

INI CET Forensic Medicine

Sample Paper – 1

Duration: 14 Minutes

Maximum Marks: 15

Instructions

- This paper contains **15** Multiple Choice Questions (Single Best Response), modelled on the Forensic Medicine content of **INI CET** (Institutes of National Importance Combined Entrance Test) conducted by AIIMS New Delhi.
- The **15-question** length matches the number of Forensic Medicine items you actually meet in the real 200-question paper.
- Each correct answer carries **+1 mark**. There is **negative marking of one-third (0.33) 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 **14 minutes**, the pace the real computer-based test demands.

Thanatology

Q1. The permanent stoppage of function of the individual cells and tissues of the body, occurring a few hours after somatic (systemic) death, is termed:

- (A) Suspended animation
- (B) Molecular (cellular) death
- (C) Somatic death
- (D) Brainstem death

Q2. Brainstem death is best defined as the:

- (A) Reversible loss of consciousness with preserved brainstem reflexes



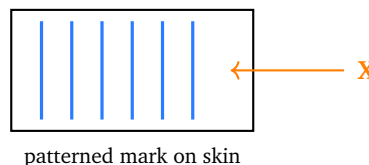
- (B) Irreversible cessation of all brainstem functions, including irreversible apnoea
- (C) Temporary cessation of the heartbeat with an intact circulation
- (D) Isolated flattening of the cortical EEG with normal spontaneous breathing

Q3. A state in which the vital functions of the body are depressed to a minimal, clinically imperceptible level so that life appears extinct, yet the person is still alive and can be revived, is called:

- (A) Molecular death
- (B) Somatic death
- (C) Suspended animation
- (D) Brainstem death

Mechanical Injuries

Q4. The abrasion illustrated on the skin faithfully reproduces the shape and pattern of the object that produced it (marked X), for example a tyre tread or radiator grille. This type of abrasion is an:



- (A) Graze (sliding) abrasion
- (B) Pressure abrasion
- (C) Imprint (patterned) abrasion
- (D) Linear scratch abrasion

Q5. The dry, parchment-like abrasion produced by sustained crushing pressure of a ligature against the skin of the neck, reproducing the weave of the ligature, is an example of a:

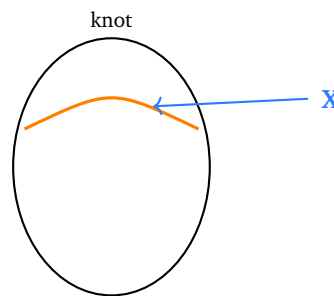
- (A) Graze abrasion



- (B) Sliding abrasion
- (C) Imprint abrasion caused by a single impact
- (D) Pressure abrasion

Asphyxial Deaths

Q6. In the neck diagram of a typical (complete) hanging, the ligature mark (marked X) most characteristically:

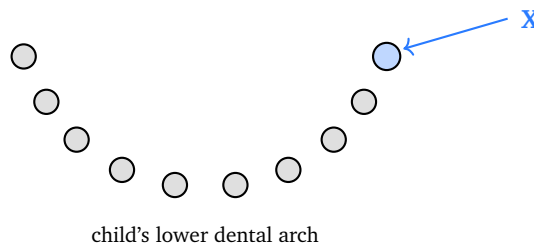


- (A) Runs obliquely, is non-continuous (interrupted at the knot) and lies high on the neck above the thyroid cartilage, directed upward towards the knot
 - (B) Runs transversely, is continuous all round the neck and lies low, below the thyroid cartilage
 - (C) Encircles the neck horizontally with uniform depth throughout
 - (D) Is placed at the root of the neck with an even, unbroken groove
- Q7.** Trickling of saliva from one angle of the mouth running down onto the chin and chest of a hanging victim is an important vital sign indicating that the hanging was antemortem. This finding is known as:
- (A) The dribbling of saliva sign
 - (B) Tardieu spots
 - (C) Paltauf haemorrhages
 - (D) Postmortem hypostasis of the face

Forensic Identification and Medical Jurisprudence



Q8. In the diagram of a child's lower dental arch, the first permanent tooth to erupt (marked X), appearing at about 6 years of age, is the:



- (A) Central incisor
- (B) Lateral incisor
- (C) First permanent molar
- (D) First premolar

Q9. Gustafson's method of estimating age from a single ground section of a tooth is based on scoring how many age-related regressive changes?

- (A) Three
- (B) Four
- (C) Five
- (D) Six

Q10. The second permanent molar tooth normally erupts at approximately what age?

- (A) 6 years
- (B) 9 years
- (C) 12 years
- (D) 18 years

General Toxicology and Corrosive Poisons

Q11. In toxicology, the "fatal period" of a poison is defined as the:

- (A) Smallest amount of the poison likely to cause death



- (B) Time interval between the intake of the poison and death
- (C) Largest dose that can be tolerated without causing death
- (D) Time taken for the poison to be completely excreted from the body

Q12. In a conscious, cooperative adult who has ingested a poison and presents early, the single most useful measure of gastrointestinal decontamination for most adsorbable poisons is:

- (A) Forced emesis with syrup of ipecac
- (B) Routine gastric lavage performed after 6 hours
- (C) Whole-bowel irrigation for every ingested poison
- (D) Oral activated charcoal

Specific Poisons

Q13. The specific pharmacological antidote that reactivates the inhibited acetylcholinesterase enzyme in organophosphorus poisoning is:

- (A) Pralidoxime (2-PAM)
- (B) Atropine
- (C) Naloxone
- (D) N-acetylcysteine

Q14. The muscarinic effects of acute organophosphate poisoning are recalled by the mnemonic DUMBELS. Which of the following is a purely muscarinic feature?

- (A) Muscle fasciculations
- (B) Excessive salivation and lacrimation
- (C) Tachycardia with widely dilated pupils
- (D) Flaccid muscle paralysis

Q15. Aluminium phosphide (celphos) poisoning liberates phosphine gas on contact with moisture and classically presents with:



- (A) A garlicky breath odour with refractory hypotension and severe metabolic acidosis, and no specific antidote
- (B) A bitter-almond breath odour with rapid loss of consciousness
- (C) Cherry-red discoloration of the skin and mucous membranes
- (D) A prompt, complete response to intravenous dimercaprol (BAL)



Detailed Solutions**Q1.****Solution**

Concept – somatic versus molecular death: Death is a process, not a single instant, and it can be graded.

Step 1 – define somatic death: Somatic (systemic) death is the permanent stoppage of the functions of the brain, heart and lungs, so the person is clinically dead.

Step 2 – define molecular death: For a few hours after somatic death individual cells and tissues keep living; their permanent stoppage of function is called **molecular (cellular) death**, and it proceeds tissue by tissue.

Why the other options are wrong:

- A, suspended animation: the person is still alive, not dead.
- C, somatic death: refers to the death of the whole organism, not of individual cells.
- D, brainstem death: irreversible loss of brainstem function, a different concept.

Final Answer: Molecular (cellular) death $\Rightarrow$ **B**

Answer: (B) [Go Back to Q1](#)

Q2.**Solution**

Concept – brainstem death: The brainstem drives breathing and the basic reflexes that sustain life.

Step 1 – state the definition: Brainstem death is the **irreversible cessation of all brainstem functions**, which includes irreversible loss of the capacity to breathe (proven by a positive apnoea test).

Step 2 – significance: Once diagnosed, it is legally equivalent to death of the person, even if the heart is kept beating on a ventilator.

Why the other options are wrong:

- A, reversible loss of consciousness with preserved reflexes: describes coma, not brain death.
- C, temporary cessation of the heartbeat: unrelated to brainstem death.
- D, isolated cortical EEG flattening with normal breathing: intact brainstem,



so not brainstem death.

Final Answer: Irreversible cessation of all brainstem function $\Rightarrow$ **B**

Answer: (B) [Go Back to Q2](#)

Q3.

Solution

Concept – apparent death: Vital functions can fall so low that they escape ordinary clinical detection.

Step 1 – name the state: When the heartbeat and breathing are so feeble that they cannot be detected clinically yet the person is alive and revivable, the condition is **suspended animation** (apparent death).

Step 2 – examples: It is seen in drowning, electrocution, hypothermia, and deep anaesthesia; the danger is a mistaken diagnosis of death.

Why the other options are wrong:

- A, molecular death: death of cells after somatic death, irreversible.
- B, somatic death: real death of the whole organism.
- D, brainstem death: irreversible, not revivable.

Final Answer: Suspended animation $\Rightarrow$ **C**

Answer: (C) [Go Back to Q3](#)

Q4.

Solution

Concept – types of abrasion: The shape of an abrasion can reveal the causative object.

Step 1 – read the figure: The mark reproduces the exact pattern of the object (parallel ridges), so the object has been stamped onto the skin.

Step 2 – name the type: This is an **imprint (patterned) abrasion**, in which the wound is a faithful negative of the object, giving it high medico-legal value for identifying the weapon or vehicle.

Why the other options are wrong:

- A, graze abrasion: caused by lateral rubbing; it does not reproduce a pattern.



- B, pressure abrasion: caused by sustained pressure (e.g. a ligature), not by a stamped pattern of impact.
- D, linear scratch abrasion: a fine line from a pointed object, again not a pattern.

Final Answer: Imprint (patterned) abrasion ⇒

Answer: (C) [Go Back to Q4](#)

Q5.

Solution

Concept – pressure abrasion: Sustained pressure, rather than a moving edge, produces a distinct abrasion.

Step 1 – identify the mechanism: A ligature crushing the neck presses the superficial skin, which dries after death into a hard, parchment-like band.

Step 2 – name the type: This is a **pressure abrasion**; the ligature mark of hanging and strangulation is its classic example.

Why the other options are wrong:

- A, graze abrasion: due to sliding friction, not sustained pressure.
- B, sliding abrasion: another term for a graze; involves movement.
- C, imprint abrasion from a single impact: results from one stamping blow, not from continuous pressure.

Final Answer: Pressure abrasion ⇒

Answer: (D) [Go Back to Q5](#)

Q6.

Solution

Concept – ligature mark in typical hanging: The direction and level of the groove separate hanging from strangulation.

Step 1 – read the figure: The groove rises on each side of the neck towards the knot, and it is interrupted where the knot lies.

Step 2 – state the features: In a typical hanging the mark is **oblique, non-continuous (broken at the knot) and high on the neck above the thyroid cartilage**, running upward towards the point of suspension.



Why the other options are wrong:

- B, transverse, continuous, low mark: this is the pattern of ligature strangulation, not hanging.
- C, uniform horizontal groove: again describes strangulation.
- D, even unbroken groove at the root of the neck: not the typical hanging mark.

Final Answer: Oblique, non-continuous, high, directed towards the knot ⇒ A

Answer: (A) [Go Back to Q6](#)

Q7.

Solution

Concept – vital signs of antemortem hanging: Certain findings prove the neck was compressed while the person was alive.

Step 1 – describe the sign: Pressure on the salivary glands makes saliva trickle from the lower angle of the mouth, running down the chin and chest along the line of gravity.

Step 2 – interpret it: This **dribbling of saliva sign** is a reliable indicator of antemortem hanging, because it needs the victim to have been upright and alive when suspended.

Why the other options are wrong:

- B, Tardieu spots: petechial haemorrhages of asphyxia, not salivary dribbling.
- C, Paltauf haemorrhages: subpleural haemorrhages seen in drowning.
- D, postmortem hypostasis: settling of blood after death, unrelated to saliva.

Final Answer: Dribbling of saliva sign ⇒ A

Answer: (A) [Go Back to Q7](#)

Q8.

Solution

Concept – eruption of permanent teeth: The eruption sequence is a standard tool for estimating a child's age.

Step 1 – read the figure: The tooth marked X sits at the back of the arch behind the deciduous teeth and erupts without any deciduous predecessor.



Step 2 – name it: The **first permanent molar** erupts at about 6 years and is the earliest permanent tooth, which is why it is called the “6-year molar”.

Why the other options are wrong:

- A, central incisor: the permanent central incisor erupts at about 7 years, after the first molar.
- B, lateral incisor: erupts later, at about 8 years.
- D, first premolar: erupts at about 9 to 10 years.

Final Answer: First permanent molar $\Rightarrow$

Answer: (C) [Go Back to Q8](#)

Q9.

Solution

Concept – Gustafson’s method: Age can be estimated from the regressive changes in a single tooth.

Step 1 – count the criteria: Gustafson scored **six** age-related changes: attrition, periodontosis, secondary dentine deposition, cementum apposition, root resorption and root (dentine) transparency.

Step 2 – how it works: Each change is graded 0 to 3, the scores are summed, and the total is fitted to a regression line to read off the age.

Why the other options are wrong:

- A, three; B, four; C, five: all understate the number; Gustafson used six distinct parameters.

Final Answer: Six $\Rightarrow$

Answer: (D) [Go Back to Q9](#)

Q10.

Solution

Concept – eruption of the second molar: Later molars mark later ages in the eruption chart.

Step 1 – place the tooth: The second permanent molar (the “12-year molar”) erupts once the first molar and the incisors, canine and premolars are already in place.



Step 2 – state the age: It erupts at about **12 years**, a widely used landmark in dental age estimation.

Why the other options are wrong:

- A, 6 years: the age of the first permanent molar, not the second.
- B, 9 years: roughly the age of the first premolar.
- D, 18 years: the approximate age of the third molar (wisdom tooth).

Final Answer: 12 years $\Rightarrow$

Answer: (C) [Go Back to Q10](#)

Q11.

Solution

Concept – toxicology definitions: Fatal dose and fatal period are separate ideas.

Step 1 – define the fatal period: The **fatal period** is the time interval between the intake of a poison and the resulting death.

Step 2 – contrast with fatal dose: The fatal dose is a quantity of poison; the fatal period is a duration of time. Both vary with the poison, the dose and the route.

Why the other options are wrong:

- A, smallest amount likely to cause death: this defines the fatal (lethal) dose.
- C, largest tolerated dose: describes a maximum safe dose, not the fatal period.
- D, time for complete excretion: relates to elimination, not the time to death.

Final Answer: Time between intake of the poison and death $\Rightarrow$

Answer: (B) [Go Back to Q11](#)

Q12.

Solution

Concept – gastrointestinal decontamination: Modern practice favours adsorption over emptying the stomach.

Step 1 – choose the measure: **Oral activated charcoal**, given early to a conscious cooperative patient, adsorbs most poisons in the gut and prevents their absorption; it is the single most useful decontamination step.



Step 2 – caveat: It does not bind iron, lithium, alcohols, strong acids or alkalis, and it must not be given if the airway is unprotected.

Why the other options are wrong:

- A, forced emesis with ipecac: now abandoned because of the aspiration risk and poor benefit.
- B, routine lavage after 6 hours: of little value so late and is not done routinely.
- C, whole-bowel irrigation for every poison: reserved for special cases such as iron or sustained-release drugs, not all poisons.

Final Answer: Oral activated charcoal $\Rightarrow$ **D**

Answer: (D) [Go Back to Q12](#)

Q13.

Solution

Concept – antidotes in organophosphate poisoning: Two drugs are used, but only one reactivates the enzyme.

Step 1 – name the reactivator: Pralidoxime (2-PAM) is an oxime that removes the phosphate group from inhibited acetylcholinesterase, regenerating the active enzyme, and it relieves both muscarinic and nicotinic effects.

Step 2 – timing: It must be given early, before “ageing” of the enzyme makes the inhibition irreversible.

Why the other options are wrong:

- B, atropine: blocks muscarinic receptors and dries secretions but does not reactivate the enzyme.
- C, naloxone: the antidote for opioid poisoning.
- D, N-acetylcysteine: the antidote for paracetamol poisoning.

Final Answer: Pralidoxime (2-PAM) $\Rightarrow$ **A**

Answer: (A) [Go Back to Q13](#)



Q14.

Solution

Concept – muscarinic versus nicotinic effects: Organophosphates flood both receptor types, but the mnemonic DUMBELS lists the muscarinic ones.

Step 1 – recall DUMBELS: Diarrhoea, Urination, Miosis, Bradycardia/Bronchorrhoea, Emesis, Lacrimation and **Salivation** are the muscarinic effects.

Step 2 – match the option: Excessive salivation and lacrimation are classic muscarinic features and are the correct answer.

Why the other options are wrong:

- A, muscle fasciculations: a nicotinic effect at the neuromuscular junction.
- C, tachycardia with dilated pupils: muscarinic stimulation causes bradycardia and pinpoint pupils, so this is the opposite pattern.
- D, flaccid muscle paralysis: a nicotinic effect from persistent depolarisation.

Final Answer: Excessive salivation and lacrimation $\Rightarrow$ **B**

Answer: (B) [Go Back to Q14](#)

Q15.

Solution

Concept – aluminium phosphide poisoning: This common agrochemical (celphos) kills by releasing phosphine gas.

Step 1 – describe the picture: On contact with moisture or gastric acid, aluminium phosphide liberates phosphine, giving a **garlicky (decaying-fish) breath odour, refractory hypotension and severe metabolic acidosis**; there is no specific antidote and treatment is supportive.

Step 2 – mechanism: Phosphine blocks mitochondrial cytochrome c oxidase, so cells cannot use oxygen, and profound shock follows.

Why the other options are wrong:

- B, bitter-almond odour: characteristic of cyanide, not phosphide.
- C, cherry-red skin: a feature of carbon monoxide poisoning.
- D, prompt response to dimercaprol: BAL is the antidote for arsenic and some heavy metals, not for aluminium phosphide, which has no specific antidote.

Final Answer: Garlicky breath with refractory hypotension and acidosis, no spe-



cific antidote $\Rightarrow$

Answer: (A) [Go Back to Q15](#)



Answer Key

Q	Ans	Q	Ans	Q	Ans	Q	Ans	Q	Ans
1	B	2	B	3	C	4	C	5	D
6	A	7	A	8	C	9	D	10	C
11	B	12	D	13	A	14	B	15	A

