GPAT Pharmaceutics

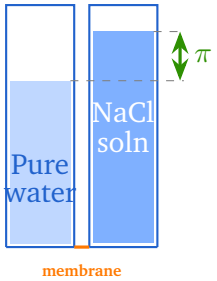
Sample Paper – 5

Duration: 36 Minutes **Maximum Marks: 100**

Instructions

- This paper contains **25** Multiple Choice Questions (Single Correct Answer), modelled on the Pharmaceutics portion of **GPAT** entrance.
- Each correct answer carries **+4 marks**. There is **a penalty of −1 mark** for each incorrect answer. Unattempted questions carry no marks.
- Only **one** option is correct per question.
- Syllabus: **GPAT Pharmaceutics — Physical Pharmacy, Physicochemical Properties, Pharmacokinetics.**
- Personal calculators, mobile phones, and electronic gadgets are strictly prohibited. A simple on-screen calculator is provided in the CBT interface; rough sheets are supplied at the centre.

Q1. The figure below shows a U-tube osmometer with a semipermeable membrane separating pure water (left arm) from a NaCl solution (right arm). A 0.9% w/v NaCl solution is equivalent to a molar concentration of 0.154 mol/L. Using the Van't Hoff equation $\pi = iMRT$ (where $i = 2$ for NaCl, $R = 0.082$ L·atm/mol·K, $T = 310$ K), calculate the osmotic pressure of this solution.



What is the osmotic pressure π of 0.9% w/v NaCl at 310 K?

(A) $\pi \approx 7.9$ atm

(B) $\pi \approx 3.9\,\text{atm}$
(C) $\pi \approx 15.8\,\text{atm}$
(D) $\pi \approx 1.3\,\text{atm}$

Q2. The partition coefficient is defined as $P = [\text{drug}]_{\text{octanol}}/[\text{drug}]_{\text{water}}$ and its logarithm $\log P$ is a measure of drug lipophilicity. A drug candidate is found to have $\log P = 2.0$.

How many times more concentrated is this drug in the octanol phase compared to the aqueous (water) phase at equilibrium?

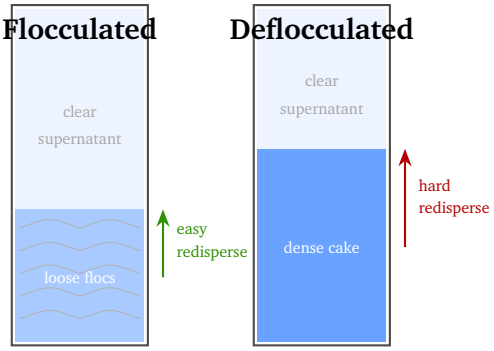
(A) $10\times$ more concentrated in octanol
(B) $100\times$ more concentrated in octanol
(C) $1000\times$ more concentrated in octanol
(D) $2\times$ more concentrated in octanol

Q3. Polymorphic systems are classified as either *enantiotropic* or *monotropic* based on the relative thermodynamic stability of the forms. In an **enantiotropic** system, both polymorphs can be the thermodynamically stable form depending on temperature.

Which statement **correctly** defines an enantiotropic polymorphic system?

(A) One polymorph is always metastable at all temperatures below the melting point; there is no reversible interconversion between forms
(B) The two polymorphs have identical solubilities and dissolution rates across all temperature ranges
(C) There exists a transition temperature below the melting points of both forms, at which the two polymorphs are in equilibrium and can reversibly interconvert
(D) The metastable form converts irreversibly to the stable form upon heating, with no accessible transition point

Q4. The diagram below compares sedimentation behaviour in flocculated (left) and deflocculated (right) pharmaceutical suspensions.



From a **patient use** perspective, which property of a **flocculated** suspension is MOST advantageous?

(A) It settles more rapidly, reducing the time the patient must wait before dosing
(B) It forms a compact, dense cake that protects the drug from degradation
(C) The packed sediment physically prevents chemical degradation of the API
(D) The loose, open flocs can be easily redispersed by gentle shaking, ensuring accurate dose delivery

Q5. The critical packing parameter (CPP) predicts the preferred self-assembled structure of amphiphiles in aqueous solution:

$$\text{CPP} = \frac{v}{a_0 \cdot l_c}$$

where v is the volume of the hydrophobic tail, a_0 is the optimal head-group area, and l_c is the critical chain length. The predicted structures are: CPP $< 1/3$ (spherical micelles); $1/3$–$1/2$ (cylindrical micelles); $1/2$–1 (bilayers/vesicles); > 1 (inverted structures).

A surfactant has a bulky polar head group relative to its tail volume, giving CPP $= 0.25$. Which structure does this surfactant preferentially form in dilute aqueous solution?

(A) Spherical micelles
(B) Cylindrical (rod-like) micelles
(C) Bilayer vesicles (liposomes)

(D) Inverted (reverse) micelles

Q6. Both the Langmuir and Freundlich adsorption isotherms describe the relationship between the amount of solute adsorbed per unit mass of adsorbent (x/m) and the equilibrium solute concentration (C) at constant temperature. The Freundlich isotherm is given by:

$$\frac{x}{m} = KC^{1/n}$$

which is linear in a $\log(x/m)$ versus $\log C$ plot.

Which feature **distinguishes** the Freundlich isotherm from the Langmuir isotherm?

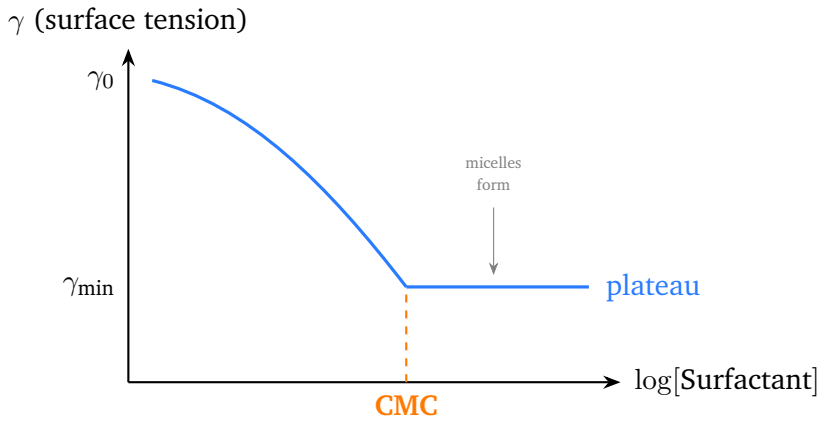
(A) The Freundlich isotherm reaches a definite saturation plateau at high solute concentrations, identical to the Langmuir maximum adsorption capacity x_m

(B) The Freundlich isotherm does **not** predict a saturation plateau; adsorption capacity continues to increase (as $x/m \propto C^{1/n}$) without an upper limit

(C) The Freundlich isotherm assumes monolayer adsorption on homogeneous sites, exactly as the Langmuir isotherm does

(D) Both isotherms are mathematically equivalent at low solute concentrations but diverge only in the linear region

Q7. The graph below shows how surface tension (γ) varies with the logarithm of surfactant concentration. Below the critical micelle concentration (CMC), surfactant monomers adsorb at the air–water interface, progressively lowering γ. At the CMC, the interface is saturated and further added surfactant forms micelles.



Based on the graph, what happens to **surface tension** when surfactant concentration is increased **above** the CMC?

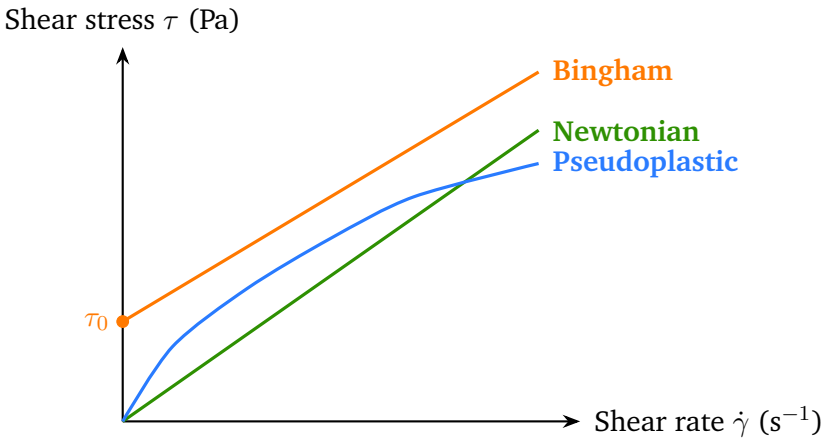
(A) Surface tension continues to decrease progressively as more micelles are formed

(B) Surface tension rises sharply above the CMC because micelles shield monomers from the interface

(C) Surface tension remains essentially constant (at γ_{min}) because the interface is already saturated; additional surfactant forms micelles instead

(D) Surface tension drops to zero above the CMC, eliminating the air–water interface

Q8. The rheogram below shows shear stress (τ, Pa) versus shear rate ($\dot{\gamma}$, s^{-1}) for three different flow behaviours.



A pharmaceutical suspension requires a minimum shear stress of 15 Pa

before it begins to flow. Above this stress, it behaves as a viscous Newtonian fluid. How is this rheological behaviour **best** described?

(A) Newtonian flow — the suspension flows immediately upon application of any non-zero shear stress
(B) Pseudoplastic (shear-thinning) — apparent viscosity decreases continuously with increasing shear rate
(C) Dilatant (shear-thickening) — apparent viscosity increases with increasing shear rate
(D) Bingham plastic — the suspension possesses a yield value ($\tau_0 = 15\,\text{Pa}$) and flows with constant plastic viscosity above this threshold

Q9. A rectal suppository formulation is required for a heat-labile pharmacological agent. The suppository must melt at body temperature ($\approx 37^\circ$C) to release the drug. Which base is **most appropriate** for this formulation?

(A) Cocoa butter (theobroma oil), which melts at 33–35°C through a polymorphic transition without chemical reaction, preserving heat-labile drugs
(B) Polyethylene glycol (PEG) 4000, which dissolves in rectal mucous secretions without requiring the drug to withstand elevated processing temperatures
(C) Glycerogelatin base, which requires preparation at 60–80°C and is therefore unsuitable for heat-labile drugs
(D) Hard fat (Witepsol H15), which melts rapidly at 34–36°C but causes oxidative degradation of heat-labile APIs

Q10. Differential Scanning Calorimetry (DSC) is used to characterise the physical form of pharmaceutical solids. When a **crystalline** drug is heated, a sharp endothermic melting peak is observed. In contrast, an **amorphous** drug exhibits a different characteristic thermal event.

In a DSC thermogram, which thermal event is **characteristic** of an amorphous drug substance?

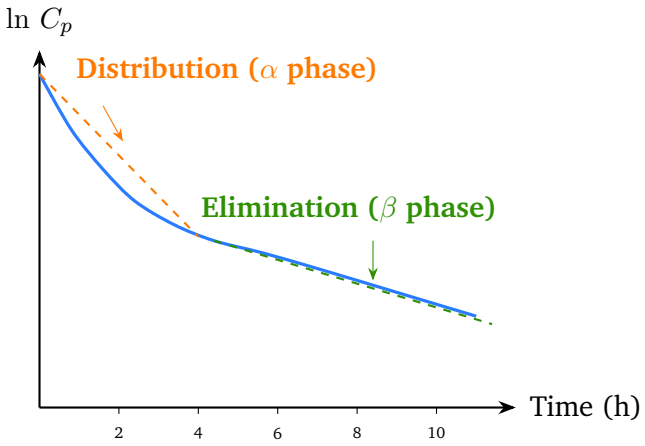
(A) A sharp, well-defined endothermic melting peak identical to the crystalline form
(B) A step change in heat flow (heat capacity) corresponding to a glass transition temperature (T_g), with no sharp melting endotherm
(C) A large exothermic peak indicating spontaneous crystallisation on heating without any prior glass transition
(D) A series of multiple sharp melting peaks representing distinct crystalline polymorphs formed during heating

Q11. X-ray powder diffraction (XRPD) is the definitive technique for identifying crystalline forms of pharmaceutical substances. Different physical states of matter produce distinctive diffraction patterns.

In an XRPD pattern, what is **characteristic** of a 100% crystalline drug substance?

(A) A broad, diffuse halo (amorphous hump) centred around $2\theta \approx 20^\circ$ with no sharp features
(B) Complete absence of any diffraction signal, confirming a non-crystalline state
(C) Sharp, intense diffraction peaks (Bragg peaks) at well-defined 2θ angles characteristic of the crystal lattice d-spacings
(D) A single broad peak whose width is inversely proportional to crystallite size according to the Scherrer equation

Q12. After intravenous bolus administration, the figure below shows a semilog plasma concentration–time profile for a drug that follows a **two-compartment** open model.



Which pharmacokinetic feature **distinguishes** a two-compartment model from a one-compartment model after IV bolus administration?

(A) Plasma concentration declines as a single monoexponential function with one rate constant k_e

(B) Clearance is time-independent and can be calculated from a single exponential slope

(C) Drug distributes instantaneously to all tissues, eliminating the need for a distribution phase

(D) Plasma concentration shows a biexponential decline ($C_p = Ae^{-\alpha t} + Be^{-\beta t}$) with an initial steep distribution phase (α) followed by a slower terminal elimination phase (β)

Q13. In non-compartmental pharmacokinetic analysis, the area under the plasma concentration–time curve (AUC) is estimated using the **linear trapezoidal rule**:

$$\text{AUC}_{[t_1 \to t_2]} = \frac{C_1 + C_2}{2} \times (t_2 - t_1)$$

In a pharmacokinetic study, the plasma concentration at $t = 2\,\text{h}$ is $C_1 = 8\,\text{mg/L}$ and at $t = 4\,\text{h}$ is $C_2 = 4\,\text{mg/L}$.

Using the linear trapezoidal rule, what is the partial AUC between $t = 2\,\text{h}$ and $t = 4\,\text{h}$?

(A) 12 mg·h/L

(B) 6 mg·h/L

(C) 24 mg·h/L

(D) 8 mg·h/L

Q14. Total body clearance (Cl) after intravenous administration is calculated using the relationship:

$$\text{Cl} = \frac{\text{Dose}_{\text{IV}}}{\text{AUC}_{0-\infty}}$$

An IV bolus dose of **400 mg** is administered to a patient, and the area under the plasma concentration–time curve extrapolated to infinity is $\text{AUC}_{0-\infty} = 800$ mg·h/L.

What is the **total body clearance** for this drug?

(A) 0.25 L/h
(B) 0.5 L/h
(C) 1.0 L/h
(D) 2.0 L/h

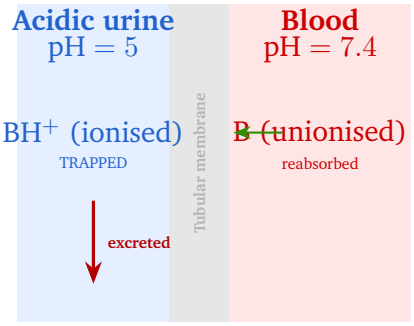
Q15. Renal clearance (Cl_R) reflects the contribution of the kidney to overall drug elimination. The glomerular filtration rate (GFR) in a healthy adult is approximately 120 mL/min.

A drug is found to have a renal clearance of $\text{Cl}_R = 400$ mL/min. Which mechanism **best explains** this observation?

(A) Passive tubular reabsorption is the dominant mechanism, which lowers Cl_R below the GFR value
(B) Glomerular filtration alone accounts for $\text{Cl}_R = 400$ mL/min; the GFR has been underestimated in this patient
(C) Active tubular secretion contributes significantly, since $\text{Cl}_R(400\,\text{mL/min}) > \text{GFR}(120\,\text{mL/min})$
(D) Protein binding reduces filterable drug fraction, paradoxically raising Cl_R above the GFR

Q16. The pH-partition theory predicts that ionised drug molecules are trapped in the renal tubular fluid (ion-trapping) and excreted, while unionised molecules are reabsorbed across the lipid tubular membrane. The diagram below illustrates how urine pH affects renal excretion of a weak

base (pKa $= 9$).



A patient is treated with ammonium chloride to **acidify the urine**. Which drug class will have **enhanced renal excretion** under these conditions?

(A) Weak acids (pK_a 3–5), which are more ionised in acidic urine and therefore excreted faster
(B) Non-ionisable drugs, since their excretion is pH-independent
(C) Strong acids with p$K_a < 1$, because they are always fully ionised regardless of urine pH
(D) Weak bases (pK_a 8–10), which become more protonated (ionised) in acidic urine and are ion-trapped, enhancing urinary excretion

Q17. Enterohepatic recirculation occurs when a drug is conjugated in the liver (e.g., glucuronidation), excreted into bile, and the conjugate is hydrolysed by intestinal bacteria to regenerate free drug, which is then reabsorbed from the gut.

Which pharmacokinetic **observation** on a plasma concentration–time curve is **most suggestive** of enterohepatic recirculation?

(A) A secondary peak (shoulder) on the plasma concentration–time curve appearing several hours after the primary absorption peak
(B) A prolonged $t_{\max}$ (time to peak concentration) with no distinct primary peak
(C) A shortened elimination half-life due to enhanced hepatic clearance
(D) A perfectly dose-proportional increase in AUC confirming linear pharmacokinetics

Q18. When a drug is administered as multiple doses at fixed intervals (τ), drug accumulates until steady state is reached. The **accumulation factor** R is defined as:

$$R = \frac{C_{\text{ss,max}}}{C_{\text{max,1}}} = \frac{1}{1 - e^{-k_e \tau}}$$

A drug has an elimination rate constant $k_e = 0.0693\,\text{h}^{-1}$ (corresponding to $t_{1/2} = 10\,\text{h}$) and is administered every $\tau = 10\,\text{h}$ (i.e., once per half-life).

What is the accumulation factor R? (*Hint*: $e^{-0.693} = 0.5$)

(A) $R = 1.5$
(B) $R = 2.0$
(C) $R = 4.0$
(D) $R = 3.0$

Q19. At steady state during multiple dosing, the maximum plasma concentration ($C_{\text{ss,max}}$) and minimum plasma concentration ($C_{\text{ss,min}}$) must both lie **within** the therapeutic window (between the minimum effective concentration, MEC, and the minimum toxic concentration, MTC).

Which factor **most directly** determines whether a multiple-dosing regimen maintains plasma drug levels within the therapeutic window between doses?

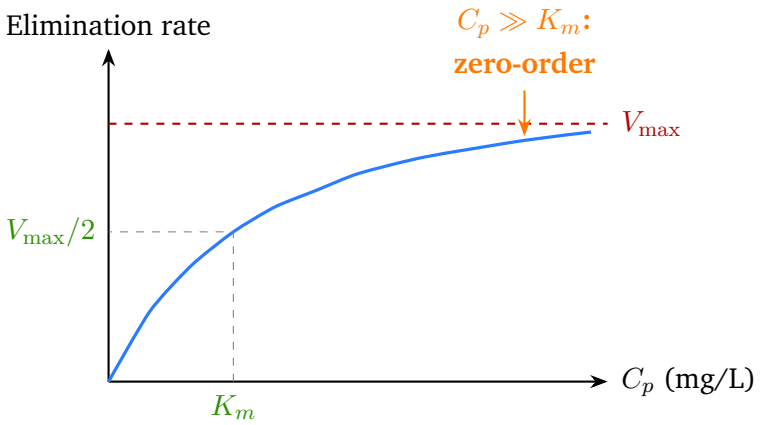
(A) The extent of plasma protein binding, which buffers free drug concentration fluctuations
(B) The route of administration, which determines whether $C_{\text{ss,max}}$ exceeds the MTC
(C) The dosing interval (τ) relative to the drug's elimination half-life, which governs the magnitude of concentration fluctuation between doses
(D) The particle size of the drug in the formulation, which controls absorption rate

Q20. Some drugs (e.g., phenytoin) exhibit Michaelis-Menten (saturable, non-

linear) pharmacokinetics, where the rate of elimination is:

$$\text{Rate} = \frac{V_{\max} \cdot C_p}{K_m + C_p}$$

The figure below shows rate of elimination versus plasma concentration C_p.



At plasma concentrations **much greater than** K_m ($C_p \gg K_m$), which statement **best describes** drug elimination?

(A) Clearance increases proportionally with plasma concentration, maintaining first-order kinetics at all concentrations

(B) The elimination half-life shortens as plasma concentration rises above K_m

(C) Elimination follows first-order kinetics, with rate proportional to plasma concentration

(D) Elimination proceeds at a constant maximal rate ($V_{\max}$), independent of further increases in plasma concentration (zero-order kinetics)

Q21. Carr's compressibility index (CI) is a simple measure of powder flowability, calculated from bulk (ρ_{bulk}) and tapped (ρ_{tapped}) densities:

$$\text{CI} = \frac{\rho_{\text{tapped}} - \rho_{\text{bulk}}}{\rho_{\text{tapped}}} \times 100$$

The flow categories are: CI $< 15\%$ (excellent); 16–20% (good); 21–25% (fair); 26–31% (poor); $> 35\%$ (very poor).

A pharmaceutical powder has $\rho_{\text{bulk}} = 0.40\,\text{g/mL}$ and $\rho_{\text{tapped}} = 0.50\,\text{g/mL}$. What is Carr's index and what **flow category** does it indicate?

(A) CI $= 20\%$, indicating **Good** powder flow
(B) CI $= 25\%$, indicating **Fair** powder flow
(C) CI $= 15\%$, indicating **Excellent** powder flow
(D) CI $= 10\%$, indicating **Excellent** powder flow

Q22. Nanoparticulate drug delivery systems can be stabilised against aggregation either by **electrostatic repulsion** (via surface charge, measured as zeta potential) or by **steric stabilisation** (via surface-grafted hydrophilic polymers such as PEG).

Which property makes **PEG-coated nanoparticles** MORE stable than electrostatically-stabilised particles in **physiological saline**?

(A) PEG coating increases the surface charge density (zeta potential) of the nanoparticle, enhancing electrostatic repulsion in saline
(B) Steric repulsion from compressed PEG chains is unaffected by ionic strength; electrostatic stabilisation is quenched in saline because counter-ions compress the electrical double layer
(C) PEG forms a crystalline shell that physically prevents particle–particle contact regardless of ionic strength
(D) PEG acts as a bioadhesive coating, anchoring particles to physiological surfaces and preventing aggregation in the bulk

Q23. Einstein's viscosity equation for dilute suspensions of hard, non-interacting spherical particles is:

$$\eta_{\text{rel}} = 1 + 2.5\,\phi$$

where $\eta_{\text{rel}} = \eta_{\text{suspension}}/\eta_{\text{medium}}$ is the relative viscosity and ϕ is the volume fraction of dispersed particles.

According to Einstein's equation, which parameter **primarily** determines the viscosity increase in a dilute hard-sphere suspension?

(A) The size (radius) of individual dispersed particles

(B) The surface charge (zeta potential) of the dispersed particles
(C) The volume fraction (ϕ) of dispersed particles
(D) The surface tension of the continuous medium

Q24. Complex coacervation is used in pharmaceutical microencapsulation (e.g., encapsulation of oily drugs). The process involves mixing two aqueous polymer solutions — gelatin (positively charged at pH below its isoelectric point) and gum arabic (negatively charged) — which undergo phase separation to form polymer-rich coacervate droplets that deposit around dispersed oil droplets.

What type of **interaction** primarily **drives** complex coacervation in the gelatin/gum arabic microencapsulation system?

(A) Hydrophobic interaction between the non-polar segments of gelatin and gum arabic
(B) Van der Waals forces between polymer chains at high polymer concentrations
(C) Hydrogen bonding between hydroxyl groups on gelatin and gum arabic backbone
(D) Electrostatic attraction between oppositely charged polyelectrolytes (cationic gelatin and anionic gum arabic)

Q25. Young's equation relates the contact angle (θ) of a liquid droplet on a solid surface to the interfacial tensions:

$$\gamma_{SV} = \gamma_{SL} + \gamma_{LV} \cos\theta$$

A water droplet placed on a solid pharmaceutical excipient surface forms a **contact angle of 130°**.

What does a contact angle of 130° indicate about the solid surface?

(A) The surface is **hydrophobic** — the liquid beads up rather than spreading ($\theta > 90°$ indicates poor wettability)
(B) The surface is completely wetted by water; the liquid spreads to a thin uniform film ($\theta = 0°$ condition)

(C) The surface is mildly hydrophilic; water partially spreads with moderate affinity for the solid

(D) The surface is amphiphilic — one region is hydrophilic and another hydrophobic, giving an average contact angle of 130°

Detailed Solutions

Q1.

Solution

Concept — Van't Hoff Osmotic Pressure: The osmotic pressure of a solution is given by the Van't Hoff equation: $\pi = iMRT$, where i is the Van't Hoff factor (number of particles per formula unit in solution), M is the molar concentration, $R = 0.082$ L·atm/mol·K, and T is the absolute temperature in Kelvin.

Step 1 — Identify all variables: NaCl is a 1:1 electrolyte that fully dissociates: NaCl $\rightarrow$ Na^+ + Cl^-, so $i = 2$.
Concentration: 0.9% w/v NaCl $= 0.9$ g per 100 mL $= 9$ g/L.
Molar mass of NaCl $= 58.5$ g/mol.
$M = 9/58.5 \approx 0.154$ mol/L.
Temperature: $T = 310$ K (physiological, 37°C).

Step 2 — Calculate π:

$$\pi = iMRT = 2 \times 0.154 \times 0.082 \times 310 \approx 7.83 \approx 7.9 \text{ atm}$$

This confirms that normal saline (0.9% NaCl) is iso-osmotic with blood ($\pi \approx 7.6$–8.0 atm at body temperature).

Why other options are wrong:

- Option A: $\pi \approx 7.9$ atm is the correctly computed value. **Correct.**
- Option B: ≈ 3.9 atm would result if $i = 1$ (non-electrolyte assumption), neglecting the dissociation of NaCl.
- Option C: ≈ 15.8 atm would require $i = 4$ or double the concentration, neither of which applies.
- Option D: ≈ 1.3 atm corresponds to the value obtained using R in SI units (8.314 J/mol·K) without converting to L·atm units.

Final Answer: $\pi = 2 \times 0.154 \times 0.082 \times 310 \approx 7.9$ atm $\Rightarrow$ A

Answer: (A) Go Back to Q1

Q2.

Solution

Concept — Partition Coefficient and Lipophilicity: The partition coefficient P is the ratio of drug concentration in the octanol phase to that in the water phase at equilibrium: $P = [\text{drug}]_{\text{oct}}/[\text{drug}]_{\text{aq}}$. The logarithmic form $\log P$ is used because P values span many orders of magnitude.

Step 1 — Convert log P to P: $\log P = 2.0$ means $P = 10^{2.0} = 100$.

Step 2 — Interpret the value: $P = 100$ means the drug is $100\times$ more concentrated in octanol than in water at equilibrium. This places the drug in the optimal lipophilicity range ($\log P = 1$–3) associated with good oral absorption, adequate membrane permeability, and reasonable aqueous solubility.

Why other options are wrong:

- Option A: $10\times$ corresponds to $\log P = 1.0$, not 2.0.
- Option B: $100\times$ corresponds to $\log P = 2.0$. **Correct.**
- Option C: $1000\times$ corresponds to $\log P = 3.0$, not 2.0.
- Option D: $2\times$ would give $\log P = \log 2 \approx 0.30$, not 2.0.

Final Answer: $P = 10^{2.0} = 100 \Rightarrow$ drug is $100\times$ more concentrated in octanol $\Rightarrow$ B

Answer: (B) Go Back to Q2

Q3.

Solution

Concept — Enantiotropic vs Monotropic Polymorphism: Polymorphic systems are governed by the Gibbs phase rule. In an **enantiotropic** system, the free energy curves of the two forms cross at a **transition temperature** T_t that lies *below* the melting points of both polymorphs. At T_t, both forms have equal free energy and are in equilibrium; the system can reversibly interconvert. In a **monotropic** system, the free energy curves never cross below the melting points, so one form is always metastable and interconversion is not thermodynamically reversible.

Step 1 — Identify the key criterion for enantiotropic behaviour: Enantiotropic $\Leftrightarrow$ accessible transition point T_t ($T_t < T_m$ for both forms) $\Leftrightarrow$ reversible solid–solid interconversion is thermodynamically possible.

Step 2 — Match to the options: Option C correctly states that a transition temperature below both melting points exists, at which the polymorphs are in equilib-

rium and can reversibly interconvert.

Why other options are wrong:

- Option A: Describes a *monotropic* system (always metastable, no reversible interconversion).
- Option B: Polymorphs have *different* solubilities and dissolution rates because they have different lattice free energies; this is incorrect for both enantiotropic and monotropic forms.
- Option C: Reversible interconversion through an accessible transition point defines enantiotropic behaviour. **Correct.**
- Option D: Irreversible conversion on heating without an accessible transition point also describes a monotropic system.

Final Answer: Enantiotropic = reversible interconversion through $T_t < T_m \Rightarrow \boxed{\text{C}}$

Answer: (C) Go Back to Q3

Q4.

Solution

Concept — Flocculated vs Deflocculated Suspensions: In a **flocculated** suspension, primary particles are held together loosely by weak interparticle forces (van der Waals, secondary minimum) forming an open, porous network (flocs). These flocs sediment relatively quickly but form a loose sediment with a high sediment volume ratio (F). Critically, the loose structure can be broken up by minimal shaking energy, ensuring the patient can easily redisperse the dose.

In a **deflocculated** suspension, individual particles settle independently and slowly, but pack tightly at the base of the container forming a hard, dense cake (caking) that is very difficult to redisperse and can lead to underdosing.

Step 1 — Evaluate from patient use perspective: The most important property for a patient is reliable **dose accuracy**, which requires complete redispersibility before each dose. Flocculated suspensions are superior in this regard because they redisperse with gentle shaking.

Step 2 — Assess the options:

- Option A: Faster sedimentation is actually a disadvantage (more frequent shaking required), not an advantage.
- Option B: Dense cake sediment is the undesirable feature of *deflocculated* suspensions, not flocculated.

- Option C: Packed sediment preventing degradation is not a recognised advantage and is pharmacokinetically irrelevant.
- Option D: Easy redispersibility of loose flocs ensures accurate dose delivery. **Correct.**

Final Answer: Easy redispersibility of loose flocs ensures accurate dosing $\Rightarrow$ $\boxed{\text{D}}$

Answer: (D) Go Back to Q4

Q5.

Solution

Concept — Critical Packing Parameter (CPP) and Aggregate Geometry: The CPP quantifies the geometric complementarity between the headgroup area and the tail volume. When the headgroup is relatively large and the tail is relatively small (inverted cone geometry), CPP $< 1/3$, which favours high positive interfacial curvature — the geometry required for **spherical micelles**.

Step 1 — Determine aggregate type from CPP = 0.25: CPP $= 0.25 < 1/3 \Rightarrow$ spherical (globular) micelles.
CPP $= 1/3$–$1/2 \Rightarrow$ cylindrical (rod-like) micelles.
CPP $= 1/2$–$1 \Rightarrow$ bilayers / vesicles.
CPP $> 1 \Rightarrow$ inverted structures (reverse micelles).

Step 2 — Confirm geometry: A bulky polar head group and small tail gives CPP $= 0.25$, well within the spherical micelle regime. The large headgroup ensures sufficient curvature to close the spherical structure with tails pointing inward.

Why other options are wrong:

- Option A: Spherical micelles $\leftrightarrow$ CPP $< 1/3$. CPP $= 0.25 < 1/3$. **Correct.**
- Option B: Cylindrical micelles require CPP $= 1/3$–$1/2$; CPP $= 0.25$ is below this range.
- Option C: Vesicles/bilayers require CPP $= 1/2$–1; CPP $= 0.25$ is far below.
- Option D: Inverted micelles require CPP > 1; the headgroup here is *larger*, not smaller, than the tail.

Final Answer: CPP $= 0.25 < 1/3 \Rightarrow$ spherical micelles $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q5

Q6.

Solution

Concept — Freundlich Adsorption Isotherm: The Freundlich isotherm $x/m = KC^{1/n}$ is an empirical equation derived from heterogeneous adsorption on sites of varying energy. Because it is a power law with no saturation term, x/m increases continuously as C increases (for $n > 1$, which is typical). It does **not** approach a plateau.

In contrast, the Langmuir isotherm $x/m = x_m KC/(1+KC)$ contains a denominator term that causes the adsorption to plateau at the monolayer maximum x_m at high concentrations. This is the defining physical difference.

Step 1 — Freundlich at high C: As $C \to \infty$: $x/m = KC^{1/n} \to \infty$ (no saturation term). The log–log plot remains linear over a wide concentration range with slope $1/n$.

Step 2 — Langmuir at high C: As $C \to \infty$: $x/m = x_m KC/(1+KC) \to x_m$ (plateaus at monolayer capacity).

Why other options are wrong:

- Option A: Reaching a saturation plateau is a *Langmuir* feature, not Freundlich.
- Option B: Freundlich does not predict a plateau; capacity increases without an upper limit. **Correct.**
- Option C: Monolayer adsorption on homogeneous sites is the *Langmuir* assumption; Freundlich assumes heterogeneous sites.
- Option D: The Freundlich isotherm diverges from Langmuir even at low C (Freundlich gives a curved log-linear plot; Langmuir gives a linear $C/(x/m)$ vs C plot).

Final Answer: Freundlich isotherm has no plateau — adsorption continues to increase $\Rightarrow$ B

Answer: (B) Go Back to Q6

Q7.

Solution

Concept — Gibbs Adsorption and Surface Tension at the CMC: The Gibbs adsorption isotherm ($\Gamma = -\frac{1}{RT}\frac{d\gamma}{d\ln c}$) describes how surfactant adsorption at the interface reduces surface tension γ. As surfactant concentration increases below the CMC, monomers progressively pack at the air–water interface, each displacing water molecules and reducing γ.

At the **CMC**, the interface is fully saturated with surfactant monomers — no additional monomers can adsorb. Any surfactant added beyond the CMC instead forms **micelles** in the bulk. Since the interfacial composition does not change, γ does not change further; it remains constant at $\gamma_{\min}$.

Step 1 — Identify what happens at the CMC: Interface saturated $\Rightarrow d\gamma/d\ln c = 0$ $\Rightarrow \gamma =$ constant.

Step 2 — Evaluate above the CMC: Excess surfactant $\rightarrow$ micelles (not interface adsorption) $\Rightarrow \gamma$ remains at $\gamma_{\min}$, as shown by the flat plateau on the γ vs $\log c$ graph.

Why other options are wrong:

- Option A: Surface tension does not continue to decrease; the interface is already saturated at the CMC.
- Option B: Surface tension does not rise; micelles form but do not displace adsorbed monomers from the interface.
- Option C: Surface tension plateaus at $\gamma_{\min}$ because the interface is saturated and further surfactant only forms micelles. **Correct.**
- Option D: Surface tension cannot drop to zero; $\gamma_{\min}$ for surfactant solutions is typically 25–40 mN/m, not zero.

Final Answer: Above CMC, interface is saturated and γ remains constant at $\gamma_{\min}$ $\Rightarrow$ C

Answer: (C) Go Back to Q7

Q8.

Solution

Concept — Bingham Plastic Rheology and Yield Value: A **Bingham plastic** is a material that behaves as a rigid body below a critical shear stress (the **yield value**, τ_0) and as a Newtonian viscous fluid above it. The constitutive equation is $\tau = \tau_0 + \eta_\infty \dot{\gamma}$ (for $\tau > \tau_0$), where η_∞ is the plastic viscosity. On a rheogram, this appears as a straight line that intercepts the stress axis at $\tau_0 > 0$.

Step 1 — Identify the defining characteristic: The suspension requires a minimum shear stress of **15 Pa** before it flows. This is the yield value $\tau_0 = 15\,\text{Pa}$. Above 15 Pa it flows with constant (Newtonian-like) viscosity. This profile matches Bingham plastic exactly.

Step 2 — Eliminate other options:

- Option A: Newtonian flow has **no yield value**; flow begins at any non-zero stress. This suspension requires 15 Pa before flowing.
- Option B: Pseudoplastic materials have no yield value and their apparent viscosity *decreases* with increasing shear rate (power law with $n < 1$).
- Option C: Dilatant materials also have no yield value; apparent viscosity *increases* with shear rate ($n > 1$).
- Option D: Bingham plastic behaviour — yield value 15 Pa, then Newtonian plastic viscosity above threshold. **Correct.**

Final Answer: Yield value $\tau_0 = 15\,\text{Pa}$ with linear flow above threshold = Bingham plastic $\Rightarrow$ $\boxed{\text{D}}$

Answer: (D) Go Back to Q8

Q9.

Solution

Concept — Suppository Base Selection for Heat-Labile Drugs: Suppository bases are classified as: (1) **fatty bases** (cocoa butter, hard fat/Witepsol, palm kernel oil) that *melt* at body temperature and release drug into rectal mucosa; and (2) **water-miscible bases** (PEG, glycerogelatin) that *dissolve* in mucous secretions.

For **rectal suppositories** containing heat-labile drugs, **cocoa butter** (theobroma oil) is the traditional choice. It melts at 33–35°C via a polymorphic transition ($\beta \rightarrow \alpha/\beta'$ forms melt at body temperature), releasing drug without any chemical reaction. The melting process itself is at low temperature (physiological), which avoids thermally degrading the heat-labile API.

Step 1 — Why cocoa butter suits heat-labile drugs: Cocoa butter melts within 15 minutes at rectal temperature (37°C). The drug does not experience temperatures above 37°C during use. However, *preparation* of cocoa butter suppositories requires the base to be melted (gently, at ∼34–36°C) and quickly cast, avoiding overheating, so the heat-labile API is incorporated just before pouring at the lowest possible temperature.

Why other options are wrong:

- Option A: Cocoa butter melts at body temperature without chemical reaction, making it suitable for rectal use with heat-labile drugs. **Correct.**
- Option B: PEG 4000 does not melt but dissolves in rectal fluid; it is suitable for water-soluble drugs but is preferred for vaginal routes, not rectal for drugs requiring local mucosal contact.
- Option C: Glycerogelatin requires preparation at 60–80°C, which would degrade a heat-labile drug during manufacturing.
- Option D: Witepsol H15 also melts at 34–36°C (similar to cocoa butter) and is a valid alternative; the claim that it causes oxidative degradation is not generally correct for Witepsol bases; however, cocoa butter is the classic pharmacopoeial answer for this context.

Final Answer: Cocoa butter melts at ≈ 35°C via polymorphic transition without chemical reaction $\Rightarrow$ A

Answer: (A) Go Back to Q9

Q10.

Solution

Concept — DSC Characterisation of Amorphous Materials (Glass Transition): In DSC, the thermal events observed depend on the physical state of the sample:

- **Crystalline drug**: well-defined, sharp *endothermic* melting peak at the melting point T_m.
- **Amorphous drug**: no long-range order, so no melting peak; instead, at the glass transition temperature (T_g), the material transitions from a glassy (rigid) to a rubbery (mobile) state. In DSC, this appears as a *step change in heat flow* (step in the baseline) corresponding to an increase in heat capacity ΔC_p — NOT a peak.

Step 1 — Physical basis of T_g: Below T_g: amorphous solid is "frozen" in a kinetically trapped state (glass); molecular mobility is very restricted. At T_g: cooperative

segmental motion is activated; heat capacity increases by $\Delta C_p \approx 0.1$–$0.5\,\text{J/g·K}$. This manifests as a step (not peak) in the DSC trace.

Step 2 — Identify the correct signature: A step change in heat capacity (baseline shift) at T_g is the hallmark of an amorphous drug on DSC. An additional exothermic recrystallisation peak may appear above T_g if the amorphous material crystallises on heating (T_{cr}).

Why other options are wrong:

- Option A: A sharp endothermic melting peak is characteristic of a *crystalline*, not amorphous, form.
- Option B: Step change in heat capacity at T_g is the characteristic signature of the amorphous (glassy) state. **Correct.**
- Option C: Spontaneous crystallisation without a T_g step would imply the material was crystalline to begin with; an amorphous drug always shows T_g first.
- Option D: Multiple sharp melting peaks indicate multiple crystalline polymorphs, not an amorphous form.

Final Answer: Amorphous drug $\Rightarrow$ glass transition (T_g) as a step change in heat capacity $\Rightarrow$ B

Answer: (B) Go Back to Q10

Q11.

Solution

Concept — XRPD Patterns and Crystallinity: X-ray powder diffraction exploits constructive interference of X-rays diffracted from ordered crystal planes, described by Bragg's law: $n\lambda = 2d\sin\theta$. Each set of parallel planes with spacing d generates a sharp peak at the corresponding 2θ angle. The resulting pattern is a unique "fingerprint" of the crystal structure.

Relationship between physical state and XRPD pattern:

- **100% crystalline**: sharp, intense peaks (Bragg peaks) at well-defined 2θ positions with narrow peak widths (broad peaks indicate small crystallites).
- **Amorphous**: no long-range order $\Rightarrow$ no Bragg peaks; instead a broad, diffuse "halo" (amorphous hump) centred around $2\theta \approx 15$–$25°$.
- **Partially crystalline**: combination of sharp peaks superimposed on a broad amorphous background.

Why other options are wrong:

- Option A: A broad amorphous halo is characteristic of an *amorphous* solid, not a crystalline one.
- Option B: Complete absence of diffraction indicates an amorphous or completely disordered material, not crystalline.
- Option C: Sharp, intense Bragg peaks at specific 2θ values are the definitive XRPD signature of 100% crystalline material. **Correct.**
- Option D: A single broad peak described by the Scherrer equation is characteristic of nanocrystalline or partially amorphous material, not a bulk 100% crystalline substance.

Final Answer: 100% crystalline material $\Rightarrow$ sharp Bragg peaks at specific 2θ angles $\Rightarrow$ C

Answer: (C) Go Back to Q11

Q12.

Solution

Concept — Two-Compartment Pharmacokinetic Model: In a **one-compartment** model, the body is treated as a single homogeneous compartment. After IV bolus, plasma concentration declines *monoexponentially*: $C_p = C_0 e^{-k_e t}$ (one straight line on a semilog plot).

In a **two-compartment** model, the body is divided into a central compartment (plasma + highly perfused tissues) and a peripheral compartment (poorly perfused tissues). After IV bolus, drug distributes between compartments while simultaneously being eliminated. The plasma concentration–time profile follows a **biexponential** equation:

$$C_p(t) = A\,e^{-\alpha t} + B\,e^{-\beta t}$$

where $\alpha > \beta$. On a semilog plot, this generates a **biphasic curve**: an initial steep slope (distribution phase, rate constant α) due to rapid tissue uptake, followed by a shallower terminal slope (elimination phase, rate constant β) after equilibration.

Step 1 — Identify the discriminating feature: The biexponential decline (two distinct slopes on a semilog C_p–t plot) is the hallmark of the two-compartment model. The distribution phase exists because the peripheral compartment acts as a drug reservoir.

Why other options are wrong:

- Option A: Monoexponential decline is the feature of a *one-compartment* model, not two-compartment.
- Option B: Time-independent clearance is a feature of *linear* PK regardless of compartment number; it does not distinguish the two models.
- Option C: Instantaneous distribution to all tissues is the assumption of the one-compartment model; the two-compartment model explicitly includes a *finite* distribution phase.
- Option D: Biexponential decline with distinct distribution (α) and elimination (β) phases defines the two-compartment model. **Correct.**

Final Answer: Biexponential $C_p = Ae^{-\alpha t} + Be^{-\beta t}$ with two slopes = two-compartment model $\Rightarrow$ D

Answer: (D) Go Back to Q12

Q13.

Solution

Concept — Linear Trapezoidal AUC Calculation: The linear trapezoidal rule estimates the area under a concentration–time curve between two adjacent data points as the area of a trapezoid:

$$\text{AUC}_{[t_1 \to t_2]} = \frac{C_1 + C_2}{2} \times (t_2 - t_1)$$

This method is accurate when the time points are closely spaced; for the rising portion of a profile, the log-trapezoidal method is sometimes preferred, but the linear method is specified here.

Step 1 — Identify the given values: $C_1 = 8\,\text{mg/L}$ at $t_1 = 2\,\text{h}$; $C_2 = 4\,\text{mg/L}$ at $t_2 = 4\,\text{h}$.
Time interval $= t_2 - t_1 = 4 - 2 = 2\,\text{h}$.

Step 2 — Apply the trapezoidal rule:

$$\text{AUC}_{[2 \to 4]} = \frac{8 + 4}{2} \times 2 = \frac{12}{2} \times 2 = 6 \times 2 = 12\,\text{mg} \cdot \text{h/L}$$

Why other options are wrong:

- Option A: $12\,\text{mg} \cdot \text{h/L}$ — correct trapezoidal calculation. **Correct.**
- Option B: $6\,\text{mg} \cdot \text{h/L}$ results from forgetting to multiply by the time interval, i.e., taking only $(C_1 + C_2)/2 = 6$ without the $\Delta t = 2$ factor.
- Option C: $24\,\text{mg} \cdot \text{h/L}$ would arise from using $C_1 \times \Delta t$ alone ($8 \times 3 = 24$, an

error in calculation).
- Option D: $8\,\text{mg}\cdot\text{h/L}$ is not the trapezoidal result for these values.

Final Answer: $\text{AUC}_{[2\to4]} = (8+4)/2 \times 2 = 12\,\text{mg}\cdot\text{h/L} \Rightarrow \boxed{\text{A}}$

Answer: (A) Go Back to Q13

Q14.

Solution

Concept — Total Body Clearance from IV AUC: Total body (systemic) clearance is the volume of plasma from which drug is completely removed per unit time. After IV bolus administration, it is calculated directly from:

$$\text{Cl} = \frac{\text{Dose}_{\text{IV}}}{\text{AUC}_{0-\infty}}$$

This relationship holds because for IV dosing, 100% of the dose enters the systemic circulation (bioavailability $F = 1$).

Step 1 — Identify given values: $\text{Dose}_{\text{IV}} = 400\,\text{mg}$; $\text{AUC}_{0-\infty} = 800\,\text{mg}\cdot\text{h/L}$.

Step 2 — Calculate clearance:

$$\text{Cl} = \frac{400\,\text{mg}}{800\,\text{mg}\cdot\text{h/L}} = 0.5\,\text{L/h}$$

Why other options are wrong:

- Option A: 0.25 L/h would result from $\text{Dose}/\text{AUC} = 200/800$, using half the actual dose.
- Option B: 0.5 L/h $= 400/800$. **Correct.**
- Option C: 1.0 L/h would result from $\text{Dose}/\text{AUC} = 400/400$, using half the actual AUC.
- Option D: 2.0 L/h would result from $\text{Dose}/\text{AUC} = 400/200$, which inverts the calculation.

Final Answer: $\text{Cl} = 400/800 = 0.5\,\text{L/h} \Rightarrow \boxed{\text{B}}$

Answer: (B) Go Back to Q14

Q15.

Solution

Concept — Renal Clearance Mechanisms: Total renal clearance reflects three processes:

$$\text{Cl}_R = \text{Cl}_{\text{filtration}} + \text{Cl}_{\text{secretion}} - \text{Cl}_{\text{reabsorption}}$$

Glomerular filtration (GFR ≈ 120 mL/min) is the baseline. Active tubular **secretion** adds to clearance (can exceed GFR for drugs that are actively pumped into the tubular lumen). Active or passive tubular **reabsorption** reduces clearance below GFR (drug is retrieved from the tubule back into blood).

Step 1 — Compare Cl_R with GFR: $\text{Cl}_R = 400$ mL/min; GFR $= 120$ mL/min.
$\text{Cl}_R \gg$ GFR $\Rightarrow$ filtration alone cannot account for this clearance. Active tubular secretion must contribute the additional $400 - 120 = 280$ mL/min.

Step 2 — Confirm mechanism: Drugs with $\text{Cl}_R >$ GFR include para-aminohippuric acid (PAH, classic secretion marker), penicillin G, and certain diuretics. The organic anion (OAT) and cation (OCT) transporters in the proximal tubule actively secrete these drugs into the lumen.

Why other options are wrong:

- Option A: Reabsorption reduces Cl_R below GFR, not above it.
- Option B: No individual patient has a GFR of 400 mL/min; GFR is physiologically capped at ~120–180 mL/min; the data require a secretory component.
- Option C: Active tubular secretion is the only mechanism that can raise Cl_R above GFR. **Correct.**
- Option D: Protein binding *reduces* the filterable fraction, which would *lower* Cl_R below GFR, not raise it.

Final Answer: $\text{Cl}_R = 400 >$ GFR $= 120$ mL/min $\Rightarrow$ active tubular secretion $\Rightarrow$ C

Answer: (C) Go Back to Q15

Q16.

Solution

Concept — Urine pH, Ion-Trapping, and Renal Excretion: The pH-partition theory predicts that only the **unionised** form of a drug can passively cross lipid membranes (e.g., the tubular epithelium). In the renal tubule, ionised drug molecules are *trapped* in the tubular fluid (ion-trapping) and excreted, while unionised drug is reabsorbed back into the blood.

For a **weak base** (pK_a = 8–10), lowering urine pH increases the ionised fraction (protonated cation, BH^+) in the tubular fluid ⇒ less reabsorption ⇒ **enhanced urinary excretion**.

For a **weak acid** (pK_a = 3–5), alkalinising urine increases the ionised fraction (anion, A^-) ⇒ enhanced excretion of weak acids in alkaline urine.

Step 1 — Effect of ammonium chloride (acidifying agent): NH_4Cl acidifies urine (pH ↓). In acidic urine, weak bases are more protonated (ionised) ⇒ ion-trapped ⇒ reduced tubular reabsorption ⇒ enhanced renal excretion of weak bases.

Why other options are wrong:

- Option A: Weak acids are *less* ionised in acidic urine (pK_a 3–5 vs urine pH < 5), so they are MORE reabsorbed (not more excreted) in acidic urine. Alkalinisation (e.g., $NaHCO_3$) enhances weak acid excretion.
- Option B: Non-ionisable drugs have no pH-dependent excretion.
- Option C: Strong acids are always fully ionised; their excretion is pH-independent.
- Option D: Weak bases (pK_a 8–10) are more ionised in acidic urine (pH 5 $\ll$ pK_a 8), leading to ion-trapping and enhanced excretion. **Correct.**

Final Answer: Acidic urine ⇒ weak bases more ionised ⇒ ion-trapped ⇒ enhanced excretion ⇒ D

Answer: (D) Go Back to Q16

Q17.

Solution

Concept — Enterohepatic Recirculation: Enterohepatic recirculation (EHC) is a cyclic process in which: (1) drug is conjugated in the liver (e.g., glucuronidation, sulfation), (2) conjugates are excreted into bile and stored in the gallbladder, (3) on meal-stimulated gallbladder contraction, conjugates enter the duodenum, (4) intestinal bacterial β-glucuronidases hydrolyse conjugates to regenerate free drug, (5) free drug is reabsorbed from the intestine, producing a secondary rise in plasma drug concentration.

Step 1 — Identify the pharmacokinetic signature: On a plasma C_p–time curve, EHC produces a characteristic **secondary peak** (or "shoulder") typically 4–8 h after the primary absorption peak, corresponding to the cycle of biliary excretion and intestinal reabsorption.

Step 2 — Compare with other options: Prolonged t_{max} (Option B) can occur with slow absorption but does not give a secondary peak. Shortened $t_{1/2}$ (Option C) is the opposite of what EHC causes (EHC prolongs apparent $t_{1/2}$ by recycling drug). Dose-proportional AUC (Option D) reflects linear PK but is not specific to EHC.

Why other options are wrong:

- Option A: A secondary concentration peak (hump/shoulder) hours after the primary peak is the classic pharmacokinetic indicator of EHC. **Correct.**
- Option B: Prolonged t_{max} reflects delayed absorption, not recirculation; there is no secondary peak.
- Option C: EHC prolongs apparent half-life (by recycling drug back into plasma), not shortens it.
- Option D: Dose-proportional AUC is a feature of linear PK; it does not specifically indicate EHC.

Final Answer: Secondary plasma peak hours after primary absorption peak = hallmark of enterohepatic recirculation $\Rightarrow$ A

Answer: (A) Go Back to Q17

Q18.

Solution

Concept — Multiple-Dose Accumulation Factor: When a drug with first-order elimination is administered as multiple equal doses at fixed intervals τ, the ratio of the steady-state peak concentration to the first-dose peak concentration is the **accumulation factor**:

$$R = \frac{C_{ss,max}}{C_{max,1}} = \frac{1}{1 - e^{-k_e \tau}}$$

Step 1 — Compute $k_e\tau$: $k_e = 0.0693\,\text{h}^{-1}$; $\tau = 10\,\text{h}$; $k_e\tau = 0.0693 \times 10 = 0.693 = \ln 2$.
Therefore $e^{-k_e\tau} = e^{-\ln 2} = 0.5$.

Step 2 — Compute R:

$$R = \frac{1}{1 - e^{-k_e \tau}} = \frac{1}{1 - 0.5} = \frac{1}{0.5} = 2.0$$

Dosing at exactly one half-life interval means drug always accumulates to twice the first-dose peak. At steady state, $C_{ss,max} = 2 \times C_{max,1}$.

Why other options are wrong:

- Option A: $R = 1.5$ would require $e^{-k_e\tau} = 1/3$, corresponding to $\tau \approx 1.58 \times t_{1/2}$, not $\tau = t_{1/2}$.
- Option B: $R = 2.0$ follows directly from dosing at one half-life. **Correct.**
- Option C: $R = 4.0$ would require $e^{-k_e\tau} = 0.75$, i.e., $\tau \ll t_{1/2}$.
- Option D: $R = 3.0$ would require $e^{-k_e\tau} = 2/3$, which is inconsistent with $\tau = t_{1/2}$.

Final Answer: $R = 1/(1 - e^{-0.693}) = 1/(1 - 0.5) = 2.0 \Rightarrow \boxed{\text{B}}$

Answer: (B) Go Back to Q18

Q19.

Solution

Concept — Dosing Interval and Steady-State Therapeutic Window: During multiple dosing at steady state, plasma concentration oscillates between $C_{ss,max}$ (immediately after each dose) and $C_{ss,min}$ (just before the next dose). The amplitude of this oscillation (the peak-to-trough ratio) is:

$$\frac{C_{ss,max}}{C_{ss,min}} = e^{k_e\tau}$$

This ratio increases as the dosing interval τ increases relative to the elimination half-life $t_{1/2}$. For the regimen to keep **both** $C_{ss,max}$ and $C_{ss,min}$ within the therapeutic window simultaneously, the ratio $\tau/t_{1/2}$ must be chosen carefully.

Step 1 — Identify the key determinant: A longer τ relative to $t_{1/2} \Rightarrow$ larger fluctuation; $C_{ss,min}$ may fall below MEC (under-dosing) while $C_{ss,max}$ may exceed MTC (toxicity). A shorter $\tau \Rightarrow$ smaller fluctuation but higher mean steady-state level. Choosing τ appropriately (typically $\tau \approx t_{1/2}$ for a 2-fold fluctuation) maintains levels within the therapeutic window.

Why other options are wrong:

- Option A: Protein binding buffers free drug to some extent but does not *primarily* determine whether trough levels stay above MEC; dosing interval is the dominant factor.
- Option B: Route of administration determines rate and extent of absorption (affecting C_{max}) but not whether trough levels stay within the window.
- Option C: Dosing interval τ relative to $t_{1/2}$ directly governs concentration fluctuation between doses. **Correct.**

- Option D: Particle size affects absorption kinetics but not the steady-state fluctuation pattern between doses.

Final Answer: Dosing interval (τ) relative to $t_{1/2}$ governs trough-to-peak fluctuation at steady state $\Rightarrow$ C

Answer: (C) Go Back to Q19

Q20.

Solution

Concept — Michaelis-Menten (Saturable) Pharmacokinetics at High C_p: The Michaelis-Menten elimination rate is: Rate $= V_{\max}C_p/(K_m + C_p)$.

At plasma concentrations **much greater than** K_m ($C_p \gg K_m$): $(K_m + C_p) \approx C_p$, so:

$$\text{Rate} = \frac{V_{\max}C_p}{K_m + C_p} \approx \frac{V_{\max}C_p}{C_p} = V_{\max}$$

The elimination rate equals $V_{\max}$ and is **independent of** C_p — this is **zero-order** (saturation) kinetics. Doubling the dose does not double the rate of elimination; instead, the metabolic enzymes are saturated and elimination proceeds at a fixed maximum rate.

Clinical importance (phenytoin example): Because elimination is zero-order at therapeutic concentrations near saturation, small dose increases produce disproportionately large rises in steady-state plasma levels. This narrow therapeutic index makes TDM (therapeutic drug monitoring) essential.

Why other options are wrong:

- Option A: Clearance does *not* increase with C_p in saturable kinetics; it actually *decreases* as concentration rises towards saturation.
- Option B: Half-life is not a constant concept in non-linear kinetics; apparent $t_{1/2}$ *increases* (not shortens) at high concentrations because the zero-order rate constant is less efficient at clearing high drug levels.
- Option C: First-order kinetics apply when $C_p \ll K_m$ (low concentration limit), not when $C_p \gg K_m$.
- Option D: At $C_p \gg K_m$, elimination is zero-order at rate $V_{\max}$, independent of concentration. **Correct.**

Final Answer: $C_p \gg K_m \Rightarrow$ rate $\approx V_{\max}$ (constant, zero-order) $\Rightarrow$ D

Answer: (D) Go Back to Q20

Q21.

Solution

Concept — Carr's Compressibility Index and Powder Flow: Carr's index (CI) quantifies the compressibility of a powder, which correlates with its interparticulate cohesion and hence flowability:

$$\text{CI} = \frac{\rho_{\text{tapped}} - \rho_{\text{bulk}}}{\rho_{\text{tapped}}} \times 100$$

A higher CI means more consolidation on tapping, implying greater cohesion and *poorer* flow.

Step 1 — Calculate CI: $\rho_{\text{bulk}} = 0.40\,\text{g/mL}$; $\rho_{\text{tapped}} = 0.50\,\text{g/mL}$.

$$\text{CI} = \frac{0.50 - 0.40}{0.50} \times 100 = \frac{0.10}{0.50} \times 100 = 20\%$$

Step 2 — Assign flow category: CI $= 20\%$ falls in the range 16–20% $\Rightarrow$ **Good** powder flow.

Why other options are wrong:

- Option A: CI $= 20\%$ (Good flow) is the correct calculated value. **Correct.**
- Option B: CI $= 25\%$ would arise from $(\rho_t - \rho_b)/\rho_t \times 100 = 25$, requiring e.g. $\rho_b = 0.375\,\text{g/mL}$ and $\rho_t = 0.50\,\text{g/mL}$; this is not consistent with the data given.
- Option C: CI $= 15\%$ would require $\rho_b = 0.425\,\text{g/mL}$, not 0.40 g/mL.
- Option D: CI $= 10\%$ would require $\rho_b = 0.45\,\text{g/mL}$, not 0.40 g/mL.

Final Answer: CI $= (0.50 - 0.40)/0.50 \times 100 = 20\% \Rightarrow$ Good flow $\Rightarrow \boxed{\text{A}}$

Answer: (A) Go Back to Q21

Q22.

Solution

Concept — Steric Stabilisation vs Electrostatic Stabilisation: Electrostatic stabilisation (DLVO theory) depends on the electrical double layer surrounding charged nanoparticles creating repulsive forces. However, in physiological saline (NaCl $\approx 150\,\text{mmol/L}$, ionic strength $\approx 0.15\,\text{M}$), counter-ions compress the electrical double layer (Debye length κ^{-1} shrinks), reducing electrostatic repulsion and making electrostatically-stabilised particles prone to aggregation.

Steric stabilisation by PEG chains works differently: when two PEG-coated par-

ticles approach, the compressed PEG brush generates an *osmotic* repulsive force (due to increased local polymer concentration) and an elastic/entropic repulsion. These steric forces are **independent of ionic strength** — they are not affected by salt concentration. Therefore, PEG-coated nanoparticles remain stable in physiological saline while electrostatically-stabilised particles may flocculate.

Why other options are wrong:

- Option A: PEG does not significantly increase surface charge; its stabilisation mechanism is steric, not electrostatic.
- Option B: Steric repulsion from compressed PEG chains is salt-independent; electrostatic repulsion is quenched by saline. **Correct.**
- Option C: PEG forms a hydrated brush layer, not a crystalline shell.
- Option D: Bioadhesion is a property of some polymers (e.g., chitosan, hyaluronic acid) but not PEG; PEGylation is anti-adhesive (anti-fouling).

Final Answer: Steric PEG repulsion is ionic-strength-independent; electrostatic repulsion is quenched by saline $\Rightarrow$ B

Answer: (B) Go Back to Q22

Q23.

Solution

Concept — Einstein Viscosity Equation for Dilute Suspensions: Einstein (1906) derived the viscosity of a dilute suspension of rigid, uncharged, non-interacting spheres:

$$\eta_{\text{rel}} = \frac{\eta_{\text{suspension}}}{\eta_{\text{medium}}} = 1 + 2.5\,\phi$$

where ϕ is the **volume fraction** (dimensionless) of dispersed spheres.

Key insight from the equation: The viscosity increase ($\eta_{\text{rel}} - 1 = 2.5\phi$) depends *only* on the volume fraction ϕ of the dispersed particles — not on their size, charge, or surface chemistry (in the dilute hard-sphere limit). Doubling ϕ doubles the viscosity increment, regardless of whether this is achieved with large or small particles.

Numerical example: At $\phi = 0.01$ (1% v/v): $\eta_{\text{rel}} = 1 + 2.5 \times 0.01 = 1.025$ (2.5% increase).
At $\phi = 0.10$ (10% v/v): $\eta_{\text{rel}} = 1 + 2.5 \times 0.10 = 1.25$ (25% increase).

Why other options are wrong:

- Option A: Particle radius does not appear in Einstein's equation; viscosity is

size-independent for hard spheres at constant ϕ.

- Option B: Zeta potential (surface charge) does not appear; Einstein's equation is for uncharged hard spheres.
- Option C: Volume fraction ϕ is the sole structural parameter in the equation. **Correct.**
- Option D: Surface tension of the medium is relevant to emulsions/wetting, not to suspension viscosity in Einstein's framework.

Final Answer: $\eta_{\text{rel}} = 1 + 2.5\phi \Rightarrow$ volume fraction ϕ solely determines viscosity increase $\Rightarrow$ C

Answer: (C) Go Back to Q23

Q24.

Solution

Concept — Complex Coacervation and Electrostatic Driving Force: Complex coacervation is a liquid–liquid phase separation triggered by mixing two aqueous polyelectrolyte solutions of **opposite charge**. The process for gelatin/gum arabic microencapsulation proceeds as follows:

(a) Gelatin is positively charged at pH $<$ pI ($\approx$ pH 9 for gelatin Type A; pH ≈ 5 for gelatin Type B); below pI, amino groups ($-NH_3^+$) are protonated.
(b) Gum arabic carries negatively charged carboxylate ($-COO^-$) and sulfate groups at normal processing pH.
(c) When the two solutions are mixed at the optimal pH, **electrostatic attraction** between the oppositely charged macromolecules drives their interaction and phase separation into a polymer-rich (coacervate) phase.
(d) The coacervate droplets deposit around dispersed oil droplets, forming the microcapsule shell.

Why other options are wrong:

- Option A: Hydrophobic interactions play a minor supporting role but are NOT the primary driving force; complex coacervation requires charged (not hydrophobic) polymers.
- Option B: Van der Waals forces are too weak and non-specific to drive the sharp phase separation seen in coacervation.
- Option C: Hydrogen bonding contributes to network stability but is not the *primary* driver; electrostatic forces are orders of magnitude stronger at polyelectrolyte interfaces.

- Option D: Electrostatic attraction between cationic gelatin and anionic gum arabic is the primary driving force for complex coacervation. **Correct.**

Final Answer: Electrostatic attraction between oppositely charged polyelectrolytes drives complex coacervation $\Rightarrow$ D

Answer: (D) Go Back to Q24

Q25.

Solution

Concept — Contact Angle and Surface Wettability: Young's equation, $\gamma_{SV} = \gamma_{SL} + \gamma_{LV}\cos\theta$, relates the contact angle θ of a liquid droplet on a flat solid surface to the three interfacial tensions (solid–vapour, solid–liquid, liquid–vapour).

Interpretation of contact angle values:

- $\theta = 0°$: complete wetting (liquid spreads to thin film); highly hydrophilic surface.
- $\theta < 90°$: partial wetting; hydrophilic surface; $\cos\theta > 0$.
- $\theta > 90°$: poor wetting; hydrophobic surface; liquid beads up; $\cos\theta < 0$.
- $\theta \to 180°$: superhydrophobic; nearly spherical droplet.

Step 1 — Evaluate $\theta = 130°$: $130° > 90° \Rightarrow$ hydrophobic surface. $\cos 130° \approx -0.64 < 0$. The solid–vapour interfacial tension is much lower than the sum of solid–liquid and liquid–vapour tensions; water (a polar liquid) does not wet the surface and beads up.

Pharmaceutical relevance: Contact angle $> 90°$ for tablet excipients (e.g., hydrophobic lubricants like magnesium stearate or stearic acid) indicates poor wettability, which can retard tablet disintegration and drug dissolution. Wetting agents (surfactants) are added to reduce θ and improve dissolution.

Why other options are wrong:

- Option A: $\theta = 130° > 90°$ indicates a hydrophobic, poorly wetted surface. **Correct.**
- Option B: Complete wetting ($\theta = 0°$) is the opposite condition; $130°$ is far from zero.
- Option C: Mildly hydrophilic surfaces have $\theta \approx 30$–$60°$; $130°$ is clearly hydrophobic.
- Option D: A single contact angle value does not indicate amphiphilicity; amphiphilic character is an intrinsic molecular property, not a macroscopic surface angle measurement.

Final Answer: $\theta = 130^\circ > 90^\circ \Rightarrow$ hydrophobic surface, poor wettability $\Rightarrow$ A

Answer: (A) Go Back to Q25

Answer Key

Q	Ans	Q	Ans	Q	Ans	Q	Ans	Q	Ans
1	A	2	B	3	C	4	D	5	A
6	B	7	C	8	D	9	A	10	B
11	C	12	D	13	A	14	B	15	C
16	D	17	A	18	B	19	C	20	D
21	A	22	B	23	C	24	D	25	A