Form & Function — Physiology

IB HL Study Guide

Contents

Topics Covered in This Guide

- **A4.1 Gas Exchange** — surfaces, alveoli, ventilation, Fick's law, gills, stomata
- **A4.2 Transport Systems** — blood composition, haemoglobin, heart structure, cardiac cycle, blood vessels, xylem and phloem
- **A4.3 Defense Against Disease** — innate and adaptive immunity, antibody structure, vaccination, ABO blood types
- **MCQ Practice** — styled like real IB Paper 1 questions
- **Exam Alerts** — the exact traps that cost marks in A4 questions

Aligned to IB Biology 2025 syllabus — A4.1 Gas Exchange — A4.2 Transport Systems — A4.3 Defense Against Disease

Jump to section: Gas Exchange · Transport Systems · Defense Against Disease · MCQ Practice

Section 1: Gas Exchange (A4.1)

Features of a Good Gas Exchange Surface

For efficient diffusion, a gas exchange surface must possess four key properties. These properties can be linked directly to Fick's law (see below).

MEMORISE THIS

The four features of an efficient gas exchange surface:

1. **Large surface area** — more membrane available for diffusion simultaneously
2. **Thin** — short diffusion distance minimises time for molecules to cross
3. **Moist** — gases dissolve before crossing; maintains integrity of epithelium
4. **Good blood (or fluid) supply** — maintains a steep concentration gradient by removing O_2 and delivering CO_2 continuously

Mnemonic: "Large Thin Moist Blood" — LTMB

Fick's Law of Diffusion

The rate of diffusion across a gas exchange surface is described by Fick's law:

$$\text{Rate of diffusion} \propto \frac{\text{Surface area} \times \text{Concentration gradient}}{\text{Diffusion distance}}$$

Every structural feature of an alveolus can be explained in terms of how it affects one of these three variables.

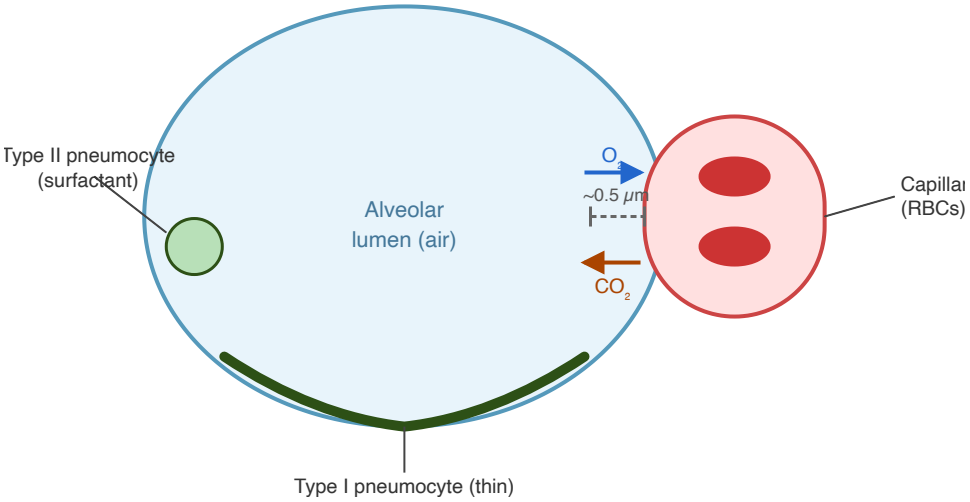
IB TIP

IB Language: Exam questions often ask you to "explain how a named feature of an alveolus increases the rate of gas exchange." Always link the structural feature to the specific variable in Fick's law it affects. For example: "Type I pneumocytes are extremely thin, which minimises the diffusion distance and therefore maximises the rate of gas exchange."

Alveoli Structure

The alveolus is the functional gas exchange unit of the mammalian lung. Each lung contains approximately 300–500 million alveoli, providing an enormous collective surface area (approximately 70 m^2 in an adult).

Cell / Structure	Role
Type I pneumocytes	Squamous (extremely flat) cells that form the alveolar wall; minimise diffusion distance (~0.5 μm)
Type II pneumocytes	Rounded secretory cells; produce **surfactant** (phospholipid mixture that reduces surface tension, preventing alveolar collapse)
Capillary endothelium	Single-cell-thick wall immediately adjacent to alveolar epithelium; brings blood rich in CO_2 and depleted of O_2
Surfactant layer	Lines the alveolar surface; allows the moist lining without causing collapse



Alveolus showing gas exchange between alveolar epithelium and capillary (not to scale)

⚠ EXAM ALERT

Exam Alert — Fick's Law and Alveoli:

In IB exams you must be able to explain how each feature of an alveolus relates to Fick's law. Use this pattern for full marks:

- "Type I pneumocytes are very thin, minimising the diffusion distance and maximising the rate of gas exchange."
- "The extensive capillary network maintains a steep concentration gradient for O_2 by removing it continuously from the blood side."
- "The large number of alveoli (~300–500 million) provides a very large surface area, increasing the rate of diffusion."

Do NOT simply list features — you must link each feature to its effect on a specific variable in Fick's law.

Ventilation

Ventilation is the movement of air into and out of the lungs to maintain the concentration gradients needed for gas exchange.

Phase	Diaphragm	External Intercostals	Internal Intercostals	Chest Volume	Air Flow
Inspiration	Contracts (flattens)	Contract (ribs up + out)	Relax	Increases	Air flows in
Expiration (quiet)	Relaxes (domes up)	Relax (ribs down + in)	Passive recoil	Decreases	Air flows out
Forced expiration	Relaxes	Relax	Contract	Decreases rapidly	Forced out

Key lung volumes:

- **Tidal volume** — volume of air inhaled or exhaled in one normal breath (~0.5 L at rest)
- **Vital capacity** — maximum volume that can be exhaled after a maximum inhalation (~4.5–5 L in adults)

IB TIP

IB Tip — Pressure and Flow: Air moves from high pressure to low pressure. Inspiration increases chest volume, which decreases pressure inside the lungs below atmospheric pressure, drawing air in. Expiration reverses this. You may be asked to explain this in terms of pressure changes rather than muscle movements.

Counter-Current Exchange in Fish Gills

Fish gills use a **counter-current exchange system** in which water flows over lamellae in the opposite direction to blood flow within the lamellae. This maintains a

concentration gradient for O_2 along the entire length of the gill lamella, allowing up to ~80% of dissolved O_2 to be extracted.

In a **parallel flow** system, blood and water would flow in the same direction. As O_2 transfers from water to blood, the concentration gradient would decrease and eventually reach equilibrium — extraction efficiency is much lower (~50%).

MEMORISE THIS

Counter-current vs Parallel flow:

- Counter-current: gradient maintained along the entire surface → efficient (~80% extraction)
- Parallel flow: gradient equalises early → inefficient (~50% extraction)

Stomata and Guard Cells HL

Stomata are pores in the leaf epidermis (mainly underside) through which CO_2 enters for photosynthesis and water vapour is lost by transpiration. Each stoma is flanked by two **guard cells**.

Mechanism of stomatal opening:

1. Light triggers K^+ ions to be actively pumped into guard cells.
2. Water follows by osmosis, increasing turgor pressure in guard cells.
3. The thickened inner wall of guard cells causes them to bow outward, opening the pore.

Abscisic acid (ABA) is a plant hormone released during water stress (drought). ABA causes guard cells to lose K^+ and water, reducing turgor and closing stomata to prevent further water loss.

CAM plants (e.g., cacti, agave) open stomata only at night to fix CO_2 as organic acids, then close them during the day. This minimises water loss in arid environments while still allowing photosynthesis.

Section 2: Transport Systems (A4.2)

Blood Composition

Blood is a connective tissue consisting of a liquid plasma and formed elements (cells and cell fragments).

Component	Structure	Function
Plasma	~92% water; contains proteins, glucose, hormones, ions, CO_2	Transport medium; carries dissolved substances
Red blood cells (RBCs / erythrocytes)	Biconcave disc; no nucleus; filled with haemoglobin; carbonic anhydrase inside	Transport O_2; central role in CO_2 transport — carbonic anhydrase inside RBCs converts ~70% of CO_2 to HCO_3^- (transported in plasma); ~20–25% carried as carbaminohaemoglobin
White blood cells (leucocytes)	Various; have nucleus	Immune defence (phagocytes, lymphocytes)
Platelets (thrombocytes)	Cell fragments from megakaryocytes; no nucleus	Blood clotting (haemostasis)

> **IB TIP**
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> **IB Tip — Why biconcave?** The biconcave disc shape of RBCs maximises surface area relative to volume, shortening the diffusion distance for O_2 to haemoglobin. The lack of a nucleus and organelles maximises the volume available for haemoglobin (~280 million molecules per RBC).

Haemoglobin and the Oxygen Dissociation Curve

Haemoglobin (Hb) is a quaternary protein with four polypeptide chains, each containing a **haem** group with an iron (Fe^{2+}) ion that can bind one O_2 molecule. Each Hb molecule therefore carries up to four O_2 molecules.

Cooperative binding: When the first O_2 binds to haem, it causes a conformational change that increases the affinity of the remaining haem groups for O_2. This produces the characteristic **sigmoid (S-shaped)** dissociation curve — haemoglobin loads O_2 rapidly in the steep middle section of the curve.

The Bohr effect — rightward shift of the dissociation curve at higher CO_2 / lower pH:

- Actively respiring tissues produce CO_2, which dissolves in plasma to form carbonic acid, lowering pH.
- Lower pH decreases Hb's affinity for O_2 (right shift).
- More O_2 is released where it is needed most.

Foetal haemoglobin (HbF) has a higher affinity for O_2 than adult Hb (left shift). This allows the foetus to extract O_2 from maternal blood across the placenta even when maternal Hb saturation is not maximal.

WORKED EXAMPLE

Worked Example — Bohr Effect at Respiring Tissues

A muscle cell is producing CO_2 rapidly during exercise. Explain how the Bohr effect enables oxygen delivery:

1. CO_2 from respiration diffuses into the plasma and combines with water: $CO_2 + H_2O \rightleftharpoons H_2CO_3 \rightleftharpoons H^+ + HCO_3^-$
2. Increased $[H^+]$ (decreased pH) causes a conformational change in haemoglobin, reducing its affinity for O_2.
3. The oxygen dissociation curve shifts to the **right** — at any given O_2 partial pressure, less O_2 is bound.
4. Haemoglobin releases O_2 to the tissues.

Conclusion: The Bohr effect is a self-regulating mechanism — tissues that produce the most CO_2 (i.e., respire fastest) automatically receive the most O_2 from the blood.

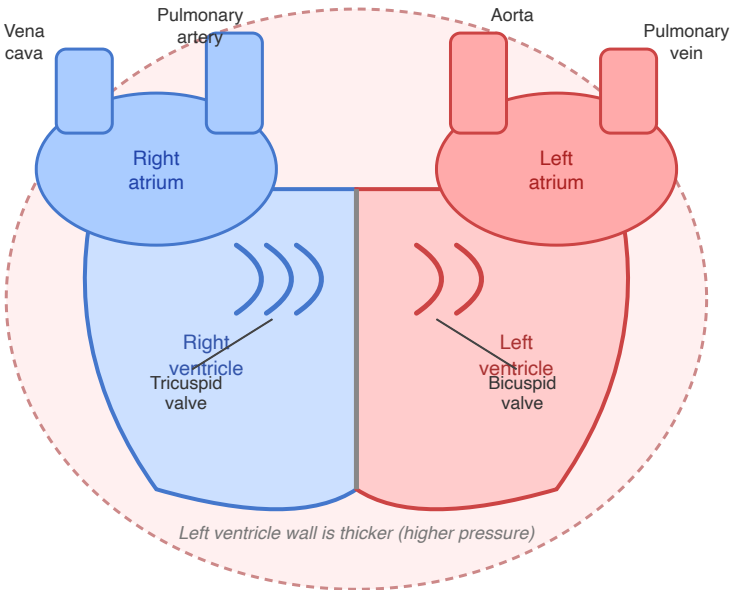
Heart Structure

The mammalian heart is a double pump. The right side pumps deoxygenated blood to the lungs (pulmonary circuit); the left side pumps oxygenated blood to the body (systemic circuit).

Chamber	Receives blood from	Pumps blood to
Right atrium	Vena cava (body)	Right ventricle
Right ventricle	Right atrium	Pulmonary artery → lungs
Left atrium	Pulmonary veins (lungs)	Left ventricle
Left ventricle	Left atrium	Aorta → body

Valves prevent backflow:

- **Atrioventricular (AV) valves** — between atria and ventricles: **bicuspid** (left) and **tricuspid** (right)
- **Semilunar valves** — at the base of the aorta (aortic) and pulmonary artery (pulmonary); prevent blood returning to ventricles after systole



Heart cross-section (anterior view) showing four chambers, major vessels, and valve positions (not to scale)

⚠ EXAM ALERT

Exam Alert — Left vs Right:

In a diagram of the heart viewed from the front (as above), the patient's **left** ventricle appears on the **right side** of the diagram. The left ventricle has a much **thicker muscular wall** than the right ventricle — it must generate higher pressure to pump blood around the entire body versus just to the nearby lungs.

Do NOT state "the left atrium receives deoxygenated blood." The left atrium receives **oxygenated** blood from the pulmonary veins (lungs have oxygenated it). A common exam trap reverses this.

Cardiac Cycle

The cardiac cycle is the sequence of contraction (systole) and relaxation (diastole) that produces one heartbeat.

Phase	Event	Pressure Change
Atrial systole	Both atria contract; blood pushed into ventricles	Atrial pressure > ventricular pressure briefly
Ventricular systole	Both ventricles contract; AV valves close (lub); semilunar valves open	Ventricular pressure rises sharply above aortic/pulmonary
Ventricular diastole	Ventricles relax; semilunar valves close (dub); AV valves open	Pressure falls; atria fill again

ECG interpretation:

- **P wave** — atrial depolarisation (atrial systole begins)
- **QRS complex** — ventricular depolarisation (ventricular systole)

- **T wave** — ventricular repolarisation (ventricles relax)

MEMORISE THIS

Heart sounds — "lub-dub":

- **Lub** (first sound, S1) — AV valves closing at start of ventricular systole
- **Dub** (second sound, S2) — semilunar valves closing at end of ventricular systole

Blood Vessels

Vessel	Wall Structure	Function
Arteries	Thick: elastic tissue + smooth muscle + collagen	Carry blood away from heart under high pressure; elastic walls buffer pressure waves
Capillaries	Single endothelial cell layer (one cell thick)	Site of substance exchange (gases, nutrients, wastes) between blood and tissues
Veins	Thin: less elastic tissue and smooth muscle; **valves** present	Return blood to heart under low pressure; valves + skeletal muscle pump prevent backflow

IB TIP

IB Tip — Capillary exchange: Only capillaries have walls thin enough to allow exchange. The combination of thin wall (short diffusion distance) and large total cross-sectional area (slow blood flow) maximises time and efficiency of exchange with tissues.

Xylem and Phloem (Plant Transport)

Plants have two vascular tissues that carry different substances.

Tissue	Cells	Contents Transported	Direction
Xylem	Dead cells; lignified walls; hollow vessels and tracheids	Water + mineral ions	Always upward (roots → leaves)
Phloem	Living sieve tube elements + companion cells	Sucrose (and other organic solutes)	Source to sink (bidirectional)

Transpiration and cohesion-tension theory:

1. Water evaporates from mesophyll cells through stomata (transpiration).
2. This creates a **tension** (negative pressure) in leaf xylem.
3. Water molecules are held together by **cohesion** (hydrogen bonding) and to xylem walls by adhesion.
4. A continuous column of water is pulled up from roots — no active energy required at the plant level.

Section 3: Defense Against Disease (A4.3)

Types of Pathogens

Pathogen	Structure	Example Disease
Bacteria	Prokaryote; may produce toxins or invade tissues	Tuberculosis, cholera
Viruses	Non-cellular; RNA or DNA core + protein capsid; hijack host cell machinery	Influenza, HIV
Fungi	Eukaryote; chitin cell wall; usually surface infections in healthy individuals	Tinea (athlete's foot), candidiasis
Protists	Eukaryote; diverse; often vector-transmitted	Malaria (*Plasmodium*)

Innate (Non-Specific) Immunity

Innate immunity is the first line of defence — it responds immediately and non-specifically to any foreign material.

Physical and chemical barriers:

- Skin — physical barrier; low pH (acidic); antimicrobial peptides
- Mucus membranes — trap pathogens in respiratory/digestive tracts; cilia sweep mucus away (mucociliary escalator)
- Stomach acid — low pH destroys most ingested pathogens

Cellular innate responses:

- **Phagocytosis** — phagocytes (neutrophils, macrophages) engulf pathogens by endocytosis; lysosomal enzymes digest them
- **Inflammation** — damaged cells release histamine; capillaries dilate and become more permeable; phagocytes migrate to site
- **Fever** — raised temperature inhibits pathogen replication; enhances immune cell activity
- **Interferon** — proteins released by virus-infected cells that warn neighbouring cells to activate antiviral defences

> **IB TIP**
>
> **IB Tip — Phagocytosis steps:** IB often asks for a sequential description. The full sequence is: (1) chemotaxis — phagocyte moves toward pathogen; (2) attachment — phagocyte membrane binds pathogen; (3) engulfment — pseudopodia extend around pathogen forming a **phagosome**; (4) fusion with lysosome forming a **phagolysosome**; (5) digestion by hydrolytic enzymes.

Adaptive (Specific) Immunity

Adaptive immunity is slower but highly specific and generates immunological memory.

Key principle — clonal selection theory:

1. Each B cell and T cell has unique surface receptors for one specific antigen.
2. When an antigen binds to the matching lymphocyte, that cell is selected and undergoes **clonal expansion** (rapid mitotic division).
3. The clone differentiates into **effector cells** (immediate response) and **memory cells** (long-term protection).

B cells — humoral immunity:

Stage	Cell	Action
Antigen activation	B cell	Antigen binds to B cell receptor; B cell activated (helped by helper T cells)
Clonal expansion	B cell clones	Rapid mitosis produces many identical B cells
Differentiation	Plasma cells	Secrete large quantities of antibodies (immunoglobulins) specific to the antigen
Memory	Memory B cells	Persist for years; respond rapidly on re-exposure

T cells — cell-mediated immunity:

T cell type	Function
Helper T cells (CD4+)	Activated by antigen-presenting cells; secrete cytokines that activate B cells and cytotoxic T cells; coordinate the entire adaptive response
Cytotoxic T cells (CD8+)	Kill body cells infected with viruses or abnormal cells (cancer) by inducing apoptosis
Memory T cells	Persist after infection; enable rapid response on re-exposure

⚠ EXAM ALERT

Exam Alert — B cells vs T cells:

Do NOT confuse B cells and T cells:

- **B cells** produce antibodies → **humoral immunity** (works in blood/lymph)
- **T cells** kill infected cells or coordinate the response → **cell-mediated immunity**
- Both types are produced in **bone marrow**; T cells mature in the **thymus** (T = thymus)
- Both produce memory cells after a primary immune response

A common wrong answer is “B cells destroy infected cells” — they do not. Cytotoxic T cells do that.

Antibody Structure

Antibodies (immunoglobulins) are Y-shaped glycoproteins consisting of four polypeptide chains:

- **2 heavy chains** (long)
- **2 light chains** (short)
- Chains held together by **disulfide bonds**
- **Variable region** (tips of the Y) — unique amino acid sequence that forms the **antigen-binding site** (specific to one antigen)
- **Constant region** (stem of the Y) — same across all antibodies of a class; interacts with immune cells and complement proteins

MEMORISE THIS

Antibody structure quick recall:

- 4 chains: 2 heavy + 2 light
- Variable region → antigen-specific binding
- Constant region → effector functions (opsonisation, complement activation)
- Two identical antigen-binding sites per antibody (bivalent)

Antigen-Antibody Interactions

Mechanism	Description	Effect
Neutralisation	Antibody binds to pathogen/toxin surface, blocking its interaction with host cells	Pathogen/toxin rendered harmless
Agglutination	Antibodies cross-link multiple pathogens (bivalent structure) into clumps	Immobilises pathogens; easier for phagocytes to engulf
Opsonisation	Antibodies coat pathogen surface	Phagocytes have receptors for constant region → enhanced phagocytosis

Vaccination

Vaccination stimulates an **active primary immune response** without causing disease, generating memory cells for rapid protection upon subsequent exposure.

Primary vs secondary immune response:

- **Primary response** — first exposure to antigen; slow (days to weeks); antibody titre rises gradually; effector and memory cells produced
- **Secondary response** — re-exposure to same antigen; **faster** (hours to days); **stronger** (much higher antibody titre); due to rapid clonal expansion of memory cells

Herd immunity — when a sufficiently large proportion of a population is immune, transmission chains are broken and even unvaccinated individuals are protected. The threshold varies by pathogen's transmissibility (R_0).

⚠ EXAM ALERT

Exam Alert — Vaccination mechanism:

Vaccination works by stimulating a **primary immune response** without disease — memory cells persist so a secondary response is rapid and robust if the real pathogen is encountered.

Do NOT write "antibodies from the vaccination persist and fight the disease." Circulating antibodies from a vaccination do wane over time. What persists are **memory cells**, not the antibodies themselves, and these generate a rapid new antibody response on real exposure.

ABO Blood Types and Transfusions HL

The ABO blood group system is determined by glycoprotein antigens on the surface of red blood cells and corresponding antibodies naturally present in plasma.

Blood Type	RBC Antigens	Plasma Antibodies	Can Donate To	Can Receive From
A	A antigen	Anti-B	A, AB	A, O
B	B antigen	Anti-A	B, AB	B, O
AB	A and B antigens	Neither	AB only	A, B, AB, O (universal recipient)
O	Neither	Anti-A and Anti-B	A, B, AB, O (universal donor)	O only

Rhesus (Rh) factor: An additional antigen. Rh^+ individuals have the D antigen on RBCs; Rh^- individuals do not. O^- is the universal donor because it lacks A, B, and Rh-D antigens — compatible with any recipient.

Transfusion reactions: If incompatible blood is transfused, the recipient's plasma antibodies bind the donor's RBC antigens, causing **agglutination** (clumping) and subsequent haemolysis (RBC destruction) — a potentially fatal reaction.

⚠ EXAM ALERT

Exam Alert — Why O– is Universal Donor:

O– RBCs carry **neither A, B, nor Rh-D antigens**, so they will not trigger antibody reactions in any recipient. This is the correct explanation. Do not simply say "O– has no antigens" — be specific: it lacks the A, B, and Rh-D antigens relevant to transfusion compatibility.

MCQ Practice — A4 Form & Function

IB Paper 1 style — one best answer.

Question 1. Which feature of an alveolus most directly increases the rate of gas exchange according to Fick's law?

A. Presence of surfactant produced by Type II pneumocytes

B. Large collective surface area provided by ~300 million alveoli

C. Moist lining that allows gases to dissolve

D. Rich capillary blood supply maintaining a concentration gradient

▶ Reveal answer

Question 2. The Bohr effect shifts the oxygen dissociation curve to the right. What is the immediate consequence for tissues with high metabolic activity?

A. Less O_2 is loaded onto haemoglobin in the lungs

B. More O_2 is released from haemoglobin to the tissues

C. CO_2 transport from tissues to lungs is impaired

D. Haemoglobin affinity for O_2 increases in respiring tissues

▶ Reveal answer

Question 3. Blood entering the left atrium comes from which vessel?

A. The vena cava

B. The coronary arteries

C. The pulmonary veins

D. The aorta

▶ Reveal answer

Question 4. Which statement correctly distinguishes B cells from T cells in the adaptive immune response?

A. B cells are produced in the thymus; T cells mature in bone marrow

B. B cells produce antibodies; T cells destroy infected cells or coordinate the immune response

C. B cells form memory cells; T cells do not form memory cells

D. B cells are part of innate immunity; T cells are part of specific immunity

▶ Reveal answer

Question 5. Why does a second exposure to the same antigen produce a faster and stronger immune response?

A. More antigen is present at second exposure, activating more lymphocytes

B. Memory cells from the primary response undergo rapid clonal expansion upon re-exposure

C. Antibodies from the first response persist at high levels and immediately neutralise the antigen

D. Innate immunity is more strongly activated at the second exposure

▶ Reveal answer

Question 6. A patient with blood type A receives a transfusion of type B blood. What would occur and why? HL

A. No reaction — A and B antigens are compatible with each other

B. Agglutination — the recipient's anti-B antibodies bind to B antigens on the donor's red blood cells

C. Haemolysis without prior agglutination, because A and B antigens cancel each other out

D. The transfusion is safe provided the Rh factor of donor and recipient match

▶ Reveal answer

Exam Strategy — A4 Top Mistakes

1. **Confusing the Bohr effect direction.** High CO_2 = lower pH = right shift = lower Hb affinity = more O_2 released. The curve shifts right (not left) in active tissues.
2. **Stating that veins always carry deoxygenated blood.** The pulmonary veins are a classic exception — they carry oxygenated blood from the lungs to the left atrium. Similarly, the pulmonary artery carries deoxygenated blood despite being an artery.
3. **Writing that vaccination provides antibodies that protect you.** Vaccination generates **memory cells**. Subsequent antibody production occurs rapidly when memory cells are re-activated by real pathogen exposure. The antibodies from the initial vaccine response are not what provides lasting immunity.
4. **Reversing B cell and T cell roles.** B cells → antibodies (humoral). T cells → cell killing and coordination (cell-mediated). Both originate from bone marrow; only T cells mature in the thymus.

5. **Applying the wrong Fick's law relationship.** Surface area and concentration gradient are in the **numerator** (increasing them increases rate). Diffusion distance is in the **denominator** (decreasing it increases rate). A thinner Type I pneumocyte increases rate because it decreases the denominator.

MEMORISE THIS

Fast-Recall Checklist — A4 Key Facts:

- Fick's law: Rate $\propto$ (SA × concentration gradient) / diffusion distance
- Alveolus: Type I (thin, gas exchange), Type II (surfactant, prevents collapse)
- Bohr effect: ↑CO_2 → ↓pH → right shift → ↑O_2 released to tissues
- Heart: right side = pulmonary circuit (deoxygenated); left side = systemic (oxygenated)
- Valves: bicuspid (left AV), tricuspid (right AV), semilunar (aortic + pulmonary)
- Innate immunity: fast, non-specific (phagocytosis, inflammation, fever, interferon)
- Adaptive immunity: slow, specific, generates memory (B cells → antibodies; T cells → coordination + killing)
- Vaccination: primary response + memory cells → rapid secondary response on re-exposure
- ABO: type O– = universal donor (no A, B, or Rh-D antigens)