NEET MDS General Pathology and Microbiology

Sample Paper – 9

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 words **granulation tissue** and **granuloma** sound alike but belong to two entirely different processes. The correct statement about the pair is:

(A) Both are focal collections of epithelioid macrophages, and they differ only in size

(B) Granulation tissue is the new fibrovascular tissue of **repair**, whereas a granuloma is a focal aggregate of epithelioid macrophages of **chronic inflammation**

(C) Granulation tissue is a lesion of chronic inflammation, whereas a granuloma is the tissue that fills a healing extraction socket

(D) Granulation tissue occurs only in tuberculosis, whereas a granuloma occurs only in clean surgical wounds

Q2. Once a neutrophil has ingested a bacterium, the phagosome fuses with its lysosomal granules and the respiratory burst floods the vacuole with hydrogen peroxide. The **most potent** bactericidal system inside that phagolysosome depends on a lysosomal enzyme. That enzyme and the product it generates are:

(A) Myeloperoxidase, which uses hydrogen peroxide and chloride to generate hypochlorous acid

(B) Catalase, which breaks hydrogen peroxide down into water and oxygen

(C) Superoxide dismutase, which converts superoxide anion into hydrogen peroxide

(D) Lysozyme, which generates nitric oxide from arginine

Q3. A patient has a painful lesion of the buccal mucosa. The statement that correctly defines an **ulcer** and describes how a small oral ulcer heals is:

(A) It is a local defect of a surface produced by sloughing of inflamed necrotic tissue, so the full thickness of the epithelium is lost and the connective tissue is exposed; a small oral ulcer heals from its margins by re-epithelialisation, usually without a scar

(B) It is a fluid-filled elevation of the epithelium more than 5 mm across, which heals when the fluid is resorbed

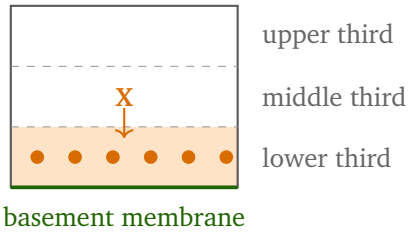
(C) It is a circumscribed collection of pus lying within a tissue, which heals only after surgical drainage

(D) It is a loss of the superficial keratin alone with the basal layer intact, which heals by desquamation

Neoplasia

Q4. The figure shows oral epithelium reported as dysplastic. The cellular atypia is confined to the zone marked **X**, the lower third of the epithelium

immediately above the basement membrane, while the middle and upper thirds mature normally. This is graded as:



(A) Moderate dysplasia

(B) Severe dysplasia

(C) Hyperkeratosis with no dysplasia

(D) Mild dysplasia

Q5. A 58-year-old man who has chewed tobacco and kept the quid in the buccal sulcus for 30 years has a bulky, white, exophytic warty growth in that sulcus. Biopsy shows a very well differentiated squamous lesion with thick club-shaped rete ridges advancing on a **broad pushing front** rather than infiltrating cords, and the neck is node negative. The lesion is:

(A) Squamous papilloma

(B) Conventional moderately differentiated squamous cell carcinoma

(C) Pyogenic granuloma

(D) Verrucous carcinoma

Q6. Tumour nomenclature normally gives the suffix *-oma* to a **benign** tumour. The group in which every member carries the *-oma* suffix yet is in fact **malignant** is:

(A) Lipoma, chondroma, osteoma

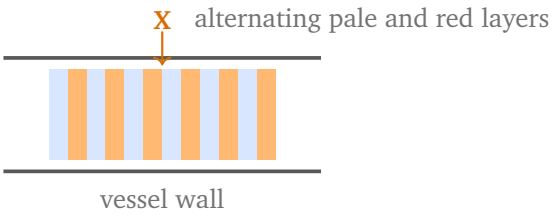
(B) Melanoma, lymphoma, seminoma

(C) Fibroma, adenoma, papilloma

(D) Leiomyoma, rhabdomyoma, haemangioma

Haemodynamic Disorders and Wound Healing

Q7. A firm mass is removed from a vessel at autopsy. The cut surface shows the alternating pale and red laminations marked **X**, and the mass is attached to the wall. These lines of Zahn tell you that the mass is:



(A) A thrombus that formed during life, because the laminations of platelets and fibrin alternating with red cells can only be laid down in flowing blood

(B) A post-mortem clot, because settling of the blood after death is what produces the layers

(C) A post-mortem clot, because it is firm and attached to the vessel wall

(D) An embolus of tumour, because only tumour tissue produces a laminated cut surface

Q8. Three days after a difficult lower third molar extraction a smoker returns with severe throbbing pain radiating to the ear. The socket is empty and foul, with bare bone visible and no granulation tissue on its walls. The underlying pathology is:

(A) An acute periapical abscess draining into the socket

(B) Osteoradionecrosis of the mandible

(C) Cervicofacial actinomycosis of the socket

(D) Loss or failed organisation of the blood clot, so the exposed bone cannot be granulated over: alveolar osteitis, or dry socket

General Microbiology and Sterilisation

Q9. *Candida albicans* and the viridans streptococci live harmlessly in the mouth of a healthy person, yet the first causes thrush in a patient on

inhaled steroids and the second causes endocarditis once it reaches a damaged heart valve. The correct description of such an organism is:

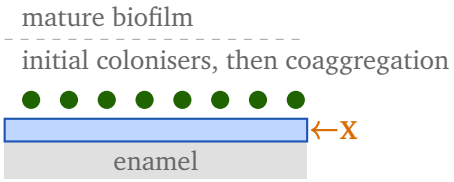
(A) An obligate pathogen, since it causes disease in every host it enters

(B) A saprophyte, since it lives only on dead organic matter and never on a host

(C) A commensal of the normal oral flora that behaves as an opportunistic pathogen when host defence falls or when it is carried away from its normal site

(D) A laboratory contaminant of no clinical significance whenever it is isolated

Q10. The figure shows dental plaque building up on a cleaned enamel surface. The layer marked **X** is the very first stage, formed within minutes of polishing and before any organism attaches. Layer **X** is:



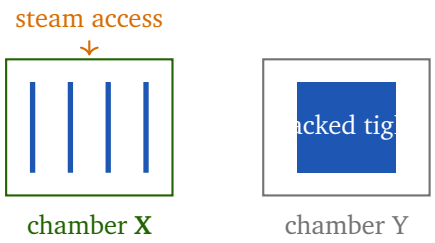
(A) A mat of filamentous organisms coaggregating with the cocci already present

(B) A fully mature biofilm with its fluid channels and anaerobic depths

(C) The acquired pellicle, a cell-free film of salivary glycoproteins adsorbed onto enamel, which then supplies the receptors the initial colonisers bind to

(D) Calculus, that is, plaque already mineralised by salivary calcium and phosphate

Q11. The two autoclave chambers below have been given the same set of wrapped instrument packs. Chamber **X** sterilises reliably and chamber Y repeatedly fails its spore test. The feature that makes chamber **X** correctly loaded is:



(A) It is packed as tightly as the chamber allows, so that more instruments are processed in each cycle

(B) Packs are stood loosely on edge with a gap between them and the walls, so air can drain out and saturated steam can touch every surface of every item

(C) The instruments are sealed inside airtight metal containers, so that no steam is wasted on the chamber

(D) The packs are wrapped in impermeable aluminium foil, so that they come out perfectly dry

Systemic Bacteriology and Virology

Q12. A patient attends with a deep, ragged, soil-contaminated laceration of the lip and chin sustained in a fall, and cannot recall any immunisation. The organism of greatest concern, with its correct mechanism, is:

(A) *Clostridium perfringens*, whose tetanospasmin causes sustained muscle spasm

(B) *Clostridium tetani*, an anaerobic spore-forming bacillus whose tetanospasmin blocks the release of the inhibitory transmitters glycine and GABA, producing trismus and spastic paralysis

(C) *Clostridium botulinum*, whose toxin blocks acetylcholine release at the neuromuscular junction and causes spastic paralysis

(D) *Staphylococcus epidermidis*, a spore former whose exotoxin causes tetanus in deep wounds

Q13. A 45-year-old man who has never smoked or chewed tobacco has a carcinoma of the tonsil. The tumour is non-keratinising and stains strongly

and diffusely for **p16**. Compared with the tobacco-related, HPV-negative oral carcinoma, this tumour:

(A) Is driven by a high-risk human papillomavirus, most often type 16, whose E6 and E7 proteins inactivate p53 and Rb, and it carries a **better** prognosis with a better response to radiotherapy

(B) Is driven by the low-risk types HPV 6 and 11, and carries a distinctly worse prognosis

(C) Has no relation to HPV, since p16 positivity merely reflects heavy tobacco exposure

(D) Is driven by HPV 16 but behaves identically to the HPV-negative tumour, so the p16 result changes nothing

Immunology and Hypersensitivity

Q14. The statement that correctly compares a **live attenuated** vaccine with a **killed (inactivated)** vaccine is:

(A) The killed vaccine produces the stronger and longer lasting immunity, because the dead organism presents more antigen at once

(B) The live vaccine is the safer choice in pregnancy and in immunodeficiency, because the organism has been weakened

(C) The live vaccine replicates in the host and so gives strong, long lasting humoral *and* cell-mediated immunity, often after a single dose, but it can revert or disseminate and is contraindicated in the immunocompromised; the killed vaccine cannot replicate, is therefore safer, but gives mainly humoral immunity and needs an adjuvant and booster doses

(D) Both types work by identical mechanisms and differ only in the temperature at which they are stored

Detailed Solutions

Q1.

Solution

Concept – two similar words, two opposite processes: Granulation tissue belongs to **repair**. A granuloma belongs to **chronic inflammation**. The similarity of the words is the whole trap.

Step 1 – define granulation tissue: It is the soft, pink, granular tissue that fills a healing defect. Its two components are **new capillaries** formed by angiogenesis and **proliferating fibroblasts** in a loose oedematous matrix, with scattered inflammatory cells.

Step 2 – explain where its name comes from: Seen with the naked eye in a healing socket or an ulcer base, the surface looks red and granular because of the loops of new capillaries pushing up. The name describes an appearance, not a cell type.

Step 3 – define a granuloma: It is a microscopic focus of **epithelioid cells**, which are activated macrophages, usually with multinucleated giant cells and a surrounding collar of lymphocytes. It is a pattern of chronic inflammation seen when the agent resists phagocytic killing.

Step 4 – state the purpose of each: Granulation tissue is the body *rebuilding* a lost piece of tissue, and it later matures into a fibrous scar. A granuloma is the body *walling off* something it cannot destroy, such as the tubercle bacillus or a suture fragment.

Step 5 – match to the option: Option B pairs granulation tissue with repair and the granuloma with chronic inflammation, which is exactly the contrast, so it is the answer.

Why the other options are wrong:

- A, both are epithelioid collections differing in size: is false. Granulation tissue contains **no** epithelioid cells at all; its cells are fibroblasts and endothelial cells.
- C, the two definitions swapped: reverses the truth. This is the option that catches candidates who remember only that the terms are related.
- D, granulation tissue only in tuberculosis and granulomas only in surgical wounds: is wrong twice over. A clean sutured wound heals with minimal granulation tissue and no granuloma, and tuberculosis produces granulomas, not granulation tissue.

Final Answer: Granulation tissue is repair; a granuloma is chronic inflammation

$\Rightarrow$ B

Answer: (B) Go Back to Q1

Q2.

Solution

Concept – the neutrophil kills mainly with a halogenating system: The respiratory burst supplies the raw material, and one lysosomal enzyme turns that raw material into the actual poison.

Step 1 – follow the respiratory burst: NADPH oxidase, assembled on the phagosome membrane, reduces oxygen to the **superoxide anion**. Superoxide is then dismutated to **hydrogen peroxide**.

Step 2 – note that hydrogen peroxide alone is not enough: The amount of hydrogen peroxide produced is not by itself sufficient to kill most bacteria efficiently. It has to be amplified.

Step 3 – bring in the lysosomal granule: The **azurophilic (primary) granules** of the neutrophil fuse with the phagosome and deliver **myeloperoxidase**.

Step 4 – state the reaction: Myeloperoxidase uses hydrogen peroxide plus a halide, in practice **chloride**, to generate **hypochlorous acid**, which is household bleach. This MPO–H_2O_2–halide system is the most efficient bactericidal mechanism the neutrophil owns.

Step 5 – name the damage it does: Hypochlorous acid halogenates and oxidises bacterial proteins and lipids, destroying the organism inside the sealed phagolysosome so the host tissue is spared.

Step 6 – note the clinical proof: In chronic granulomatous disease NADPH oxidase is defective, no hydrogen peroxide is made, the system cannot run, and the patient suffers recurrent catalase-positive infections. That disease is the direct evidence that this pathway is the killing pathway.

Why the other options are wrong:

- B, catalase: **destroys** hydrogen peroxide. It is a protective enzyme, and in catalase-positive bacteria it actually helps the organism **resist** killing, so it is the opposite of a bactericidal enzyme.
- C, superoxide dismutase: does convert superoxide to hydrogen peroxide, so it sits *upstream* in the burst, but it is not a lysosomal granule enzyme and its product still needs myeloperoxidase to become lethal. This is the closest distractor.
- D, lysozyme generating nitric oxide: is a false pairing. Lysozyme is real and

does hydrolyse the peptidoglycan of the bacterial cell wall, but it makes no nitric oxide. Nitric oxide comes from arginine through nitric oxide synthase, mainly in macrophages.

Final Answer: Myeloperoxidase generating hypochlorous acid $\Rightarrow$ A

Answer: (A) Go Back to Q2

Q3.

Solution

Concept – an ulcer is defined by full thickness loss of epithelium: Every other surface lesion is told apart from an ulcer by how deep the loss goes.

Step 1 – state the definition: An ulcer is a **local defect or excavation of the surface** of an organ or tissue, produced by the sloughing of inflamed necrotic tissue.

Step 2 – state the depth criterion: The loss must be **full thickness** through the epithelium, so the underlying connective tissue lies exposed. If the basal layer survives, the lesion is an erosion, not an ulcer.

Step 3 – describe the floor: The floor of an established ulcer carries a slough of fibrin and neutrophils, and beneath it **granulation tissue** with new capillaries and fibroblasts.

Step 4 – describe how a small oral ulcer heals: The slough is cleared by neutrophils and macrophages, granulation tissue fills the shallow defect, and epithelium then migrates in **from the intact margins** across that bed until the sheets meet and stop.

Step 5 – explain why no scar results: Oral epithelium is a labile tissue with a very fast turnover, and a minor aphthous ulcer is shallow, so little collagen is needed. Healing is complete in about 7 to 14 days with no scar. A major aphthous ulcer, being deep, can scar.

Step 6 – note the examination point: Any oral ulcer that fails to heal within about 3 weeks must be biopsied, because failure to re-epithelialise is a cardinal warning of malignancy.

Why the other options are wrong:

- B, a fluid-filled elevation over 5 mm: defines a **bulla**. It is an elevation, not a defect, and it is the lesion that may later break down *into* an ulcer.
- C, a circumscribed collection of pus within a tissue: defines an **abscess**. An abscess sits inside the tissue, whereas an ulcer is a loss of the surface.

- D, loss of keratin only with an intact basal layer: is not full thickness, so by definition it is an **erosion**, not an ulcer.

Final Answer: Full thickness surface defect healing by re-epithelialisation from the margins ⇒ A

Answer: (A) Go Back to Q3

Q4.

Solution

Concept – dysplasia is graded by how high the atypia climbs: The pathologist mentally divides the epithelium into thirds and asks how far up the disordered cells reach.

Step 1 – state the grading rule: Atypia confined to the **lower third** is **mild** dysplasia. Atypia reaching into the **middle third** is **moderate**. Atypia extending into the **upper third** is **severe**.

Step 2 – read the figure: The shaded zone marked X, holding the atypical cells, sits between the basement membrane and the first dashed line. That is the lower third only.

Step 3 – read what the rest of the epithelium is doing: The question states that the middle and upper thirds mature normally, that is, the cells above X flatten and keratinise in the usual orderly way. Maturation is preserved above the lower third.

Step 4 – apply the rule: Atypia in the lower third alone, with normal maturation above it, is **mild dysplasia**.

Step 5 – note what the atypia consists of: The features counted are nuclear hyperchromatism, an increased nuclear to cytoplasmic ratio, pleomorphism, loss of polarity, and mitoses appearing above the basal layer. In mild dysplasia these are all crowded into the bottom third.

Step 6 – note why the grade matters: The grade predicts the risk of malignant transformation. Mild dysplasia carries the lowest risk and is usually managed by removing the cause, such as tobacco, and reviewing the patient, whereas severe dysplasia is excised.

Why the other options are wrong:

- A, moderate dysplasia: would require the atypia to reach into the **middle** third. The figure shows the middle third clear.
- B, severe dysplasia: would require the atypia to extend into the **upper** third. That is two grades beyond what is shown.

- C, hyperkeratosis with no dysplasia: cannot be right, because atypical cells are present. Hyperkeratosis is a thickened keratin layer with no cellular atypia at all.

Final Answer: Atypia in the lower third only ⇒ mild dysplasia ⇒ D

Answer: (D) Go Back to Q4

Q5.

Solution

Concept – verrucous carcinoma is the low-grade carcinoma that pushes instead of infiltrating: It is malignant, but it behaves far better than the conventional tumour, and its margin is the key.

Step 1 – use the clinical picture: A bulky, white, **exophytic warty** growth with a shaggy pebbly surface, sitting exactly where the tobacco quid is held, is the classic Ackerman tumour of the buccal mucosa and sulcus.

Step 2 – use the aetiology given: Verrucous carcinoma is strongly associated with prolonged **tobacco chewing** and quid holding, and in India the buccal mucosa and gingivobuccal sulcus are its favourite sites for that reason.

Step 3 – use the histology given: It is **very well differentiated**, with abundant keratin and thick club-shaped rete ridges. Cytological atypia is minimal, which is why a superficial biopsy can be reported as benign and the biopsy must be deep enough to include the interface.

Step 4 – use the decisive feature, the margin: The lesion advances on a **broad pushing front**. It does not send out the thin infiltrating cords and islands of conventional squamous cell carcinoma.

Step 5 – link the margin to the behaviour: Because it pushes rather than infiltrates, it rarely invades lymphatics and **almost never metastasises**. The node-negative neck in the question follows from that. Treatment is wide surgical excision, and the prognosis is good.

Step 6 – confirm it is still malignant: It is locally destructive and will burrow into the underlying bone if neglected, so it is a true carcinoma despite its bland appearance.

Why the other options are wrong:

- A, squamous papilloma: is a small, soft, benign pedunculated cauliflower growth a few millimetres across, linked to HPV 6 and 11. It is not bulky, not tobacco related, and it does not destroy bone.

- B, conventional squamous cell carcinoma: would show an **infiltrating** margin with irregular cords and islands, more atypia, and a real risk of cervical nodes. The question explicitly excludes infiltration.
- C, pyogenic granuloma: is a red, soft, bleeding vascular swelling of granulation tissue, usually on the gingiva after irritation. It is neither white nor warty nor keratinised.

Final Answer: Verrucous carcinoma $\Rightarrow$ D

Answer: (D) Go Back to Q5

Q6.

Solution

Concept – the *-oma* rule has famous exceptions: The suffix normally means benign, but a handful of malignant tumours were named before the rule existed, and their names stuck.

Step 1 – state the general rule: Benign tumours are named by adding *-oma* to the cell of origin: lipoma from fat, chondroma from cartilage, osteoma from bone.

Step 2 – name the exceptions: **Melanoma** is a highly aggressive malignant tumour of melanocytes. **Lymphoma** is a malignant tumour of lymphoid tissue. **Seminoma** is a malignant germ cell tumour of the testis.

Step 3 – explain why the names persist: These tumours were described and named before the modern convention was fixed, and the names were too well established to change. Some texts write "malignant melanoma" to remove the ambiguity.

Step 4 – add the other exceptions worth knowing: Hepatoma means hepatocellular carcinoma, and it is malignant. Mesothelioma is malignant. Glioma is malignant. Neuroblastoma, retinoblastoma and medulloblastoma all end in *-oma* and are malignant tumours of childhood.

Step 5 – note the mirror-image traps: Hamartoma and choristoma end in *-oma* but are not neoplasms at all. A hamartoma is a disorganised mass of tissue native to the site, and a choristoma is normal tissue in the wrong place.

Why the other options are wrong:

- A, lipoma, chondroma and osteoma: are the **benign** mesenchymal tumours that obey the rule perfectly. They are the standard against which the exceptions are measured.
- C, fibroma, adenoma and papilloma: are all **benign**. Fibroma is benign

fibrous tissue, adenoma is benign glandular epithelium, papilloma is benign surface epithelium.

- D, leiomyoma, rhabdomyoma and haemangioma: are all **benign** tumours of smooth muscle, skeletal muscle and blood vessels. Their malignant counterparts have to be renamed leiomyosarcoma, rhabdomyosarcoma and angiosarcoma, which again shows the rule holding rather than breaking.

Final Answer: Melanoma, lymphoma and seminoma ⇒ B

Answer: (B) Go Back to Q6

Q7.

Solution

Concept – lines of Zahn prove that blood was still flowing: The single question at autopsy is whether the mass formed before death or after it, and the laminations settle it.

Step 1 – say what the lines of Zahn are: They are **alternating pale layers of platelets and fibrin** and **darker layers rich in red cells**, giving the laminated appearance marked X in the figure.

Step 2 – explain why they need flow: Platelets can only be swept out of the axial stream and deposited on the wall in successive waves while blood is **moving**. Each wave lays down a pale layer, and red cells caught between the waves form the dark layer. Motionless blood cannot build this pattern.

Step 3 – conclude the timing: Laminations therefore mean the mass was built up in flowing blood, that is, **during life**. It is a thrombus, and it is **ante-mortem**.

Step 4 – describe the post-mortem clot for contrast: A post-mortem clot forms in still blood after death. It is **soft, gelatinous and rubbery**, it is **not attached** to the vessel wall and slides out easily, and it has **no lines of Zahn**.

Step 5 – give the post-mortem clot's own sign: Instead of laminations it shows gravity settling: a dark red dependent portion of sedimented red cells with a yellow "chicken fat" supernatant of plasma and white cells above it.

Step 6 – add the attachment clue: The question says the mass is **attached to the wall**. A thrombus is anchored at its point of endothelial injury; a post-mortem clot is not attached at all. Attachment and laminations point the same way.

Why the other options are wrong:

- B, a post-mortem clot with layers from settling: confuses the two patterns. Settling after death gives the currant-jelly and chicken-fat separation, not

the fine alternating laminations of Zahn.

- C, a post-mortem clot because it is firm and attached: reasons backwards. Firmness and attachment are features of a **thrombus**; a post-mortem clot is soft and unattached.
- D, a tumour embolus: is unfounded. Nothing in the description is cellular tumour, and laminated pale and red layers are the signature of platelet and fibrin deposition, not of tumour tissue.

Final Answer: A thrombus formed during life $\Rightarrow$ A

Answer: (A) Go Back to Q7

Q8.

Solution

Concept – dry socket is a healing failure, not an infection: The socket clot is the scaffold on which healing is built. Lose the clot and healing simply cannot start.

Step 1 – recall the normal sequence: The socket fills with blood, the clot organises as capillaries and fibroblasts grow into it from the socket walls, granulation tissue replaces the clot in about a week, and woven bone follows.

Step 2 – state what goes wrong: In alveolar osteitis the clot is **lost or disintegrates**, usually through excessive local fibrinolysis, before it can be organised. The scaffold is gone.

Step 3 – explain the consequence: With no clot there is nothing for the capillaries and fibroblasts to grow into, so the socket cannot granulate. The **bare bony walls** stay exposed, which is exactly what the question describes.

Step 4 – explain the pain: Denuded alveolar bone has almost no soft tissue cover and is directly exposed to oral fluids and food. The severe throbbing pain radiating to the ear is the classic result, and it characteristically starts on **day 2 to 4**, after an initially comfortable period, exactly as here.

Step 5 – fit the risk factors: Smoking, a difficult or traumatic extraction, the lower third molar site, vigorous rinsing and oral contraceptive use are the recognised risks. The question supplies smoking, difficulty and the lower third molar.

Step 6 – note why it is not an infection: There is no pus, no swelling and usually no fever, and the socket is foul only because of stagnating debris. Treatment is irrigation and a sedative obtundent dressing to cover the bone, not primarily antibiotics.

Why the other options are wrong:

- A, an acute periapical abscess: would give pus, swelling, tenderness and often fever, and the tooth causing it has already been removed. Bare non-granulating bone is not an abscess.
- B, osteoradionecrosis: needs a history of **radiotherapy** to the jaws. None is given, and it develops over months, not in 3 days.
- C, actinomycosis: is a chronic infection producing a board-like swelling with multiple sinuses discharging sulphur granules over weeks. The 3-day timing rules it out.

Final Answer: Failed clot organisation leaving bare bone, that is dry socket $\Rightarrow$ D

Answer: (D) Go Back to Q8

Q9.

Solution

Concept – pathogenicity is a relationship, not a fixed property: Whether an organism causes disease depends on the host and the site as much as on the organism.

Step 1 – define a commensal: A commensal is a member of the **normal flora**. It lives on the host, benefits from it, and causes no harm in health. The mouth carries hundreds of such species, including viridans streptococci and *Candida albicans* in roughly half of healthy people.

Step 2 – note the benefit the commensal gives: Normal flora is not merely tolerated. It occupies binding sites and consumes nutrients, so it resists colonisation by true pathogens. That is why broad spectrum antibiotics, by wiping it out, allow candidal overgrowth.

Step 3 – define an opportunistic pathogen: The same commensal causes disease only when the **opportunity** arises: when host defence is lowered, or when it is displaced from its normal site into a sterile one.

Step 4 – apply this to the first example: Inhaled or systemic steroids depress local and cell-mediated immunity, so *Candida albicans* overgrows the mucosa and produces thrush. The organism has not changed; the host has.

Step 5 – apply this to the second example: Viridans streptococci enter the blood during dental procedures or even toothbrushing. In a normal heart they are cleared harmlessly, but on a **damaged valve** they adhere and grow into vegetations, giving subacute infective endocarditis. Here the organism has been carried out of its normal site.

Step 6 – draw the conclusion: An organism can be a harmless commensal in one

situation and a lethal pathogen in another, which is exactly what option C states.

Why the other options are wrong:

- A, an obligate pathogen: is an organism that causes disease in essentially every host it infects, such as *Mycobacterium tuberculosis* or the rabies virus. It is never a harmless resident, so the term contradicts the stem.
- B, a saprophyte: lives on **dead** organic matter in the environment and does not colonise a living host as normal flora. That is not what these two organisms do.
- D, a contaminant of no significance: is dangerously wrong. In the settings described these isolates are the actual cause of disease, and dismissing viridans streptococci from a blood culture in a patient with a damaged valve could cost the patient's life.

Final Answer: A commensal acting as an opportunistic pathogen $\Rightarrow$ C

Answer: (C) Go Back to Q9

Q10.

Solution

Concept – plaque forms in a fixed sequence of stages: Nothing sticks to bare enamel first. A protein film must be laid down before any organism can attach.

Step 1 – stage one, the acquired pellicle: Within minutes of polishing, salivary glycoproteins, proline-rich proteins, statherin and mucins **adsorb** onto the enamel by electrostatic attraction to hydroxyapatite. The result is a thin, **cell-free** film called the acquired pellicle. It is the layer marked X.

Step 2 – state what the pellicle does: It changes the surface the bacteria see. It presents **receptors** that bacterial adhesins can bind to, so it is the essential first step of every later stage.

Step 3 – stage two, the initial colonisers: Within hours the pioneer organisms, mainly *Streptococcus sanguinis*, *S. oralis* and *S. mitis*, attach reversibly by weak long-range forces, then irreversibly through adhesin-to-receptor binding on the pellicle. These are the cocci drawn just above X.

Step 4 – stage three, coaggregation: Later organisms cannot bind the pellicle directly, so they bind the bacteria already there. *Fusobacterium nucleatum* is the classic bridging organism, linking early colonisers to late Gram-negative anaerobes. The species diversity rises sharply.

Step 5 – stage four, maturation: The mass grows, the cells secrete an extracellu-

lar polymeric matrix, fluid channels form, and the depths turn anaerobic. Within 1 to 3 weeks the flora shifts to filaments, spirochaetes and Gram-negative anaerobes, and the mature biofilm resists antimicrobials far better than free-floating cells.

Step 6 – read the figure against the sequence: X sits directly on the enamel and beneath the first organisms, and the stem states it forms before any organism attaches. Only the acquired pellicle fits.

Why the other options are wrong:

- A, a mat of filaments coaggregating with cocci: is stage three. It requires organisms to be present already, but X is stated to form before any organism attaches.
- B, a mature biofilm with channels and anaerobic depths: is the final stage after days to weeks, and the figure places the mature biofilm at the top, well above X.
- D, calculus: is plaque that has **already** mineralised, so it is later still, and it certainly is not a cell-free film formed within minutes on polished enamel.

Final Answer: The acquired pellicle of salivary glycoproteins $\Rightarrow$ C

Answer: (C) Go Back to Q10

Q11.

Solution

Concept – an autoclave sterilises only what its steam can touch: The cycle parameters can be perfect and the load still fail, because steam must physically reach every surface.

Step 1 – state how steam kills: Saturated steam kills by condensing on a cool surface, giving up its latent heat there and coagulating microbial proteins. **No contact means no condensation, and no condensation means no kill.**

Step 2 – identify the enemy, which is air: Air is a poor conductor and, being denser, forms cold pockets that steam cannot displace. Any trapped air pocket is an unsterilised pocket. Correct loading exists to let air out and steam in.

Step 3 – state the loading rules: Load packs **loosely**, filling no more than about three quarters of the chamber. Stand packs **on edge** and keep them separated and clear of the chamber walls and door, so steam can circulate on every face. Place bowls and containers **tilted or inverted** so air drains out instead of pooling.

Step 4 – state the packaging rules: Wrap in a **steam-permeable** material such as

autoclave paper, muslin or a paper and plastic pouch. Wrap firmly but not tightly, and never in an impermeable foil or a sealed airtight tin. Open hinged instruments and unlock ratchets so the joint surfaces are exposed.

Step 5 – read chamber X in the figure: Its packs stand upright on edge with clear gaps between them. Steam can flow between the packs and contact every face, so the load sterilises.

Step 6 – read chamber Y: Its packs are jammed into a solid block. The centre of that block never sees steam, so a spore strip placed there survives and the cycle fails, even though the chamber gauge read 121°C throughout.

Why the other options are wrong:

- A, packing as tightly as possible: is the exact fault shown in chamber Y. Overloading is the commonest reason a correctly working autoclave fails to sterilise.
- C, sealing in airtight metal containers: guarantees failure, because steam cannot enter a sealed container at all and the trapped air inside is never displaced.
- D, wrapping in impermeable aluminium foil: is wrong for the same reason. Foil blocks steam. Dryness at the end of the cycle is achieved by the drying phase, not by keeping steam out.

Final Answer: Loose packs on edge with gaps, so steam contacts every surface $\Rightarrow$ B

Answer: (B) Go Back to Q11

Q12.

Solution

Concept – a deep, dirty, devitalised wound is a tetanus wound: The organism is an obligate anaerobe, so the wound must supply low oxygen tension before it can germinate.

Step 1 – read the wound: Deep, ragged and contaminated with **soil**. Soil is the reservoir of *Clostridium tetani* spores, which also occur in dust and in animal and human faeces.

Step 2 – describe the organism: *C. tetani* is a Gram-positive, **obligately anaerobic**, spore-forming bacillus. Its terminal spore gives the classic drumstick or tennis-racket shape.

Step 3 – explain why this wound permits it: Spores germinate only where the

redox potential is low. A deep, crushed, dirty wound with necrotic tissue and foreign material provides exactly that, whereas a clean superficial cut does not.

Step 4 – state the mechanism precisely: The vegetative organism stays local and produces **tetanospasmin**. The toxin travels by retrograde axonal transport to the spinal cord, where it cleaves synaptobrevin and so **blocks the release of the inhibitory transmitters glycine and GABA** from Renshaw cells and other inhibitory interneurons.

Step 5 – link mechanism to signs: Losing inhibition leaves motor neurons firing unopposed, giving **spastic** paralysis: trismus or lockjaw, risus sardonicus, opisthotonus and lethal laryngeal and respiratory spasm.

Step 6 – state the dental relevance and the management: Trismus is often the first sign, so a dentist may see the patient first, and this wound is on the face. Management is wound debridement, human tetanus immunoglobulin for immediate passive protection, tetanus toxoid to start active immunity, and metronidazole. This is the case that makes an unknown immunisation history a genuine emergency.

Why the other options are wrong:

- A, *C. perfringens* with tetanospasmin: names the wrong toxin. *C. perfringens* causes gas gangrene through its **alpha toxin**, a lecithinase that lyses membranes. Tetanospasmin belongs only to *C. tetani*.
- C, *C. botulinum* causing spastic paralysis: reverses the effect. Botulinum toxin does block acetylcholine release, but the result is **flaccid** descending paralysis, the opposite of tetanus, and botulism follows ingestion of preformed toxin rather than a soil-contaminated wound.
- D, *Staphylococcus epidermidis* as a spore former causing tetanus: is false on every count. It is a Gram-positive coccus, it forms **no spores**, it is a skin commensal, and it produces no tetanospasmin.

Final Answer: *Clostridium tetani* and tetanospasmin blocking inhibitory transmitters $\Rightarrow$ B

Answer: (B) Go Back to Q12

Q13.

Solution

Concept – HPV-positive oropharyngeal carcinoma is a separate disease: It has a different cause, a different patient, a different biology and, most importantly for the examination, a better outcome.

Step 1 – read the patient: A relatively young **non-smoker** with a **tonsillar** carcinoma. The oropharynx, meaning tonsil and base of tongue, is where HPV-driven tumours arise, because the tonsillar crypt epithelium is the virus's target.

Step 2 – read the marker: Strong diffuse **p16** overexpression is the accepted surrogate for transcriptionally active high-risk HPV in the oropharynx. The commonest type by far is **HPV 16**.

Step 3 – explain why p16 goes up rather than down: The viral **E7** protein binds and inactivates **Rb**. Free E2F then drives the cell cycle, and the cell responds by pushing p16 up in a futile attempt to brake a pathway that is already broken downstream. So p16 accumulates and is easily stained.

Step 4 – complete the mechanism: The viral **E6** protein binds **p53** and directs it to proteasomal degradation. So both major tumour suppressors are knocked out by viral proteins, not by mutation. In the HPV-negative tumour p53 is instead **mutated** by tobacco carcinogens.

Step 5 – explain the better prognosis: Because p53 is degraded rather than mutated, the gene itself is intact. These tumours also carry far fewer background mutations and arise in mucosa that has not undergone tobacco-driven field change. They are markedly more **radiosensitive and chemosensitive**, so survival is substantially better stage for stage.

Step 6 – note the practical consequence: HPV status is now a formal part of staging for oropharyngeal carcinoma, and p16-positive tumours are staged separately because their outcome is so much better. The histology given, **non-keratinising**, is itself typical of the HPV-driven tumour.

Why the other options are wrong:

- B, HPV 6 and 11 with a worse prognosis: is wrong twice. HPV 6 and 11 are the **low-risk** types, causing squamous papilloma and condyloma, and the prognosis here is better, not worse.
- C, p16 merely reflecting tobacco: is wrong. The patient is a non-smoker, and in the tobacco-related, HPV-negative tumour p16 is typically **lost** through deletion or methylation, so the staining would be negative.
- D, HPV 16 but identical behaviour: contradicts the clinical evidence. The whole reason p16 is tested is that outcome differs so much, and de-escalation

of treatment for these patients is built on that difference.

Final Answer: High-risk HPV 16, E6 and E7 on p53 and Rb, better prognosis ⇒ A

Answer: (A) Go Back to Q13

Q14.

Solution

Concept – one question decides everything: can the organism still replicate? Every difference between a live attenuated vaccine and a killed vaccine follows from that single fact.

Step 1 – define the live attenuated vaccine: It contains the whole organism, still **alive** but weakened by repeated passage or genetic modification so that it has lost virulence while keeping its antigens. Examples are BCG, oral polio, and measles, mumps and rubella.

Step 2 – work out its immunity from its ability to replicate: Because it multiplies in the host, it mimics natural infection. The antigen dose amplifies itself, so the response is **strong and long lasting**, often lifelong after a single dose.

Step 3 – explain the cell-mediated arm: Since the organism actually enters host cells and produces antigen inside them, its peptides are presented on **MHC class I**. That primes **cytotoxic T cells**, so live vaccines give solid cell-mediated immunity as well as antibody. Given by the natural route, as with oral polio, they also raise local **IgA**.

Step 4 – state the price of replication: The same replication makes it unsafe in some hosts. It can **revert** to virulence, as vaccine-associated paralytic polio does, and it can **disseminate** in a patient who cannot control it. It is therefore **contraindicated in immunodeficiency, in patients on high-dose steroids or chemotherapy, and in pregnancy**. It is also heat labile, so it needs a strict cold chain.

Step 5 – define the killed vaccine and derive its properties: It contains organisms inactivated by heat or chemicals. It **cannot replicate**, so it cannot revert or disseminate, and it is **safe** even in the immunocompromised and in pregnancy. It is also more stable.

Step 6 – derive its weaknesses from the same fact: Because the antigen cannot amplify itself, the whole dose must be injected, it is handled through the **MHC class II** exogenous pathway, and the response is mainly **humoral** with weak cell-mediated immunity. It therefore needs an **adjuvant**, a larger antigen mass, and

booster doses, and immunity wanes. Examples are the Salk polio vaccine, hepatitis A and rabies vaccines.

Step 7 – match to the option: Option C states each of these points correctly and in the right direction, so it is the answer.

Why the other options are wrong:

- A, killed vaccines giving stronger and longer immunity: reverses the truth. The live vaccine gives the stronger and more durable response precisely because it replicates, which is why the killed vaccine is the one that needs boosters.
- B, live vaccines safe in pregnancy and immunodeficiency: is the most dangerous error in the set. These are exactly the two groups in which live vaccines are **contraindicated**, because a weakened organism can still disseminate in a host who cannot contain it or cross to the fetus.
- D, identical mechanisms differing only in storage: is false. They differ in replication, in the arms of immunity produced, in dosing, and in safety. Storage stability is a consequence of the difference, not the whole of it.

Final Answer: Live replicates and gives stronger, longer immunity but is unsafe in the immunocompromised; killed is safer but needs adjuvant and boosters $\Rightarrow$ C

Answer: (C) Go Back to Q14

Answer Key

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