Introduction

The FRCR viva, together with its sibling, the rapid reporting session, is responsible for the majority of failures in the exam. Like all postgraduate exams, the key to passing the viva is to play the game wisely and to know what to expect.

Pre-preparation

Before preparing for the viva, read the exam guidelines published by the Royal College of Radiologists. Many candidates are unaware of what to expect at the exam until just before the exam. Do not be one of them.

Reporting

This book is a good starting point. Look at the images. Present the cases to yourself (or even better, a study partner). Working 'templates' to generate a radiology report are given below.

The ideal situation

1. Pause for a couple of seconds to look at the film and think before talking.
2. 'This is a [type of radiological investigation] of a [male/female] [adult/child/infant/newborn].' (*This gives you a couple more seconds to look at the film and think.*)
3. 'The investigation shows [findings]. The diagnosis/differential diagnoses is/are …' (Give a *maximum* of 3–4.)
4. 'I would like to …' (Tell them what you wish to do to confirm your diagnosis or your further management plan.)
5. Answer any questions.
6. Film comes down.

Of course, things are often more complicated. If you see no obvious abnormality, try the following:

1 and 2 as above.

3. Go through the imaging systematically, while presenting the normal findings. For example, on a chest x-ray, say 'There are no obvious lung abnormalities. I will now look at the review areas carefully …'
4. Most of the time, you will see the abnormality. Continue as above. If not, ask for additional history (which might prompt the examiner to help you) and look at the film again.
5. If you can see the abnormality but do not know what it is, describe it and give a list of possible differential diagnoses.
6. If appropriate, you could suggest further investigation based on the patient's presenting complaint (although this can be risky strategy). For example, if you cannot see an abnormality in the chest x-ray of someone with progressive shortness of breath, you may try to get the examiner to show you a CT by suggesting that this is what you would do in your clinical practice. However, do not use this trick more than a couple of times, and do not suggest an outlandish or non-indicated test.

6. If you *really* cannot see the abnormality, cut your losses and say you cannot see it and try to move on (if the examiner lets you). It is better to score badly on one image and make up for it on later images than fail the session completely by not moving on.

Other preparation

1. Be proactive. Organize formal FRCR viva sessions with your consultants.
2. Go through the local film collection with a study partner. Practise reporting the images found in major textbooks. As a rule, diseases found in Grainger & Allison and Sutton are considered fair game for the viva.
3. Have a book of lists like Chapman & Nakielny or Dähnert to come up with a reasonable list of differential diagnoses.
4. Go on at least one course. There are countless FRCR courses on offer in the UK and abroad. These courses may seem expensive, but they are certainly cheaper than having to resit the exam. This advice is especially true for candidates from outside the UK.
5. Do not ignore the other components of the exam. The key to passing the long cases is time management, and for the rapid reporting it is getting at least 27/30 correct. Practice is crucial to passing these two components.
6. Do not lose track of the fact that revising for this exam will make you a better radiologist.

Things to do during the exam

1. Be polite to everyone. Smile. Greet the examiner and thank them at the end.
2. Dress appropriately. This means a dark suit, a tie (for men) and long skirt/trousers for women. Not only will the examiners take you more seriously if you look the part, you will feel more confident. (Before your exam, perhaps you can tell yourself that although you're the one being examined, at least your suit is of a better cut than those of the examiners – a great confidence booster!)
3. Talk clearly.
4. Follow instructions.
5. Think before you speak.
6. Always listen to the examiner. They are trying to pass you. If they ask a question like 'Are you sure?', it is not to trip you up but because the answer you gave is not the one they expected.
7. Remember that examiners could be impassive, friendly or somewhere in between. The examiner's affect does not really reflect your performance.
8. Remember that you might actually know more than the examiner (*great confidence booster*). You have just spent months preparing for this exam and are at the peak of your powers. The examiner may be a subspecialist who passed the exams in 1980 and is showing you films outside his/her subspecialist area.
9. Remember to play it safe. Only say and do what you would reasonably do in real life. If asked to describe a procedure that you do not do, always start by saying you do not do it yourself before giving the description. If asked about small radiological minutiae that you do not know anything about, either say you do not know or say you will look it up on PubMed or Google, or in the local medical library.
10. If you get asked about something completely esoteric – e.g. 'Can you elaborate on what you mean by a true FISP MRI sequence?' – it often means that you are doing well.

Things not to do during the exam

1. Smoke before the viva (unless you can make absolutely sure that you do not smell of cigarettes).
2. Chew gum.
3. Go to pieces if a case goes badly – a.k.a. 'the downward spiral syndrome' (see scenarios below). Even when things appear to be going wrong, it is still possible to pass the exam.
4. Argue with the examiner. You will lose.
5. Regurgitate the 10 differential diagnoses given in Dähnert. If the examiner wishes to hear more than the 3–4 differentials, he/she will ask you for it.
6. Answer a question you do not know the answer to. It could lead to a bottomless pit of further questions on the subject. It is often better to say you do not know and move on.
7. 'Kill' the patient. The examiners are here to make sure you are a safe general radiologist.

The following two scenarios are 'fly on the wall' accounts of actual FRCR vivas.

Scenario 1

EXAMINER1: Hello Dr _____, I'm Dr _____ and this is Dr _____. Please have a seat.

CANDIDATE: Hello. Pleased to meet you.

EXAMINER1: I will start. Please report this investigation. (*Puts up a pelvic x-ray*)

CANDIDATE: This is a pelvic x-ray of a male patient. There are multiple lucencies with well-defined mildly sclerotic borders in the proximal right femur. The lesions have a narrow zone of transition. The areas of lucency appear to have a 'ground-glass appearance'. The diagnosis is fibrous dysplasia. (*Expecting the film to go down, which it doesn't*)

EXAMINER1: Anything else?

CANDIDATE: Oh … There is a pathological fracture through the femur. (*Starting to feel stressed*)

EXAMINER1: (*Removes film*) This infant has abdominal pain. (*Puts up paediatric abdominal x-ray*)

CANDIDATE: This is a paediatric abdominal radiograph. There is gross dilatation of the stomach and the duodenum. The distal small bowel and colon are not dilated. The diagnosis is consistent with proximal small bowel obstruction, for which the differential diagnoses are wide. An upper GI barium study would be useful for further investigation.

EXAMINER1: Exactly … (*Puts up small bowel FT*)

CANDIDATE: The barium study shows a corkscrew appearance of the proximal small bowel. (*Suddenly realises he cannot remember the diagnosis associated with corkscrew small bowel. Starts getting more stressed*) There is complete proximal small bowel obstruction. The patient requires a surgical referral.

EXAMINER1: What causes of paediatric proximal small bowel obstruction do you know of?

CANDIDATE: Annular pancreas, small bowel hypoplasia/atresia. I cannot remember the exact cause of a corkscrew small bowel …

EXAMINER1: (*Smiles*) … Calm down. (*Takes film down and puts up a CXR*)

CANDIDATE: Oh … the previous case was malrotation.

EXAMINER1: (*Smiles*) … OK. Look at this chest radiograph.

CANDIDATE: This is a PA chest radiograph of an adult patient. The NG tube tip is above the diaphragm and pointing towards the left. This needs to be repositioned and a repeat chest radiograph obtained. The heart is enlarged. There are a couple of linear lines in the lung bases, which could suggest heart failure.

EXAMINER1: I would actually call those linear atelectasis … Where do you think the NG tube is?
CANDIDATE: It is above the diaphragm and could be in either the distal oesophagus or the left brochial tree.
EXAMINER1: Look again and tell me where it is.
CANDIDATE: It's in the left lower lobe bronchus.
EXAMINER1: (*Film comes down*) Relax … (*Shows another CXR*)
CANDIDATE: This is a PA chest radiograph of an adult female. There is upper lobe blood diversion. There are linear shadows in both lower zones. The heart is enlarged. The diagnosis is … (*Pauses*)
EXAMINER1: Tell me the diagnosis.
CANDIDATE: Heart failure.
EXAMINER1: (*Smiles. Film comes down*)
At this point, the candidate started to relax a bit more and the rest of the viva passed smoothly. The candidate went on to pass the exam.

Scenario 2

CANDIDATE: Good morning.
EXAMINER1: Hello, I'm Dr _____.
EXAMINER2: And I'm Dr _____. Please have a seat.
CANDIDATE: Thank you.
EXAMINER1: I will start. (*Puts up a CXR*)
CANDIDATE: This is an AP radiograph of an adult. There is obscuration of the retrocardiac left hemidiaphragm, along with a triangular left retrocardiac shadow. There is no associated volume loss. No pleural effusion. The findings are consistent with left lower lobe consolidation, most likely due to pneumonia. I would like to confirm this with a history.
EXAMINER1: Patient has a fever and raised white cell count. What else would you recommend?
CANDIDATE: Treat with antibiotics and chest x-ray follow-up in 4 weeks.
EXAMINER1: (*Film comes down*) This man has abdominal pain. (*Puts up a CT scan*)
CANDIDATE: This is a CT scan of an adult male. There are multiple cavitating lymph nodes in the retroperitoneum and porta hepatis. The pancreas is abnormal with a heterogeneous mass in the pancreatic head. The findings are consistent with a pancreatic tumour with lymph node spread. No intraperitoneal free fluid. The liver, adrenals and kidneys are normal. I would like to look at the lung windows to look for pulmonary spread. The patient will need to be discussed at the MDT for further management.
EXAMINER1: (*Film comes down*) This patient has abdominal pain. (*Puts up an AXR*)
CANDIDATE: There is gross large and small bowel dilatation. Hernial orifices appear normal. No radiological evidence of surgery. Findings are consistent with large bowel obstruction of uncertain cause. A history would be helpful. Further investigation by CT or barium enema can be arranged, as guided by clinical history. (*Expecting to be shown more investigations*)
EXAMINER1: What else does this film show?
CANDIDATE: There is no obvious free gas but I will confirm this by obtaining an erect chest radiograph …
EXAMINER1: Are you sure there is no free gas?
CANDIDATE: (*Starting to sweat*) Uh … there is no lucency over the liver. Rigler's sign is negative. No obvious triangular gas shadows … I don't see any free gas.
EXAMINER1: Look again.
CANDIDATE: The falciform ligament is evident, consistent with free gas … I will inform the surgeons about this finding. (*Sweating even more*)

EXAMINER1: (*Film comes down*) Tell me about this foot. (*Puts up radiographs of the left foot*)
CANDIDATE: I cannot see any obvious abnormality … There are no fractures …

The film showed a subtle Lisfranc fracture dislocation, which the candidate eventually got. The rest of the viva continued to go badly and the candidate failed the examination. Unlike in the first scenario, the examiners were impassive throughout the viva.

The take-home messages from these two scenarios are:
1. Have a system when reporting and stick to it no matter how stressed or confident you are.
2. Missing radiological findings does not necessarily mean failure.

Cardiothoracic Case 1

KIAT T. TAN AND PATRICIA DUNLOP

Clinical history

No history.

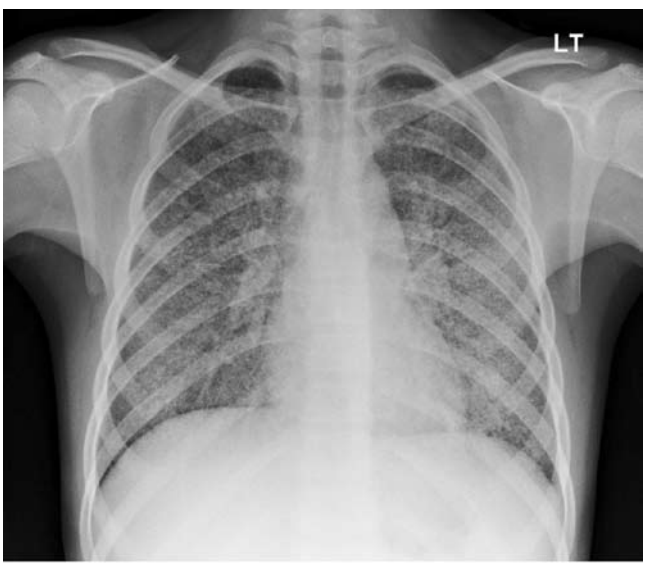

Figure 1.1

Model answer

This is a good-quality frontal chest radiograph of a young patient (Figure 1.1). There are multiple small well-defined nodules that are around 1–2 mm in diameter, spread evenly throughout both lungs with no zonal predominance. No calcification and no pleural effusion. The heart and mediastinal contours are within normal limits. No bony abnormality. The two main differential diagnoses in this case would be infection (such as miliary tuberculosis, fungal and viral pneumonia) or metastatic disease. Less common differential diagnoses would include pneumoconiosis (such as silicosis and coal worker's pneumoconiosis) or atypical sarcoidosis. I would want to look at previous chest radiographs to look for evidence of prior TB or lung nodules and speak to the clinician to obtain a full history as well as to discuss further management. Further investigations may include bronchoscopy with acid- and alcohol-fast bacilli (AAFB)/cultures and CT chest/abdomen/pelvis to look for multiorgan involvement.

Questions

1. **How do you define miliary shadowing?**
   Multiple small (less than 1–2 mm) nodular shadows scattered evenly throughout the lungs.
2. **What are the typical causes of malignant miliary shadows?**
   Thyroid and renal cancer.

3. Is miliary TB seen in primary or post-primary tuberculosis?
Both.

Key points

- Miliary nodules are named after their resemblance to millet seeds. It is due to spread of the free organisms in the blood rather than infected thrombi (as occurs with septic emboli).
- Miliary disease complicates up to 3% of cases of tuberculosis.
- The vast majority of patients have multiorgan involvement.

Further reading

Jeong YJ, Lee KS. Pulmonary tuberculosis: up-to-date imaging and management. *AJR Am J Roentgenol* 2008; **191**: 834–44.

Miller WT. Chest radiographic evaluation of diffuse infiltrative lung disease: review of a dying art. *Eur J Radiol* 2002; **44**: 182–97.

Tables of differential diagnosis. In Fraser RS, Müller NL, Colman N, Paré PD, eds., *Fraser and Paré's Diagnosis of Diseases of the Chest*, 4th edn. Philadelphia, PA: Saunders, 1999; 3144–9.

Cardiothoracic Case 2

KIAT T. TAN AND PATRICIA DUNLOP

Clinical history

Chest pain.

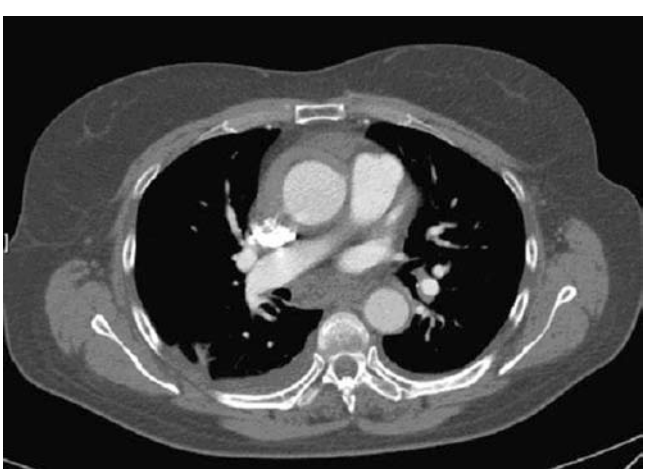

Figure 1.2

Model answer

This is a single image from a contrast-enhanced CT scan of the chest (Figure 1.2). The ascending aorta is dilated and the aortic wall is thickened and of high attenuation. Bilateral pleural effusions and a pericardial effusion are present. The findings

are strongly suggestive of a type A intramural haematoma (IMH). I would like to review the images on a workstation to assess the size and density of the pericardial and pleural effusions as well as the extent of aortic involvement. If possible, I would also like to view the pre-contrast and delayed scans to assess for the degree of contrast enhancement. The presence or absence of concurrent aortic dissection, atherosclerosis and aortic valve involvement should be determined. The cardiothoracic surgeons and cardiologists need to be informed immediately of this finding, as a type A intramural haematoma can progress to frank aortic dissection and/or rupture.

Questions

1. **What is an intramural haematoma?**
   Haematoma within the aortic wall with no intimal flap, intimal tear or direct communication of flow with the lumen.
2. **What is the aetiology?**
   Bleeding from vasa vasorum, penetrating atherosclerotic ulcer.
3. **How do you classify thoracic aortic intramural haematomas?**
   According to the Stanford system, in which an intramural haematoma involving the ascending aorta is classified as type A, and, regardless of the extent, all others are type B.
4. **What are the adverse radiological features associated with intramural haematoma?**
   Adverse features include maximal diameter, thickness of the haematoma, presence of aortic ulceration and location in the ascending aorta. Type A IMH has an early mortality rate of up to 55% if treated medically alone.

Key points

- IMH is part of a spectrum of aortic diseases that include aortic dissection and penetrating aortic ulcer.
- Ideal investigation is by triple-phase ECG-gated CT scan (pre-contrast, arterial phase, delayed phase).

Further reading

Lee YK, Seo JB, Jang YM, *et al*. Acute and chronic complications of aortic intramural hematoma on follow-up computed tomography: incidence and predictor analysis. *J Comput Assist Tomogr* 2007; **31**: 435–40.

Macura KJ, Corl FM, Fishman EK, Bluemke DA. Pathogenesis in acute aortic syndromes: aortic dissection, intramural hematoma, and penetrating atherosclerotic aortic ulcer. *AJR Am J Roentgenol* 2003; **181**: 309–16.

Nienaber CA, Richartz BM, Rehders T, Ince T, Petzsch M. Aortic intramural haematoma: natural history and predictive factors for complications. *Heart* 2004; **90**: 372–4.

Cardiothoracic Case 3

KIAT T. TAN AND PATRICIA DUNLOP

Clinical history

Shortness of breath.

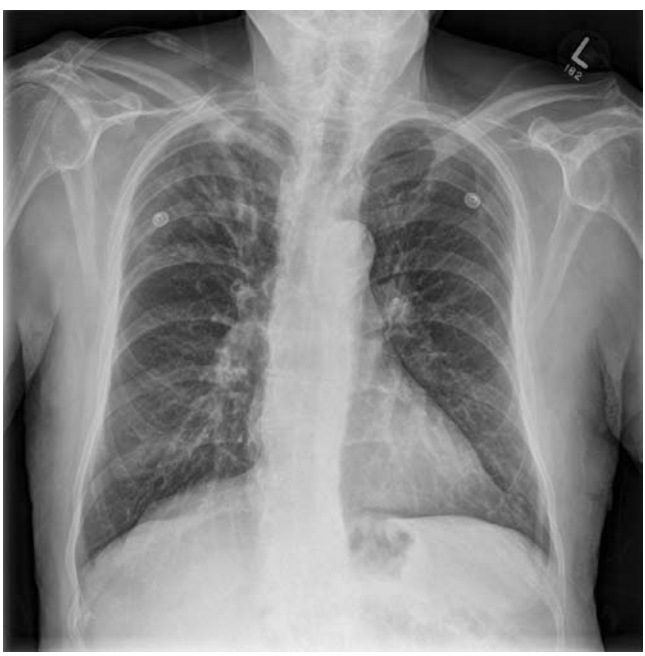

Figure 1.3

Model answer

This is a frontal chest radiograph of an adult (Figure 1.3). The right upper lobe bronchi are thick-walled and dilated. This sign is often referred to as 'tramline' opacities. A 'signet ring' shadow is seen adjacent to the right hilum. There is also a tubular opacity in a right apical bronchus due to mucus plugging (ancillary finding). Less severe changes are present in the left upper lobe bronchi. The finding of bronchial dilatation is consistent with bronchiectasis.

Possible underlying causes of the bronchiectasis include infection (such as tuberculosis, viral pneumonia, aspiration), genetic susceptibility (such as cystic fibrosis, alpha-1-antitrypsin deficiency), immunological deficiencies, defects of mucociliary clearance (which could be congenital, such as in Kartagener's syndrome, or acquired, such as those occurring with aspirated foreign bodies and slow-growing tumours) and immunological (as in cases of allergic bronchopulmonary aspergillosis or rheumatoid disease).

Potential complications of bronchiectasis include recurrent infections, deteriorating pulmonary function, cor pulmonale and haemoptysis (sometimes torrential).

The patient should have an HRCT to assess the severity and distribution of disease. He or she should also be referred to the chest physician for assessment and follow-up.

Questions

1. **What is the definition of bronchiectasis?**
   Abnormal, permanent dilatation of the bronchi, usually associated with inflammation.
2. **What are the HRCT findings of bronchiectasis?**
   - Bronchial lumen diameter greater than accompanying artery (bronchoarterial ratio > 1): high sensitivity, low specificity.
   - Absence of bronchial tapering (seen on longitudinal sections through bronchi).
   - Presence of a visible bronchus within 1 cm of the pleura or mediastinum.
3. **What are the morphological types of bronchiectasis?**
   Classically, bronchiectasis has been classified on morphology into cylindrical (less severe), cystic and varicose (more severe). Cylindrical bronchiectasis refers to disease where there are thick-walled dilated bronchi that have regular diameters. In cystic bronchiectasis, the bronchi are grossly dilated with a 'ballooned' appearance. Varicose bronchiectasis refers to disease that contains interspersed bronchial dilatation and constriction.

Key points

- A widespread pattern of bronchial dilatation affecting both lungs is an indication of systemic disease, such as cystic fibrosis, immunodeficiency states and alpha-1-antitrypsin deficiency. Localized bronchiectasis is usually caused by a focal lesion, e.g. infection and obstruction.
- Certain diseases also tend to affect particular parts of the lung. For example, in tuberculosis bronchiectasis involves mainly the upper lobes, whereas *Mycobacterium avium-intracellulare* (MAI) in non-immunocompromised individuals tends to involve most of the lungs without a predilection for a specific lobe(s). It has been stated that bronchiectasis with a middle lobe and lingular prominence is seen in elderly women with the non-classic form of MAI infection, 'Lady Windermere syndrome'.
- Cystic fibrosis has an upper lobe predominance, and allergic bronchopulmonary aspergillosis a central and upper lung zone predominance.
- Localized bronchiectasis may be surgically curable, but disease with a diffuse pattern is not.

Further reading

Hansell DM. Bronchiectasis. *Radiol Clin North Am* 1998; **36**: 107–28.

Koh WJ, Lee KS, Kwon OJ, *et al*. Bilateral bronchiectasis and bronchiolitis at thin-section CT: diagnostic implications in nontuberculous mycobacterial pulmonary infection. *Radiology* 2005; **235**: 282–8.

Primack SL, Logan PM, Hartman TE, Lee KS, Müller NL. Pulmonary tuberculosis and Mycobacterium avium-intracellulare: a comparison of CT findings. *Radiology* 1995; **194**: 413–17.

Reich JM, Johnson RE. Mycobacterium avium complex pulmonary disease presenting as an isolated lingular or middle lobe pattern: the Lady Windermere syndrome. *Chest* 1992; **101**: 1605–9.