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# Advice on Answering Clinical Science Oral Questions

## Structured Oral Examination (SOE) (Formerly the Clinical Science Viva)

As the prefaces to successive editions of this book suggest, the Final Fellowship of the Royal College of Anaesthetists (FRCA) examination has continued to evolve since its inception in 1996, now almost three decades ago. The perception that the basic science components were little different from those examined in the Primary FRCA led to a change in emphasis from 'basic' to 'clinical' science. Some saw the change as little more than cosmetic, albeit that the College asserted that the structured orals were intended to test 'the understanding of basic science to the practice of anaesthesia, intensive therapy and pain management'. The College has always included the proviso that 'it is accepted that candidates will not have acquired a detailed knowledge of every topic during the period of recognised training', and the examination is linked specifically to the curriculum, but hitherto this statement on occasion contrasted uneasily with the bitter perception of some candidates that they had been examined almost to destruction on scientific minutiae. This perception, against a background of muted unease about this section of the exam, has long since been acknowledged by the College, which decided therefore to introduce greater clinical emphasis into the science oral. The change of emphasis initially was relatively subtle, but in the current iteration of the SOE, it is much more explicit, with a clinical scenario usually prefacing discussion of a basic science topic. That change notwithstanding, you will be expected to discuss the clinical science subject in some depth because both the College and its examiners are reluctant to dilute the rigour of what for most candidates will be the last examination in anaesthesia that they are likely to take.

As the name of this part of the exam implies, the FRCA has a highly structured format. The material on which candidates are to be tested is made available to the examiners only on each morning of the exam. The questions are changed after each session to avoid any possibility of later candidates obtaining an unfair advantage. Each pair of examiners will decide between themselves which of the questions they are going to ask. This is largely the

extent of the choice that they can make, because the scope of each question is limited both by the guidance answer and by the relatively short time available for each topic. At the end of the oral, each examiner will mark each of the questions independently, without conferring and without discussion. This removes any accusation that one examiner may exert undue pressure on the other during the marking process.

The first part of the structured oral (SOE 1) is in two parts, A and B, which follow consecutively. Each part consists of two short clinical cases with a clinical science question linked to each of the cases. Each short case and science question is intended to last 13 minutes, with each part lasting 26 minutes in total. The amount of time available for the science component therefore is limited to six or seven minutes, which in turn limits the amount of information that can be exchanged during the questioning. So although much of the material in this book is relatively simplified, it should still provide enough detail for a pass.

## The Marking System

The examiners can each give a mark of 2 (pass), 1 (borderline) or 0 (fail) for each separate part. This system is important because it means that you can perform poorly in one or more of the questions yet still be able to pass the exam overall. Let us assume for the sake of argument that your clinical anaesthesia is stronger than your basic science and that you have obtained two marks for each part. The first science question is, say, about the anatomy of the coeliac plexus, followed by a discussion in the physiology section about cytochrome P450. You are then asked rather more forgiving questions about propofol and the safety features of the anaesthetic machine. In theory, you could actually respond to the first two questions with complete silence (and be awarded four 0's) before dealing confidently with the second two questions, receive a total of eight marks and still pass the overall exam. It is an improbable scenario, but it does make the point that even if you feel that you have done very badly on a particular question (and you may have performed better than you think, because most candidates are notoriously pessimistic when assessing their own performance), you must not let it affect your approach to the next topic. If you allow yourself to become demoralised, then you will enter a downward spiral from which it may be difficult to recover. You must leave the question behind you, cognisant of the fact that the four subjects are entirely unrelated and that your other answers may well redeem it.

## Why Do They Have to Ask These Kinds of Questions?

When your examiner looks up benignly from the question paper and invites you to discuss 'cytochrome P450' or 'chirality', your heartfelt sigh may be difficult to suppress.

Some examiners will ask these questions with at least a hint of apology, which may raise your spirits marginally as you sense that these individuals might be on your side. Other examiners, alas, will be completely bereft of irony. The difference between them should be obvious, but it might be of interest, if little consolation, were you to be aware of some of the reasons why such questions can arise.

## A Brief History of Anaesthesia's Inferiority Complex

Anaesthesia had its humble origins in mid-nineteenth-century dentistry, and although hospital-based anaesthesia later became much more sophisticated, in the early twentieth century, simple general anaesthesia in the United Kingdom was still being delivered by individuals who were not only without medical qualifications but in many instances were without even a rudimentary education. In contrast, physicians and surgeons of that era had a high social and intellectual standing that had been established for a century or more. As the specialty evolved over succeeding decades, it continued to enjoy only very modest status. There were, however, some politically astute anaesthetists, amongst them Macintosh and Magill, who recognised the potential perils of anaesthetic humility and who thought it unwise to succumb to anaesthesia's inferiority complex. In particular, they recognised the reality that anaesthetists could achieve equality of status with surgeons only if they had a qualification that was equivalent to the Fellowship of the Royal College of Surgeons, the FRCS. It was this realisation which explained the early two-part exams, first the Diploma of Anaesthesia and then the FFARCS (Fellowship of the Faculty of Anaesthetists of the Royal College of Surgeons), which was the immediate forerunner of the FRCA. These examinations were modelled on the FRCS, had a low pass mark in the region of 25–30% and, by including in the syllabus detailed anatomy and pathology, established the precedent for rigour in the basic sciences.

The establishment of a difficult anaesthetic exam with a low pass rate played a crucial role in the development of the specialty. When you are tempted therefore to curse the College for erecting the hurdles of the Primary and Final FRCA, you can at least reflect on the fact that the difficulty of these examinations may in some oblique way ensure that you get paid the same as your colleagues in surgery and medicine. Anaesthesia has a reputation for having amongst the most difficult postgraduate exams, and superficial though this may sound, it does remain one of the ways in which the specialty safeguards its standing. Did this attempt to mirror the FRCS take the process too far? At times, it can certainly seem so, and you may have to console yourself with the familiar, yet no less true, observation that 'Examinations are formidable even to the best prepared . . . for the greatest fool may ask more than the wisest man can answer' (Rev. Charles Colton (1780–1832)). A more recent perspective was provided by a distinguished scientist and professor of medicine from the University of Oxford. During his valedictory speech to the faculty of medicine, he commented that in 30 years of clinical

medicine, his intimate research knowledge of the Krebs cycle had influenced his management 'of not one single patient'. Medicine is as often pragmatic and empirical as it is intellectual. Most, but not all, examiners agree with that view, and do not accept that a detailed knowledge of scientific minutiae is necessary for the safe and effective practice of clinical anaesthesia. It may be obvious at your oral into which category the examiner falls.

## Oral Questions

On average, you will have around six–seven minutes on each science topic. Should a question have limited scope, or if your knowledge is thin, you may spend a bit less time on it, but consistency and fairness demand that the examiners divide the time more or less equally. As explained earlier, these orals are structured, and the examiners have no choice of question. Although it would be logical, given the avowed purpose of the clinical science oral, to subdivide the questions into anaesthesia, intensive therapy and pain management, in practice they do not fit readily into these categories. In the past, the four questions could be somewhat random; it is now usual to have one question which relates to applied anatomy, one to physiology, one to pharmacology and one to physics, clinical measurement, equipment and statistics. This classification is not absolute (topics such as jaundice or latex allergy do not fit strictly into any one of these groups), but it does indicate the broad division of the available questions. The structured nature of the exam minimises the likelihood of an examiner being able to question you in excessive depth on a subject which happens to be an area of special interest or expertise. It also increases the likelihood of an examiner having to ask questions about a subject in which they do not have a current generalist interest.

The subspecialty interests of examiners change as retiring examiners are replaced, but, at any one time, only about 15–20% will have an interest in intensive care medicine, in paediatric anaesthesia or in neuroanaesthesia, and a much smaller number will work in chronic pain management. Thus, a paediatric cardiac anaesthetist may have to ask about adult ophthalmic applied anatomy, a neuroanaesthetist about neonatal fluid requirements or an obstetric anaesthetist about intensive therapy ventilatory strategies. These examiners will not necessarily be ignorant on these topics, but it is certainly possible that your own clinical experience will be more recent and well informed than theirs. This should give you confidence, and you should not let the stress of the exam situation override it. As an extreme example, in recent memory there was an examiner who as a paediatric cardiac anaesthetist had never inserted a supraglottic airway. In some clinical areas, therefore, your experience may be greater and more recent than your examiner's. Draw reassurance from this, and do not be intimidated. So, if you do get the sense that the examiner is unhappy with your answer mainly because it does not accord with what is written on their sheet, then have the confidence to explain the current thinking. Do not be argumentative but simply offer your considered reasoning of the issue. So, if you have recently seen an innovative technique

used in the operating theatre, in the chronic pain clinic or in critical care, do not be hesitant about citing it during the discussion.

The other consequence of the format of the structured oral is that it may lack fluency. It is partly a reflection of examining technique. Some examiners simply introduce the question before initiating a discussion, with only occasional reference to their paperwork. This is usually because they are familiar with the material and can allow the oral to run a more spontaneous course because they have confidence enough in their own ability to assess the answers. An examiner who is less comfortable with the topic and who is less certain of the criteria against which the answers are to be judged is likely to spend much more time referring to the answer sheet. Alternatively, of course, they might just be particularly pedantic in their interpretation of how the structured oral should be conducted. You may get a clue as to which of these you are facing by the way that they introduce the topic.

The structured nature of the examination question and marking system, however, does mean that you may get no chance to exhibit your fluency, and the oral may have a very staccato and rather disjointed feel as the examiners move rapidly on to the next part of the topic. Do not be disconcerted by this; it does not mean that you are doing poorly. It simply reflects the marking system, and so it is much more likely that you are doing well as the examiner in effect ticks off the question that has been answered and goes on to the next. You do not need to worry about trying to pace the oral. It is the responsibility of the examiners to ensure that the requisite points are covered, and the guided answer sheets from which they are working contain more information than all but the most exceptional candidate will cover in the time.

## And Finally, Information, Understanding and 'Buzzwords'

It was not that long ago when one particularly ferocious examiner, having encountered some hapless candidate or other, argued that no one should be allowed to pass the FRCA if they did not know the structure of ether. Although she said 'structure', it is likely that she really meant 'formula' (which as it happens is $CH_3$-$CH_2$-O-$CH_2$-$CH_3$). Either way, the proposition is absurd. Yet it does raise interesting issues in relation to postgraduate examinations. What is their primary purpose? What are they for? Some have argued that, in addition to providing a test of knowledge and a core syllabus, examinations also act as an incentive to learn and, perhaps less urgently, as an incentive to teach. They are used as a hurdle to promotion, and success indicates to colleagues that a standard of training has been achieved. This may also offer a measure of reassurance to an increasingly suspicious public, particularly if the examination is perceived as conferring a title of distinction.

Only two of these functions are of immediate relevance to you. The first is the suggestion that the possession of the diploma of FRCA is a title of distinction. That may sound somewhat grandiose, but in fact it is in everyone's interest that it should be such.

The diploma should not be easily won; it should feel like an exam that is difficult to pass yet one that is worth passing. Were it not so, then examiners and candidates alike would rapidly become demotivated and the standing of the specialty would slide. This thought may offer some solace as you lose many months of your life to the work that is necessary. The second relevant factor is the exam's function as a test of knowledge. It is relatively simple to test for information, harder to assess understanding and more difficult still to provide an objective test of judgement. Hence, as a particular exam evolves, its structure and content elide to create what in effect becomes an examination game. Yet it is a game whose rules curiously do seem to become clear both to candidates and to examiners, as independently they develop a broad appreciation of the level of knowledge that the exam expects.

But the cold reality is that science has outstripped the format of this exam, particularly in respect of pharmacology and, to a lesser extent, physiology. Take, for example, the action of dexamethasone, a glucocorticoid that has become something of an all-purpose anti-emetic, analgesic and anti-inflammatory anaesthetic adjunct. There was a time when it would have been enough to describe its effects and to answer that the drug was given empirically because its mode of action remained unclear. No longer. It is now known that there is a glucocorticoid receptor with diverse isoforms, which is a:

> modular protein comprised of three functional domains: an N-terminal transactivation domain; a central DNA binding domain; and a C-terminal ligand-binding domain. Two nuclear localisation signals are situated within the ligand-binding domain and at the DNA-binding domain-hinge region. The variants are derived from a single gene by alternative splicing and alternative translation initiation mechanisms. Post-translational modifications of these receptor isoforms further expand the heterogeneity of glucocorticoid signaling.

That was just the introduction to a paper which went on to delineate in molecular detail the mechanisms of glucocorticoid actions. You might or might not agree that this extract approaches incomprehensibility, but I think that it is clear, even for those who do not need a translator, that this level of information by far exceeds anything that could be asked or expected in the context of a short structured oral exam. So, as a generalisation, with many topics which appear as examination questions, there is what could be described as a hierarchy of information. Take the slightly more straightforward example of 5-hydroxytryptamine (5-HT). At one end of its continuum of knowledge is the basic fact that it is an aminergic neurotransmitter. At the much more difficult end are details such as the significance of the inositol triphosphate pathway for 5-$HT_2$ receptor function. In between these two extremes is the information about drugs which act at 5-HT receptors, the classes of 5-HT receptors, the subsets of those receptors and the physiological functions that they mediate. Somewhere along that scale is the boundary between a pass and a fail. So how much do you have to know about 5-HT to pass the question? Ask yourself. Should you know that ondansetron is a 5-$HT_3$ antagonist? Probably. Should you know the exact details of the 14 5-HT receptors that have been identified? Probably not, particularly as their functions have not been fully elaborated. Should you know that all bar 5-$HT_3$ receptors are coupled to G proteins? Possibly. Should you know that cerebrospinal fluid production is mediated via 5-$HT_{2C}$ receptors? Not unless you are heading for the prize.

Strange to say most examiners would probably give much the same replies. Both parties seem to understand the rules which dictate that the oral will start at the simpler end of the spectrum and move towards that fail/pass boundary. It is inevitable that it will take some time to cover the basic information, so how do you then convince the examiners that you deserve to pass? Facile though it may seem, some of the time you do it by producing the appropriate buzzwords. They can be described as buzzwords because, unless you are a potential prize winner who has swept the core knowledge aside, there is unlikely to be much time to discuss the more complex information in any detail. By producing the key words and phrases, however, you will have given the examiner at least the subliminal impression that you know more about the subject than just basic information.

So, what are the buzzwords in the example above? One of them would be G protein-coupling. This has a nice echo of Primary FRCA basic science about it, and its mention alone may well satisfy the examiner who is unlikely then to explore your knowledge of ligand-gated ion channels ($5\text{-}HT_3$ only). Similarly, it might help were you to mention that there were seven main 5-HT receptor types. With regard to the actions of corticosteroids, it would probably be more than sufficient to answer that these are mediated by the glucocorticoid receptor, which is a modular protein with three functional domains that enhance or repress the transcription of nuclear target genes. What about a simpler question, say, on atracurium or sevoflurane? How much should you know? Clearly, you will have to display sufficient knowledge to show that your use of these agents is safe and effective. But beyond that, it will help if you happen to refer to atracurium as a 'benzylisoquinolinium' and sevoflurane as a 'halogenated ether'. The examiners are not going to start asking about benzylisoquinolinium chemistry, although they might perhaps want to know what you mean by a 'halogenated ether'. Were you to reply that it is a hexafluorinated methyl isopropyl ether, then that line of questioning would cease. That is because it is clearly a complete dead end down which, were you to have the knowledge, you could continue with the information that sevoflurane is fluoromethyl 1,1,1,3,3,3–hexafluoroisopropyl ether, and that it can be synthesised by a reaction that involves formaldehyde and hydrogen fluoride. By this point, even the most astringent examiner would recognise that you had left anaesthesia far behind in the hot pursuit of irrelevant facts. So, as you revise topics, it is worth bearing this advice in mind because it should not be too difficult to identify those small additional pieces of information that may add further credibility to your answers. This analysis may seem dispiritingly reductive, if not intellectually disreputable, but it is an inevitable consequence of the nature of a standardised exam in which knowledge of the relevant basic sciences has to be explored in a relatively rigid way. If, however, your grasp of that basic knowledge is sound, then you deserve to pass, and it would be unfortunate to fail the examination for want of a few of these simple strategies.

And finally, of course, the best of luck.

# 2

# Anatomy and Its Applications

## The Cerebral Circulation

### Preamble

This is a standard question, but one which contains a lot of anatomical detail. It may be useful to practise drawing a simple explanatory diagram. The oral may be linked to intracranial aneurysms and their management, and it may also include physiological aspects of cerebral perfusion, the problem of cerebral vasospasm following subarachnoid haemorrhage or briefly the subject of intracranial pressure (ICP).

### Core Information

#### Arterial Supply (Figure 2.1)

- The brain is supplied by four major vessels: two internal carotid arteries which provide two-thirds of the arterial supply, and the two vertebral arteries which deliver the remaining third. (Some texts quote an 80:20 distribution.)
- The vertebral arteries give off the posterior inferior cerebellar arteries, before joining to form the basilar artery. This also provides the anterior inferior cerebellar and the superior cerebellar arteries.
- The basilar artery then gives off the two posterior cerebral arteries, which supply the medial side of the temporal lobe and the occipital lobe.
- The artery anastomoses subsequently with the carotid arteries via two posterior communicating arteries.
- The internal carotid arteries meanwhile give rise to the middle cerebral arteries which supply the lateral parts of the cerebral hemispheres. They also provide much of the supply to the internal capsule, through which pass a large number of cortical afferent and efferent fibres, which is why disruption causes such significant neurological damage.

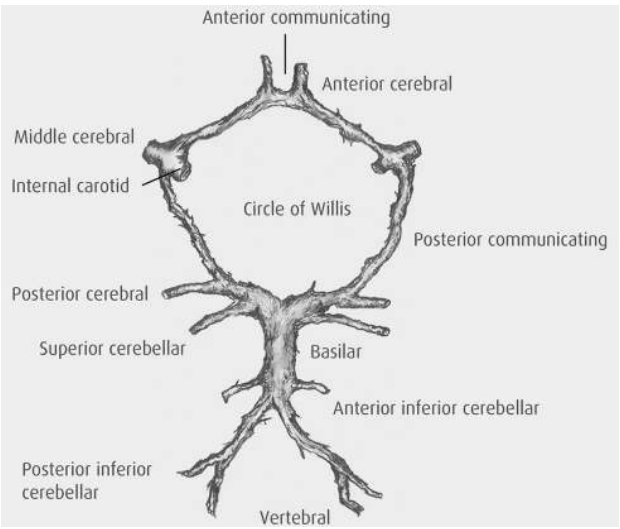


**Figure 2.1** Arterial supply of the brain

- The carotid arteries also give rise to the anterior cerebral arteries, which are connected by the anterior communicating artery, and which supply the medial and superior aspects of the hemispheres.
- The three arterial stems (basilar and carotid arteries), linked by the anterior and posterior communicating arteries, comprise the arterial Circle of Willis. This is said to be incomplete in up to 15% of normal asymptomatic subjects. It provides effective collateral blood supply in the presence of arterial occlusion. Three out of four of the main arteries can be occluded without producing cerebral ischaemia, provided that the process is gradual.

## Venous System

- The cerebral and cerebellar cortices, which are relatively superficial structures, drain into the dural sinuses. These venous sinuses lie between the two layers of the cranial dura mater. The superior sagittal sinus lies along the attached edge of the falx cerebri, dividing the hemispheres, and usually drains into the right transverse sinus.
- The inferior sagittal sinus lies along the free edge of the falx and drains via the straight sinus into the left transverse sinus. (The straight sinus lies in the tentorium cerebelli.) The transverse sinuses merge into the sigmoid sinuses before emerging from the cranium as the internal jugular veins.
- Deeper cranial structures drain via the two internal cerebral veins, which join to form the great cerebral vein (of Galen). This also drains into the inferior sagittal sinus.
- The cavernous sinuses lie on either side of the pituitary fossa and drain eventually into the transverse sinuses.
- The superficial areas of the cerebral (and cerebellar) cortex drain to the venous sinuses via thin-walled veins. These are vulnerable to rupture, with the formation of subdural haematomata, particularly in the elderly in whom there is a loss of brain mass.

## Supplementary Information

### Aneurysmal Subarachnoid Haemorrhage (SAH)

- Intracranial aneurysms account for about 85% of cases of spontaneous SAH; the incidence is 1 in 10–12,000 individuals per year. The overall mortality rate approaches 50%, and morbidity amongst survivors is high. Aneurysms are associated with a weakening of the tunica media of the arterial wall and develop most commonly at vascular bifurcations. Only 10–20% of aneurysms form in the posterior vertebrobasilar circulation. Most are found in the anterior carotid circulation, in the middle cerebral artery and in the anterior and posterior communicating arteries.
- Initial management is as for any other acute cerebral injury, with the emphasis on cardiorespiratory stabilisation and the prevention of secondary brain injury. (There are different grading systems which classify the severity of the injury, but their details are beyond the scope of this oral.)
- Treatment is either with endovascular occlusion using coils or by aneurysm clipping via a direct neurosurgical approach. The cumulative risk of rebleeding approaches 20% at 14 days.
- **Cerebral vasospasm:** this is the major cause of morbidity and mortality following SAH and occurs in up to 70% of cases. Its peak onset is at 7–10 days, may manifest as early as day 3 and usually resolves by 21 days. There are various theories for its aetiology on which the oral may touch, but their complexity precludes excessive detail. Acutely, there is an increase in intracellular calcium which follows exposure to haemoglobin. This produces contraction (via phosphorylation of myosin light chains). However, prolonged vasoconstriction is independent of intracellular calcium levels and may be due to an increase in calcium responsiveness induced by endothelin.
- **Endothelin-1 (ET-1)** is a potent vasoconstrictor whose receptors are upregulated in response to cerebral ischaemia. There is also a general increase in the density of both ET-1 and 5-$HT_{1B}$ receptors. Other factors include the production of reactive oxygen species and lipid peroxidation secondary to haemoglobin autoxidation and changes in the scavenging or production of nitric oxide (NO). A large volume of subarachnoid blood (as seen on CT scanning) is a consistent predictor of the development of vasospasm.
- **Management:** the broad principles include the prophylactic use of the dihydropyridine calcium channel blocker nimodipine, which improves outcome (typical dose regimen is 60 mg 4-hourly for 21 days). Nimodipine blocks the slow calcium channel of vascular smooth muscle and cardiac muscle but has no effect on skeletal muscle. The historical British Aneurysm Trial (1989) demonstrated a 40% reduction in poor outcomes (mortality and neurodisability).
- Established or incipient cerebral vasospasm has been managed with so-called triple-H therapy, consisting of **H**ypertension, **H**ypervolaemia and **H**aemodilution, the combination of which aims to increase perfusion pressure, decrease blood viscosity and maximise cerebral blood flow. (The normal intracranial blood volume is around 100–130 ml.) While it is important to avoid hypotension, hypovolaemia and haemoconcentration, triple-H therapy lacks evidence from controlled trials and its use remains contentious. A low haematocrit, for example, may improve cerebral blood flow but may reduce oxygen delivery.