On Call
Principles & Protocols
AUSTRALIAN AND NEW ZEALAND 3RD EDITION

Australian adaptation by
Anthony FT Brown MB ChB, FRCP, FRCS (Ed), FACEM, FRCEM
Professor, Discipline of Anaesthesiology and Critical Care,
School of Medicine MD Program, University of Queensland, Brisbane.
Senior Staff Specialist (Pre-Eminent Status), Department of Emergency
Medicine, Royal Brisbane and Women’s Hospital, Brisbane.

Mike Cadogan MA (Oxon), MB ChB, FACEM
Staff Specialist in Emergency Medicine, Department of
Emergency Medicine, Sir Charles Gairdner Hospital, Perth.
Team Physician, Wallabies and the Western Force.

Antonio Celenza MBBS, MClinEd, FACEM, FRCEM
Professor of Emergency Medicine and Medical Education,
Faculty of Medicine, Dentistry and Health Sciences,
University of Western Australia, Perth. Staff Specialist,
Department of Emergency Medicine, Sir Charles Gairdner
Hospital, Perth.

Original edition by
Shane A Marshall MD, FRCPC; Director of Cardiac Care,
Chief of Medicine, King Edward the VIIth Memorial Hospital,
Paget, Bermuda

John Ruedy MDCM, FRCPC, LL.D (Hons); Professor (Emeritus)
of Pharmacology, Faculty of Medicine, Dalhousie University,
Halifax, Canada
# Contents

Foreword ix  
Preface to the third edition xi  
About the authors xiii  
Dedication xiv  
Acknowledgements xv  
Reviewers xvi  
Abbreviations xvii

## Section A – General principles
1. Approach to the diagnosis and management of on-call problems 2  
2. Professionalism and teamwork 5  
3. Documentation and communication 12  
4. Ethical and legal considerations 16  
5. Death, dying and breaking bad news 21  
6. Preparation of patients for transfer 30

## Section B – Emergency calls
7. The critically ill patient 36  
8. Cardiac arrest 42  
9. Acute airway failure 50  
10. Acute respiratory failure 59  
11. Acute circulatory failure 65  
12. Disability: acute neurological failure 80  
13. Environment, exposure and examination 83  
14. Hospital-based emergency response codes 85

## Section C – Common calls
15. Shortness of breath, cough and haemoptysis 89  
16. Chest pain 117  
17. Heart rate and rhythm disorders 135  
18. Hypotension 157  
19. Hypertension 171  
20. Altered mental status 181  
21. Collapse, syncope and mechanical falls 193  
22. Headache 203  
23. Seizures 215  
24. Weakness and dizziness 225  
25. Abdominal pain 237  
26. Altered bowel habit 255
Contents

27 Gastrointestinal bleeding 267
28 Haematuria 278
29 Decreased urine output and acute kidney injury 283
30 Frequency and polyuria 291
31 Leg pain 298
32 Febrile patient 311
33 Skin rashes including allergic reactions 322
34 Transfusion reactions 334

Section D – Investigations
35 Electrocardiogram 341
36 Chest X-ray 349
37 Abdominal X-ray 355
38 CT head scan 360
39 Urinalysis 365
40 Acid–base disorders 372
41 Glucose disorders 382
42 Sodium disorders 392
43 Potassium disorders 399
44 Calcium disorders 406
45 Anaemia 413
46 Coagulation disorders 419

Section E – Practical procedures
47 General preparation for a practical procedure 434
48 Infection control and standard precautions 438
49 Venepuncture 442
50 Blood cultures 445
51 Peripheral venous cannulation 448
52 Arterial puncture 454
53 Administering an injection 458
54 Local anaesthetic infiltration 465
55 Nasogastric tube insertion 468
56 Urinary catheterisation 471
57 Paracentesis 476
58 Pleural tap 480
59 Chest drain insertion and removal 484
60 Lumbar puncture 488
61 Joint aspiration 493
62 Cardiac monitoring and the electrocardiograph 496
63 Defibrillation 501
64 Electrical cardioversion (DC reversion) 505
65 Transthoracic cardiac pacing 507
66 Central venous cannulation 509

Section F – Formulary
67 On-call formulary 519

Section G – Laboratory values
68 Normal laboratory ranges 625
Foreword

This book is a treasure trove of useful, up-to-date, practical information for newly qualified doctors responding to hospital ward calls. Indeed, such is the scope of its content, many senior doctors in various fields within acute medicine will find it an invaluable resource to have on hand for everyday practice. All three authors are among the finest teachers of emergency medicine in Australasia, with complementary and widely recognised experience in translating knowledge into the clinical performance of students and junior doctors. The book is remarkably well organised, with a clear and easy-to-follow structure that belies the great depth of information provided. It is so relevant to the concerns of junior doctors, and so full of concise clinical wisdom, that it is frankly a joy to read. The book is a source of excitement for those of us who have spent our careers in acute medicine and watched junior staff come and go in the sometimes chaotic and confusing hospital environment, and wished for some more structure and consistency in their teaching.

The authors provide clear guidelines on how to respond to a range of acute emergencies, illuminating the decision-making process in what can be difficult and challenging situations. Few textbooks discuss what might go through one’s mind on the way to an emergency; this one does. Similarly, there is often little attention given to what does not need to be done in such emergencies and what is frankly wasting valuable time; this book teaches students and young doctors how to prioritise clinical assessments so that the important issues are addressed in a logical and timely sequence.

The table of contents gives a welcome indication of the importance and priority assigned to highlighting professional, ethical and end-of-life issues before any discussion of managing the critically ill patient. Junior doctors would do well to follow this lead in the development of their careers. The authors have done a great service to acutely ill hospital patients and their attending medical staff by producing this wonderful book. It should make the hospital experience a whole lot better for all concerned! If only a book like this could have been around when I was a junior doctor.

Professor George A Jelinek, MD, DipDHM, FACEM
Professor and Head
Neuroepidemiology Unit
Melbourne School of Population and Global Health
The University of Melbourne, Victoria
Preface to the third edition

Purpose
This new edition provides a structured approach to the initial assessment, resuscitation, differential diagnosis and short-term management of common on-call problems. It is designed to help junior doctors and senior medical students acquire a logical, practical and efficient system, which is essential for problem-based learning and acute management. The entire text has been standardised and updated to include the latest evidence-based guidelines, to optimise both the internal consistency and the external validity.

Clinical problem-solving is a fundamental skill for the doctor on call. Traditionally, the diagnosis and management of a patient’s problems are approached with an ordered, structured and sequential system (e.g. history-taking, physical examination and review of available investigations) before formulating the provisional and differential diagnoses and the management plan.

In an emergency, doctors proceed concurrently with resuscitation, history, examination, investigation and definitive treatment. Stabilisation of the airway, breathing, circulation and neurological disability must occur in the first few minutes to avoid death and disability.

This book provides a focused approach to many clinical problems in order to increase efficiency and improve time management.

Structure
Additional reading material, high-quality images, procedural videos and references have been integrated online at http://lifeinthefastlane.com/book/oncall

The book is divided into seven main sections:
A. General principles
   An overview of the knowledge and skills that are required to deal with undifferentiated on-call problems.
B. Emergency calls
   Life-threatening, time-critical problems involving airway, breathing, circulation, neurological disability and environmental factors (ABCDE). This section outlines a structured approach to managing these emergency situations.
C. Common calls
   These are the calls associated with changes in symptoms or signs
   that most commonly require review while on call.

D. Interpretation of common investigations

E. Practical procedures

F. Formulary
   A compendium of commonly used medications that are likely to
   be prescribed by the doctor on call. It is a quick reference for dosages,
   routes of administration, adverse effects, contraindications and
   modes of actions.

G. Laboratory values
   A list of the normal reference ranges for all common laboratory
   investigations.

Within Section C – Common calls, the chapters are further subdivided
into:
• Phone call (pertinent questions to ask the ward)
• Corridor thoughts (differential diagnosis)
• Major threat to life (now highlighted in red)
• Bedside (first actions)
• Management (with immediate management also now highlighted
   in red).

This practical guide to rapid, efficient and effective clinical problem-
solving is described in more detail in Chapter 1.

**Being a doctor on call**
Being ‘on call’ is an invaluable part of medical training and practice,
even if only appreciated in retrospect! It undoubtedly grows the doc-
tor’s maturity, competence and confidence by:
• Obtaining experience in rapid, focused patient assessment and
  emergency treatment
• Honing clinical skills assessing patients with acute pathology
• Encouraging independent thought and actual decision making
• Improving procedural competence
• Providing increased responsibility.

*Anthony Brown*
*Mike Cadogan*
*Antonio Celenza*
Chest pain

Coronary artery disease (CAD) is the leading cause of death in developed countries. A patient with CAD may present critically ill with an arrhythmia, heart failure or hypotension (cardiogenic shock), or may be stable and appear deceptively well, yet still be at risk of sudden death. Thus, when a patient complains of ‘chest pain’, a cardiac cause due to acute coronary syndrome (ACS), which includes the spectrum from acute myocardial infarction (AMI) to angina, must be considered first.

There are several other equally serious causes of chest pain, such as PE or aortic dissection, that may go undiagnosed if they are not also specifically looked for. The history is the most important aspect in the differential diagnosis of chest pain.

**Phone call**

**Questions**
1. What is the character of the pain? Does it change with breathing?
2. Where is the pain maximal and does it radiate?
3. How severe is the pain?
4. What are the vital signs?
5. What was the reason for admission?
6. Does the patient have a history of ischaemic heart disease? If ‘Yes’, is the pain similar to their usual angina or a previous MI?

**Instructions**

If you suspect AMI or angina (heavy, crushing, tight pain radiating to jaw or left or right arm):
- Ask the nurse to stay by the patient’s bedside and call for additional nursing staff if necessary.
- Give oxygen if hypoxic or shocked to maintain saturation $\geq 94\%$, after attaching a pulse oximeter and cardiac monitor to the patient.
- Request an urgent 12-lead ECG.
- Give aspirin 150–300 mg PO unless contraindicated by hypersensitivity.
• Administer GTN SL (0.6 mg tablets or 0.4 mg spray) and repeat every 5–10 minutes if the pain persists, provided SBP remains >90 mmHg.
• Request an IV trolley for the patient’s bedside, with a range of cannulae ready for insertion.

Prioritisation
Attend any patient with chest pain immediately, especially those with abnormal vital signs, particularly an arrhythmia or hypotension.

Corridor thoughts

What causes chest pain? (* = major threat to life)
• **Cardiac**
  • Acute coronary syndrome (ACS)*
  • Acute aortic dissection (AAD)*
  • Pericarditis
• **Respiratory**
  • Pulmonary embolism (PE)*
  • Pleurisy
  • Pneumonia
  • Pneumothorax
  • Pneumomediastinum
• **Gastrointestinal tract**
  • Oesophagitis
  • Oesophageal spasm
  • Ruptured oesophagus*, including Boerhaave’s syndrome due to vomiting
  • Hiatus hernia
  • Peptic ulcer disease
  • Biliary colic/cholecystitis
  • Subdiaphragmatic irritation
• **Musculoskeletal**
  • Costochondritis
  • Muscular pain
  • Fractured rib
  • Referred pain from spinal crush fracture or spinal disc disease
  • Herpes zoster or postherpetic neuralgia
• **Psychiatric**
  • Anxiety and panic disorders

Major threat to life
• **Acute coronary syndrome (ACS)**, including ST-elevation myocardial infarction (STEMI), non-ST-elevation myocardial infarction (NSTEMI) and unstable angina (UA).
• Heart failure, cardiogenic shock or ventricular or supraventricular arrhythmias may occur suddenly as a result of ACS, which can be fatal.
• **Acute aortic dissection (AAD)** may cause acute aortic incompetence, pericardial tamponade or aortic rupture, and involve other organs such as the CNS, renal and GI tracts by acute vascular occlusion (which can be intermittent).
• **Pulmonary embolus (PE)** causes hypoxia and, potentially acute right ventricular failure with obstructive shock and sudden death.

**Bedside**

**Quick-look test**

Does the patient look well (comfortable), sick (uncomfortable or distressed) or critical (about to die)?

- Patients with chest pain from ACS may look anxious and pale, or are shocked, breathless and clearly unwell.
- Patients with aortic dissection have severe pain and are restless and agitated.
- Patients with PE, pericarditis or pneumothorax are dyspnoeic and breathe with shallow, painful respirations (pleuritic pain).
- However, even if patients look well and appear comfortable at rest, they may still have a life-threatening underlying cause such as ACS, aortic dissection or PE.
- Oesophagitis, or a musculoskeletal cause such as costochondritis, is a diagnosis of exclusion, and is only made when all potentially life-threatening causes of chest pain have been considered and actively excluded.

**Airway and vital signs**

What is the blood pressure?

- Most patients with chest pain have a normal BP. Take the BP in both arms if the pain is suspicious of aortic dissection (sudden onset, sharp, tearing or migratory), and look for a difference of >15 mmHg.
- Hypotension occurs in ACS, massive PE and AAD with cardiac tamponade.
- Hypertension in association with ACS or aortic dissection should be treated urgently in a monitored resuscitation area (see Chapter 19).

What is the heart rate?

- If the HR is >150 beats/min or <40 beats/min, obtain a 12-lead ECG or rhythm strip to help diagnose the arrhythmia.
- Sinus tachycardia may result from chest pain of any cause. A heart rate >140 beats/min raises the possibility of atrial fibrillation or ventricular tachycardia, which require urgent cardioversion,
especially if associated with chest pain and or hypotension (‘symptomatic’ arrhythmias).

- Bradycardia with chest pain may represent sinus or atroventricular (AV) nodal ischaemia associated with ACS, or a drug effect such as calcium-channel blocker or beta-blocker use.
- Immediate treatment of bradycardia is not required unless the patient is hypotensive or syncopal and/or the rate is extremely slow (<40 beats/min).

**What is the respiratory rate?**

- Tachypnoea may accompany any type of chest pain.
- Shallow, painful breathing suggests a pleuritic or musculoskeletal cause.
- Dyspnoea from increased work of breathing occurs with respiratory causes of chest pain or acute LVF, and may lead to respiratory failure.

**What does the ECG show?** (see Chapter 35)

- Review the ECG immediately after assessing the vital signs. Compare with an old tracing if possible.
- ST-segment elevation or depression, T wave changes and the presence of new Q waves suggest myocardial ischaemia from ACS.
  - Only 50% of patients with ACS will have a diagnostic ECG, with non-specific or no initial ECG changes in the remainder (the diagnosis is then made on a rise in cardiac biomarker levels such as troponin).
- The most common ECG finding in a patient with PE is a sinus tachycardia. Additional changes of right-axis deviation, right bundle branch block, atrial fibrillation with rapid ventricular response or the SI QIII TIII phenomenon should be looked for.
  - **Note:** SI QIII TIII is well known, but is neither sensitive nor specific for PE!
- The ECG in a patient with pericarditis shows diffuse, concave ST-segment elevation with PR depression.
- Left ventricular hypertrophy with R wave and S wave in V1 > 35 mm may give a clue to long-standing hypertension, which is a risk factor for aortic dissection in particular, as well as ACS.
- A normal initial ECG does not rule out sinister pathology.
  - If the first ECG is non-diagnostic, repeat after 15 minutes if the chest pain is continuing to exclude evolving changes.
  - Repeat the ECG whenever the chest pain recurs, stops or changes in severity or character, as well as after 6–8 hours with repeat cardiac biomarkers (troponin) to rule out ACS.
  - Accelerated chest pain rule-out protocols now include repeat ECG and troponin testing within 2–3 hours. Check your local process.

If the patient has normal vital signs, the chest pain has resolved and the ECG is normal or only has non-specific changes, proceed to further history and examination.
Management

Immediate management

- If the patient still has chest pain or has respiratory distress, hypotension or an altered mental state:
  - Attach continuous non-invasive ECG, BP and pulse oximeter monitoring to the patient.
  - Commence oxygen if hypoxic or shocked to maintain oxygen saturation >94%. Give high-dose 40–60% oxygen unless there is a prior history of obstructive airways disease, in which case give 28% oxygen via a Venturi mask.
  - Establish IV access with two cannulae in peripheral veins. Draw and send bloods for FBC, U&E, cardiac biomarkers (troponin I or T) and coagulation profile.
  - Request a portable CXR.
- If the patient has chest pain and SBP >90 mmHg:
  - Give a second dose of GTN 300–600 micrograms SL, and a third after an additional 5 minutes if pain is still present.
  - Give morphine 2.5–5 mg IV with metoclopramide 10–20 mg IV for nausea, if pain persists despite two to three doses of GTN.
    — Note: morphine may also cause hypotension, as well as respiratory depression and drowsiness. Monitor the patient’s BP and respiratory rate carefully after each dose administered.
  - Maintain SBP >100 mmHg and avoid excessive hypotension.
- If the patient has chest pain and SBP <90 mmHg:
  - Check that the lung bases are clear to auscultation and give a bolus of 125–250 mL IV normal saline.
  - However, if the patient has signs of pulmonary oedema, with an S3 gallop, tachypnoea and basal crackles, treat as cardiogenic shock (see Chapter 11).
  - Call senior staff immediately, especially if there is:
    - Persistent hypoxia, hypotension or altered mental status despite the above measures.
    - HR >150 beats/min or <40 beats/min associated with hypotension, as cardioversion or pacing may be required.
    - Persistent pain and ST-segment changes on ECG, as urgent treatment for ACS is required (see below).

Selective history and chart review

A careful history of the pain is essential if the initial ECG is non-diagnostic.

How did the chest pain evolve?

- Crescendo, build-up of pain suggests ACS.
- Sudden, precipitate onset suggests PE, aortic dissection or oesophageal rupture.
Onset of pain with physical exertion or emotional stress suggests ACS.

Onset with coughing suggests a pneumothorax or a mechanical cause of pain (see Chapter 15).

Sudden pain following vomiting suggests oesophageal rupture (Boerhaave’s syndrome).

How does the patient describe the pain?

• Determine the degree of discomfort for the patient (e.g. score the pain out of 10, with zero being no pain at all and 10 being the worst pain the patient has ever experienced). This allows ongoing evaluation of the chest pain to determine the response to treatment.

• If the patient recognises the current discomfort as usual angina pain, assume this is correct.

• Crushing, vice-like or squeezing pain is characteristic of ACS, but note that ‘atypical’ pain still due to ACS occurs in women, elderly people and those with diabetes and renal impairment.

• Tearing or ripping pain is characteristic of an AAD. Likewise, severe pain that is poorly relieved by large doses of morphine suggests an aortic dissection or an oesophageal rupture.

• Sharp, well-localised pain suggests a pleuritic or musculoskeletal origin, but may occasionally occur with ACS or PE.

Where is the pain? Does the pain radiate?

• Central, retrosternal chest pain indicates a mediastinal or gastrointestinal origin.

• Lateral pain suggests lung, pleura, chest wall or neurological referred pain.

• Radiation of the pain to the jaw, shoulders or arms is suggestive of ACS.

• Radiation of the pain to the back suggests ACS, or less commonly an AAD distal to the left subclavian artery.

• Dissection proximal to the left subclavian artery more characteristically causes non-radiating anterior chest pain.

• Burning retrosternal chest pain radiating to the neck and throat suggests oesophageal reflux, but ACS must always be excluded first (mistaking ACS chest pain for indigestion is a common error in both patients and medical staff).

Are there any related symptoms?

• Nausea, sweating and light-headedness are non-specific but must be taken seriously.

• Collapse, or a syncopal episode, can indicate a sinister cause such as ACS, PE or AAD—again this must be taken seriously.

• Dyspnoea is usually related to a cardiorespiratory cause of pain or a resultant metabolic acidosis (such as a lactic acidosis in shock).
Does anything make the pain worse or better?

- Is the chest pain worse on coughing or deep breathing? Pleuritic chest pain suggests pneumothorax, pericarditis, PE, pneumonia, pleurisy, rib fracture or costochondritis.
- Pain worse on deep inspiration, when lying flat or when raising both legs (increases venous return) is suggestive of pericarditis.
- Pain worse with swallowing suggests an oesophageal source or pericarditis.
- Pain worse on particular movements, especially when muscular actions are resisted, suggests musculoskeletal pain or sometimes pneumothorax from rib injury.
- Relief of pain with antacids or GTN does not aid in the differential diagnosis. These should not be used as diagnostic tests, but as specific therapy once you have a working diagnosis.

Review past medical history and risk factors

Confirm a risk-factor profile or past medical history that may increase the likelihood (pre-test probability) of a particular cause of the chest pain:

- **ACS** is more likely with prior known ischaemic heart disease, increasing age >65 years, diabetes, hypertension, smoking, hyperlipidaemia, a family history of premature ischaemic heart disease in first-degree relative under age 50 years, or end-stage renal disease. Cocaine use and HIV are less common risk factors.
- **Aortic dissection** occurs in patients aged 60–80 years, usually males with chronic hypertension, or in younger patients with a connective tissue disorder, such as Marfan’s or Ehlers–Danlos syndrome, or a family history.
- **PE** is more likely with recent surgery, immobilisation, malignancy, prior thromboembolic episode (PE or DVT) or history of first-degree relative with thromboembolism, malignancy or chronic cardiorespiratory disease. Thus, a PE is common in hospital patients, with the possible exception of the paediatric ward.

Check the medication list

- Confirm prescribed antianginal medications have been given.
- Check for thromboembolic prophylaxis in immobilised or postoperative patients.
- Check for corticosteroids or NSAIDs as a possible cause of gastritis or oesophagitis.
## Selective physical examination

<table>
<thead>
<tr>
<th>Vitals</th>
<th>Repeat now</th>
</tr>
</thead>
<tbody>
<tr>
<td>Appearance</td>
<td>Does the patient look Marfanoid? A tall, thin patient with long limbs and arachnodactyly (&quot;spider fingers&quot;) may have a connective tissue disorder predisposing to aortic dissection</td>
</tr>
<tr>
<td>HEENT</td>
<td>Xanthelasma around the eyelids (may indicate hypercholesterolaemia, especially if familial) White exudate in oral cavity or pharynx (thrush with possible oesophageal candidiasis)</td>
</tr>
<tr>
<td>Resp</td>
<td>Asymmetrical expansion of the chest (pneumothorax, large pleural effusion or massive haemothorax) <strong>Tracheal deviation:</strong> Pushed away from large pneumothorax, large effusion and large haemothorax Pulled towards by collapse and consolidation <strong>Percussion:</strong> Hyperresonance (pneumothorax) Dullness to percussion (pleural effusion, haemothorax and consolidation) <strong>Auscultation:</strong> Diminished breath sounds (on the side of a pneumothorax) Crackles and/or wheezes (pulmonary oedema, PE or pneumonia) Pleural rub (PE or pneumonia)</td>
</tr>
<tr>
<td>Chest wall</td>
<td>Tender costal cartilage (costochondritis) <strong>Note:</strong> does not exclude another more serious diagnosis, as a patient with ACS may have chest wall tenderness Localised rib pain (rib fracture)</td>
</tr>
<tr>
<td>CVS</td>
<td>Unequal carotid pulses (aortic dissection) Unequal upper limb BP or diminished or absent radial or femoral pulse (aortic dissection) Elevated JVP (biventricular failure with CCF; right ventricular failure secondary to PE, tension pneumothorax or cardiac tamponade) Left ventricular heave (LVF) Right ventricular heave (acute RV failure secondary to PE) Displaced apical impulse (away from the side of pneumothorax; COPD) <strong>Auscultation:</strong> S₃ gallop (LVF), loud P₂ (acute RV failure secondary to cor pulmonale) Muffled heart sounds (cardiac tamponade, pericarditis)</td>
</tr>
</tbody>
</table>
Systolic murmur:
- Aortic stenosis (angina)
- Mitral regurgitation (acute papillary muscle or chordae tendinae dysfunction secondary to ACS)
- Late (mitral valve prolapse [Barlow’s syndrome] associated with systolic click and non-anginal chest pain)

Diastolic murmur:
- Early (aortic regurgitation associated with proximal aortic root dissection)
- Pericardial rub—biphasic systolic and diastolic scratching ‘squeaky new leather’ sound that varies with position (pericarditis)

<table>
<thead>
<tr>
<th>GIT</th>
<th>Guarding, rigid abdomen (perforated peptic ulcer)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Epigastric tenderness (PUD)</td>
</tr>
<tr>
<td></td>
<td>Generalised abdominal pain (mesenteric infarction from aortic dissection)</td>
</tr>
</tbody>
</table>

| CNS       | Hemiplegia or paraparesis (aortic dissection involving carotid or spinal arteries) |

| Skin      | Unilateral, vesicular maculopapular rash, dermatomal distribution (herpes zoster) |

**Investigations**

**Laboratory**
- Take blood for FBC, coags, BSL, U&E, LFTs and cardiac enzymes (troponin I or T). Send a D-dimer only for low pre-test probability PE (see Chapter 15).
- Anaemia may be associated with angina.
- BSL, U&E, LFTs will show evidence of diabetes and chronic renal disease (risk of ACS) and may be abnormal in cholecystitis.
- Cardiac biomarkers to rule out ACS—these must be repeated after 6–8 hours. Never send just one sample for troponin testing following chest pain.
- Accelerated chest pain rule-out protocols now include repeat ECG and troponin testing within 2–3 hours. Check your local process.

**Chest X-ray**

Review the CXR as soon as possible (see Chapter 36). The CXR is apparently normal in many conditions, such as uncomplicated ACS, PE and pericarditis. However, abnormal findings may include:
- LVF with upper lobe pulmonary venous congestion, bat-wing peri-hilar haze, Kerley B lines (complicating ACS).
- Aortic dissection—widened mediastinum or prominent aortic knuckle with a ‘double calcium’ shadow >6 mm.
Common calls

- Pneumothorax—peripheral hyperlucent area indicating free air in the pleural cavity, with partial or complete collapse of the affected lung.
- Pericarditis—if significant fluid (>250 mL) has accumulated, the cardiac silhouette may be symmetrically enlarged.
- Pneumonia—lobar or diffuse infiltrate changes.
- Pulmonary embolism—blunted costophrenic angle, raised hemidiaphragm, linear atelectasis or infarction, or an area of oligaemia.

Specific management

Acute coronary syndrome

- Classic features of the pain of ACS and its complications are described above.
- However, some patients have atypical pain, especially women, elderly people and those with diabetes and renal disease.
- ACS may also present with dyspnoea, orthopnoea or paroxysmal nocturnal dyspnoea (PND), lethargy, an arrhythmia or confusion (delirium, especially in the elderly), rather than chest pain.
- If the patient has chest pain and the ECG does not demonstrate significant ST elevation (≥1 mm in two or more contiguous limb leads or ≥2 mm in two or more contiguous chest leads):
  - Administer oxygen if shocked or hypoxic with saturation <93%, and give aspirin 300 mg PO.
  - Try to relieve the pain with GTN SL (0.6 mg tablets or 0.4 mg spray). Do not administer GTN if SBP <90 mmHg.
  - Give morphine if the pain continues despite 2–3 doses of GTN. Administer morphine IV in 2.5–5 mg aliquots until the pain is relieved.
  - Send blood for a cardiac troponin level immediately, and after 6–8 hours. Accelerated protocols now allow repeat troponin testing after 2–3 hours.
  - Perform serial ECGs immediately and after 6–8 hours, or every 15 minutes if the pain is continuing. Accelerated protocols now allow repeat ECG testing after 2–3 hours.
  - Determine the patient’s disposition and further management. This is based on their ACS risk-profile stratification.

High-risk features

- The patient has:
  - Repetitive or prolonged (>10 min) ongoing chest pain.
  - Elevated troponin level on initial or repeat testing.
  - Persistent or dynamic ECG changes (ST depression or T wave inversion).
• Haemodynamic compromise with SBP <90 mmHg, heart failure, ventricular tachycardia, syncope or new-onset mitral regurgitation.
• Recent PCI (within last 6 months) or prior coronary bypass grafting (CABG).
• Diabetes, or chronic renal impairment (eGFR <60 mL/min) with ‘typical’ chest pain.
• Commence high-flow oxygen if shocked or hypoxic, give aspirin 300 mg PO and clopidogrel 300 mg PO.
• Contact your senior doctor immediately, then the cardiology registrar. Organise for transfer to CCU for consideration of angiography within next 48 hours.
• Note: chest pain with ST elevation on ECG indicates a STEMI and requires immediate reperfusion therapy—see later.

Intermediate-risk features
• The patient has:
  • Resolved chest pain consistent with ACS that occurred at rest, was prolonged (>10 min) or was not controlled with GTN (i.e. not their usual angina).
  • Age >65 years.
  • Known previous AMI or significant coronary artery stenosis >50%. Regular aspirin use.
  • Two or more of cardiac risk factors (hypertension, smoking, hyperlipidaemia or significant family history).
  • ‘Atypical’ chest pain in a patient with diabetes or chronic renal impairment (eGFR <60 mL/min).
  • First episode of chest pain.
• Give aspirin 300 mg PO as above, call your senior doctor and refer to the inpatient medical registrar. These patients should ideally have continuous ECG monitoring in the CCU (if available), or at least a repeat ECG and troponin in 6–8 hours.
• Accelerated protocols now allow repeat troponin testing after 2–3 hours.
• If still negative, they then require exercise stress testing (EST), ideally within 72 hours, to further categorise them as high-risk (EST positive) or low-risk (EST negative). An alternative test such as a myocardial perfusion scan, stress echo or even a CT coronary angiogram is indicated in those unable to do an EST.

Low-risk features
• The patient has:
  • Chest pain consistent with ACS and no high-risk or intermediate-risk features.
  • A known diagnosis of angina and the current symptoms resolve promptly with 1–3 GTN doses.
Review the precipitating cause and ECG, and determine if the threshold for angina has decreased or the severity of pain increased. Consult with your senior, as adjustment to antianginal medication may be all that is required.

However, if this episode of pain was more prolonged, was worse or occurred with a lower threshold than normal with no easily remediable precipitant, send blood for cardiac biomarkers and repeat them and the ECG at 6–8 hours.

Accelerated protocols now allow repeat ECG and troponin testing after 2–3 hours.

If both are negative, organise an outpatient cardiology or usual physician referral.

ST-elevation myocardial infarction
A patient with chest pain and ST elevation on ECG must be assessed urgently with a view to immediate reperfusion therapy with thrombolysis or PCI.

Call your senior doctor and obtain an urgent cardiology consult, as the outcome is optimised if reperfusion occurs within 30 minutes (thrombolysis) or 60 minutes (PCI).

Evaluate the ECG changes in comparison with previous ECGs.

Check if there are any contraindications to thrombolysis (see Table 16.1) before the decision on which type of reperfusion therapy is made.

Indications for reperfusion:
- Typical chest pain (or equivalent) in preceding 12 hours and
- Persistent ST elevation >2 mm in two contiguous chest leads or >1 mm in two contiguous limb leads, or new onset of left bundle branch block (LBBB).

Commence high-flow oxygen, give aspirin 300 mg PO and clopidogrel 300–600 mg PO or prasugrel 60 mg PO.

Give LMWH or IV heparin on discussion with the cardiologist and in the light of which reperfusion therapy is to be used.

Aortic dissection
Predisposed to by hypertension, especially males over 60 years, bicuspid aortic valve, coarctation or iatrogenic (i.e. cardiac catheterisation). When younger than 40 years consider Marfan’s or Ehlers-Danlos syndrome. Ask about a family history or a previous dissection.

There is sudden onset of severe pain that is sharp or tearing in nature, retrosternal to interscapular and may migrate. Patients with dissection typically have pain that is out of proportion to other clinical findings and difficult to relieve, even with large doses of IV opioid.
Table 16.1 Contraindications to thrombolysis

<table>
<thead>
<tr>
<th>Absolute contraindications</th>
<th>Relative contraindications</th>
</tr>
</thead>
<tbody>
<tr>
<td>✆ Risk of bleeding</td>
<td>✆ Risk of bleeding</td>
</tr>
<tr>
<td>Active bleeding or bleeding</td>
<td>Current use of anticoagulants—the higher the INR, the higher the risk of bleeding</td>
</tr>
<tr>
<td>diathesis (excluding menses)</td>
<td>Non-compressible vascular puncture sites</td>
</tr>
<tr>
<td>Significant closed head injury or facial trauma within past 3 months</td>
<td>Recent major surgery (&lt;3 weeks)</td>
</tr>
<tr>
<td>Suspected aortic dissection (including new neurological symptoms)</td>
<td>Traumatic or prolonged CPR (&gt;10 min)</td>
</tr>
<tr>
<td></td>
<td>Recent (&lt;4 weeks) internal bleeding (e.g. GI bleed or urinary tract haemorrhage)</td>
</tr>
<tr>
<td></td>
<td>Active peptic ulcer</td>
</tr>
<tr>
<td></td>
<td>Pregnancy</td>
</tr>
<tr>
<td></td>
<td>✆ Risk of intracranial haemorrhage</td>
</tr>
<tr>
<td>Any previous intracranial haemorrhage</td>
<td>History of chronic, severe, poorly controlled hypertension</td>
</tr>
<tr>
<td>Ischaemic stroke in previous 3 months</td>
<td>Severe uncontrolled hypertension on presentation (SBP &gt;180 mmHg, DBP &gt;110 mmHg)</td>
</tr>
<tr>
<td>Known structural cerebral vascular lesion (e.g. arteriovenous malformation [AVM])</td>
<td>Ischaemic stroke &gt;3 months ago, dementia or known intracranial abnormality not covered in contraindications</td>
</tr>
<tr>
<td>Known malignant intracranial neoplasm (primary or metastatic)</td>
<td></td>
</tr>
</tbody>
</table>

- Look for unequal or absent pulses, a difference of >20 mmHg BP between arms or other complications such as aortic incompetence, pleural effusion or any new focal neurology.
- Call your senior doctor if you suspect an aortic dissection, and investigate and treat urgently:
  - Give oxygen to maintain saturation >95%.
  - Insert two large IV cannulae (14–16G) and send blood for an immediate cross-match of six units packed RBCs.
  - Relieve pain with titrated IV morphine 2.5–5 mg repeated as needed.
- Review the CXR (see Figure 16.1)—if suggestive of dissection or if the clinical suspicion is still high:
  - Arrange for an urgent helical CT angiogram (CTA) of the chest, a transoesophageal echocardiogram (TOE) if expertise is available or, in their absence, a transthoracic echo, which may demonstrate some of the complications of a dissection, such as cardiac tamponade or acute aortic regurgitation.
  - Arrange admission to ICU and contact the cardiothoracic or vascular surgical team.
• Give an IV beta-blocker such as esmolol or metoprolol slowly until the pulse is <60 beats/min or the SBP is <120 mmHg.
• If the BP is not controlled at this level, despite a pulse <60 beats/min, add sodium nitroprusside or a GTN infusion until the SBP is 100–120 mmHg. Start these in an intensive care area with intra-arterial BP monitoring.
• Prepare the patient for an operation if the CTA or TOE confirms an ascending AAD (type A). If there is a descending acute aortic dissection (type B), the patient should be managed in ICU with careful control of BP and observation for complications.
• Endovascular stent repair is becoming more common, particularly in descending acute aortic dissection.

Pericarditis
Pericarditis may be postviral (coxsackievirus, echovirus, adenovirus, influenza, mumps, herpes virus); associated with AMI or 2–6 weeks post-MI (Dressler’s syndrome), uraemia, malignancy or a connective tissue disorder (SLE, RA, scleroderma or polyarteritis nodosa [PAN]); TB; or follow trauma, radiation therapy or cardiac surgery; and occurs in AAD. There is no discernible cause in 10–20% of cases.
• The pain is sharp, retrosternal and radiates to the back; is worse on inspiration, swallowing or lying down; and is relieved by sitting up.
• A pericardial rub is best heard along the left sternal edge in expiration with the patient sitting up, but may be transient.
• Pericarditis related to TB, uraemia or neoplastic disease is usually more insidious and pain is often mild or absent, with the patient presenting with insidious pericardial tamponade.
• Most patients with pericarditis do not have a haemodynamically significant pericardial effusion or myocardial inflammation. The main issue is control of pain.

**Diagnosis and management:**
• Attach a cardiac monitor and pulse oximeter to the patient.
• Send blood for FBC, U&E, LFTs, troponin and viral serology.
• Perform an ECG, which may show sinus tachycardia alone, widespread concave ST elevation or PR-segment depression.
  • Late in the course T waves may flatten or become symmetrically inverted, sometimes permanently. Decreased voltages are suggestive of a pericardial effusion, and electrical alternans (components of the ECG such as the QRS axis alternate between beats) is a rare finding in pericardial effusion.
• **Note:** distinguishing pericarditis from STEMI is critical, as thrombolysis is contraindicated in pericarditis due to the risk of bleeding from haemorrhagic transformation.
• Request a CXR, which is usually normal even when a pericardial effusion is present. Apparent cardiomegaly only occurs once 250 mL of pericardial fluid have accumulated.
• Give an NSAID as the drug of choice. If there are contraindications to NSAIDs, colchicine 0.5 mg BD may be used.
• Prednisolone 50 mg PO or dexamethasone 4 mg PO or IV may be tried if intolerant of NSAIDs, or diarrhoea with colchicine.
• Arrange an urgent echocardiogram followed by pericardiocentesis if signs of cardiac tamponade such as tachycardia, hypotension, pulsus paradoxus and a raised JVP that rises on inspiration (known as Kussmaul’s sign) occur.
• Otherwise, echocardiography may be organised electively for a stable patient suspected of an effusion.

**Pleuritic causes of chest pain**
Pleuritic pain is a sharp, well-localised pain that is worse with inspiration. Often patients cannot take a deep breath in because it ‘catches’ and prevents full inspiration. Radiation to the shoulder or abdomen occurs with diaphragmatic involvement.
• Important causes of pleuritic pain include:
  • Pericarditis (see above)
  • Pneumothorax
• Pneumomediastinum
• Pneumonia
• Pulmonary embolus with a pulmonary infarction
• Autoimmune disorders (SLE, RA)
• Malignancy
• Musculoskeletal pain.

‘Pleurisy’ is a diagnosis of exclusion when none of the above can be identified. It may be due to viruses, such as enteroviruses, including epidemic myalgia (Bornholm’s disease) due to coxsackie B virus.

Examine the patient for features suggestive of an underlying cause such as fever and bronchial breathing in pneumonia etc. Listen to the chest for a pleural rub, although a rub may be inaudible if pain limits deep breathing, and disappears as an effusion develops.

Perform an ECG, which should be normal, but may show non-diagnostic abnormalities in PE.

Request a CXR, which may suggest an underlying cause or show non-specific basal atelectasis that may occur in any patient with restricted inspiration. A normal-looking CXR is more likely with PE.

If no significant underlying cause is found:
• Give oxygen to maintain oxygen saturation >95%
• Give NSAIDs (e.g. ibuprofen 400 mg PO TDS) plus paracetamol 1 g PO 4-hourly.

Pneumomediastinum

A patient with a spontaneous pneumomediastinum presents with sudden onset of chest, neck or throat pain. The CXR shows mediastinal air as a dark line outlining a heart border. The pathogenesis is similar to spontaneous pneumothorax and includes inhalational drug use. Most patients remain systemically well and can be treated with oral analgesia alone.

Gastrointestinal causes of chest pain

Suggested by heartburn, burning retrosternal or epigastric pain, worse on stooping or lying flat, exacerbated by swallowing, hot drinks or food and relieved by antacids.

Oesophagitis or oesophageal spasm may mimic cardiac pain and be relieved by sublingual GTN. ACS must always be excluded first if there is any doubt about the diagnosis.

The pain of oesophagitis, gastro-oesophageal reflux, gastritis or peptic ulcer may be temporarily relieved with oral antacids, but requires acid suppression therapy:
• Give an antacid 15–20 mL every 2 hours in the acute phase, then TDS after meals and once before bedtime.
• Prescribe a proton-pump inhibitor such as omeprazole 20–40 mg PO daily or an H2-receptor blocker such as ranitidine 150 mg PO BD.
• Advise lifestyle modification such as stopping smoking, reducing alcohol consumption, omitting hot or spicy food from the diet, eating small meals regularly during the day and not eating immediately before sleep.
• Refer for endoscopic evaluation and Helicobacter pylori testing.
• Immunocompromised patients may experience severe chest pain from oesophageal candidiasis that does not respond to antacids. Diagnosis should be confirmed by endoscopy. Fluconazole 100 mg PO daily is the treatment of choice.

Oesophageal rupture is associated with sudden onset of severe chest pain, which may follow vomiting (Boerhaave’s syndrome). It can also occur after endoscopy, foreign body impaction, caustic ingestion or trauma. Pain is persistent and difficult to relieve, and often associated with dyspnoea, diaphoresis, tachycardia and shock.
• The patient looks unwell and may have palpable subcutaneous emphysema or crunching sounds on auscultation of the heart (Hamman’s crunch due to pneumomediastinum).
• ECG may be normal or have non-specific changes.
• CXR shows mediastinal air, left pleural effusion or left-sided pneumothorax.
• Call your senior regarding further investigation—usually by watersoluble gastrografin contrast oesophogram, CT scan or endoscopy.
• If oesophageal rupture is confirmed, obtain large-bore IV access, commence fluid resuscitation and titrated opioid analgesia and broad-spectrum antibiotics. Admit to ICU and arrange immediate cardiothoracic or general surgical consult.

Musculoskeletal causes of chest pain
• Musculoskeletal disorders cause pain that is worse not only with breathing but also with movement. There may have been a preceding bout of coughing, strenuous exercise or a history of minor trauma.
• Palpate and compress the chest wall to identify a tender muscle, fractured rib(s) or tender costochondral junction. Palpate and gently percuss the spine to identify a crush fracture, which may be causing referred dermatomal pain.
• The ECG is normal and other investigations are unhelpful.
• Treat musculoskeletal causes of pain with an NSAID, such as ibuprofen 400 mg PO TDS or naproxen 250–500 mg PO TDS.

Herpes zoster
• Unilateral chest pain in a dermatomal distribution may precede the typical skin lesions of herpes zoster (‘shingles’) by 2 or 3 days.
The rash begins as a reddened, maculopapular area that rapidly evolves into vesicular lesions.

- Give opioid analgesia to patients with severe pain and commence famciclovir 250 mg or valaciclovir 1 g given orally 8-hourly for 7 days, if seen within 72 hours of vesicle eruption. Combining antiviral therapy with steroids (e.g. prednisolone 50 mg PO daily for 3 days) may provide added pain relief.

- Postherpetic neuralgia occurs after the acute episode and is difficult to treat. Potential therapies include anticonvulsant, antidepressant or antiarrhythmic drugs. Specialist input is required, arranged by the usual medical care team.

**Panic and anxiety disorders**

Panic attacks are discrete periods of intense fear or discomfort associated with the abrupt onset of four or more symptoms that include palpitations, sweating, trembling, sensation of shortness of breath or choking, chest pain or discomfort, dizziness, weakness. Hyperventilation causes perioral, hand or feet parasthesiae or muscular spasm related to acute hypocalcaemia from a respiratory alkalosis, even light-headedness to the point of unconsciousness.

Due to the possibility of a life-threatening cause of chest pain, panic and anxiety disorders are *always* a diagnosis of exclusion. Discuss the patient with your senior.

Attempt to control the hyperventilation by reassurance. Alternatively, ask the patient to re-breathe into a paper bag or mask to control the ventilatory loss of CO₂. Give diazepam 2–5 mg PO or lorazepam 1–2 mg SL acutely if there is no response to initial treatment or the attack recurs.