INI CET Paediatrics

Sample Paper – 5

Duration: 14 Minutes **Maximum Marks: 15**

Instructions

- This paper contains **15** Multiple Choice Questions (Single Best Response), modelled on the Paediatrics content of **INI CET** (Institutes of National Importance Combined Entrance Test) conducted by AIIMS New Delhi.
- The **15-question** length matches the number of Paediatrics 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.

Neonatology

Q1. In a healthy term newborn managed with dry cord care, the umbilical cord stump normally separates and falls off by about:

(A) 5 to 15 days after birth

(B) Within the first 24 hours of life

(C) 6 to 8 weeks after birth

(D) Only after 3 months of age

Q2. Routine intramuscular administration of which vitamin at birth prevents haemorrhagic disease of the newborn (vitamin K deficiency bleeding)?

(A) Vitamin D

(B) Vitamin K

(C) Vitamin C

(D) Vitamin B12

Q3. Gonococcal ophthalmia neonatorum characteristically presents with a hyperacute, profusely purulent conjunctivitis appearing within:

(A) Only after 3 weeks of life

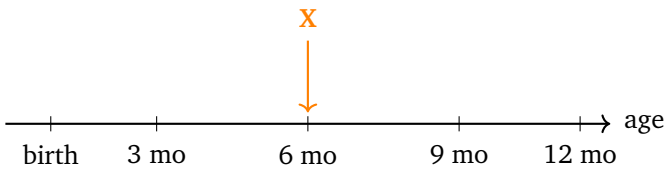
(B) At the moment of birth only

(C) The first 2 to 5 days of life

(D) Only after 6 weeks of age

Growth, Development and Nutrition

Q4. On the developmental timeline below, the arrow **X** marks the age at which a normal infant first *sits without support*. That age is:



(A) About 3 months

(B) About 4 months

(C) About 6 months

(D) About 12 months

Q5. Infantile beriberi, presenting with a hoarse aphonic cry, cardiac failure and peripheral neuropathy in a breastfed infant of a malnourished mother, is caused by deficiency of:

(A) Vitamin B12 (cobalamin)

(B) Vitamin C (ascorbic acid)

(C) Vitamin D (cholecalciferol)

(D) Thiamine (vitamin B1)

Q6. In a child aged 6 to 59 months, the mid-upper-arm circumference (MUAC) cut-off that by itself diagnoses severe acute malnutrition is a value less than:

(A) 15.0 cm

(B) 13.5 cm

(C) 11.5 cm

(D) 9.0 cm

Immunisation and Infectious Disease

Q7. Under India's Universal Immunisation Programme (UIP), the first dose of the measles-rubella (MR) vaccine is scheduled to be given:

(A) At birth, along with BCG

(B) At 9 completed months (with a second dose at 16 to 24 months)

(C) At 6 weeks of age

(D) At 5 years of age

Q8. An unimmunised child has a low-grade fever with a tough, grey, adherent membrane over the tonsils and pharynx that bleeds on attempted removal, plus a 'bull-neck' appearance from cervical swelling. The systemic toxicity is driven by an exotoxin. The diagnosis is:

(A) Streptococcal pharyngitis

(B) Infectious mononucleosis

(C) Oral candidiasis

(D) Diphtheria

Respiratory and Cardiovascular Paediatrics

Q9. A 6-year-old with episodic wheeze is diagnosed with asthma. The recommended first-line *reliever* for the acute relief of his symptoms is:

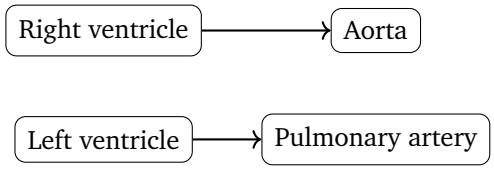
(A) An inhaled short-acting beta-2 agonist (salbutamol)

(B) A course of oral antibiotics

(C) Oral montelukast used as the sole reliever

(D) Daily systemic (oral) corticosteroids

Q10. A term neonate is deeply cyanosed from the first hours of life; the cyanosis does not improve with oxygen. Echocardiography shows the aorta arising from the right ventricle and the pulmonary artery from the left ventricle, as sketched below. An infusion of prostaglandin E1 keeps the child pink until surgery. The diagnosis is:



(A) Transposition of the great arteries

(B) Isolated ventricular septal defect

(C) Atrial septal defect

(D) Patent ductus arteriosus

Q11. A toddler with Tetralogy of Fallot suddenly becomes deeply cyanosed, irritable and hyperpnoeic during a 'tet' spell. The single most useful immediate positioning manoeuvre by the bedside is to:

(A) Lay the child completely flat and supine

(B) Give an intravenous diuretic at once

(C) Stand the child upright and walk him about

(D) Place the child in the knee-chest position

Haematology, Genetics and Systemic Disease

Q12. A 4-year-old boy develops sudden pallor, jaundice and dark (cola-coloured) urine two days after eating fava beans; the blood film shows bite cells and Heinz bodies. This X-linked, oxidant-induced haemolysis is due to deficiency of:

(A) Glucose-6-phosphate dehydrogenase (G6PD)

(B) Spectrin (hereditary spherocytosis)

(C) Iron (iron deficiency anaemia)

(D) Beta-globin chains (beta-thalassaemia)

Q13. A boy has moderate intellectual disability, a long narrow face with a prominent jaw, large protruding ears and, after puberty, macro-orchidism; testing shows an expanded CGG trinucleotide repeat. This commonest *inherited* cause of intellectual disability is:

(A) Down syndrome (trisomy 21)

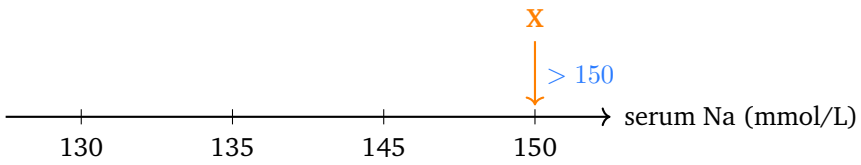
(B) Klinefelter syndrome (47,XXY)

(C) Fragile X syndrome

(D) Turner syndrome (45,X)

Emergency, Fluids and Electrolytes

Q14. On the serum-sodium scale below, the shaded band to the right of the arrow **X** defines *hypernatraemic* dehydration. By definition, hypernatraemic dehydration is present when the serum sodium is:



(A) Below 130 mmol/L

(B) Above 150 mmol/L

(C) Between 135 and 145 mmol/L

(D) Exactly 140 mmol/L

Q15. A toddler swallows a mouthful of kerosene (a hydrocarbon). Forced vomiting and gastric lavage are **avoided** in this poisoning chiefly because:

(A) Emesis chemically neutralises the swallowed kerosene

(B) Vomiting risks aspiration of the volatile hydrocarbon, causing a severe chemical pneumonitis

(C) Kerosene is strongly corrosive and would burn the oesophagus on the way up

(D) Kerosene is very well absorbed from the gut and must be removed systemically

Detailed Solutions

Q1.

Solution

Concept – normal separation of the umbilical cord: After birth the cord stump dries, mummifies and then detaches as a line of demarcation forms at its base.

Step 1 – recall the timing: With clean, dry cord care the stump typically separates by about **5 to 15 days** (roughly one to two weeks) after birth.

Step 2 – the practical point: Nothing need be applied to the cord; keeping it clean and dry is enough, and delayed separation beyond three weeks may hint at a neutrophil defect such as leucocyte adhesion deficiency.

Why the other options are wrong:

- B, within 24 hours: far too early; the cord has not yet demarcated.
- C, 6 to 8 weeks: this is abnormally delayed separation.
- D, only after 3 months: grossly delayed and pathological.

Final Answer: About 5 to 15 days $\Rightarrow$ A

Answer: (A) Go Back to Q1

Q2.

Solution

Concept – prevention of vitamin K deficiency bleeding: Newborns have low vitamin K stores and immature clotting, putting them at risk of bleeding.

Step 1 – name the vitamin: A single intramuscular dose of **vitamin K** given at birth prevents haemorrhagic disease of the newborn (vitamin K deficiency bleeding).

Step 2 – why it works: Vitamin K is the cofactor for hepatic activation of clotting factors II, VII, IX and X; supplying it corrects the deficiency and prevents early, classic and late bleeding.

Why the other options are wrong:

- A, vitamin D: prevents rickets, not neonatal bleeding.
- C, vitamin C: its lack causes scurvy, not vitamin K deficiency bleeding.
- D, vitamin B12: its lack causes megaloblastic anaemia, not a bleeding tendency.

Final Answer: Vitamin K $\Rightarrow$ B

Answer: (B) Go Back to Q2

Q3.

Solution

Concept – timing of gonococcal ophthalmia neonatorum: The age at which a neonatal conjunctivitis begins is a strong clue to its cause.

Step 1 – recall the window: Gonococcal ophthalmia neonatorum is hyperacute and appears **within the first 2 to 5 days** of life, with copious purulent discharge and marked lid swelling.

Step 2 – why it matters: It is a sight-threatening emergency because *Neisseria gonorrhoeae* can penetrate an intact cornea; it needs urgent systemic ceftriaxone and saline eye lavage.

Why the other options are wrong:

- A, after 3 weeks: this is the timing of chlamydial conjunctivitis, not gonococcal.
- B, at birth only: chemical conjunctivitis from prophylaxis appears on day 1, but gonococcal disease evolves over the first few days.
- D, after 6 weeks: far too late for gonococcal ophthalmia.

Final Answer: First 2 to 5 days of life $\Rightarrow$ C

Answer: (C) Go Back to Q3

Q4.

Solution

Concept – the gross-motor milestone of sitting: Motor milestones follow a fixed cephalocaudal sequence with predictable ages.

Step 1 – read the timeline: The arrow **X** points to the 6-month mark on the age axis.

Step 2 – match the milestone: A normal infant **sits without support** at about **6 months** of age (having sat with support at about 5 months).

Why the other options are wrong:

- A, 3 months: at this age the baby only achieves steady head control.

- B, 4 months: the baby rolls and reaches but cannot yet sit unsupported.
- D, 12 months: by now the child stands and walks with support, well beyond independent sitting.

Final Answer: About 6 months $\Rightarrow$ C

Answer: (C) Go Back to Q4

Q5.

Solution

Concept – infantile beriberi and thiamine: A specific water-soluble vitamin underlies the cardiac and neurological picture of beriberi.

Step 1 – name the vitamin: Infantile beriberi is caused by deficiency of **thiamine (vitamin B1)**, classically in a breastfed infant whose mother is thiamine-deficient.

Step 2 – the mechanism: Thiamine is a cofactor for pyruvate dehydrogenase and transketolase; its lack impairs aerobic energy production, producing high-output cardiac failure (wet beriberi), a hoarse aphonic cry and neuropathy.

Why the other options are wrong:

- A, vitamin B12: its lack causes megaloblastic anaemia and neuropathy, not beriberi.
- B, vitamin C: its lack causes scurvy.
- C, vitamin D: its lack causes rickets.

Final Answer: Thiamine (vitamin B1) $\Rightarrow$ D

Answer: (D) Go Back to Q5

Q6.

Solution

Concept – MUAC as a diagnostic criterion for severe acute malnutrition: Mid-upper-arm circumference is a quick, age-independent screen in young children.

Step 1 – recall the cut-off: In a child aged 6 to 59 months, a MUAC **less than 11.5 cm** on its own diagnoses severe acute malnutrition (SAM).

Step 2 – the context: A MUAC of 11.5 to 12.5 cm indicates moderate acute malnutrition; SAM is also defined by weight-for-height below -3 SD or by bilateral pitting oedema.

Why the other options are wrong:

- A, 15.0 cm: this is a normal, reassuring arm circumference.
- B, 13.5 cm: this lies within the normal range, above the malnutrition thresholds.
- D, 9.0 cm: well below the cut-off; SAM is already diagnosed by 11.5 cm, so this is not the defining value.

Final Answer: Less than 11.5 cm $\Rightarrow$ C

Answer: (C) Go Back to Q6

Q7.

Solution

Concept – timing of the MR vaccine in the UIP schedule: Each vaccine has a fixed due age in the national programme.

Step 1 – recall MR timing: The first dose of measles-rubella (MR) vaccine is given **at 9 completed months**, with a second dose at 16 to 24 months.

Step 2 – the rationale: Maternal antibody wanes by around 9 months, so vaccinating then gives a good response while covering the child before the peak age of measles complications.

Why the other options are wrong:

- A, at birth: this is the timing of BCG, OPV-0 and hepatitis B birth dose, not MR.
- C, 6 weeks: this is when the first pentavalent, OPV, rotavirus and PCV doses are due.
- D, 5 years: around this age the second DPT booster is given, not MR.

Final Answer: At 9 completed months $\Rightarrow$ B

Answer: (B) Go Back to Q7

Q8.

Solution

Concept – the pseudomembrane of diphtheria: A tough grey membrane plus systemic toxicity from an exotoxin points to one organism.

Step 1 – name the disease: A grey, adherent pseudomembrane over the tonsils

and pharynx that bleeds on removal, with a bull-neck, is characteristic of **diphtheria** caused by *Corynebacterium diphtheriae*.

Step 2 – the mechanism: The exotoxin inhibits protein synthesis (by ADP-ribosylating elongation factor 2) and can damage the heart and nerves; treatment is prompt antitoxin plus antibiotics.

Why the other options are wrong:

- A, streptococcal pharyngitis: gives a red throat with exudate that wipes off easily, without a true adherent membrane.
- B, infectious mononucleosis: causes tonsillar exudate and lymphadenopathy but no toxin-mediated bull-neck membrane.
- C, oral candidiasis: produces white plaques that scrape off leaving a raw base, not a grey bleeding membrane.

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

Answer: (D) Go Back to Q8

Q9.

Solution

Concept – first-line reliever in childhood asthma: Asthma treatment separates quick relievers from long-term controllers.

Step 1 – name the reliever: The first-line reliever for the acute relief of asthma symptoms is an **inhaled short-acting beta-2 agonist** such as salbutamol.

Step 2 – how it works: It relaxes bronchial smooth muscle within minutes, relieving wheeze and breathlessness; regular symptom control instead relies on a low-dose inhaled corticosteroid controller.

Why the other options are wrong:

- B, oral antibiotics: asthma is not a bacterial infection, so antibiotics do not relieve an attack.
- C, montelukast alone as reliever: it is a slow-acting controller, useless for immediate relief.
- D, daily systemic steroids: reserved for severe disease, not first-line relief, and carry major side effects.

Final Answer: Inhaled short-acting beta-2 agonist $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q9

Q10.

Solution

Concept – transposition of the great arteries (TGA): Cyanosis from birth with discordant great vessels defines this lesion.

Step 1 – read the sketch: The aorta arising from the right ventricle and the pulmonary artery from the left ventricle is **transposition of the great arteries**, giving two parallel circulations.

Step 2 – the emergency management: Survival depends on mixing between the circuits, so prostaglandin E1 is infused to keep the ductus arteriosus open until a balloon atrial septostomy or arterial switch operation is done.

Why the other options are wrong:

- B, isolated VSD: an acyanotic left-to-right shunt, not cyanosis from birth.
- C, atrial septal defect: an acyanotic shunt with fixed splitting of S2.
- D, patent ductus arteriosus: an acyanotic shunt with a continuous machinery murmur.

Final Answer: Transposition of the great arteries $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q10

Q11.

Solution

Concept – managing a cyanotic (tet) spell: The first bedside step aims to raise systemic vascular resistance and cut the right-to-left shunt.

Step 1 – name the manoeuvre: Placing the child in the **knee-chest position** is the key immediate step in a tet spell.

Step 2 – why it works: Flexing the hips and knees increases systemic vascular resistance, which reduces the right-to-left shunt across the VSD and drives more blood through the lungs, improving oxygenation; oxygen, calming the child and morphine follow.

Why the other options are wrong:

- A, lay flat and supine: this does not raise systemic resistance and fails to abort the spell.
- B, intravenous diuretic: diuretics reduce preload and can worsen a spell, not relieve it.
- C, stand the child up: standing lowers systemic resistance and worsens the

right-to-left shunt.

Final Answer: Knee-chest position ⇒ D

Answer: (D) Go Back to Q11

Q12.

Solution

Concept – G6PD deficiency and oxidant haemolysis: An X-linked enzyme defect leaves red cells unable to withstand oxidant stress.

Step 1 – name the deficiency: Acute haemolysis after fava beans, with bite cells and Heinz bodies on the film, is due to deficiency of **glucose-6-phosphate dehydrogenase (G6PD)**.

Step 2 – the mechanism: G6PD generates NADPH, which keeps glutathione reduced; without it, oxidant stress (fava beans, antimalarials, sulfonamides, infection) denatures haemoglobin into Heinz bodies and the spleen bites them out, causing haemolysis.

Why the other options are wrong:

- B, spectrin defect: hereditary spherocytosis gives spherocytes and a chronic haemolysis, not fava-bean-triggered bite cells.
- C, iron deficiency: causes microcytic anaemia, not acute oxidant haemolysis.
- D, beta-globin deficiency: beta-thalassaemia gives chronic transfusion-dependent microcytic anaemia, not episodic haemolysis.

Final Answer: G6PD deficiency ⇒ A

Answer: (A) Go Back to Q12

Q13.

Solution

Concept – fragile X syndrome: A trinucleotide-repeat disorder is the commonest inherited cause of intellectual disability.

Step 1 – name the syndrome: A long face with a prominent jaw, large ears, macro-orchidism and an expanded CGG repeat defines **fragile X syndrome**, the commonest *inherited* cause of intellectual disability.

Step 2 – the mechanism: Expansion of the CGG repeat in the FMR1 gene silences it, so the FMRP protein needed for normal synaptic development is lost.

Why the other options are wrong:

- A, Down syndrome: the commonest *overall* genetic cause of intellectual disability, but it arises from sporadic trisomy, not inheritance of a repeat.
- B, Klinefelter syndrome: causes tall stature and hypogonadism with only mild learning difficulty, and no CGG repeat.
- D, Turner syndrome: causes short stature and ovarian failure in girls, usually with normal intelligence.

Final Answer: Fragile X syndrome $\Rightarrow$ C

Answer: (C) Go Back to Q13

Q14.

Solution

Concept – classifying dehydration by serum sodium: Dehydration is grouped by the measured serum sodium concentration.

Step 1 – read the scale: The arrow **X** sits at the 150 mmol/L mark, and the shaded band beyond it defines **hypernatraemic** dehydration.

Step 2 – apply the definition: Hypernatraemic dehydration is present when the serum sodium is **above 150 mmol/L**; hyponatraemic dehydration is a sodium below 130 mmol/L, and isonatraemic dehydration lies between 130 and 150.

Why the other options are wrong:

- A, below 130 mmol/L: this defines hyponatraemic dehydration, the opposite category.
- C, 135 to 145 mmol/L: this is the normal range, seen in isonatraemic dehydration.
- D, exactly 140 mmol/L: a normal value, not a hypernatraemic one.

Final Answer: Above 150 mmol/L $\Rightarrow$ B

Answer: (B) Go Back to Q14

Q15.

Solution

Concept – why emesis is avoided after hydrocarbon ingestion: Kerosene is a volatile, low-viscosity hydrocarbon, and its main danger is to the lungs.

Step 1 – state the reason: Inducing vomiting is avoided because it risks **aspiration of the hydrocarbon into the airway, causing a severe chemical (aspiration) pneumonitis**.

Step 2 – the principle: Kerosene is poorly absorbed from the gut but very damaging if it reaches the lungs; management is therefore supportive, keeping the airway safe and avoiding both emesis and gastric lavage.

Why the other options are wrong:

- A, neutralises the poison: vomiting does not neutralise kerosene.
- C, corrosive to the oesophagus: kerosene is an irritant but is not a strong corrosive like acids or alkalis; aspiration, not oesophageal burn, is the danger.
- D, well absorbed systemically: on the contrary, hydrocarbons are poorly absorbed from the gut, so systemic toxicity from ingestion is limited.

Final Answer: Vomiting risks aspiration and chemical pneumonitis $\Rightarrow$ $\boxed{\text{B}}$

Answer: (B) Go Back to Q15

Answer Key

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