarised light into two components, producing the green shift.

Step 4 – read the clinical setting: The patient has **multiple myeloma**, a plasma cell neoplasm. The malignant clone pours out excess immunoglobulin light chains, and these are deposited as **AL** (amyloid light chain) type amyloid. Myeloma is the classic cause of AL amyloidosis.

Step 5 – link to the dental relevance: Amyloid deposits in the tongue produce **macroglossia** with lateral tooth indentations, and gingival or rectal biopsy is a standard, easy way to obtain tissue for diagnosis. That is why a gingival biopsy was taken here.

Why the other options are wrong:

- A, haemosiderin: is a golden-brown **granular, intracellular** iron pigment. It is demonstrated by the **Perls Prussian blue** reaction, not by Congo red, and it shows no birefringence.
- C, dystrophic calcium salt: stains deep **basophilic (blue-purple)** with haematoxylin, not eosinophilic, and is confirmed with **von Kossa** or alizarin red. It also has no apple-green birefringence.
- D, lipofuscin: is a brown **intracytoplasmic** wear-and-tear pigment, whereas the deposits here are described as extracellular. It is highlighted by PAS and by its own autofluorescence, not by Congo red.

Final Answer: Amyloid $\Rightarrow$ $\boxed{\text{B}}$

Answer: (B) Go Back to Q1

Q2.

Solution

Concept – each pigment carries its own message: Pigment questions are answered by matching three things, the colour, the location and the process the pigment reports on.

Step 1 – read the "ageing" clue: The pigment accumulates in **ageing** hearts, livers and neurons. That is the description of **lipofuscin**, the wear-and-tear or lipochrome pigment.

Step 2 – read the "lipid peroxidation" clue: Lipofuscin is the insoluble residue of **free radical damage to membrane lipids**. Peroxidised lipid cross-links with protein to form a polymer that no lysosomal enzyme can digest, so it simply piles up inside residual bodies.

Step 3 – state what it signifies: Lipofuscin itself is **harmless**. It is a marker, not a cause, telling you that the cell has sustained years of free radical injury. Its presence is not evidence of any current disease.

Step 4 – name the associated appearance: When many cells of a shrunken organ are loaded with it, the organ looks brown, and the combination of atrophy with lipofuscin is called **brown atrophy**, classically of the heart in the elderly and the cachectic.

Step 5 – classify it correctly: Lipofuscin is an **endogenous** pigment, made by the body itself, which is what the question asked for.

Why the other options are wrong:

- B, melanin: is endogenous, but it is **black-brown**, is made by melanocytes from tyrosine through tyrosinase, and reports on **pigment production**, not on lipid peroxidation. Orally it appears as racial pigmentation, the melanotic macule, and in Addison's disease or Peutz-Jeghers syndrome. It also has nothing to do with ageing.
- C, haemosiderin: is endogenous but it is an **iron storage** pigment derived from ferritin. It indicates **local haemorrhage** (as in a bruise or a haemangioma) or **systemic iron overload**. Its message is iron, not ageing.
- D, anthracotic carbon: is an **exogenous** pigment inhaled as soot, so it fails the "endogenous" requirement of the stem. It marks the lungs and hilar nodes of city dwellers and smokers.

Final Answer: Lipofuscin $\Rightarrow$ A

Answer: (A) Go Back to Q2

Q3.

Solution

Concept – the morphological patterns of acute inflammation are named after the exudate: Look at what comes out of the vessels, and the pattern names itself.

Step 1 – read the description of the fluid: It is **thin, clear, watery, low in protein and almost acellular**.

Step 2 – match it to the pattern: That is **serous** inflammation, the mildest pattern, produced by outpouring of a watery fluid from plasma or from mesothelial secretion.

Step 3 – explain what serous inflammation means physically: The vascular leak is modest. Only water and small solutes escape, because the endothelial gaps are not wide enough to let large molecules such as fibrinogen through, and leucocyte emigration has not become prominent.

Step 4 – give the familiar examples: A skin blister after a burn, and the fluid of an early viral vesicle, are the standard serous examples. The lip lesion here yields the same thin clear fluid.

Step 5 – place the patterns on a scale of severity: Serous is the mildest; **fibrinous** means a bigger leak that lets fibrinogen through; **suppurative** means neutrophils and liquefactive necrosis, that is pus; **ulcerative** means the surface has actually been shed. The exudate described sits at the mildest end.

Why the other options are wrong:

- A, fibrinous inflammation: needs a leak large enough for **fibrinogen** to escape and clot into a sticky meshwork, as on the pericardium in bread-and-butter pericarditis. The fluid here is explicitly **low in protein**, so no fibrin is present.
- B, suppurative inflammation: means **pus**, that is thick, creamy, neutrophil-rich fluid with liquefied debris, as in a periapical abscess. The aspirate here is clear and nearly acellular, the exact opposite.
- D, ulcerative inflammation: requires **loss of the surface epithelium** leaving a crater, as in an aphthous ulcer. A mucocele is a fluid-filled swelling with the epithelium intact over it, so no ulcer exists.

Final Answer: Serous inflammation $\Rightarrow$ C

Answer: (C) Go Back to Q3

Q4.

Solution

Concept – limitless replicative potential is a hallmark of cancer: A cell that must count its divisions cannot form a tumour, so the tumour has to disable the counter.

Step 1 – state the normal problem: Chromosome ends carry **telomeres**, repeats of the sequence TTAGGG. DNA polymerase cannot copy the very end of a linear molecule, so a little telomere is lost at each round of replication. This is the end-replication problem.

Step 2 – state the normal consequence: Once telomeres shorten to a critical length the exposed ends look like broken DNA. **p53** is activated and the cell enters permanent **replicative senescence**. Roughly 60 to 70 doublings are possible, which is the Hayflick limit named in the stem.

Step 3 – name the enzyme that solves it: Telomerase is a ribonucleoprotein. It carries its own RNA template and uses reverse transcriptase activity to add fresh TTAGGG repeats back on to the chromosome end, so the counter never runs down.

Step 4 – link it to cancer: Telomerase is active in germ cells and stem cells but is nearly silent in normal somatic cells. It is re-expressed in about **90 percent of human cancers**, including oral squamous cell carcinoma, and that re-expression is what confers immortality on the clone.

Step 5 – note why this hallmark needs a partner: Escaping senescence alone is not enough. The cell must usually also have lost p53 or Rb function first, so that it can survive long enough for telomerase to be switched back on. That is why immortality is listed as a separate hallmark from evading growth suppressors.

Why the other options are wrong:

- A, topoisomerase II: relieves supercoiling ahead of the replication fork. It is essential to ordinary DNA replication in every dividing cell and does nothing to lengthen telomeres. It is a drug target (etoposide, doxorubicin), which is why it is a tempting distractor.
- C, DNA ligase IV: seals the ends in **non-homologous end joining** of double-strand breaks. It repairs breaks; it does not extend chromosome ends, and losing it causes radiosensitivity, not immortality.
- D, poly ADP-ribose polymerase: senses **single-strand breaks** and recruits base excision repair. Its famous role is as the target of PARP inhibitors in BRCA-mutant tumours. It has no telomere-lengthening function.

Final Answer: Telomerase $\Rightarrow$ $\boxed{\text{B}}$

Answer: (B) Go Back to Q4

Q5.

Solution

Concept – the radiograph is the answer here: Two periosteal signs together, sunray spiculation and Codman's triangle, point to an aggressive bone-forming malignancy.

Step 1 – read the first radiographic sign: Spicules of new bone running **perpendicular** to the cortex give the **sunray** or sunburst appearance. The tumour raises the periosteum, and the Sharpey fibres stretched between the periosteum and the cortex become the scaffold along which reactive bone is laid down. Only a fast, bone-forming lesion does this.

Step 2 – read the second radiographic sign: Codman's triangle, marked X, is the triangular cuff of reactive periosteal bone at the edge of the lesion, where the tumour has lifted the periosteum clear of the cortex but has not yet destroyed it. It signals rapid growth breaking out of bone.

Step 3 – read the clinical picture: A rapidly enlarging, painful swelling of the mandible in a young adult fits **osteosarcoma of the jaw**, which characteristically presents a decade later than long-bone osteosarcoma, in the third to fourth decade.

Step 4 – add the third jaw-specific sign: Symmetrical widening of the **periodontal ligament space** around an involved tooth is the early and highly suggestive jaw sign, from tumour infiltration along the ligament. Numbness of the lower lip may follow inferior alveolar nerve involvement.

Step 5 – confirm on histology: The diagnosis rests on malignant sarcomatous stroma directly producing **osteoid**. Jaw osteosarcomas are more often chondroblastic than long-bone ones, and they metastasise late but recur locally, which is why wide resection is the treatment.

Why the other options are wrong:

- B, ossifying fibroma: is a **benign** fibro-osseous neoplasm. It grows slowly and painlessly, is **well demarcated** and often corticated, and it displaces rather than destroys. It never gives sunray spicules or Codman's triangle.
- C, compact osteoma: is a benign, slow, dense mass of mature bone, usually painless and often an incidental finding. Radiographically it is a uniformly radiopaque lump with a smooth border, with no periosteal reaction at all.
- D, odontogenic keratocyst: is a **radiolucent** cystic lesion that grows along the marrow of the ramus with a scalloped, corticated margin. The stem de-

scribes bone **formation** and periosteal reaction, which excludes a cyst outright.

Final Answer: Osteosarcoma of the mandible $\Rightarrow$ A

Answer: (A) Go Back to Q5

Q6.

Solution

Concept – the jaw as a recipient of metastasis: Here the jaw is not the source of the tumour but the destination, so you must recall which distant primaries send deposits to bone.

Step 1 – recall the general rule for bone metastasis: The tumours that metastasise to bone are those of the **breast, lung, prostate, kidney and thyroid**. The jaws follow the same list, because deposits reach them haematogenously from the same primaries.

Step 2 – apply the rule by sex: In women the commonest primary sending deposits to the jaw is the **breast**. In men it is the **lung**, with prostate close behind. Kidney and thyroid are less common but well recognised.

Step 3 – explain the site preference: Deposits favour the **posterior body, angle and ramus of the mandible**. That region retains active haematopoietic marrow in the adult, and its rich sinusoidal blood supply lets circulating tumour cells lodge. The maxilla is involved far less often.

Step 4 – explain the classic presenting sign: A deposit expanding in the mandible compresses the **inferior alveolar nerve**, producing numbness of the lower lip and chin. This is the **numb chin syndrome**, and in an adult with no dental cause it must be treated as metastatic disease until proved otherwise.

Step 5 – note the diagnostic importance: In roughly a third of cases the jaw deposit is the **first** manifestation of the disease, appearing before the primary is known. So an ill-defined, ragged, moth-eaten radiolucency with teeth apparently floating in it demands biopsy and a search for a primary.

Why the other options are wrong:

- A, brain, retina and spinal cord: central nervous system tumours essentially **do not metastasise outside the CNS**. They spread locally and through cerebrospinal fluid, so they never seed the jaw.
- B, skin, lip and floor of mouth: these are **local** primaries. Their spread to the jaw is by **direct invasion** or to the cervical **lymph nodes**, not by distant

blood-borne metastasis to bone, which is what the stem describes.

- C, stomach, pancreas and gall bladder: gastrointestinal carcinomas drain by the **portal vein** and metastasise first to the **liver**. Jaw deposits from them are rare curiosities, not the common primaries.

Final Answer: Breast, lung, prostate, kidney and thyroid $\Rightarrow$ D

Answer: (D) Go Back to Q6

Q7.

Solution

Concept – four mechanisms produce oedema, and the history tells you which one: Fluid stays in the capillary because of a balance between the pressure pushing it out and the protein holding it in. Break either arm, or block the drain, and oedema follows.

Step 1 – list the four mechanisms: (i) increased capillary hydrostatic pressure, **(ii)** reduced plasma oncotic pressure, **(iii)** lymphatic obstruction, and **(iv)** sodium and water retention. Increased vascular permeability is the fifth, inflammatory mechanism.

Step 2 – read the key laboratory clue: The child **loses albumin in the urine**. Albumin is the main protein generating plasma colloid osmotic pressure, contributing roughly 80 percent of it.

Step 3 – follow the physiology: Once serum albumin falls, the inward oncotic pull weakens. The Starling balance now favours net outward filtration at the capillary all along its length, so fluid accumulates in the interstitium. This is mechanism (ii).

Step 4 – explain why the oedema is generalised and facial: Because the defect is in the blood itself and not in one vascular bed, every capillary in the body leaks, so the oedema is **generalised (anasarca)**. It appears first where tissue pressure is lowest, that is the **periorbital** tissue and eyelids on waking, exactly as described.

Step 5 – explain why sodium retention is secondary here: The oedema reduces the effective circulating volume, which triggers renin, aldosterone and antidiuretic hormone. Sodium and water retention then **worsens** the oedema, but it is a consequence of the hypoalbuminaemia, not the initiating event. The stem asks for the mechanism operating **first and principally**, which is the oncotic one.

Why the other options are wrong:

- A, increased hydrostatic pressure: is the mechanism of **congestive cardiac**

failure and of venous obstruction or deep vein thrombosis. It produces **dependent** oedema of the ankles, and it does not fit a child losing albumin in urine.

- C, lymphatic obstruction: produces **localised, non-pitting** lymphoedema in the territory of the blocked channels, as after neck dissection or in filariasis. It could never cause generalised anasarca.
- D, increased vascular permeability: is the **inflammatory** mechanism, and the fluid it produces is a protein-rich **exudate**. Nephrotic oedema is a low-protein **transudate**, and the child has no inflammatory illness.

Final Answer: Reduced plasma oncotic pressure $\Rightarrow$ B

Answer: (B) Go Back to Q7

Q8.

Solution

Concept – first intention healing runs to a fixed timetable: The examiner wants the day-by-day order of events in a sutured incision, so recite the calendar.

Step 1 – within 24 hours: A **fibrin clot** fills the narrow gap and dries to a scab. **Neutrophils** appear at the incision margins and migrate towards the clot. Basal epithelial cells at the cut edges start to divide.

Step 2 – by 24 to 48 hours: Epithelial cells from both edges migrate beneath the scab and **fuse in the midline**, laying down basement membrane as they go. A thin continuous epithelial layer now closes the surface. This is why a sutured wound is watertight so early.

Step 3 – by day 3: Macrophages replace the neutrophils. Granulation tissue begins to invade the incisional space, and **collagen fibres** appear at the margins, still vertical and not yet bridging the gap. Epithelial proliferation thickens the cover.

Step 4 – by day 5: The gap is **filled with granulation tissue** and **neovascularisation is maximal**, so the wound looks its reddest. Collagen fibrils now **bridge** the incision. The epidermis regains its **normal thickness** with surface keratinisation.

Step 5 – through week 2: Fibroblast proliferation and collagen accumulation continue while **leucocytes, oedema and vessels regress**. The wound therefore begins to **blanch**, changing from red to pale.

Step 6 – by the end of month 1: An **intact epidermis** covers a scar of cellular connective tissue with essentially no inflammatory cells left. The dermal appendages destroyed by the cut are gone permanently. Option B recites exactly this sequence.

Why the other options are wrong:

- A, macrophages first then neutrophils, clot on day 5: reverses the order. The **clot forms within minutes to hours**, **neutrophils** come first and **macrophages** replace them by day 3, not the other way round. Every element of this sequence is inverted.
- C, epithelial continuity only after 3 weeks and no clot: is false on both counts. Re-epithelialisation across an approximated incision is complete within **24 to 48 hours**, and clot formation is the obligatory first step of any healing.
- D, contraction to a tenth of the area: describes **second intention** healing of a large open defect, where myofibroblasts contract the wound. In first intention the edges are already apposed, so there is nothing to contract and contraction plays no meaningful part.

Final Answer: The clot to neutrophil to macrophage to granulation tissue to scar sequence $\Rightarrow$ B

Answer: (B) Go Back to Q8

Q9.

Solution

Concept – an autoclave kills with steam contact, not with a number on a gauge: Sterilisation happens only where saturated steam actually touches the surface. Anything that keeps steam off a surface makes the cycle fail, however good the readings look.

Step 1 – state how moist heat kills: Steam condenses on the cooler instrument, releases its large **latent heat** of vaporisation directly on to that surface and coagulates microbial protein. No condensation means no latent heat delivered and no kill.

Step 2 – explain why trapped air is fatal: Air is **denser than steam** and does not condense. A pocket of residual air, marked X in the figure, sinks and sits as an insulating blanket over the packs. Steam cannot displace it, so the surfaces under it never receive latent heat and stay effectively in a dry-air oven at 121°C, a condition that is not sporicidal in 15 minutes.

Step 3 – explain why the gauge still reads correctly: The temperature probe and the gauge sample the **chamber**, not the inside of the pack. An air-steam mixture at the same pressure reads a plausible temperature while delivering far less lethal energy. This is precisely why chamber readings alone cannot validate a

load.

Step 4 – explain the overloading half: Packing the chamber tightly, as sketched, leaves no channels for steam to circulate and for air to be pushed out. Packs must be loose, on edge, and never touch the chamber wall, and the chamber should not be filled beyond about three quarters of its volume.

Step 5 – state the remedy: Air must be removed before the holding phase, by downward displacement in a simple autoclave or by a **pre-vacuum** pump in a modern class B machine. Correct wrapping in a steam-permeable material, correct loading, and never over-filling complete the answer. Option C states the trapped air plus overloading combination, so it is correct.

Why the other options are wrong:

- A, distilled water: is the **recommended** feed water, not a fault. Tap water leaves mineral scale that damages the machine and stains instruments, so this option reverses the truth.
- B, pressure marginally above 15 psi: is harmless and is exactly what a 134°C cycle uses. Higher pressure means hotter steam and a **safer** kill, so it cannot explain a failure.
- D, opening the door too early: is a real error, but its consequences are wet packs and burns, and a wet pack fails because it **wicks** contaminants afterwards. It does not explain unsterile packs at the centre of a correctly run cycle, and it is not the commonest cause of failure.

Final Answer: Trapped air shielding the load, worsened by overloading $\Rightarrow$ C

Answer: (C) Go Back to Q9

Q10.

Solution

Concept – segregate biomedical waste at the point of generation, by colour: The colour code is fixed by rule, and every item in a dental clinic belongs to exactly one bin. Segregation must happen at the chair, because once categories mix the whole bag must be treated as the most hazardous category present.

Step 1 – classify the extracted tooth: A tooth is a body part removed from the patient, so it is **human anatomical waste**. Human anatomical waste is a **yellow** category item.

Step 2 – classify the blood-soaked gauze: Cotton and gauze contaminated with blood or body fluids is **soiled waste**, and soiled waste is also a **yellow** category

item.

Step 3 – confirm both go to the same bin: Both belong to yellow, so a single yellow bag takes them. This is convenient in dentistry, since extraction produces both together.

Step 4 – state the fate of yellow waste: Yellow bags go for **incineration**, plasma pyrolysis or deep burial. That is the correct end point for anatomical and soiled waste, and it is why they must not be mixed with recyclables.

Step 5 – recall the rest of the code for a dental clinic: Red takes contaminated recyclable plastic, that is gloves, suction tips, saliva ejectors, IV tubing and syringes without needles, for autoclaving or shredding and recycling. **White** translucent puncture-proof containers take sharps, that is needles, scalpel blades, burs and endodontic files. **Blue** puncture-proof boxes take broken or discarded glass, glass carpules and metallic implants. Yellow also carries expired medicines, chemical waste and discarded linen.

Why the other options are wrong:

- A, red: is for contaminated **recyclable plastics** only. Neither a tooth nor blood-soaked gauze is a plastic, and neither can be shredded and recycled, so red is wrong.
- B, white translucent: is reserved for **sharps**, and its whole purpose is puncture resistance to prevent needlestick injury. Gauze is not a sharp, and though a tooth is hard, it is classified by its origin as anatomical waste, not by its edge.
- C, blue: is for **broken glass and metallic implants**, which are disinfected then recycled. Neither item in the stem is glass or metal.

Final Answer: Yellow $\Rightarrow$ $\boxed{\text{D}}$

Answer: (D) Go Back to Q10

Q11.

Solution

Concept – disc diffusion gives a category, not a concentration: Kirby-Bauer answers one clinical question, will this drug work on this organism, and it answers it as susceptible, intermediate or resistant.

Step 1 – describe what happens on the plate: The organism is spread as a confluent **lawn** on Mueller-Hinton agar. Each paper disc holds a fixed amount of one antibiotic. The drug **diffuses radially**, so its concentration is highest at the

disc and falls with distance.

Step 2 – explain the edge of the zone: The plate is then incubated. The bacteria grow everywhere the drug concentration is too low to inhibit them. The circular clear area around the disc, where no growth occurs, is the **zone of inhibition**, and its edge marks the point at which the diffused concentration has fallen to the inhibitory level for that organism.

Step 3 – explain how the diameter is used: The diameter is measured in millimetres across the whole zone, including the disc. It is then compared against a published **interpretive breakpoint table** (CLSI or EUCAST) that is specific to **that organism, that drug and that disc content**. The table converts the millimetre figure into **susceptible, intermediate or resistant**, which is what the laboratory reports to the clinician. That is option A.

Step 4 – explain why the tables are drug and organism specific: Zone size depends on how fast the molecule diffuses through agar as well as on how potent it is. A large molecule diffuses slowly and gives a small zone even when the drug is highly active, so a raw millimetre number is meaningless until it is read against its own table.

Step 5 – read the plate in the figure: Disc X gives the widest zone and, once checked against its table, would most likely be reported susceptible. The disc with **no zone at all** means growth right up to the paper, so that isolate is **resistant** to that drug.

Why the other options are wrong:

- B, reading the zone directly as the MIC: is wrong. Disc diffusion is **qualitative**, giving only a category. The MIC is a number in micrograms per millilitre and needs broth dilution, agar dilution or an E-test gradient strip, where the MIC is read where the ellipse meets the strip.
- C, widest zone equals most potent drug: is the classic trap. Zones from **different drugs cannot be compared with one another**, because each drug has its own diffusion rate and its own disc content and breakpoint. A drug with a 22 mm zone may be resistant by its own table while a drug with 15 mm is susceptible by its own.
- D, confirming a bactericidal action: is wrong, because the zone shows only that growth was **inhibited**. Distinguishing killing from inhibition needs the minimum bactericidal concentration, obtained by subculturing from clear tubes.

Final Answer: Compare with the interpretive breakpoint table and report S, I or R $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q11

Q12.

Solution

Concept – sulphur granules plus sinuses at the angle of the jaw: This triad names one organism, and the important trap is that the disease sounds fungal but is not.

Step 1 – read the granule clue: Hard yellow granules in the pus are **sulphur granules**. They are not sulphur at all. They are colonies of the organism, a tangled mass of filaments with the ends clubbed by host protein, and they are named only for their yellow colour.

Step 2 – read the staining clue: Crushing a granule shows **Gram-positive branching filaments**. That is **Actinomyces israelii**, a Gram-positive, **strictly anaerobic**, non-acid-fast filamentous bacterium.

Step 3 – read the site and the trigger: This is **cervicofacial actinomycosis**, the commonest form, accounting for over half of cases. *A. israelii* is a normal commensal of the mouth, living in tonsillar crypts and plaque. It becomes pathogenic only when a breach lets it into deep tissue with a low oxygen tension, which is exactly what a difficult extraction, trauma or jaw fracture provides.

Step 4 – explain the clinical picture given: The lesion is a chronic suppurative and fibrosing infection. It spreads by **direct extension** across tissue planes, ignoring anatomical boundaries and **not using lymphatics**. Dense fibrosis gives the woody, board-like induration, and the abscesses point to the skin as multiple **discharging sinuses**, classically at the angle of the mandible.

Step 5 – settle the fungus trap and the treatment: Actinomycosis was long thought fungal, because the organism forms branching filaments and the name ends in -myces. It is a **bacterium**, and that is why the treatment is antibiotic, not antifungal: **high-dose penicillin for a prolonged course**, typically weeks of intravenous therapy followed by months orally, because the fibrosis and the granule itself keep the drug out. Surgical drainage and excision of sinus tracts assist.

Why the other options are wrong:

- B, Nocardia brasiliensis: is the near-identical distractor. It is also a Gram-positive branching filamentous bacterium, but it is **aerobic** and **partially acid-fast** on a modified Ziehl-Neelsen stain, whereas *Actinomyces* is anaerobic and **not** acid-fast. Nocardia typically causes mycetoma of the foot or pulmonary and brain disease, not a cervicofacial lesion after extraction, and

it is treated with cotrimoxazole rather than penicillin.

- C, Blastomyces dermatitidis: is a true **fungus** and would show broad-based budding yeasts on KOH or histology, not Gram-positive filaments in a granule. It causes pulmonary and cutaneous disease and needs antifungal therapy.
- D, Mycobacterium tuberculosis: can cause a chronic neck swelling with a discharging sinus, so the shape of the illness overlaps, but the organism is an **acid-fast bacillus** that does not branch and produces no sulphur granules. Its pus is caseous, and its histology shows caseating granulomas.

Final Answer: Actinomyces israelii $\Rightarrow$ A

Answer: (A) Go Back to Q12

Q13.

Solution

Concept – viral versus bacterial parotitis is decided at the duct orifice: Both swell the gland and both hurt, but only one produces pus.

Step 1 – read the pus clue: Massaging the gland yields **no pus** from Stensen's duct, although the orifice is red. Bacterial suppurative parotitis, by definition, expresses frank pus on milking. Its absence points away from bacteria.

Step 2 – read the laterality clue: The swelling is **bilateral**. Mumps is bilateral in about 70 percent of cases, because the virus reaches both glands through the bloodstream. Acute bacterial parotitis is almost always **unilateral**, since it ascends one duct.

Step 3 – read the amylase clue: The **serum amylase is raised**, reflecting salivary amylase released from inflamed acini. This is characteristic of mumps parotitis and is a useful confirmatory test.

Step 4 – read the epidemiological and immunisation clues: The boy is **unimmunised** and **two classmates have the same illness**. Mumps is a highly contagious **droplet-spread** paramyxovirus with an incubation of 16 to 18 days, so clusters in a class are typical. Bacterial parotitis is not contagious and does not cluster.

Step 5 – confirm the physical sign and complete the picture: Swelling that **lifts the ear lobe** upwards and outwards, and obliterates the hollow behind the angle of the jaw, is the classic mumps sign. The virus has a prodrome of fever, headache and malaise, and complications include orchitis in postpubertal males, aseptic meningitis, pancreatitis and sensorineural deafness. Management is supportive, and the MMR vaccine is the prevention.

Step 6 – state the contrast fully: Bacterial parotitis is caused chiefly by *Staphylococcus aureus* ascending the duct in a dehydrated, debilitated or postoperative patient with reduced salivary flow. It is unilateral, exquisitely tender, gives a red duct orifice with **expressible pus**, and needs antibiotics, hydration, sialagogues and sometimes drainage.

Why the other options are wrong:

- A, acute bacterial suppurative parotitis: fails on three counts. It gives **pus** from the duct, it is **unilateral**, and it strikes dehydrated or debilitated patients, not a well-nourished school child, and it never comes in classroom clusters.
- B, sialolithiasis: causes **intermittent** swelling and pain that peak **at mealtimes** and settle afterwards, and it is unilateral. It also favours the **submandibular** gland, whose secretion is viscid and whose duct runs uphill. It causes no fever and no cluster.
- C, Sjogren's syndrome: is a chronic autoimmune exocrinopathy of **middle-aged women**, presenting with **dry mouth and dry eyes** and painless recurrent gland enlargement over years. It has no place in an acute febrile illness in a 9-year-old.

Final Answer: Mumps $\Rightarrow$ D

Answer: (D) Go Back to Q13

Q14.

Solution

Concept – three pathways, three triggers, one common end: The complement pathways differ only in what starts them. From C3 onwards they are identical.

Step 1 – read the trigger given: A plasma protein binds **terminal mannose residues** on a microbial surface, and **no antibody** is involved.

Step 2 – name the protein and the pathway: The protein is **mannose-binding lectin (MBL)**, an acute phase collectin made by the liver. It defines the **lectin pathway**, so X is the lectin pathway.

Step 3 – follow the lectin mechanism: MBL is structurally like C1q. When it binds mannose it activates the associated serine proteases **MASP-1 and MASP-2**, which cleave C4 and C2 exactly as C1s does. The lectin pathway therefore uses the classical pathway's own machinery but is switched on by sugar rather than by antibody.

Step 4 – state the other two triggers for contrast: The **classical** pathway is triggered by **C1q binding the Fc of IgG or IgM** that is already bound to antigen, so it needs an antibody and belongs to adaptive immunity. The **alternative** pathway is triggered by **spontaneous C3 hydrolysis (tickover)** that is stabilised on any surface lacking regulators, such as bacterial lipopolysaccharide, with factors B and D and **properdin** completing it.

Step 5 – state the common terminal path shown in the figure: All three converge on a **C3 convertase** that cleaves C3 into C3a and C3b. C3b then builds the **C5 convertase**, which cleaves C5. **C5b** recruits C6, C7 and C8 and multiple C9 molecules to form the **membrane attack complex (C5b-C9)**, a pore that lyses the cell.

Step 6 – note why the lectin pathway matters: Like the alternative pathway it is part of **innate** immunity and works immediately, before any antibody exists. MBL deficiency is a common cause of recurrent infection in infants, exactly in the window after maternal antibody wanes and before the child's own antibody develops.

Why the other options are wrong:

- A, classical pathway: requires an **antigen-antibody complex** recognised by C1q. The stem says explicitly that no antibody is involved, so this is excluded by the stem itself.
- B, alternative pathway: is antibody-independent, which makes it the tempting option, but it is triggered by **spontaneous C3b deposition** on a permissive surface, not by a lectin binding mannose. Its distinctive components are factors B and D and properdin.
- D, properdin-independent coagulation pathway: does not exist as a complement pathway. Properdin is a **stabiliser** of the alternative pathway C3 convertase, and while complement and coagulation do cross-talk, no such named pathway is recognised.

Final Answer: Lectin pathway $\Rightarrow$ C

Answer: (C) Go Back to Q14

Answer Key

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