OSCEs

FOR MEDICAL FINALS

HAMED KHAN
IQBAL KHAN
AKHIL GUPTA
NAZMUL HUSSAIN
SATHIJI NAGESHWARAN

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OSCEs for Medical Finals

Hamed Khan

MBBS DGM MRCGP MRCP (London)
GP Principal and Undergraduate Tutor
Oxted, Surrey

Iqbal Khan

BSc MBBS
FY2 Doctor
Homerton University Hospital NHS Foundation Trust

Akhil Gupta

BSc MBBS
Specialist Registrar in Anaesthetics
London Deanery

Nazmul Hussain

MBBS MRPharmS
GP ST3
Newham GP Vocational Training Scheme

Sathiji Nageshwaran

BSc MBBS
FY2 Doctor
Royal Free London NHS Foundation Trust

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The Atrium, Southern Gate, Chichester, West Sussex, PO19 8SQ, UK
111 River Street, Hoboken, NJ 07030-5774, USA

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Contributors

We are grateful to the following doctors and medical students for their contributions to this book.

Contributors to the chapters

Shifa Rahman
Manpreet Sahamey
Ruth-Mary deSouza
Gillian Landymore
Ravi Naik

Contributors to the medical school tables

Saba Ali
Ali Alidina
Nina Arnesen
Svitlana Austin
James Best
Kerry Bosworth
Lisa Burton
Sangeetha Chandragopal
Emily Clark
Laura Clarke
Rebecca Critchley
Nicola Davis
Ruth-Mary deSouza
Pippa Dwan
Matthew Everson
Martin Fawcett
Clare Fernandes
Lyndsey Forbes
Rachel Friel
Ushma Gadhvi
Harminder Gill
Catherine Hatzantonis
Elizabeth Hockley
Laura Hopkins
Towhid Imam
Zara Jaulim
Michelle Kameda
Jennifer Kelly
Pamini Ledchumykanthan
Almas Malik
Sathiji Nageshwaran
Ravi Naik
Sania Naqvi
Siva Nathan
Allan Nghiem
Gary Nicholson
Clarissa Perks
Anna Rebowska
Elissa Scotland
Charly Sengheiser
Nadir Sohail
Charlotte Spilsbury
Sarah Thompson
Elizabeth Khadija Tissingh
Christine Wahba
John Wahba
Siobhan Wild
Anna Willcock
Ahila Yogendra

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Preface

The student begins with the patient, continues with the patient, and ends his studies with the patient, using books and lectures as tools, as means to an end.

Sir William Osler

Few will disagree that the recent overhauls in medical training, together with higher numbers of medical students being trained, has made medicine far more competitive than before. Medical students today have to make definitive career choices much earlier on than they would have had in years gone by, and to start building a portfolio of achievements such as audits and publications very early on at medical school. Time has become even more precious than it was before, and it is understandable that medical students today will opt for concise focused textbooks rather than sprawling prosaic texts, some of which have been used over many generations and gained an almost legendary status.

This book is perhaps unique in that it has been written by a group of doctors who range from those in career-grade posts who have completed postgraduate training and have been OSCE examiners themselves, to those who have very recently sat their finals. We have collated our experiences to create a textbook that we have made as focused, easy to read and, above all, as exam-orientated as possible. While doing this, we have worked hard to ensure that we include everything necessary not only to pass finals, but also to achieve excellent marks and hopefully merits and distinctions.

The structure is based on four sections – clinical examinations, histories, communication skills and procedures. At the beginning of each of these sections, there is a 'Top Tips' page that has generic advice for any OSCE station of that section which would help you boost your marks and performance regardless of what the station is.

Each section is divided into chapters based on the stations we feel are most likely to appear in OSCEs at medical schools. Practice makes perfect – and more so in OSCEs than in any other form of assessment. That is why we have started each chapter with a checklist of items reflecting the areas you are likely to be marked on. You should use these to perfect and consolidate your routines, and also when practising OSCEs with friends and on patients. You should ideally do this in a pair or a group of three, with one student doing the station as a candidate and one allocating mock 'marks' using the checklists to assess the candidate's performance.

Following this in each section, we have included tables that summarise the most common conditions that are likely to present in finals OSCEs. We have ensured that the information on the conditions in these tables is as focused and exam-oriented as possible. There is also a 'Hints and tips for the exam' section in which we have summarised key advice and common pitfalls that finalists tend to make.

We hope that this book will make your revision not only thorough and focused, but also enjoyable. We have spent a lot of time working with our publishers to make the text as vibrant, colourful and easy to read as possible, with a plethora of tables, illustrations and photos that will not only make it easy to remember key ideas and principles, but also make the topic more interesting.

We wish you the very best of luck with your finals OSCEs, and hope that you find this book both enjoyable and useful.

Hamed Khan

Part 1: Examinations

Top tips

Do:

• **Memorise the steps:** The most important thing that OSCE examiners are looking for is an ability to carry out a full examination with reasonable technique and speed. At finals level, you will be forgiven for missing a few signs, and the vast majority of the marks on the mark schemes are allocated for going through the motions and doing all the 'steps'. In contrast, at postgraduate level, for example for the MRCP exam, you would be expected to pick up all the major signs, and be penalised heavily for missing them.

• **Always suggest a number of possible differential diagnoses:** Very few doctors will be able conclusively to put their finger on a diagnosis after examining a patient for 10 minutes without a history. Offering a number of differentials means that you have a higher chance of at least mentioning the correct one, even if it is not at the top of your list. It will also show a healthy awareness of your own limitations.

• **Practise, practise and practise:** The best way to do this is by seeing patients, having a friend to assess you using our checklists and then getting critical (but constructive) feedback from them. Swapping roles and watching colleagues examine is more useful than most students think, as it will reinforce the steps of the examination, and you may see them use techniques and skills that you would not otherwise have thought of. Doing all the major examinations should become such a normal routine for you that you can do it without thinking about what the next step will be – just like riding a bicycle or driving a car.

Don't:

• **Don't be nervous:** Most people have problems in OSCEs not because of poor technique or knowledge, but because of anxiety and nervousness. Don't be overwhelmed by the occasion, and don't be intimated by an examiner's grilling. You will find it much easier to focus on your technique and findings if you are relaxed, and most examiners only grill students who are doing well, as they do not waste their breath on those whom they have decided are a lost cause!

• **Don't worry about minutiae:** Medicine is not an exact science, and different doctors have different ways of examining patients, most of which yield the right conclusions. At undergraduate level, all the examiners are looking for is a decent, fluent technique that appears to be well practised. Don't spend ages trying to figure out exactly how much the chest should expand, or whether the cricoid–sternal notch distance is three finger breadths or four.

• **Don't hurt the patient:** This is the only unforgivable sin in the OSCE. Its always a good idea to start your examination by asking if the patient is in pain anywhere, and reassuring them that if you unintentionally cause pain during the examination, you will be happy to stop. Students often have a tendency to ignore patients saying 'Ouch!' and pretending that they have not heard it, but this is definitely the worst thing you can do. If you do cause pain, acknowledge it immediately, apologise unreservedly and offer to stop – both examiners and patients will appreciate your honesty and professionalism.

Generic points for all examination stations

HELP:
H: 'Hello' (introduction and gains consent)
E: Exposure (nipples to knees/down to groins)
L: Lighting
P: Positions correctly (supine), asks if patient is in any pain
Washes hands
Inspects from end of bed for paraphernalia
Inspects patient (scars, etc.)
Thanks patient
Offers to help patient get dressed
Washes hands
Presents findings
Offers appropriate differential diagnosis
Suggests appropriate further investigations and management
For joints only: Look → Feel → Move → Active/passive/resisted

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1 Cardiovascular

Checklist	P	MP	F
HELP:			
H: 'Hello' (introduction and gains consent)			
E: Exposure			
L: Lighting			
P: Positions at 45 degrees, asks if patient is in any pain			
Washes hands			
Inspection:			
• From end of bed: ECG, GTN spray			
• Scars: thoracotomy, mitral valvotomy			
• Pacemaker			
Hands:			
• Clubbing (infective endocarditis, cyanotic heart disease, atrial myxoma)			
• Signs of infective endocarditis (splinter haemorrhages, Janeway lesions, Osler's nodes)			
Radial pulse:			
• Rate			
• Rhythm (regular or irregular)			
• Character (collapsing, slow rising)			
• Radial–radial delay			
Requests blood pressure			
Eyes:			
• Xanthelasma			
• Corneal arcus			
• Anaemia			
Face:			
• Malar flush			
Mouth			
• 'CDD' (central cyanosis, dental hygiene, dehydration)			

Neck:			
• Jugular venous pressure (raised >4 cm)			
• Palpates carotid pulse (character)			
Palpates apex beat			
Checks if apex beat is displaced in axilla			
Palpates sternal edges and subclavicular areas for thrills			
Auscultates chest:			
• Mitral area/apex beat (5th intercostal space [ICS], midclavicular line) • Tricuspid area (4th ICS, right sternal edge) • Pulmonary area (2nd ICS, right sternal edge) • Aortic area (2nd ICS, left sternal edge) • Palpate carotid or brachial pulse simultaneously to time murmur			
Cardiac manoeuvres:			
• Auscultates mitral area with patient lying on left side and in expiration for murmur of mitral stenosis			
• Auscultates aortic area with patient sitting forward and in expiration for murmur of aortic regurgitation			
Auscultates lung bases for pulmonary oedema			
Palpates shins or ankles for peripheral oedema			
Thanks patient			
Offers to help patient get dressed			
Washes hands			
Presents findings			
Offers appropriate differential diagnosis			
Suggests appropriate further investigations and management			
OVERALL IMPRESSION:			

Summary of common conditions seen in OSCEs

	Aortic stenosis	Aortic regurgitation	Mitral stenosis	Mitral regurgitation	Tricuspid regurgitation
Location of murmur (loudest heard)	Aortic area	Aortic area	Mitral area	Mitral area	Tricuspid area
Type of murmur	Ejection **systolic** Radiating to carotids	End-**diastolic**	Mid-**diastolic**	Pan-**systolic** Radiating to axilla	**Systolic**
Manoeuvres to enhance murmur	None	Sit forward and expirate	Roll on left side and expirate	None	None
Pulse	Slow rising	Collapsing Carotid pulsations	Irregular if atrial fibrillation	Normal	Normal
Peripheral features	Narrow pulse pressure Commonly have CABG scar	Quincke's sign Corrigan's pulsation De Musset's sign	Atrial fibrillation on auscultation Right-sided heart failure	None	JVP increased to earlobes
Key management points	Aortic valve replacement if severe Beta-blockers Diuretics Treat heart failure Antibiotic prophylaxis for gastrointestinal/genitourinary/dental procedures	Aortic valve replacement Treat any heart failure Antibiotic prophylaxis for gastrointestinal/genitourinary/dental procedures	Mitral valvotomy Treat atrial fibrillation and heart failure Antibiotic prophylaxis for gastrointestinal/genitourinary/dental procedures Anticoagulate (with warfarin)	Mitral valve replacement Treat any heart failure Antibiotic prophylaxis for gastrointestinal/genitourinary/dental procedures	Tricuspid valvotomy Treat right heart failure

Hints and tips for the exam

Identifying valvular lesions

Trying to learn all the murmurs and all the conditions associated with them is futile and only really necessary if you are a cardiologist. Trying to correctly differentiate whether murmurs are ejection systolic or pansystolic, end-diastolic rather than mid-diastolic, is also difficult and is not necessary for finals and perhaps even PACES.

The easiest and most logical way of diagnosing the correct valvular lesion from the murmur is by answering the following two questions:

1. Where is the murmur?

Murmurs can frequently be heard throughout the chest, but the area where a murmur is loudest is usually where the murmur is – so a murmur heard loudest in the aortic area will probably be aortic regurgitation (AR) or aortic stenosis (AS), and a murmur heard loudest in the mitral area will probably be mitral regurgitation (MR) or mitral stenosis (MS). Exceptions to this include Gallavardin's phenomenon, in which an AR murmur is heard loudest in the tricuspid area; however, from the perspective of passing an exam, you would not be penalised for missing that, and in any case it is extremely rare.

2. Is it systolic or diastolic?

In other words, does the murmur correspond with the pulse (systolic) or not (diastolic)?

Murmurs will only be produced if the natural flow of the blood is opposed. In the case of valves through which the blood leaves the heart (such as the aortic valve), systolic murmurs will only be produced when the outflow of blood is hindered, which can only happen in AS (as opposed to AR, which would not hinder the outflow of blood).

In the case of valves where the blood flows into the heart in diastole, the natural flow of blood in diastole is against the aortic valve, as the purpose of the aortic valve is to stop blood flowing into the aorta during diastole. Hence blood hits the aortic valves and stops there when the cardiac muscles relax in diastole. This natural flow would be impaired by AR as the blood flows into the aorta when it should not, which is why a diastolic murmur in the aortic area can only be AR.

If this seems too complex, remember that diastolic murmurs are usually 'ARMS' (AR or MS), and the area where it is loudest is probably where the murmur is.

Right versus left

- LEFT-sided murmurs are louder in EXPIRATION.
- RIGHT-sided murmurs are louder in INSPIRATION.

This is because more blood flows into the intrathoracic cavity and lungs on inspiration, and hence more blood flows through the right-sided heart valves as these supply the lungs. The converse is true for left-sided murmurs.

It is vital to ask patients to hold their breath when using this test, but you must not ask them to do this for too long as this can cause the patient pain and you will fail the exam. Its often a good idea to hold your own breath at the same time so that you will know when it is getting too long to allow your patient to breath normally.

Timing the murmur

Timing murmurs is something that both students and experienced doctors have difficulty with. Just remember to palpate the pulse when listening to the heart sound, and see if you hear the murmur at the same time as you feel the pulse.

- If the murmur is WITH the pulse, it is a SYSTOLIC murmur.
- If the murmur if NOT WITH the pulse, it is a DIASTOLIC murmur.

Use a central pulse such as the carotid or brachial to do this, otherwise it will not be accurate.

Diastolic murmurs

A number of conditions can cause diastolic murmurs, but the most common ones are AR and MS – this can be easily memorised using the mnemonic 'ARMS'.

Diastolic murmurs are very difficult to elicit for even the most experienced doctors, and if you can hear a murmur easily, it is most likely to be systolic. However, if you do manage to identify a diastolic murmur, it is handy to remember that MS murmurs are much quieter than AR murmurs, and if you can auscultate a diastolic murmur throughout the chest, it is much more likely to be AR than MS.

Valve replacements

If you see a midline sternotomy scar, you should immediately bring your ear close to the patient's chest and listen carefully for the clicking noise that is indicative of the closing of a metallic valve replacement – this can easily be heard without a stethoscope.

Also remember that you should not hear a murmur with a replaced valve unless it is leaking.

Identifying which valve has been replaced

Remember that the pulse correlates with the first heart sound, which is the mitral valve closing. (The second heart sound is the aortic valve closing.)

- If the loudest sound of the valve closing correlates with the pulse, it is the first heart sound, indicating that the mitral valve has been replaced.
- If the loudest sound of the valve does not correspond with the pulse, it is the second heart sound, indicating that the aortic valve has been replaced.
- The location of the loudest sounds may also be helpful. Bioprosthetic valves sound the same as normal heart valves, so it would be unfair for examiners to expect you to identify them.

Apex beat

The apex beat is palpable in the 5th intercostal space, and is displaced to the apex in MR. Various characters of the apex beat have been described, such as 'heaving' and 'thrusting'; differentiating between them is extremely difficult and probably beyond the scope of a 10-minute OSCE. Other than this, it is more likely to cause confusion than add anything substantive.

The best course of action is to describe where the apex beat it, and whether it is palpable or not. An impalpable apex beat is often caused by obesity, hyperinflation of the lungs, dextrocardia or poor technique.

Scars

Figures 1.1–1.5 show scars and other signs that you will need to note on your examination of the patient.

Questions you could be asked

Q. Which organism causing infective endocarditis is associated with underlying bowel cancer?

A. *Streptococcus bovis* – a colonoscopy should be considered in all patients presenting who are found to have *Streptococcus bovis*.

Q. What is the most common cause of tricuspid regurgitation?

A. Most cases of tricuspid regurgitation are 'functional', due to dilatation of the right ventricle (so that the tricuspid valves flop downwards). This could arise for a number of reasons, such as right heart failure, congestive heart failure and pulmonary hypertension.

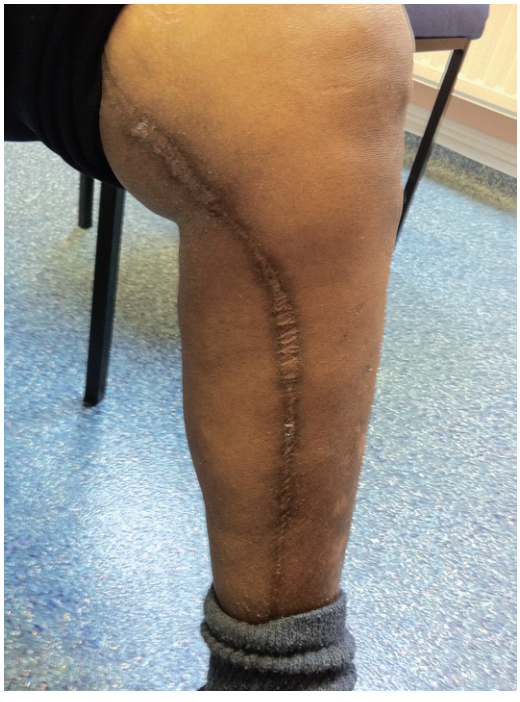

Figure 1.1 Graft scar from leg vein removal in coronary artery bypass grafting

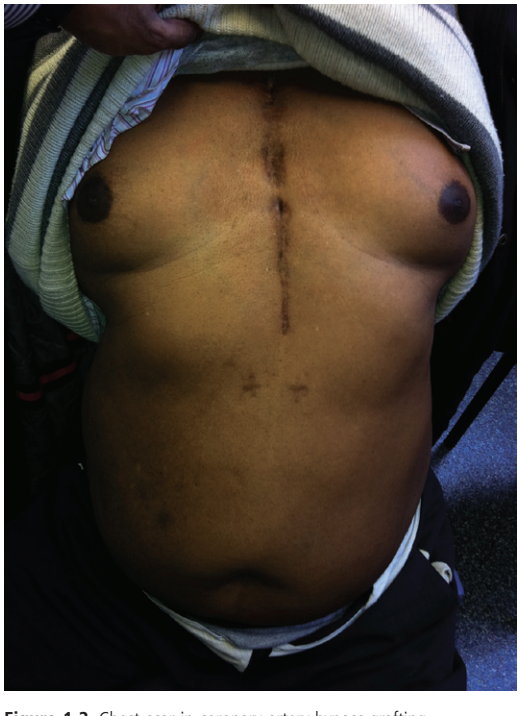

Figure 1.2 Chest scar in coronary artery bypass grafting

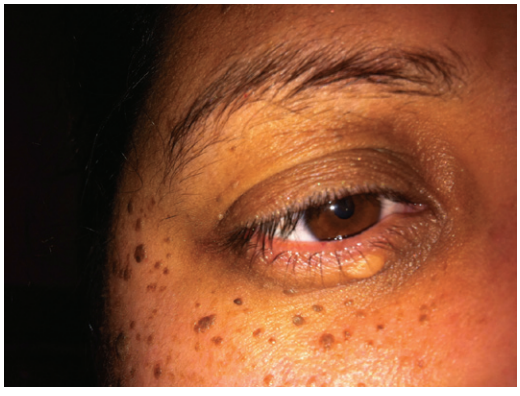

Figure 1.3 Xanthelasma

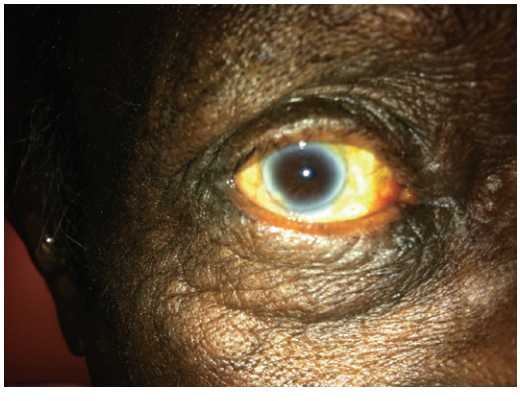

Figure 1.4 Corneal arcus

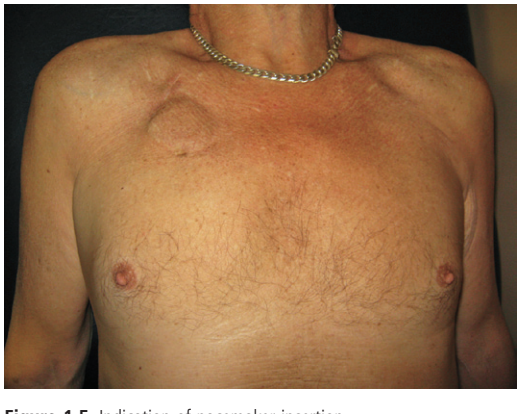

Figure 1.5 Indication of pacemaker insertion

Q. How should a patient with suspected heart failure be investigated in primary care?

A. According to the NICE guidelines (NICE, 2010), the primary investigation of choice is the blood level of brain natriuretic peptide (BNP)– patients with normal results are unlikely to have heart failure, and those with a BNP level >400 pg/mL should be investigated urgently (within 2 weeks).

Reference

National Institute for Health and Clinical Excellence (2010) Chronic heart failure: Management of chronic heart failure in adults in primary and secondary care. Available from http://www.nice.org.uk/nicemedia/live/13099/50526/50526.pdf (accessed June 2012).

2 Respiratory

Checklist	P	MP	F
HELP:			
H: 'Hello' (introduction and gains consent)			
E: Exposure			
L: Lighting			
P: Positions at 45 degrees, asks if the patient is in any pain			
Washes hands			
Inspects from end of bed: • Looks at the front and back for thoracotomy scars • Sputum pots (bronchiectasis, COPD) • Oxygen cylinders (COPD) • Inhalers (COPD, asthma) • Immuosuppressants (pulmonary fibrosis) • Nebulisers (COPD) • Peak flow charts (asthma)			
Hands:			
• Clubbing (suppurative conditions, lung cancer, fibrosis)			
• Tar staining			
• Wasting of small muscles			
Tremor + CO_2 retention flap			
Radial pulse			
Respiratory rate			
Eyes:			
• Horner syndrome (Pancoast syndrome)			
• Anaemia			
Face:			
• Plethora (polycythaemia)			
Mouth:			
• 'CDD' (central cyanosis, dental hygiene, dehydration)			
Neck:			
• JVP (raised >4 cm) in cor pulmonale			
• Palpates lymph nodes			
• Tracheal deviation			
• Cricoid–suprasternal notch distance (<three finger breadths in hyperinflation)			
Palpates:			
• Palpates apex beat			
• Measures chest expansion (6–8 cm is normal) at three places on the anterior and three on the posterior chest			
Percusses:			
• Percusses at three positions on the anterior and three on the posterior chest			
Auscultates:			
• Auscultates at three positions on the anterior and three on the posterior chest			
• Auscultates axillae to listen for right middle lobe signs			
• Auscultates for vocal fremitus			
Palpates shins or ankles for peripheral oedema			
Thanks patient			
Offers to help patient get dressed			
Washes hands			
Presents findings			
Offers appropriate differential diagnosis			
Suggests appropriate further investigations and management			
OVERALL IMPRESSION:			

Summary of common conditions seen in OSCEs

Condition	Key finding	Chest expansion	Percussion	Auscultation	Vocal fremitus
Pulmonary fibrosis	**Fine end-inspiratory crackles**	Decreased bilaterally	Normal/mild decrease at bases	Fine end-inspiratory crepitations	Normal/increased at bases
Pneumothorax	**Increased resonance**	Decreased unilaterally	Increased resonance	Decreased breath sounds at site of pneumothorax	Decreased
Pleural effusion	**Stony dull bases**	Normal	Dull on side of effusion	Dull base(s)	Decreased at base
COPD/asthma	**Wheeze**	Normal	Normal	Wheeze + scattered crepitations in COPD	Normal
Lobectomy	**Scar**	Normal	Dull at site of lobectomy	Normal	Normal
Pneumonectomy	**Scar**	Decreased unilaterally	Dull on side of pneumonectomy	Absent on side of pneumonectomy	Decreased
Bronchiectasis	**Sputum pot, crackles, clubbing**	Normal	Normal	Normal	Normal
Consolidation	**Crackles concentrated in one area**	Normal	Normal	Normal/increased at site of consolidation	Normal/increased at site of consolidation

Hints and tips for the exam

Inspection

Inspection can often provide the diagnosis at the respiratory station. There are some key stereotypical features of a few conditions that can give the case away.

Findings	Condition
Young, thin, short patient with a PEG site near the umbilicus and a tunnelled catheter at the axilla or on the chest	Bronchiectasis secondary to cystic fibrosis
Middle-aged patient with full sputum pot	Bronchiectasis
Cushingoid features (high BMI, bruising, striae) and bruising (from steroid use)	Pulmonary fibrosis
Features of rheumatological disease, e.g. rheumatoid hands (ulnar deviation, swollen metacarpophalangeal joints, swan neck deformity) or scleroderma (beak-shaped nose, small mouth, tight skin, telangiectasia)	Pulmonary fibrosis
Elderly patient with tar-stained fingernails and an oxygen cylinder at the bedside	COPD
Characteristic scars (with pictures)	Lobectomy/ pneumonectomy

Timing

A common problem at the respiratory station is timing as students find it difficult to listen to carefully all the breath sounds in enough places during the 5–10 minutes they have.

Once you have completed your inspection, start examining from the back. Most physicians will agree that it is easier to percuss and auscultate at the back as you have more surface area available. In addition, the position of the heart often makes it difficult to establish findings in the left lower zone of the lung anteriorly.

One of the ways you can minimise collateral time losses is by reducing the time spent in changing the patient's position. When the patient is lying down, palpate, percuss and auscultate the anterior aspect of the chest. When he or she is sitting forwards, palpate, percuss and auscultate the posterior aspect, and examine for lymphadenopathy at the same time.

Lobectomies and pneumonectomies

These are very common in OSCEs as patients are usually stable and ambulant, and the examination findings are obvious. Students are often surprised when they do not hear decreased breath sounds at the site of lobectomy scars, which they may have done during their ward attachments. This is because, after a few months or years, patients with lobectomies develop

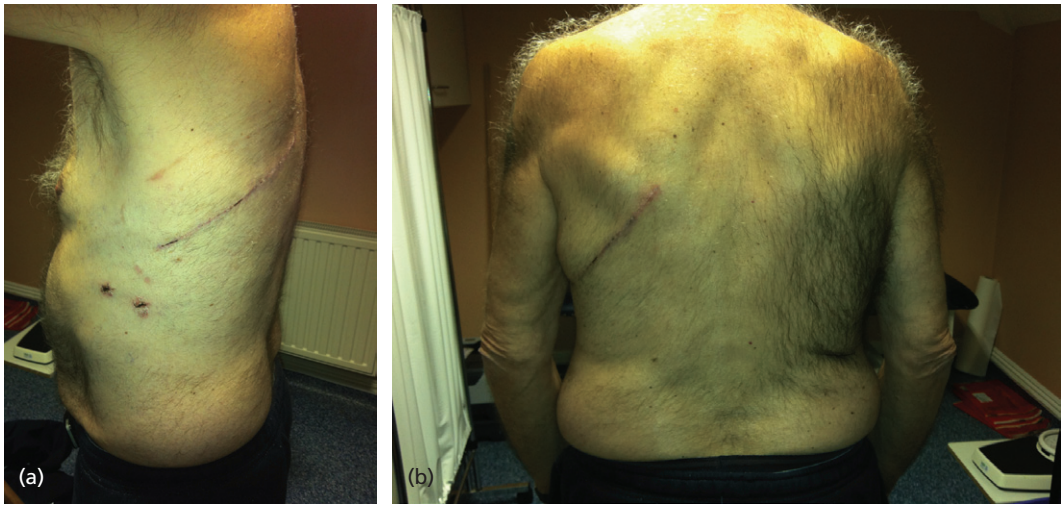

Figure 2.1 Lobectomy scar: side view (a) and back view (b)

compensatory hyperinflation, and lung tissue fills up areas it was removed from. This will not be the case immediately after lobectomy surgery as sufficient time has not surpassed for compensatory hyperinflation to occur.

The scar from a pneumonectomy can be very similar to the scar from a lobectomy (Figure 2.1), although they can immediately be distinguished by the fact that chest expansion and breaths sounds are usually completely absent on the side of a chest that has undergone a pneumonectomy.

'Creps and clubbing'

Remember that bilateral crepitations and clubbing that occur together most commonly present in patients with bronchiectasis or pulmonary fibrosis.

Questions you could be asked

Q. Why are spontaneous pneumothoraces more common in tall men?

A. There are a number of theories for this. One is that the difference between the intrapleural pressure of the apex and the base is greater in taller people, making it easier for a pneumothorax to form spontaneously. Another is that any anatomical defects or blebs will become more stretched if the length if the lung is longer, as is the case in taller individuals.

Q. Why might you hear breath sounds over an area of the lung that has been excised in a lobectomy?

A. See 'Lobectomies and pneumonectomies' above.

Q. Name three causes of bibasal crepitations with clubbing in a patient.

A. See 'Creps and clubbing' above.

3 Abdominal

Checklist	P	MP	F
HELP:			
H: 'Hello' (introduction and gains consent)			
E: Exposure (nipples to knees/down to groins)			
L: Lighting			
P: Positions correctly (supine), asks if patient is in any pain			
Washes hands			
Inspects from end of bed for relevant paraphernalia (e.g. nutritional supplements, CAPD device)			
Inspects patient:			
• Body habitus (BMI, Cushingoid from immunosuppressants following organ transplant)			
• Pallor (anaemia)			
• Jaundice			
• Pigmentation (Addison's disease, Peutz–Jeghers syndrome, 'bronze'/slate grey in haemochromatosis, drugs)			
• Bruising			
• Tattoos			
• Peripheral skin lesions associated with IBD (erythema nodosum, pyoderma gangrenosum)			
Hands:			
• Clubbing (IBD, malignancy, malabsorption states such as coeliac disease, liver cirrhosis)			
• Dupuytren contracture			
• Palmar erythema			
• Leukonychia (iron deficiency)			
• Koilonychia			
• Liver flap			
Arms:			
• Arteriovenous fistula (for dialysis) – auscultate for bruit			
• Tattoos			
Eyes:			
• Jaundice			
• Anaemia			
• Xanthelasmata			
Face:			
• Parotid enlargement (alcohol excess)			
Mouth:			
• Angular stomatitis (iron/vitamin B deficiency)			
• Glossitis (vitamin B deficiency)			
• Peri-oral pigmentation (Peutz–Jeghers syndrome), telangiectasia			
• Ulcers (IBD)			
• Dehydration			
• Dental hygiene			
• Smell of breath (hepatic fetor, uraemia)			
Supraclavicular lymph nodes (Virchow's node/ Troisier's sign for stomach cancer)			
Chest:			
• Gynaecomastia • Spider naevi (more than five is significant)			
Inspects abdomen:			
• Scars (see Figure 3.4) • Drain insertion sites • Peristalsis/pulsations • Caput medusae • Distension • Masses/swellings • Stretch marks/striae			
Palpates abdomen (ideally kneeling down): • Superficial palpation in nine quadrants for masses and tenderness • Deep palpation in nine quadrants for masses and tenderness • Hepatomegaly • Splenomegaly • Ballots kidneys • Abdominal aortic aneurysm			
Percusses abdomen: • Liver • Spleen • Ascites with shifting dullness • Bladder (dull if full, e.g. in urinary retention)			
Auscultates for bowel sounds, renal bruits, abdominal aortic aneurysm			
Examines for shifting dullness/ascites			
Examines lower legs for oedema			

Tells examiner he would like to complete the examination by examining the following: • Hernial orifices (with cough/sitting up) • Genitalia • Rectum • Lymph nodes • Urine dipstick			
Thanks patient			
Offers to help patient get dressed			

Washes hands			
Presents findings			
Offers appropriate differential diagnosis			
Suggests appropriate further investigations and management			
OVERALL IMPRESSION:			

Summary of common findings seen in OSCEs

- Chronic liver disease
- Hepatomegaly
- Splenomegaly
- Nephrectomy scar/features of end-stage renal failure (ESRF)
- Enlarged kidneys
- Transplanted kidneys
- Ascites
- Hernia
- Stoma
- Surgical scars

Summary of common conditions seen in OSCEs

Common chronic conditions	Chronic liver disease	Inflammatory bowel disease	Renal disease/ESRF
Examination findings			
General inspection	Malnourished Bruising (impaired clotting)	Cushingoid appearance (from steroids)	Cushingoid appearance (from steroids) CAPD paraphernalia
Hands/arms	Clubbing Palmar erythema Dupuytren contracture Liver flap (in hepatic encephalopathy) Leukonychia (due to hypoalbuminaemia)	Clubbing Tubing for total parenteral nutrition Leukonychia/ koilonychia	Arteriovenous fistula (listen to bruit) Elevated blood pressure Renal osteodystrophy
Face	Jaundiced sclera (if decompensated) Parotid enlargement (if liver failure caused by excess alcohol intake)	Mouth ulcers Temporalis muscle wasting	Gum hypertrophy (ciclosporin) Anaemia Collapsed nasal bridge (Wegener's granulomatosis) Molluscum (immunosuppression) Viral skin warts/skin cancers Butterfly rash (if SLE) Hearing aid (if Alport syndrome)
Neck	Raised JVP (if fluid overload secondary to hypoalbuminaemia)		Parathyroidectomy scar (after tertiary hyperparathyroidism) Raised JVP Cushingoid neck

(*Continued*)

Common chronic conditions	Chronic liver disease	Inflammatory bowel disease	Renal disease/ESRF
Chest	Reduced hair Gynaecomastia Spider naevi		Right internal jugular/subclavian tunnelled intravenous line/scar CABG scar (may indicate atherosclerosis causing renovascular disease)
Abdomen	Jaundice (if decompensated) Ascites (if portal hypertension) Hepatomegaly Splenomegaly (in portal hypertension) Caput medusae	Surgical scars Liver transplant scar (from primary sclerosing cholangitis) Fistulas Stomas	Nephrectomy scars (if renal transplant/dialysis) Enlarged kidneys (if adult polycystic kidney disease) Transplanted kidney palpable in iliac fossa/near groin CAPD scars Injection sites (from subcutaneous insulin) Cushingoid features (if immunosuppression with steroids)
Legs	Peripheral oedema (hypoalbuminaemia)	Pyoderma gangrenosum Erythema nodosum	Peripheral oedema
Key investigations	Liver function tests Clotting and albumin (for synthetic liver function) Alcohol screen Abdominal ultrasound Viral hepatitis screen Autoimmune hepatitis screen Viral serology screen Liver biopsy Oesophago-gastro-duodenoscopy (to look for varices if portal hypertension suspected)	Inflammatory markers Colonoscopy Stool microscopy, culture and sensitivity	Urinalysis (including albumin creatinine ratio) Us+Es and glomerular filtration rate Nephritic/vasculitic screen Renal ultrasound IVU/CT kidneys, ureter and bladder Renal biopsy
Key management principles	Treat underlying cause Stop all hepatotoxic medications Nutritional support Salt restriction Monitor fluid status and input/output Vitamin B/folate supplements Lactulose Monitor blood glucose Monitor Glasgow Coma Scale score Treat clotting abnormalities Assess for portal hypertension (splenomegaly/ascites/caput medusae) → if present do oesophago-gastro-duodenoscopy for varices	Steroids (topical/enema/oral) Mesalazine/ azathioprine/ anti-TNF Assess for toxic megacolon Monitor inflammatory markers Metronidazole for perianal disease Nutritional support/ elemental diet Surgery	Treat underlying cause Stop all nephrotoxic medications Nutritional support Salt restriction Monitor fluid status and input/output Calcium supplements (if hypocalcaemic) Phosphate binders (if high phosphate) Monitor parathyroid hormone level (consider parathyroidectomy if tertiary hyperparathyroidism) Monitor blood gases and treat acidosis Monitor Hb (consider erythropoietin/iron if anaemic) Optimise blood pressure (ACE inhibitor) and cholesterol

Common conditions leading to chronic liver disease

To make things easier, we have summarised here the key clinical features and investigations of chronic liver disease that you can use in the viva/questions part at the end of the OSCE generically, regardless of what the cause of the liver disease is. Table 3.1 outlines common conditions leading to chronic liver disease – the most common ones are marked with an asterisk. This will be especially useful for students aiming for a merit or distinction, as it helps to diagnose not only chronic liver disease, but also the underlying cause.

Hints and tips for the exam

Hepatomegaly and splenomegaly

Hepatomegaly and splenomegaly are also very common findings at this station in finals. We have discussed various key tips below to help you in both the diagnosis and the discussion.

Examining large livers and spleens

• Start low in the right iliac fossa, so that you do not miss giant organomegaly.

Table 3.1 Common conditions leading to chronic liver disease

Common causes of chronic liver disease	Key points in history	Collateral 'clues'	Specific investigations to identify cause
*Alcohol	Alcohol intake CAGE	Rib fractures on chest X-ray	High AST:ALT ratio High MCV
*Hepatitis B and C	Sexual history Intravenous drug abuse Blood transfusions Travel abroad	Tattoos Scars from intravenous access	Hepatitis serology
Primary biliary cirrhosis	Xanthelasmata Pigmentation Clubbing Excoriation marks	Female (>90%) Middle-aged Features of autoimmune/connective tissue/rheumatological diseases Features of immunosuppression (Cushing's disease, molluscum contagiosum)	↑ IgM Antimitochondrial antibodies Cholestatic liver profile (↑ ALP) Liver biopsy
Autoimmune hepatitis	Musculoskeletal pain	Features of autoimmune/connective tissue/rheumatological diseases Features of immunosuppression (Cushing's disease, molluscum)	↑ IgG Antinuclear antibodies Anti-smooth muscle antibodies Liver biopsy
Primary sclerosing cholangitis	Past medical history of or active IBD	Features of IBD (usually ulcerative colitis) Bowel surgery scars Stoma	pANCA ERCP/MRCP Liver biopsy Cholestatic liver profile (↑ ALP)
Wilson's disease	Family history (autosomal recessive inheritance)	'Bronze' skin pigmentation Marked tremor Kayser–Fleischer rings in iris Dysarthria/cognitive impairment	Serum copper 24-hour urinary copper excretion
Haemochromatosis	Family history (autosomal recessive inheritance) Diabetes Arthritis Hypopituitarism	'Slate grey' pigmentation 'Bronzing' of the skin Gonadal atrophy Gynaecomastia	Serum iron studies Liver biopsy
Fatty liver/non-alcoholic steatohepatitis)	Xanthelasmata	Hypertension CABG scar	Ultrasound Lipids
Heart failure	Past medical history of heart disease/hypercholesterolaemia	Signs of heart failure	Echo

• Use the radial aspect of your index finger – but if that doesn't work, use your finger with your hands pointing up towards the patient's head.
• Keep your fingers absolutely still as the patient breathes in and out.
• Make sure that you move your hand upwards superiorly by no more than 2 cm as the patient breathes in and out. If you leave too large a distance as you move up, there is a risk that you may miss the edge of the liver or spleen.
• For the liver, percussion is almost as discriminatory as palpation. It is also useful to differentiate between lung hyperinflation pushing the liver down, and true hepatomegaly. The superior aspect of the liver usually lies between the 4th and 6th ribs, and continues down to the last rib at the inferior border of the rib cage; hence, there should be dullness in all of this area. Hyperinflation pushing down the liver is confirmed if percussion is resonant significantly below the 6th rib.
• For the spleen, use your left hand to stabilise the left ribs in order to prevent them from being pushed towards the left as you palpate the spleen with your right hand. If you still have difficulty, roll the patient on to the right side and repeat this.
• When you do find an enlarged liver or spleen, estimate the size of hepatomegaly in centimeters rather than 'finger breadths', which vary from person to person (depending on how big their fingers are!).
• Avoid the business of trying to identify the liver characteristics (e.g. whether it 'firm', 'hard' or 'soft', or pulsatile, or nodular or smooth). Doing this in an exam will make the patient uncomfortable and use up your valuable time without achieving very much. Once a large liver or spleen has been identified, the most logical way of defining its characteristics would be to carry out some sort of imaging – usually an ultrasound of the abdomen.

Systematic differentiation of the underlying causes of hepatomegaly and splenomegaly

• A large liver and/or spleen is a very common finding at finals OSCE stations. Make sure that you have a generic system for categorising the causes, so that you can reel off a list of differential diagnoses quickly, confidently and systematically.
• Always try to use all the signs to help you devise a differential diagnosis. However, if you find an enlarged spleen or liver and have no clue what the cause is, go for conditions that can cause hepatomegaly and splenomegaly either individually or together – the first column of Table 3.2 summarises these.
• Don't be too pedantic when distinguishing between gigantic, moderate and mild splenomegaly. Identifying splenomegaly and giving a reasonable list of differential diagnoses and investigations will usually be enough to score a decent pass. Distinguishing between mild/moderate and gigantic splenomegaly will help to get you into the merit/distinction range. Remember that the spleen has to be at least double or triple its normal size to be palpable.
• Remember to piece the other parts of your examination together to complete the diagnostic jigsaw. All the conditions that cause hepatomegaly or splenomegaly have several peripheral signs so look out for these and use them to support your differential diagnosis.

Renal cases

Although students often worry about getting a 'renal case' in finals, it can often be a blessing in disguise. The differential diagnosis is relatively straightforward, and the signs are easy to elicit.

Fundamentally, there are only two findings in renal cases – those of ESRF, and ballotable enlarged kidneys.

End-stage renal failure

There are potentially three findings that are all attributable to ESRF:
• **Nephrectomy scar** (Figure 3.2)**:** Inspect carefully for this, making sure that you look all the way around the lumbar/flank regions through to the back. Finding a nephrectomy scar is alone sufficient to devise a full list of differential diagnoses and a management plan.
• **Palpable transplanted kidney:** This is usually near the groin/iliac fossa with a small scar at the site.
• **Signs of dialysis use** (arteriovenous fistula, right internal jugular vein line, CAPD scars; Figure 3.3)**:** A slicker way of describing this is 'renal replacement therapy', which covers them all – and also sounds more impressive!

Whichever of these signs the patient has, the underlying condition is always ESRF.

The four most common causes of ESRF are as follows:
1) Diabetes
2) Hypertension
3) Adult polycystic kidney disease (APKD)
4) Glomerulonephritis

Once you have got to this stage, your investigations and management should be guided by your differential diagnosis. However, if you are still struggling, merely discuss the generic investigations and management strategies for patients with ESRF, as discussed in the summary table above.

Table 3.2 Causes of hepatomegaly and splenomegaly

	Hepatosplenomegaly	Hepatomegaly only (without splenomegaly)	Splenomegaly only (without hepatomegaly)			Peripheral signs
			Gigantic splenomegaly (palpable in right lower quadrant)	Moderate splenomegaly (5–10 cm)	Mild splenomegaly (2–5 cm)	
Malignancy	All haematological malignancies (myeloproliferative and lymphoproliferative)	Hepatocellular carcinoma Secondary metastases	Chronic myelogenous leukaemia Myelofibrosis	All haematological malignancies (myeloproliferative and lymphoproliferative)	All haematological malignancies (myeloproliferative and lymphoproliferative)	Lymphadenopathy Cachexia Anaemia Bruising and purpura
Infective	Viral hepatitis CMV Toxoplasmosis Malaria Schistosomiasis Histoplasmosis Brucellosis Leptospirosis Kala-azar Weils disease Hydatid disease	Viral hepatitis	Chronic malaria Visceral leishmaniasis	All infectious causes of hepatosplenomegaly	Glandular fever Brucellosis Viral hepatitis Early sickle cell disease HIV	Pyrexia Recent foreign travel Tattoos/intravenous drug abuse scars (viral hepatitis)
Infiltrative	Sarcoidosis Amyloidosis Gaucher's disease	Fatty liver/NASH Haemochromatosis	Gaucher's disease	Gaucher's disease	Sarcoidosis Amyloidosis Gaucher's disease	Sarcoid skin disease
Inflammatory	–	–	–	Felty's syndrome (rheumatoid arthritis, neutropenia, splenomegaly)	Rheumatoid arthritis SLE	Arthropathy Butterfly rash of SLE
Liver disease	Liver disease with portal hypertension	Any cause of chronic liver disease (as above)	–	Portal hypertension	Liver cirrhosis with portal hypertension	Signs of chronic liver disease (as above)
Cardiovascular	–	Right heart failure Tricuspid regurgitation	–	–	Infective endocarditis Constrictive pericarditis	Ankle oedema, raised JVP Haematuria and peripheral signs of endocarditis
Miscellaneous	–	Polycystic kidney disease (causing liver cysts)	–	–	Haemolytic anaemias (autoimmune, hereditary spherocytosis) Thalassaemia	Ballotable kidneys/ nephrectomy scar Jaundice (from haemolysis)

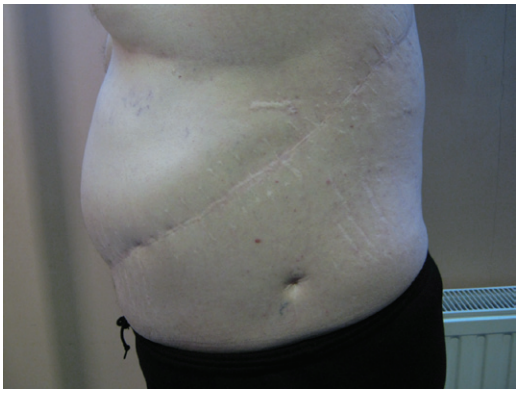

Figure 3.1 Scar from splenectomy after a road traffic accident, also showing the drain insertion site

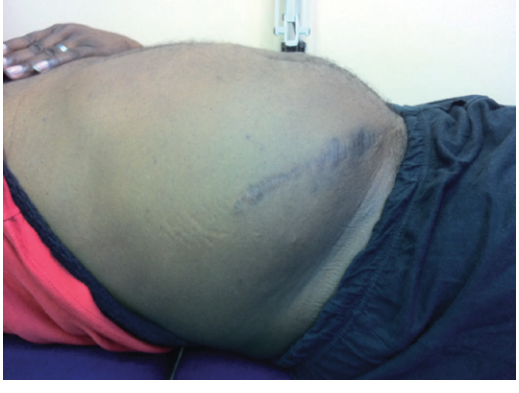

Figure 3.2 Nephrectomy scar

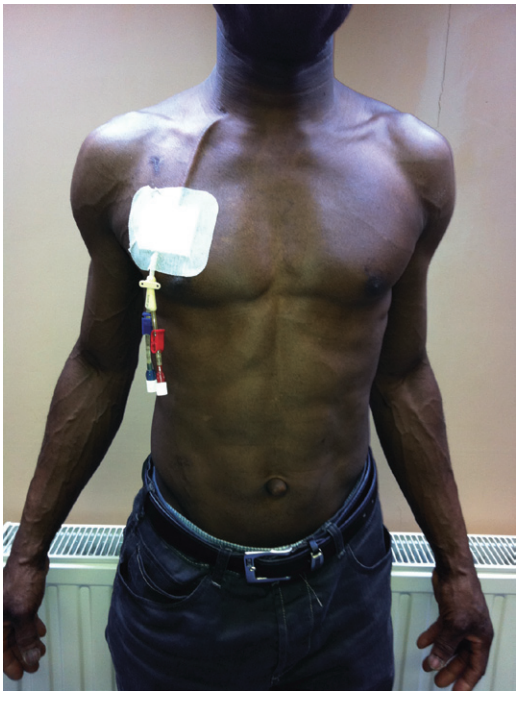

Figure 3.3 Right internal jugular tunnelled catheter (for dialysis)

Ballotable/ enlarged kidneys

Ballotable enlarged kidneys can be palpated in the lateral lumbar regions. As with ESRF, you only need to remember a short list of differential diagnosis:

- APKD
- Renal cell carcinoma
- Bilateral hydronephrosis (secondary to obstruction, e.g. by an external mass, prostate enlargement, etc.)
- Amyloidosis (primary or secondary)

The key investigations with all of these are imaging (CT of the kidney, ureter and bladder/IVU) and renal biopsy, with the management depending on the underlying cause.

Rare findings

- **Clubbing versus pseudoclubbing:** Although these conditions look similar on examination, the underlying causes are fundamentally different. Pseudoclubbing occurs because of tertiary hyperparathyroidism and although it looks like clubbing, with prominence of the distal phalanges, what actually happens is that the proximal phalanges become narrow, and this makes the distal phalanges look prominent despite being normal.

  Pseudoclubbing is common after renal replacement therapy – patients with long-standing secondary hyperparathyroidism (due to low calcium levels) develop parathyroid hyperplasia, leading to increased parathyroid hormone production that becomes autonomous of the negative feedback system. Once a patient is undergoing renal replacement therapy and their calcium levels normalise, the parathyroid continues producing excess parathyroid hormone, which results in hypercalcaemia and resorption of bone from the proximal phalanges, causing them to narrow.
- **Chronic liver disease and features of ESRF in the same patient:** This is rare, but don't let it put you off. The most likely cause is hepatitis C (leading to chronic liver disease), which also causes membranous glomerulonephritis (leading to ESRF).
- **Spleen versus kidney:** When palpating the left side of the abdomen, it can sometimes be difficult to distinguish a ballotable kidney from a spleen. Table 3.3 below summarises the key differences.

Theoretically, the spleen should be dull while the kidney has traditionally been documented in most texts to be 'resonant'. This is, however, more theoretical than realistic as in practice both kidneys and spleens feel dull on percussion.

Abdominal scars

As with all OSCEs, the key findings in abdominal examination are often established on inspection (Figure 3.4):

1) Rooftop scar
   - Partial hepatectomy
   - Pancreatic surgery
   - Accessing aorta
2) Kocher incision
   - Cholecystectomy
3) 'Mercedes-Benz' scar
   - Liver transplant
   - Gastric surgery
   - Oesophageal surgery
4) Midline laparotomy
   - Colon surgery
   - Aortic abdominal aneurysm surgery
5) Nephrectomy scar (rarely adrenalectomy scar)
   1. Classic caesarean section scar/hysterectomy scar
   2. Appendicectomy scar: at McBurney's point
   3. Caesarean section scar (suprapubic)
   4. Inguinal hernia scar
   5. Femoral hernia scar

Table 3.3 Spleen or kidney?

Spleen	Kidney
Cannot get above the spleen	Should be able to get above the kidney
Moves downwards and medially with inspiration	No movement with breathing
Not ballotable	Ballotable
Palpable notch (medial aspect)	No notch

Abdominal masses

If you find a mass, try to answer two questions in your mind.

1. Where is the mass?

First identify the quadrant where the mass is located, and then think of the organs in that quadrant from which the mass might originate (Figure 3.5).

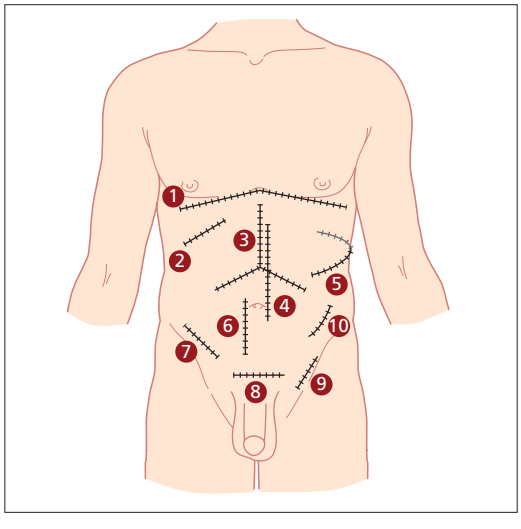

Figure 3.4 Common abdominal scars

Lung Liver Gallbladder	Stomach Pancreas Abdominal aorta	Lung Spleen Pancreas (rarely)
Liver Kidney Ureter	Stomach Small intestine/ transverse colon Abdominal aorta	Spleen Kidney Ureter
Ureter Ovary Fallopian tube Caecum (appendix)	Bladder Uterus Cervix (referred pain from testicles)	Ureter Ovary Fallopian tube Sigmoid colon

Figure 3.5 Location of organs in the abdomen

Remember to describe the mass accurately and logically – see Chapter 10 on breast examination for a table of characteristics that you should aim to describe.

2. What is the lesion?

As with everything in OSCEs, the key is to have a generic method of categorising potential differential diagnoses. The categories below can be used to devise a differential diagnosis for a mass in almost any of the nine quadrants:

- Tumour
  - Benign
    - **i.** Cyst (liver, renal)
    - **ii.** Fibroids (in the pelvic area in women)
    - **iii.** Vascular (abdominal aortic aneurysm)
  - Malignant
    - **i.** Primary
    - **ii.** Secondary
    - **iii.** Lymphoma
- Infection
  - Abscess
  - Tuberculosis (usually ileocaecal)
- Inflammatory bowel disease
  - Crohn's disease (in right iliac fossa)
  - Diverticular disease (left iliac fossa)

Key investigations

The crux of investigating a mass is to visualise it and to get a tissue sample from it. Hence the following investigations are most important:

- **Imaging:** CT/MRI scan
- **Endoscopy:** colonoscopy for colon, oesophago-gastro-duodenoscopy for oesophagus/stomach, cystoscopy for bladder
- **Biopsy:** for any non-vascular mass

Stomas

Stomas (Figures 3.6 and 3.7) feature more commonly in finals than most students think, and they are actually quite easy to examine and talk about. The most common stomas are ileostomies and colostomies, and the key feature that distinguishes them is their location. Table 3.4 summarises the key features.

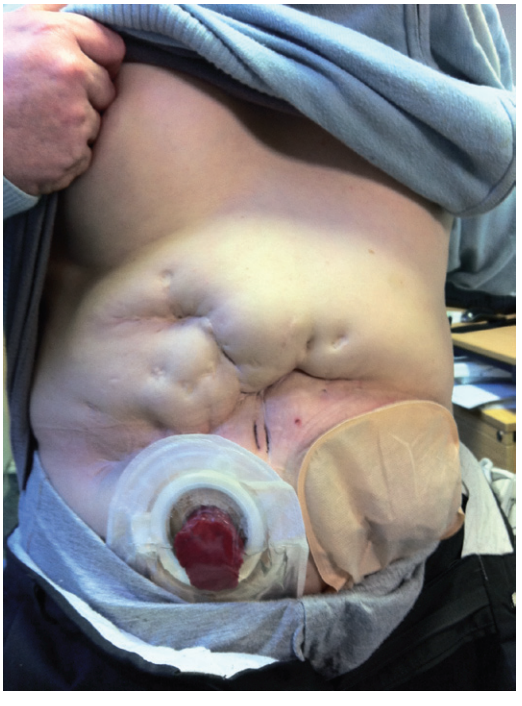

Figure 3.6 Stoma

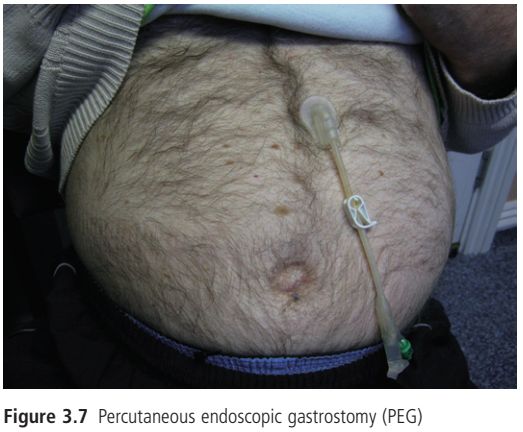

Figure 3.7 Percutaneous endoscopic gastrostomy (PEG)

Questions you could be asked

Q. What is the one investigation you would do in a patient with known portal hypertension in order to reduce mortality?

A. The key features of portal hypertension are:

- Splenomegaly
- Ascites
- Caput medusae
- Oesophageal varices

Although the answer to this is debatable, the most important investigation would be an oesophago-gastro-duodenoscopy to identify varices, and more importantly to band them and prevent torrential acute severe gastrointestinal bleeding.

Q. How big does the spleen have to be before it is palpable?

A. About twice its normal size.