NEET MDS General Pathology and Microbiology

Sample Paper – 1

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 change that marks the point of no return, where reversibly injured cells become irreversibly injured, is:

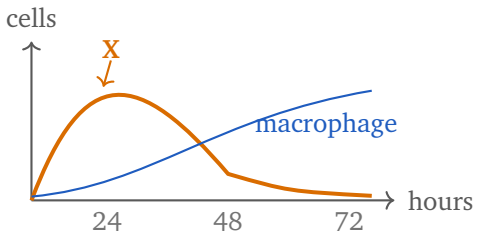
(A) Cellular swelling from failure of the sodium-potassium pump

(B) Fatty change within the cytoplasm

(C) Severe mitochondrial damage together with profound membrane defects

(D) Detachment of ribosomes from the rough endoplasmic reticulum

Q2. The graph below plots the cells arriving at a site of acute inflammation against time. The cell marked **X**, which predominates during the first 24 hours, is the:



(A) Lymphocyte

(B) Plasma cell

(C) Eosinophil

(D) Neutrophil

Q3. A granuloma, the histological hallmark of chronic granulomatous inflammation as seen in tuberculosis, is a focal collection of:

(A) Neutrophils surrounding a necrotic core

(B) Epithelioid cells, which are activated macrophages, often with Langhans giant cells and a rim of lymphocytes

(C) Plasma cells actively producing antibody

(D) Fibroblasts laying down dense collagen

Neoplasia

Q4. The single most reliable feature that distinguishes a malignant tumour from a benign one is:

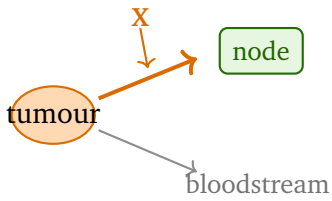
(A) Invasion of surrounding tissue and the capacity to metastasise

(B) A large size at presentation

(C) A rapid rate of growth

(D) The presence of a well formed capsule

Q5. The diagram below shows the routes by which a tumour may spread. Oral squamous cell carcinoma, like carcinomas generally, spreads preferentially by the route marked **X**, reaching the submandibular and deep cervical nodes. Route **X** is:



(A) Lymphatic spread

(B) Haematogenous spread

(C) Transcoelomic spread

(D) Perineural spread, and by no other route

Q6. A malignant tumour arising from mesenchymal tissue such as bone, cartilage, muscle or fibrous tissue is termed a:

(A) Carcinoma

(B) Sarcoma

(C) Adenoma

(D) Papilloma

Haemodynamic Disorders and Wound Healing

Q7. An extraction socket cannot have its margins approximated, so the defect fills with granulation tissue from its base and contracts as it heals. This is healing by:

(A) Second intention

(B) First intention

(C) Primary union

(D) Pure regeneration, with no granulation tissue formed

Q8. Virchow's triad names the three broad factors that predispose to thrombosis. They are:

(A) Fever, leucocytosis and a raised ESR

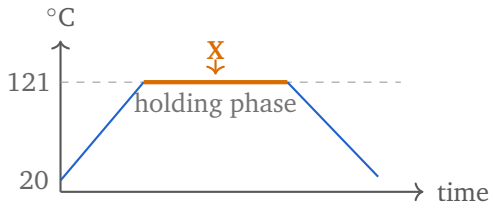
(B) Hypertension, hyperlipidaemia and smoking

(C) Anaemia, neutropenia and thrombocytopenia

(D) Endothelial injury, abnormal blood flow and hypercoagulability

General Microbiology and Sterilisation

Q9. The chart below shows the holding phase of the standard autoclave cycle used for wrapped dental instruments, marked **X**. The parameters at **X** are:



(A) 121°C at 15 psi for 15 minutes

(B) 160°C at atmospheric pressure for 2 hours

(C) 100°C at 15 psi for 5 minutes

(D) 180°C at 30 psi for 30 minutes

Q10. A heat sensitive item such as a plastic or rubber component, which would be destroyed by autoclaving, is best sterilised by:

(A) A hot air oven

(B) Boiling for 30 minutes

(C) Ethylene oxide gas

(D) Autoclaving at a reduced temperature

Q11. The biological indicator used to confirm that an autoclave cycle has actually achieved sterilisation contains the spores of:

(A) Bacillus atrophaeus (formerly Bacillus subtilis)

(B) Geobacillus stearothermophilus

(C) Clostridium tetani

(D) Escherichia coli

Systemic Bacteriology and Virology

Q12. The organism most strongly implicated in the initiation of dental caries, because it is acidogenic, aciduric and synthesises extracellular polysaccharide from sucrose, is:

(A) Lactobacillus acidophilus

(B) Actinomyces viscosus

(C) Streptococcus salivarius

(D) Streptococcus mutans

Q13. The hepatitis virus that carries the greatest occupational risk to a dentist from a needlestick injury, and against which vaccination is required before clinical work, is:

(A) Hepatitis A virus

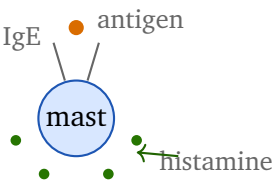
(B) Hepatitis B virus

(C) Hepatitis E virus

(D) Hepatitis D virus, acting on its own

Immunology and Hypersensitivity

Q14. Minutes after an injection of penicillin a patient develops bronchospasm, laryngeal oedema and hypotension. The mechanism, shown below, involves antigen cross-linking **IgE** bound to mast cells, which degranulate and release histamine. This reaction is:



(A) Type II hypersensitivity

(B) Type III hypersensitivity

(C) Type I hypersensitivity

(D) Type IV hypersensitivity

Detailed Solutions

Q1.

Solution

Concept – the point of no return in cell injury: Reversible injury is a cell in trouble; irreversible injury is a cell that cannot recover even if the stimulus is removed.

Step 1 – name the two hallmarks of irreversibility: They are the **inability to reverse mitochondrial dysfunction** and **profound disturbance of membrane function**.

Step 2 – explain the mitochondrial half: Severe mitochondrial damage means ATP cannot be regenerated. Without ATP no pump can run, so the cell has lost the very machinery it would need to repair itself.

Step 3 – explain the membrane half: Membrane defects let lysosomal enzymes escape into the cytoplasm and digest the cell, and let calcium flood in, which activates further degradative enzymes.

Step 4 – match to the option: Option C names both hallmarks together, so it is the answer.

Why the other options are wrong:

- A, cellular swelling: is the **first** and the classic **reversible** change. Restore the oxygen supply and the pump restarts and the swelling resolves.
- B, fatty change: is also **reversible**. It is seen in the liver, heart and kidney and regresses once the insult is removed.
- D, detachment of ribosomes: is a **reversible** ultrastructural change reflecting reduced protein synthesis.

Final Answer: Severe mitochondrial damage with membrane defects $\Rightarrow$ $\boxed{\text{C}}$

Answer: (C) Go Back to Q1

Q2.

Solution

Concept – acute inflammation has a fixed cellular timetable: The cells arrive in a set order, and the graph is really asking you to recall that order.

Step 1 – read the timing given: Cell X predominates in the **first 24 hours**.

Step 2 – recall who arrives first: The **neutrophil** is the first cell to reach the site,

dominating the first 6 to 24 hours.

Step 3 – explain why it is first: Neutrophils are the most numerous leucocyte in blood, they respond fastest to chemotactic signals, and they adhere and emigrate more readily than other cells.

Step 4 – read the second curve: Neutrophils are short lived and die by apoptosis within about 24 to 48 hours, and **macrophages** then take over from 24 to 48 hours onwards. The rising second curve in the figure is labelled macrophage, which confirms the first curve must be the neutrophil.

Why the other options are wrong:

- A, the lymphocyte: is the hallmark of **chronic** inflammation, arriving days later, not in the first 24 hours.
- B, the plasma cell: is a terminally differentiated B cell of **chronic** inflammation, and it takes days to appear.
- C, the eosinophil: predominates only in **parasitic** infections and **allergic** reactions, not in ordinary acute inflammation.

Final Answer: Neutrophil $\Rightarrow$ D

Answer: (D) Go Back to Q2

Q3.

Solution

Concept – what a granuloma actually is: A granuloma is the body walling off something it cannot digest.

Step 1 – name the central cell: The **epithelioid cell**, which is an activated macrophage that has flattened out and come to resemble an epithelial cell.

Step 2 – add the giant cells: Epithelioid cells fuse to form multinucleated **giant cells**. In tuberculosis the nuclei arrange peripherally in a horseshoe, giving the **Langhans** giant cell.

Step 3 – add the rim: A collar of **lymphocytes**, mostly CD4 T cells, surrounds the aggregate and keeps the macrophages activated through interferon gamma.

Step 4 – explain why it forms: When an agent resists phagocytic killing, such as the mycobacterium, acute inflammation cannot clear it. The body instead isolates it inside this cellular wall. Caseous necrosis at the centre is the additional feature specific to tuberculosis.

Why the other options are wrong:

- A, neutrophils around a necrotic core: describes an **abscess**, which is suppurative acute inflammation, not a granuloma.
- C, plasma cells producing antibody: is a feature of chronic inflammation generally, and it defines no granuloma.
- D, fibroblasts laying down collagen: describes **fibrosis** or scarring. Fibrosis may surround an old granuloma, but it is not the granuloma itself.

Final Answer: Epithelioid cells with Langhans giant cells $\Rightarrow$ B

Answer: (B) Go Back to Q3

Q4.

Solution

Concept – the one criterion that never fails: Most features that "look" malignant are only tendencies. Only one is definitive.

Step 1 – state the definitive criterion: Invasion of surrounding tissue, and the capacity to **metastasise**, are the two features unique to malignancy.

Step 2 – explain why metastasis is decisive: No benign tumour ever metastasises. If a deposit is found at a distant site, the tumour is malignant, with no further argument needed.

Step 3 – explain why invasion is decisive: Benign tumours push tissue aside and stay within a capsule. Malignant tumours cross the basement membrane and infiltrate, which is why they cannot be shelled out.

Step 4 – note why the others are only tendencies: Size and growth rate are relative. Malignant tumours *tend* to be larger and faster, but a small slow carcinoma is still a carcinoma and a large fast benign tumour is still benign.

Why the other options are wrong:

- B, large size: is unreliable. A lipoma can grow very large and stay entirely benign, while a small oral carcinoma is already lethal.
- C, rapid growth: is unreliable. A rapidly growing pyogenic granuloma is benign, and some carcinomas grow slowly over years.
- D, the presence of a capsule: points the **opposite** way. A capsule is characteristic of a **benign** tumour, because it reflects the absence of invasion.

Final Answer: Invasion and the capacity to metastasise $\Rightarrow$ A

Answer: (A) Go Back to Q4

Q5.

Solution

Concept – carcinomas and sarcomas choose different roads: The route of spread follows the tissue of origin, and this single rule answers most spread questions.

Step 1 – state the rule: Carcinomas spread preferentially by **lymphatics**. **Sarcomas** spread preferentially by the **bloodstream**.

Step 2 – apply it to the tumour named: Oral squamous cell carcinoma is a carcinoma of epithelial origin, so it takes the lymphatic route.

Step 3 – confirm with the destination given: The question says the tumour reaches the **submandibular and deep cervical nodes**. Those are lymph nodes, which only lymphatic spread can reach directly.

Step 4 – note the clinical importance: Because spread is lymphatic and orderly, cervical node status is the strongest single prognostic factor in oral cancer, and it is why neck dissection is part of treatment.

Why the other options are wrong:

- B, haematogenous spread: is the preferred route of **sarcomas**, and typically reaches the lungs and liver. Oral carcinoma can eventually spread this way, but it is not the preferential route and it does not explain cervical nodes.
- C, transcoelomic spread: is seeding across a **body cavity**, such as the peritoneum in ovarian carcinoma. The oral cavity is not a coelomic cavity.
- D, perineural spread and no other route: is a real feature of some oral carcinomas, particularly adenoid cystic carcinoma, but the wording "by no other route" makes the statement false.

Final Answer: Lymphatic spread $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q5

Q6.

Solution

Concept – tumour nomenclature follows the tissue of origin: Name the germ layer, then apply the suffix.

Step 1 – read the tissue given: Bone, cartilage, muscle and fibrous tissue are all **mesenchymal** (connective) tissues.

Step 2 – apply the malignant suffix for mesenchyme: A malignant mesenchy-

mal tumour is a **sarcoma**, for example osteosarcoma, chondrosarcoma or rhabdomyosarcoma.

Step 3 – contrast with epithelium: A malignant **epithelial** tumour is a carcinoma. That is the distinction being tested.

Step 4 – complete the rule for benign tumours: A benign mesenchymal tumour simply takes the suffix *-oma* on its tissue name, such as osteoma, chondroma or lipoma.

Why the other options are wrong:

- A, carcinoma: is malignant but arises from **epithelium**, which is the wrong tissue of origin here.
- C, adenoma: is a **benign** tumour of **glandular epithelium**, so it is wrong on both counts.
- D, papilloma: is a **benign** tumour of surface epithelium forming finger-like projections, again wrong on both counts.

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

Answer: (B) Go Back to Q6

Q7.

Solution

Concept – first versus second intention is decided by the gap: Whether the wound edges meet decides which kind of healing occurs.

Step 1 – define first intention: A clean surgical incision with edges **approximated** by sutures. The gap is minimal, so little granulation tissue is needed and the scar is thin.

Step 2 – define second intention: A wound with a **large tissue defect** whose edges cannot be brought together. It fills with abundant granulation tissue from the base upwards.

Step 3 – read the socket described: The question says the margins cannot be approximated and the defect fills with granulation tissue from its base, which is the second intention description exactly.

Step 4 – confirm with contraction: The question also mentions contraction. Wound contraction by myofibroblasts is a distinctive feature of **second** intention healing, and it does not feature meaningfully in first intention.

Why the other options are wrong:

- B, first intention: requires approximated edges, which the question explicitly rules out.
- C, primary union: is simply another name for first intention, so it fails for the same reason.
- D, pure regeneration with no granulation tissue: does not occur in a socket. Regeneration alone happens only in labile tissues with an intact framework, whereas a socket involves a true tissue defect and must granulate.

Final Answer: Second intention ⇒ A

Answer: (A) Go Back to Q7

Q8.

Solution

Concept – Virchow's triad: Three broad categories cover every predisposing cause of thrombosis.

Step 1 – the first arm: Endothelial injury. A damaged endothelium exposes subendothelial collagen and tissue factor, so platelets adhere and the cascade fires.

Step 2 – the second arm: Abnormal blood flow, meaning stasis or turbulence. Normal laminar flow keeps platelets central and away from the wall; stasis lets them contact the endothelium and lets clotting factors accumulate instead of being washed away.

Step 3 – the third arm: Hypercoagulability, an altered blood constitution. This may be inherited, such as factor V Leiden, or acquired, such as pregnancy, malignancy or oral contraceptive use.

Step 4 – match to the option: Option D lists exactly these three, so it is the answer.

Why the other options are wrong:

- A, fever, leucocytosis and raised ESR: are non-specific markers of **inflammation**, not a description of thrombogenesis.
- B, hypertension, hyperlipidaemia and smoking: are risk factors for **atherosclerosis**. They work partly by causing endothelial injury, which makes this the tempting distractor, but they are not the triad itself.
- C, anaemia, neutropenia and thrombocytopenia: are cytopenias. Thrombocytopenia in fact predisposes to **bleeding**, the opposite of thrombosis.

Final Answer: Endothelial injury, abnormal flow and hypercoagulability ⇒ D

Answer: (D) Go Back to Q8

Q9.

Solution

Concept – the standard autoclave cycle: Moist heat under pressure is the workhorse of dental sterilisation, and its numbers must be known exactly.

Step 1 – recall the standard parameters: 121°C, at **15 psi** (about 1 atmosphere above ambient), for a holding time of **15 minutes**.

Step 2 – explain why pressure is needed: Water boils at 100°C at atmospheric pressure, and 100°C does not kill spores. Raising the pressure raises the boiling point, which lets steam reach 121°C.

Step 3 – explain why steam rather than dry heat: Steam kills by coagulating proteins and carries far more latent heat than dry air, so it sterilises at a much lower temperature and in a fraction of the time.

Step 4 – note the holding time: The 15 minutes is the **holding** time at temperature, which the figure marks as X. It excludes the heat-up and cool-down limbs, a distinction the examiner likes to test.

Step 5 – note the alternative cycle: 134°C at 30 psi for 3 minutes is the accepted faster alternative, used in most modern vacuum autoclaves.

Why the other options are wrong:

- B, 160°C for 2 hours at atmospheric pressure: is the **hot air oven** cycle, which is dry heat and not an autoclave.
- C, 100°C for 5 minutes: is boiling. It kills vegetative bacteria but **not spores**, so it disinfects rather than sterilises. The stated 15 psi is also internally inconsistent, since at 15 psi steam would be at 121°C, not 100°C.
- D, 180°C at 30 psi for 30 minutes: mixes numbers from different methods and corresponds to no recognised cycle.

Final Answer: 121°C at 15 psi for 15 minutes $\Rightarrow$ A

Answer: (A) Go Back to Q9

Q10.

Solution

Concept – match the method to the material: When heat would destroy the item, you must sterilise chemically at low temperature.

Step 1 – read the constraint: The item is **heat sensitive**, so every heat-based method is ruled out at once.

Step 2 – name the low-temperature option: Ethylene oxide gas sterilises at 55 to 60°C, which plastics and rubber tolerate.

Step 3 – explain how it works: Ethylene oxide is an alkylating agent. It alkylates the nucleic acids and proteins of the organism, and it is **sporicidal**, so it achieves true sterilisation rather than disinfection.

Step 4 – note its cost: It is slow, taking hours, and it is toxic and carcinogenic, so the load must be aerated afterwards to drive off residual gas. That is the price paid for low-temperature sterilisation.

Why the other options are wrong:

- A, the hot air oven: runs at 160°C, which is **hotter** than the autoclave the item already could not survive. It is used for glass, oils and sharp instruments.
- B, boiling: does not kill spores at all, so it is **disinfection**, not sterilisation, whatever the material.
- D, autoclaving at a reduced temperature: is not a recognised method. Below 121°C the cycle stops being sporicidal, so it would fail to sterilise.

Final Answer: Ethylene oxide gas ⇒ C

Answer: (C) Go Back to Q10

Q11.

Solution

Concept – a biological indicator uses the toughest available spore: If the hardest organism to kill is dead, everything easier is dead too.

Step 1 – state the principle: The indicator carries spores that are **more resistant** to the process than any pathogen likely to be present.

Step 2 – name the autoclave indicator: Geobacillus stearothermophilus, whose spores are the most heat resistant available and are killed only by a fully effective moist heat cycle.

Step 3 – explain why it is the gold standard: Chemical indicators and autoclave tape only confirm that a temperature was reached. A biological indicator confirms that **killing** actually occurred, which is the only direct proof of sterilisation.

Step 4 – pair each method with its indicator: Autoclave uses *G. stearothermophilus*; hot air oven and ethylene oxide use *Bacillus atrophaeus*. That pairing is what the question tests.

Why the other options are wrong:

- A, Bacillus atrophaeus: is the correct indicator for the **hot air oven** and for **ethylene oxide**, not for the autoclave. This is the intended trap.
- C, Clostridium tetani: is a spore-forming pathogen, but its spores are less heat resistant than those of *G. stearothermophilus*, and deliberately culturing a pathogen for routine testing would be unsafe.
- D, Escherichia coli: does not form spores at all. It is killed easily, so it could never validate a sporicidal cycle.

Final Answer: Geobacillus stearothermophilus $\Rightarrow$ B

Answer: (B) Go Back to Q11

Q12.

Solution

Concept – initiation of caries versus progression of caries: Two different organisms dominate the two different stages, and the question specifies initiation.

Step 1 – read the three properties given: Acidogenic (makes acid), aciduric (survives in acid) and able to make extracellular polysaccharide from sucrose.

Step 2 – name the organism: All three define **Streptococcus mutans**.

Step 3 – explain the polysaccharide clue: *S. mutans* uses glucosyltransferase to convert sucrose into insoluble glucans. Those glucans let it stick to enamel and build plaque, which is exactly why it can **initiate** a lesion where other acid producers cannot.

Step 4 – explain the aciduric clue: Having made the acid, it survives the low pH it created, so demineralisation continues below the critical pH of about 5.5.

Why the other options are wrong:

- A, Lactobacillus acidophilus: is strongly acidogenic and aciduric, but it adheres poorly to smooth enamel. It is associated with the **progression** of an established lesion in dentine, not with initiation. This is the main trap in the

question.

- B, Actinomyces viscosus: is implicated in **root surface** caries and in plaque formation, not in the initiation of enamel caries.
- C, Streptococcus salivarius: colonises the **tongue** and oral mucosa rather than enamel, and it is not cariogenic in the way *S. mutans* is.

Final Answer: Streptococcus mutans $\Rightarrow$ D

Answer: (D) Go Back to Q12

Q13.

Solution

Concept – occupational risk in dentistry is decided by route and transmissibility: The virus that matters is the one that travels in blood and transmits easily.

Step 1 – use the route clue: A needlestick is a **parenteral, blood-borne** exposure, so the answer must be a blood-borne hepatitis virus. That leaves B and D, since A and E are faeco-oral.

Step 2 – use the transmissibility clue: Hepatitis B is the most transmissible of the blood-borne viruses. The risk from a single needlestick from a positive source is roughly **30 percent**, against about 3 percent for hepatitis C and about 0.3 percent for HIV.

Step 3 – use the vaccination clue: Hepatitis B is the only hepatitis virus in the list with a vaccine that is mandatory before clinical work. That clue alone settles the answer.

Step 4 – note why the risk is so high: HBV survives on surfaces for up to 7 days and is present in blood at very high titre, so a small inoculum is enough.

Why the other options are wrong:

- A, hepatitis A virus: spreads by the **faeco-oral** route, causes no chronic carrier state, and is not a needlestick risk.
- C, hepatitis E virus: is also **faeco-oral**, typically waterborne, and again not transmitted by needlestick.
- D, hepatitis D acting on its own: cannot happen. HDV is a defective virus that requires the HBsAg coat of **hepatitis B** to infect at all, so it never acts alone. The phrase "on its own" makes the option false.

Final Answer: Hepatitis B virus $\Rightarrow$ B

Answer: (B) Go Back to Q13

Q14.

Solution

Concept – the four Gell and Coombs types are told apart by their mediator: Name the antibody or cell doing the damage, and the type follows.

Step 1 – read the mediator given: The question states that **IgE** bound to **mast cells** is cross-linked by antigen.

Step 2 – match mediator to type: IgE on mast cells is the definition of **Type I** hypersensitivity, the immediate or anaphylactic type.

Step 3 – follow the mechanism: Cross-linking triggers degranulation, releasing **histamine**, leukotrienes and prostaglandins. These cause bronchoconstriction, increased vascular permeability and vasodilation.

Step 4 – match to the clinical picture: Bronchospasm, laryngeal oedema and hypotension within minutes are exactly those mediator effects. The speed is the giveaway, since only Type I acts within minutes.

Step 5 – note the treatment that confirms it: Adrenaline is the first-line treatment, because it reverses each of those mediator effects directly.

Why the other options are wrong:

- A, Type II: is antibody-dependent cytotoxicity, in which **IgG or IgM** binds antigen on a cell surface, as in a mismatched transfusion. The antibody class is wrong.
- B, Type III: is **immune complex** deposition, as in serum sickness. Complexes take hours to days to deposit and activate complement, so the timing is wrong.
- D, Type IV: is **cell-mediated** and driven by T cells, as in contact dermatitis or the tuberculin test. It is delayed by 24 to 72 hours and involves no antibody at all.

Final Answer: Type I hypersensitivity $\Rightarrow$ C

Answer: (C) Go Back to Q14

Answer Key

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