**In a Nutshell!** 

The aim of this exercise is to identify what is the most important information to put down that directly answers the question that is asked. I only want the core, essential parts of the answer, without any of the supporting details that you might put in a real SAQ answer. Rather than limiting time, I am limiting the maximum number of dot point per answer.

Example:

Explain the A-a gradient for pO2 and when it is increased. (3 – 5 points)

* Gas transfer of O2 across alveolar membrane is **perfusion limited** and alveolar and end pulmonary capillary (and hence arterial) pO2 should be equilibrated
* However, arterial pO2 is slightly lower than pAO2 in a healthy person due to a small amount of shunt and low V/Q lung units, ie **venous admixture**
* Venous admixture blood is desaturated and dilutes the O2 content in arterial blood causing the A-a gradient
* Conditions that increase **true shunt** and **V/Q mismatch** will increase the A-a gradient, such as atelectasis, CAL, old age.

1. Explain the ET-arterial pCO2 difference and when it is increased. (3 – 5 points)

2. If a healthy person is given atropine, initially cardiac output will increase, but soon drop back down to baseline. Why? (3 – 5 points)

3. What determines the speed of onset of action of a local anaesthetic? (2 – 4 points)

4. What determines the duration of action of a local anaesthetic? (2 – 4 points)

5. Why can O neg blood be safely given to most patients? (3 – 5 points)

6. Why does hypoalbuminaemia cause interstitial oedema? (2 – 4 point)

7. Why are there no factors that increase or decrease MAC? (2 points)

8. Why are sevoflurane and fentanyl a useful anaesthetic combination? (3 – 5 points)

9. Why is 5% EMLA an effective skin topical anaesthetic, while 5% lignocaine is not? (2 – 4 points)

10. How does preoxygenation prevent desaturation during induction? (3 – 5 points)

**Answers**

1. Explain the ET-arterial pCO2 difference and when it is increased. (3 – 5 points)

* Gas transfer of CO2 across alveolar membrane is **perfusion limited** and alveolar and end pulmonary capillary (and hence arterial) pCO2 should be equilibrated
* End tidal CO2 is representative of mixed alveolar gas, however ETCO2 is slightly lower than paCO2 even in healthy people, by ~3mmHg, due to a small amount of **alveolar dead space**
* Alveolar dead space are alveoli that are ventilated but not perfused and hence do not participate in gas exchange, thus they have the same composition as the inspired gas which contains no CO2\*
* During expiration, alveolar DS gas mixes with the remaining alveolar gas (which contains pCO2 that has equilibrated with arterial pCO2) and slightly dilutes the CO2 concentration
* Any conditions that **increase alveolar dead space will increase the ET-arterial CO2** difference, eg pulmonary embolus, high PEEP, low cardiac output states

\*This is not quite accurate (see Brandis) but for exam purposes, KISS

2. If a healthy person is given atropine, initially cardiac output will increase, but soon drop back down to baseline. Why? (3 – 5 points)

* Atropine is an anti-muscarinic agent that antagonises the effects of acetyl choline released by postganglionic parasympathetic nerve fibres synapsing on the heart (CN X) → ↑automaticity of the SA node →↑HR
* CO = HR x SV, hence CO will increase
* However, ↑HR affects the other determinants of myocardial performance, which result in ↓SV and normalisation of CO
	+ ↓preload due to reduced diastolic filling time (follows the vascular function curve)
	+ ↑afterload due to ↑MAP (since ↑CO and unchanged SVR)
* Over a wide range of heart rates (50 – 180 bpm), an isolated change in HR does not cause a sustained change in CO, unless there are other factors at play, such as exercise that ↑ tissue metabolic demand → alter determinants of myocardial performance (see Brandis)

3. What determines the speed of onset of action of a local anaesthetic? (2 – 4 points)

* LAs are weak bases that exist in equilibrium between their ionised and unionised states, the ratio being dependent on their pKa and the pH of the surrounding environment
* LAs which have a **higher unionised fraction**, eg ligocaine 25% unionised at pH 7.4, have a faster speed of onset as only the unionised form can diffuse through the cell membrane to reach its site of action within the Vg Na channels of nerve fibres
* LAs that have **lower potency** → faster onset due to
	+ Greater dose administered → higher concentration to drive diffusion (Fick’s Law)
	+ Potency correlates to lipid solubility and protein binding. Greater binding to extraaxonal proteins slows down entry into axons and retards onset

4. What determines the duration of action of a local anaesthetic? (2 – 4 points)

* The main determinant of duration of action is the degree of **protein binding** (which also correlates with lipid solubility and potency)
* The degree of plasma protein binding correlates to protein binding onto extracellular axonal membrane
* High protein binding retards the rate of removal by vascular absorption, providing a reservoir of slow release of LA, hence prolonging duration of action
* Other factors include: vascularity of site of injection, extrinsic/intrinsic vasoconstrictor activity, dose administered

5. Why can O neg packed cells be safely given to most patients? (3 – 5 points)

* Group O neg blood does not have A or B antigens or Rhesus D antigen expressed on the red cell membrane
* Even if the patient has anti-A antibodies in their plasma (ie blood group B or O) or anti-B (blood group A or O) there will be no ABO incompatibility reaction, nor any reaction if the patient had Rhesus D antibodies
* However, antibody reactions to other antigens on the donor red cells can still occur, such as other Rhesus subtypes, Rho, Duffy and Kelly
* Also, reactions unrelated to incompatibility can still occur such as febrile reactions, TRALI, delayed haemolytic reaction, transmission of infection

6. Why does hypoalbuminaemia cause interstitial oedema? (2 – 4 point)

7. Why are there no factors that increase or decrease MAC? (2 points)

* MAC is the minimum alveolar concentration of a volatile anaesthetic agent
	+ **expressed as a percentage**
	+ **at 1 atm ambient pressure**
	+ **after 15 min delivery, to allow equilibration of alveolar partial pressure with the brain**

that will prevent **gross, purposeful movement** in 50% of

* + **young**
	+ **healthy**
	+ **unpremedicated**

human subjects or laboratory animals, in response to a **standardised, supramaximal stimulus**, such as a surgical skin incision or tail clamping

* The factors that are commonly claimed to affect MAC, such as age, medications and pathological conditions are excluded by the very definition of MAC\*

\*Don’t be a smart arse like me, DO NOT say this is the real exam!

8. Why are sevoflurane and fentanyl a useful anaesthetic combination? (3 – 5 points)

9. Why is 5% EMLA an effective skin topical anaesthetic, while 5% lignocaine is not? (2 – 4 points)

* EMLA is a eutectic mixture of 2.5% prilocaine and 2.5% lignocaine, which has a melting point of 16C, ie the local anaesthetic mixture is a liquid at room temperature with a 100% concentration within the droplets
* It is presented as a LA droplet in water emulsion and at the droplet/skin interface, there is an effective LA concentration of 80%
* Thus the rate of diffusion through the skin is much higher than for 5% lignocaine, in accordance to Fick’s Law of diffusion, making it more effective as a skin topical anaesthetic

10. How does preoxygenation prevent desaturation during induction? (3 – 5 points)

* Preoxygenation is a procedure that aims to **denitrogenate** the lungs and fill the **functional residual capacity** with O2 (3-5mins of breathing 100% O2)
* In accordance with the **alveolar gas equation**, the pAO2 will be 663mmHg, with an O2 concentration of ~90%
* The FRC is 30ml/kg, ~ 2L in a 70kg adult, providing a **reservoir** of 2000ml x 0.9 = 1800ml of O2
* Given that the adult **basal metabolic rate** is 250ml O2/min, the patient will not begin to desaturate for ~7 minutes