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MARSHALL & RUEDY'S

On Call

Principles & Protocols

Australasian and UK edition

4E

ELSEVIER

Anthony FT Brown
Mike Cadogan
Tony Celenza
Viet Tran

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MARSHALL & RUEDY'S

On Call

Principles & Protocols

Australasian and UK edition **4E**

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Preface to the fourth edition

New edition changes and intended audience

This new edition is written for all junior doctors and senior medical students who provide after-hours ward call, in both Australasia and the UK.

There is much new, updated and refreshed content, plus the addition of the highly respected author Viet Tran. Entirely new chapters cover *Professionalism and teamwork*; *Transferring the unwell patient*; *Junior doctors' health and wellbeing*; *Falls*, with a focus on the older patient; *Postoperative ward calls*; and *Basic ultrasound*, with a focus on difficult peripheral cannulation.

The entire text has the latest up-to-date, evidence-based management related to the after-hours care of the ward patient, whether in hospitals in Australia and New Zealand, or in the United Kingdom. Updates range from the new CPR guidelines, to acid–base disorders, electrolyte disturbances and many other acute medical conditions.

Purpose

This new Australasian and UK fourth edition retains the previous edition's hugely successful structured approach to the initial assessment, resuscitation, differential diagnosis and short-term management of common on-call problems. It is designed to provide junior doctors and senior medical students with a logical, practical and efficient approach to problem-based learning and acute management.

Problem-solving is a fundamental clinical skill for the doctor on call. Traditionally, the investigation of a patient's problems follows an orderly sequence, before provisional diagnoses and management are formulated.

In an emergency, the doctor will instead need to engage concurrently with resuscitation, history, examination, investigation and definitive treatment. Stabilisation of the airway, breathing, circulation and neurological disability, as well as calling for senior help, may need to be done in the first few minutes.

This book provides a focused approach to the most important clinical problems seen on call. It starts with advice on how to be a safe and confident doctor on call, followed by how to approach the critically ill patient. There is then an extensive section on symptom-based clinical problem solving, followed by chapters on how to interpret investigations and perform a practical procedure, and an in-depth drug

formulary. The book ends with a list of normal values (see Structure below).

Structure

The book is divided into seven main sections:

A. General principles
An overview of the professional, organisational, ethical and social traits required of the junior doctor on call.

B. Emergency calls
Life-threatening airway, breathing, circulation, neurological disability and environmental factors (ABCDE) are all covered in a risk-stratified approach.

C. Common calls
These cover the most common changes in symptoms or signs that will need review while on call—see below for the structure used in the chapters of this section.

D. Investigations
Guidance on how to interpret an ECG, common imaging, acid–base, electrolyte and haematological tests when on call.

E. Practical procedures
Guidance on how to perform a large array of practical procedures that may be needed, when on call.

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

G. Laboratory values
Normal reference ranges for all common laboratory investigations.

Within **Section C – Common calls**, the chapters are each further subdivided into:

- *Phone call* (pertinent questions to ask the ward)
- *Corridor thoughts* (differential diagnosis)
- *Major threat to life* (now highlighted in red)
- *Bedside* (starting with a 'Quick-look test', then 'Airway and vital signs')
- *Management* (with 'Immediate management' also now highlighted in red, followed by 'Selective history and chart review', 'Selective physical examination', 'Investigations', and 'Selective management'; see also Chapter 1).

Anthony Brown
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Viet Tran
April 2025

Foreword

This book aimed at Australasian and UK practice 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. The authors are among the finest teachers of 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 real source of excitement for those of us who have 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 usually little attention given to what does not need to be done, 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.

Professional, ethical and end-of-life issues are highlighted before the critically ill patient is discussed. 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 by producing this wonderful book. It will 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.

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(Abridged April 2025)

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Professor Tony Brown has written extensively in the medical literature, including a bestselling handbook on emergency medicine now in its eighth edition. He holds a Professorial academic teaching title at the University of Queensland Faculty of Medicine, and still works full-time in clinical emergency medicine. He was awarded the Order of Australia (AM) in 2018, *'for significant service to emergency medicine as a clinician, author and educator'*. His teaching awards include the inaugural Teaching Excellence Award 2001 at the Australasian College for Emergency Medicine; the Excellence in Clinical Teaching Award 2001 at the Royal Brisbane Hospital; the Outstanding Teaching Award 2015 at the Royal Brisbane Clinical School, University of Queensland; and The Michael Perera Teaching Award, Royal Brisbane Clinical Unit, Phase 2 MD Program, Faculty of Medicine, University of Queensland in 2021.

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Associate Professor Viet Tran is a clinician, researcher, educator and patient advocate. He is founder and director of the Tasmanian Emergency Medicine Research (TASER) Institute, where he inspires patient care through an evidence-based and data-driven approach. He is also the founder of Doctorswriting.com. He was awarded the Early Career Research Award 2022, College of Health and Medicine, University of Tasmania; and the Vice Chancellor's Award for Teaching Excellence in 2023, University of Tasmania.

Dedication

With thanks to my beautiful wife Regina for her enduring support, our inspiring and amazing children Edward and Lucy, and to our new grandchild Camille [AFTB]

For George, Olivia, Hamish and William [MC]

Thanks to my wife, Helen, and children, Alex, Kate, Anne and Ella, for their continuing support, patience and perseverance for my academic endeavours. To colleagues and students who force me to scrutinise, organise and crystallise my thoughts with every question [TC]

To Wun, Tai, Jay, Lee and Sam—the source of my best laughs and my proudest moments [VT]

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1

Approach to the diagnosis and management of on-call problems

Clinical problem-solving is a fundamental skill for the doctor on call. Traditionally, this is approached in an orderly, systematic manner. This includes focused history-taking and physical examination, review of available investigations, formulation of the provisional and differential diagnoses, making a management plan and calling for help when required.

History-taking and physical examination may take 20–30 minutes for a patient with a single problem seeing a new doctor for the first time. Or they may take longer for an older patient with multiple complaints.

Clearly, if a patient is found unconscious in bed, the chief complaint is 'coma' and the history of the presenting illness is limited to the information provided by witnesses, nursing staff or the patient's medical records. In this situation, the doctor is trained to proceed with a simultaneous history, examination, investigation and treatment approach, often starting with treatment. The initial steps that must be completed within the first 5–10 minutes to save life are known as the DRS ABCDE approach (Danger, Response, Send for help, Airway, Breathing, Circulation, Disability, Environment), as outlined further in Chapter 8.

When on call, the trainee or junior doctor is often faced with a well-defined problem (e.g. fever, chest pain, collapse), yet may feel ill-equipped to begin clinical problem-solving unless a comprehensive history and physical examination are obtained. Anything less induces guilt over a task only partially completed. However, few on-call problems should involve more than 30 minutes of the doctor's time, because excessive time spent with one patient will deny adequate treatment time to another more seriously ill patient.

Therefore, the approach recommended in this book is based on a structured system that is adaptable. It is intended as a practical guide to aid rapid, effective and efficient clinical problem-solving on call. Each clinical symptom-based chapter in Section C is divided the same way into five parts:

1. Phone call
2. Corridor thoughts
3. Major threat to life
4. Bedside
5. Management.

Phone call

Most problems are first communicated by telephone. The on-call doctor needs to determine the severity of the problem and thus prioritise patients based on this initial telephone call. The phone call section is divided into three parts:

1. **Questions:** pertinent initial questions to help determine the urgency of the problem.
2. **Instructions:** phone orders for the nurse at the bedside to expedite the investigation and management of the patient's immediate problem.
3. **Prioritisation:** assessment of the urgency of the problem to determine which patients need to be seen immediately.

Corridor thoughts

The time spent going to the ward is used to consider the differential diagnoses and potential life-threats of the problem at hand. This 'travel time' is also useful for organising a plan of action at the bedside.

The differential diagnosis lists presented in this book are not exhaustive—they focus on the most common or most serious causes that should be considered in a hospitalised patient.

Major threat to life

Identifying a potential major threat to life follows logically from consideration of the differential diagnoses. It is more useful to appreciate the most likely threats to life and use them to focus questions and the physical examination, than to simply arrive at the bedside with a memorised list of all possible diagnoses. This risk-analysis process ensures that seeking and treating the most serious life-threatening possibilities in each clinical scenario is emphasised.

Bedside

The evaluation of the patient at the bedside is divided into the following areas:

- **Quick-look test**
- **Airway and vital signs**
- **Immediate management**
- **Selective history and chart review**
- **Selective physical examination**
- **Bedside and other investigations.**

Thus, the bedside assessment begins with the quick-look test, which is a rapid visual assessment to categorise the patient's condition in terms of severity: well (comfortable), sick (uncomfortable or distressed) or critical (about to die).

Next is an assessment of the airway and vital signs, which are critically important in the evaluation of any sick patient.

The order of the remaining parts is not uniform, due to the nature of the various problems that require assessment when on call. For example, the selective physical examination may either precede or follow the selective history and chart review, and either of these may be superseded by immediate management when the urgency of the clinical situation dictates.

Occasionally, the physical examination and management sections are further subdivided to focus on urgent, life-threatening problems, leaving the less urgent problems to be reviewed later.

Management

General supportive and specific management include monitoring, stabilisation and therapy, both pharmacological and procedural. Immediate resuscitation with attention to the DRS ABCDE approach is dealt with first. Next, disease-specific management issues are considered. In every situation, clear guidance is given on when to call your senior, and how urgently.

Conclusion

The principles and protocols offered in this book provide a logical, efficient and safe system for the assessment and management of common on-call problems. The aim is to make an already stressful situation easier to handle, for the benefit of patients and the relief of the doctor involved.

By adopting a consistent, logical and safe approach to clinical reasoning, you will ensure the experience you gain on call sets you up for a lifetime of good clinical practice.

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Hypotension

Nursing staff may call you to assess a patient with low BP. Remember that it is the threat to end-organ function that dictates the need for intervention, rather than the absolute BP level. The priority is to assess for tissue hypoperfusion, which indicates shock (see also Chapter 12).

- Not all patients with hypotension have shock, and conversely compensatory changes will maintain the SBP in early shock. Although you may be called to see many patients with hypotension, shock requires evidence of inadequate tissue perfusion.
- Thus, some healthy patients normally have a SBP of 80–100 mmHg, especially thin, young females.
- Conversely, a patient with chronic, severe heart failure has to function with a SBP of 80–90 mmHg. A higher BP precipitates an increased cardiac workload, leading to angina and worsening heart failure; lower pressures lead to light-headedness and decreasing renal function. Reviewing these patients is complex and requires senior assistance.
- Other patients with chronic hypertension require a higher-than-normal perfusing pressure, so a BP of 120/80 mmHg may be low for that patient and associated with significant tissue hypoperfusion.

Phone call

Questions

1. **What is the BP?**
2. **What is the HR?**
3. **What is the RR?**
4. **What is the patient's mental status?**
5. **Is the patient clammy or pale?**
6. **Does the patient have dyspnoea or chest pain?** (septic or cardiogenic shock)
7. **Is there evidence of bleeding?** (haemorrhagic shock)
8. **What is the temperature?** (septic shock)
9. **Has the patient been given medication in the last hour?** (anaphylaxis)

10. **Does the patient have a rash?** (anaphylaxis or septicaemia)
11. **What was the reason for admission?**

Instructions

- If there is a possibility of shock:
  - Administer at least 6 L/min oxygen by mask and attach pulse oximetry to the patient.
- Request an IV trolley at the patient's bedside with two large-bore 14–16G cannulae ready for insertion, if an IV is not in place.
  - Give 20 mL/kg 0.9% sodium chloride IV rapidly, unless the patient is SOB with pulmonary oedema from cardiogenic shock, as fluid overload will worsen the situation.
  - Request an ECG immediately and commence continuous cardiac monitoring.
- Organise cross-matching of at least four units of packed RBCs if the admission diagnosis was a GI bleed, or if there is external evidence of blood loss.
- Request 1:1000 adrenaline 0.01 mg/kg up to 0.5 mg (0.5 mL) be drawn up ready for IM injection into the upper lateral thigh if you suspect anaphylaxis.
- Arrange a portable CXR immediately if the patient has dyspnoea or chest pain.

Prioritisation

See every patient with hypotension and suspected shock immediately.

Corridor thoughts

What are the causes of hypotension? (* = major threat to life)

- Shock*:
  - Hypovolaemic, cardiogenic, distributive or obstructive (see Chapter 12)
- Medications (often postural hypotension):
  - Antihypertensives, nitrates, sedatives, analgesics
- Autonomic neuropathy:
  - Diabetes, Parkinson's disease, multiple system atrophy (MSA)
- Vasovagal attack (also known as neurocardiogenic syncope)
- Constitutional:
  - Some patients have a naturally low BP normally

Major threat to life

- **Shock**
  - Progressive hypotension leads to circulatory failure and results in hypoxia, anaerobic metabolism with a metabolic lactic acidosis,

organ hypoperfusion and tissue ischaemia that further worsen the cardiovascular status.

- **Underlying cause**
  - Hypotension may be secondary to a life-threatening condition such as ACS, cardiac arrhythmia, hypovolaemia, anaphylaxis, sepsis, massive PE, tension pneumothorax or cardiac tamponade that must be looked for and treated (see also Chapter 12).

Bedside

The immediate bedside review is aimed at quickly determining whether shock is present, rapidly isolating the potential cause(s) and instituting appropriate early management.

Quick-look test

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

A patient with hypotension but adequate tissue perfusion usually looks well and has a normal mental status. Once perfusion of vital organs is compromised, the patient starts to look pale, agitated and sweaty. Patients who are seriously volume-depleted appear haggard, drawn and exhausted.

Airway and vital signs

Is the airway clear?

- If the patient has a depressed conscious level and the airway cannot be protected or maintained, endotracheal intubation will be required.
- Roll the patient onto the left lateral decubitus position to avoid aspiration, and remove loose-fitting dentures.
- Contact your senior urgently, as well as an anaesthetist and the ICU.

What is the respiratory rate/pattern?

- Tachypnoea is a compensatory sign commonly associated with hypotension and shock. Tachypnoea may indicate lactic acidosis secondary to tissue hypoperfusion, acute LVF or an underlying PE, pneumothorax or cardiac tamponade.
- The increased RR serves to increase the thoracic pump effect, improves venous return and attempts to compensate for tissue acidosis by blowing off CO_2.

What is the blood pressure?

- Measure the patient's supine BP in both arms.
  - SBP <90 mmHg is usually associated with inadequate tissue perfusion, especially if this is markedly lower than the patient's normal readings.
  - Do not be fooled by an apparently acceptable automated non-invasive blood pressure (NIBP) measurement if the patient

looks shocked. The NIBP is notoriously inaccurate at low readings. Retake the BP manually.

- What is the pulse pressure?
  - A narrow pulse pressure (raised DBP from peripheral vasoconstriction as a compensatory mechanism) suggests a reduced LV stroke volume and shock.
  - A wider pulse pressure (lowered DBP from vasodilation) may occur with anaphylaxis or early septicaemia i.e. 'warm (distributive) shock'.
- The hypotension may have been caused by a vasovagal episode or orthostatic hypotension if the patient's BP has returned to normal lying supine.
  - If hypotension is not marked and the patient appears well, examine for postural vital sign changes. This is not indicated if the patient is already hypotensive when supine.
  - Ask for assistance if the patient is unable to stand alone, or have the patient sit up and dangle the legs over the side of the bed.
  - Repeat the HR and BP after the patient sits or stands for *at least* 2 minutes. Ask the patient if they feel light-headed.
    - An increase in HR of >15 beats/min, or the patient reporting light-headedness, is an indicator of inadequate intravascular volume.
    - A fall in SBP of >15 mmHg or any fall in DBP is less specific, as patients with autonomic dysfunction (e.g. on beta-blockers, or with diabetic neuropathy, MSA) may also have a pronounced postural fall in BP without being hypovolaemic. They will not have a compensatory rise in HR.

What is the heart rate?

- Low volume or impalpable peripheral pulses indicate hypoperfusion.
- A rough guide to the minimum underlying systolic BP is if a radial pulse is palpable = 90 mmHg; a femoral pulse is palpable = 80 mmHg; and with only a carotid pulse palpable = 70 mmHg. Check what the BP is manually.
- Most causes of hypotension are accompanied by a compensatory reflex sinus tachycardia.
- Look at the ECG or rhythm strip. HR <50 beats/min or >150 beats/min, especially if not in sinus rhythm, could be the cause of the shock (cardiogenic).
  - If the resting HR is <50 beats/min in the presence of hypotension, suspect a vasovagal episode, autonomic neuropathy, rate-slowing drugs or bradyarrhythmia. Haemorrhagic shock can cause a relative bradycardia in 10% of cases.
  - If the HR is >150 beats/min and there is any evidence of VT, rapid AF or SVT, emergency electrical cardioversion should be considered (see Chapters 18 and 67).
  - Look for ECG changes suggestive of ACS as a cause of the hypotension (cardiogenic shock). ECG changes of diffuse

myocardial ischaemia, such as widespread T inversion and/or ST-segment depression, suggest myocardial hypoperfusion as a result of the hypotension.
- ECG abnormalities can also indicate drug toxicity or electrolyte disorders.

What is the temperature?
- An elevated (>38°C) or low (<36°C) temperature suggest sepsis. However, sepsis may occur with a normal temperature in some patients, especially the elderly or immunosuppressed.
- Hypothermia will also develop in the later stages of shock because of metabolic changes from tissue hypoperfusion, exposure or fluid resuscitation (particularly cold blood in haemorrhagic shock, in the absence of a blood warmer).

Selective physical examination I

Is the patient in shock?

Vitals	Repeat now; pay particular attention to HR and BP
Skin	Cool, mottled and clammy (decreased perfusion)
	Warm and pink (adequate perfusion, or possibly distributive shock)
	Purpura (septicaemia) or urticaria/erythema (anaphylaxis)
CVS	Small pulse volume and slow capillary refill > 2 sec (hypoperfusion)
	Non-visible JVP, flat neck veins (hypovolaemic or distributive shock)
	Elevated JVP, distended neck veins (obstructive or cardiogenic shock)
	Kussmaul's sign: the JVP rises on inspiration (cardiac tamponade or RV failure)
Resp	Stridor (anaphylaxis)
	Extreme respiratory distress with unilateral hyperexpansion, hyperresonnance and absent breath sounds (tension pneumothorax)
	Crackles on chest auscultation (pneumonia or cardiogenic shock)
	Tachypnoea (Kussmaul's breathing from metabolic acidosis)
GIT	Check for tenderness or a pulsatile mass (AAA)
	Generalised abdominal tenderness with peritonism (bowel infarction, intraperitoneal haemorrhage from ectopic pregnancy or pancreatitis with third-space fluid loss)
	Bedside ultrasound will quickly show AAA, or free intraperitoneal fluid (rupture AAA or ectopic)
CNS	Alert, orientated (maintaining cerebral perfusion)
	Apprehensive, confused, agitated, delirious (decreased cerebral perfusion)
Urine	Urine output >0.5 mL/kg/h (adequate renal perfusion)
	Urine output <0.5 mL/kg/h (decreased renal perfusion) on hourly measurements, which usually necessitate an IDC

- *Note*: the urine output correlates with the renal blood flow, which in turn is dependent on cardiac output. It is an important measure of the adequacy of systemic perfusion. However, placement of an IDC does not take priority over resuscitation measures. It is best placed once resuscitation is well underway.

Management

Immediate management

Commence resuscitation while taking a focused history and performing a selective physical examination to identify the underlying cause in every patient with clinical features of shock.

- Call your senior immediately.
- Specific management of the various shock conditions is detailed later.
- **Monitor vital signs**
  - Attach continuous ECG, non-invasive BP and pulse oximeter to the patient.
  - Commence oxygen therapy to maintain saturation >94%.
  - Shocked patients require high-flow oxygen and may need assistance with ventilation if consciousness is depressed.
- **Obtain adequate IV access**
  - Insert two large-bore (14–16G) cannulae in large peripheral veins. Antecubital veins are preferred.
  - Send blood for FBC, U&E, coagulation profile, and a troponin if the ECG is abnormal. Rapidly check a venous blood gas including a lactate. Request an immediate cross-match of 2–6 units of packed RBCs if haemorrhage is suspected.
  - Send two separate sets of paired blood cultures if septic shock is possible.
  - Request a urinary or blood beta-hCG, particularly in a younger female with suspected haemorrhagic shock.
- **Resuscitate intravascular depletion**
  - Give 20 mL/kg 0.9% sodium chloride IV rapidly if the patient is hypotensive, the JVP is low and there are no crackles on chest auscultation. Elevate or squeeze the IV bag, or use IV pressure cuffs to increase the rate as necessary.
  - Observe the effect of this fluid challenge by monitoring response in HR, BP, JVP, peripheral perfusion, basal lung crackles and urine output, and repeat 20 mL/kg as clinically indicated.
  - Titrate fluid resuscitation more cautiously in a patient with a history of cardiac failure and commence with a smaller fluid bolus of 2.5 mL/kg.

- Search for the site of blood loss in the shocked patient suspected of bleeding.

- **Treat any immediately apparent cause**
  - Give adrenaline up to 0.5 mg (up to 0.5 mL 1:1000) IM for anaphylaxis every 5 minutes, according to the age, body mass, comorbidity such as cardiac disease, and clinical response.
  - Decompress a tension pneumothorax using needle thoracostomy.
  - Give broad-spectrum antibiotics for septicaemia.
- **Insert an IDC to monitor urine output**
  - A urine output of 0.5–1 mL/kg/h indicates restoration of adequate renal perfusion, and reversal of the shock state.
- **Perform a 12-lead ECG and review the rhythm strip**
  - Give IV atropine bolus (0.5–1.0 mg) if the HR <50 beats/min.
  - Follow with transcutaneous or transvenous pacing if the HR remains inappropriately low (see Chapters 18 and 68). Continuous ECG monitoring is essential in CCU or ICU.
  - Perform electrical DC cardioversion with appropriate sedation (see Chapters 18 and 67) if the HR >150 beats/min with hypotension and evidence of VT, rapid AF or SVT.
- **Request a portable CXR.**

Selective history and chart review

- Determine whether a presenting problem or the past medical history is a cause of the hypotension and quickly review the chart for recent trends or medication changes.

Does the patient have any risk factors for hypovolaemic or distributive shock?

- Bleeding (e.g. major epistaxis, haematemesis, melaena, bright-red rectal blood, menorrhagia), concealed bleeding best found on bedside ultrasound such as a ruptured AAA or ectopic pregnancy (beta-hCG positive), pelvic or long-bone fracture.
- Vomiting and/or diarrhoea.
- Bowel obstruction, ileus or pancreatitis, which lead to severe fluid sequestration in the abdomen, 'third spacing'.
- Burns or generalised erythroderma, which cause skin surface loss of fluids.
- Fever, rigors, malaise, recent contact with meningococcal disease, any of which may be associated with sepsis.
  - Ask every male or older female about sudden abdominal or back pain (ruptured AAA).
  - Ask every female patient about sudden abdominal pain and/or vaginal bleeding (ectopic pregnancy), or about the use of tampons (toxic shock syndrome).

Does the patient have any risk factors for cardiogenic or obstructive shock?

- Chest pain, dyspnoea, orthopnoea, previous ACS or cardiac interventions are associated with cardiogenic shock.
  - Diabetes or chronic renal impairment may be associated with atypical or 'silent' myocardial ischaemia and cardiogenic shock.
- Pleuritic chest pain, whole-leg swelling, postoperative patient, known malignancy and prior thromboembolism may be associated with PE.
- Malignancy, CKD or connective tissue disorder may be associated with the development of cardiac tamponade (exclude with ultrasound).

Check the observation chart

- Check previous BP measurements. Confirm that this episode is abnormally low for the patient. See whether there has been a slow deterioration or sudden change.
- Check the temperature chart for fever.
- Look for a change in weight. A loss of several kilograms since admission may indicate excessive dehydration (>5–10% body weight loss).

Check the fluid balance chart

- Look for evidence of hypovolaemia:
  - Reduced PO or IV intake
  - Excessive fluid loss from nasogastric or surgical drains or ileostomies
  - Urine output <30 mL/h.
- Check also for recent urinary loss from diuretic medications, osmotic diuresis (hyperglycaemia, mannitol administration, hypertonic IV contrast material), post-obstructive renal diuresis, arginine vasopressin disorder (diabetes insipidus), recovery phase of acute tubular necrosis, adrenal insufficiency (with vomiting). See Chapter 33.

Check the medication chart

- Recent administration of radiographic contrast or IV/IM medication may precipitate anaphylaxis.
- Note any change in dose or addition of hypotension-inducing medications.
  - Beta-blockers, calcium-channel blockers and many other antiarrhythmics cause hypotension and will counter a reflex tachycardia. Diuretics may cause hypovolaemia.
  - Excessive doses of potent analgesics or sedatives cause hypotension.
- NSAIDs and steroids can precipitate GI bleeding and anticoagulants worsen any bleeding.

Selective physical examination II

Are there any immediately obvious clues to the cause of shock?

Vital signs	Recheck HR, BP and peripheral perfusion now to assess for any improvement
HEENT	Dry mouth (hypovolaemia)
CVS	Recheck JVP to assess for any improvement
	Displaced apex beat, S_3 gallop (LVF)
	Pericardial effusion (seen on bedside ultrasound)
Resp	Crackles, pleural effusions (cardiogenic shock)
	Wheezes (anaphylaxis, LVF or PE-related bronchospasm)
GIT	Tender hepatomegaly (heart failure)
Rectal	Melaena or haematochezia (GI bleed)
Skin	Pruritic, urticarial rash with vasodilated peripheries (anaphylaxis)
	Pale skin creases and conjunctivae (occult haemorrhage)
Ext	Sacral or ankle oedema (pre-existing CCF)
	Note: patients with acute cardiogenic shock are not oedematous unless they had previous CCF. Patients with anaphylaxis or sepsis may become oedematous from capillary leakage.
Urine	Reassess urine output.

Investigations

- Check the recent Hb for any trends.
- Generalised tissue hypoperfusion causes lactic acidosis from anaerobic metabolism, with low bicarbonate plus other electrolyte abnormalities such as hyperkalaemia.
- Renal hypoperfusion causes a fall in urine output and a worsening of renal function. Look at the urea-to-creatinine ratio. A ratio >12 (calculated in SI units) is suggestive of volume depletion or a GI bleed.
- Send a urine sample for MCS if dipstick testing indicates potential urosepsis (i.e. was abnormal).
- Review the CXR as soon as possible. An abnormal CXR may be seen with:
  - Acute LVF (pulmonary venous congestion, cardiomegaly, Kerley B lines)
  - Aortic dissection (widened mediastinum)
  - Pulmonary embolism (plate atelectasis, basal effusion)
  - Pneumonia (consolidation, diffuse alveolar changes)
  - Tension pneumothorax (CXR *not* usually indicated).

- Bedside ultrasound will diagnose the presence of an aortic aneurysm, pericardial tamponade or ectopic pregnancy. Arrange this urgently.
- Echocardiography is used to determine volume status, myocardial or valvular dysfunction; it will confirm pericardial tamponade, right ventricular strain of PE, and occasionally may diagnose aortic dissection. It is harder to organise after-hours.

Specific management

Cardiogenic shock

- Make sure your senior doctor and cardiology are present.
- Cardiogenic shock has a mortality of 50–70% and is clinically manifest by hypotension, hypoxia, elevated JVP and pulmonary oedema.
- Patients with acute pulmonary oedema alone will frequently have poor peripheral perfusion and elevated filling pressures, but they are often hypertensive, so lowering their BP by decreasing preload (e.g. with GTN or CPAP) improves them.
- Treat ACS-related causes, such as STEMI or NSTEMI.
  - Give aspirin, heparin and clopidogrel or prasugrel or ticagrelor (check first with cardiology their preference). Refer for immediate PCI in STEMI (see Chapter 17).
  - Exclude other causes of hypotension with raised JVP.
    - Arrange an urgent echocardiogram to distinguish myocardial ischaemic dysfunction from pericardial tamponade, PE, acute valvular lesion or a septal perforation.
  - Thus a patient with a new murmur requires echocardiography urgently, as valve repair may be required to save the patient's life.
- Confirm RV infarction in an inferior STEMI by performing right-sided chest leads on the ECG.
  - V4R, V5R, V6R electrodes are placed in the same chest position, but on the right hemithorax.
  - ST-segment elevation indicates RV infarction.
  - These patients are dependent on preload for their cardiac output, so avoid dropping preload with GTN, morphine or diuretics.
  - They are less likely to develop pulmonary oedema; try small aliquots of 0.9% sodium chloride at 2 mL/kg.
- Exclude aortic dissection causing shock from other causes of tamponade or severe aortic incompetence.
  - Arrange a CT chest scan with IV contrast (CT angiography) or transoesophageal echocardiography (TOE) to best distinguish aortic dissection from ACS.

- Otherwise, general principles of shock management apply.
- Give maximal oxygenation, careful fluid management and commence vasopressor/inotrope infusions such as noradrenaline with or without dobutamine in the ICU.
- If these measures are unsuccessful, intra-aortic balloon pump, left ventricular assist device (LVAD) or circulatory bypass are required if a reversible pathological feature is present and the expertise is available. These all occur in CCU or ICU only.

Hypovolaemic shock

- Take measures to control external bleeding. Compress or pack any external haemorrhage such as epistaxis or an open wound.
- Consult a surgeon immediately if there is suspicion of acute blood loss causing hypotension (e.g. GI bleed, ruptured AAA, ectopic pregnancy).
- If massive bleeding is occurring, order uncross-matched blood and consider other blood products such as fresh frozen plasma (FFP) and platelets.
- Notify the blood bank that a 'massive transfusion' may be necessary. Some blood banks will then routinely provide additional FFP and platelets as part of this, often guided by ROTEM or TEG point of care viscoelastic haemostatic testing.
- Excess fluid losses via vomiting, diarrhoea, sweating, polyuria, extreme diuretic therapy and third-space losses (e.g. pancreatitis, bowel obstruction, peritonitis) will respond to intravascular volume expansion (resuscitation) with 0.9% sodium chloride.
  - Give 20 mL/kg at a time to restore the circulation.
  - Then gradually correct any dehydration (rehydration), and include daily maintenance amounts.
  - Search for the underlying cause.

Anaphylaxis

- Treat immediately with adrenaline up to 0.5 mg (up to 0.5 mL of 1:1000 solution) IM plus 0.9% sodium chloride 20 mL/kg (see Chapter 10).

Septic shock

- This is most simply defined as sepsis and, despite adequate fluid resuscitation, an inability to maintain a systolic BP >90 mmHg (or a MAP >65 mmHg) without vasopressors; and a blood lactate >2 mmol/L.
- Life-threatening organ dysfunction is indicated by an altered conscious level, tachypnoea, mottled skin, and acute oliguria.
- More than 85% of infections originate from the chest, abdominal or genitourinary systems, skin and vascular access.

- Arrange a CXR and urinalysis/MSU as initial screens. Make sure at least two sets of paired blood cultures from two different sites have been sent.
- Remember to examine all areas of the skin, looking for a source or entry site of infection, including between the toes (tinea), skin folds (intertrigo), perineum and axillae (abscess), and the ear, nose and throat for localised infections.
- Intravascular fluid resuscitation requires large volumes—up to 50–100 mL/kg—before adequate volume repletion. Ensure Hb is maintained between 70 and 90 g/L.
- Patients who are neutropenic from chemotherapy, diabetic, have HIV or are otherwise immunosuppressed may show few signs of sepsis. They also demonstrate few focal features and have non-specific inflammatory changes on blood tests.
  - Give a shocked patient, or any neutropenic patient empiric broad-spectrum antibiotics such as piperacillin 4 g with tazobactam 0.5 g IV plus gentamicin 4–7 mg/kg stat, if no source is apparent. Add vancomycin 25–30 mg/kg IV loading dose, particularly when MRSA or line sepsis are possible.
  - Otherwise, give broad-spectrum antibiotics as per hospital guidelines to cover likely pathogens depending on the presumed focus as soon as possible, within 1 hr of the onset of hypotension.
  - Continuing hypotension despite intravascular volume repletion requires admission to ICU and vasopressor support (adrenaline or noradrenaline infusion), as vasodilation and increased vascular permeability are common.

Other causes of hypotension

- **Tension pneumothorax**
  - Causes extreme respiratory distress, distended neck veins and a hyperexpanded chest on the affected side, with diminished breath sounds.
  - Do not wait for X-ray confirmation in an unstable patient. Call for senior assistance and get a 14–16G cannula ready to insert over 5th rib in the mid-axillary line on the affected side, if necessary inserting this before senior help arrives.
  - Once inserted into the pleural space, trapped air will be released with immediate improvement in blood pressure. Follow this with a formal Seldinger intercostal catheter, or tube thoracostomy (see Chapter 62).
- **Cardiac tamponade**
  - Elevated JVP, hypotension and soft heart sounds (Beck's triad) with agitation related to intracerebral venous congestion.

- Urgent bedside ultrasound is now best to confirm the diagnosis.
- Most commonly follows penetrating trauma, or is non-traumatic in a patient with chronic renal impairment, malignancy, connective tissue disorders etc.
  - Pericardiocentesis with insertion of a catheter into the pericardial sac and aspiration of fluid should result in immediate improvement in non-traumatic causes. ECG monitoring in a CCU is required.
  - Traumatic cardiac tamponade requires immediate surgery.

- **Massive pulmonary embolus**
  - Sudden hypotension, breathlessness, elevated JVP and cyanosis accompanied by additional evidence of acute RV overload such as RV dilatation or tricuspid insufficiency seen on bedside ultrasound.
  - If PE is suspected, treat as outlined in Chapter 16.
- **Drug toxicity**
  - Common causes of hypotension are GTN, vasodilators, opioids, sedatives, antiarrhythmics, beta-blockers, calcium-channel blockers and ACE inhibitors. However, hypotension is seldom accompanied by evidence of inadequate tissue end organ perfusion.
  - Place the patient flat, elevate the legs and give 5–10 mL/kg IV of 0.9% sodium chloride to support the BP until the effect of the drug wears off.
  - Bradycardia and hypotension from excessive narcotic are reversed by naloxone hydrochloride 0.1–0.2 mg IV, SC or IM every 5 minutes repeated until alert. Check the past history to avoid precipitating an acutely agitated withdrawal state in an opioid-dependent patient.
  - Following stabilisation of the patient, reduce the dose or alter the schedule of the opioid.
- **Vasovagal syncope**
  - The patient is usually normotensive by the time you arrive, having been laid flat, but will still feel nauseated and miserable. Warn the patient of continuing to feel faint for several hours afterwards.
  - Look for a precipitating stimulus such as pain or exposure to blood, with prodromal light-headedness and sweating.
  - There is no specific treatment, but ensure there was no underlying exacerbating factor such as dehydration, infection, medications that cause orthostatic hypotension, ectopic pregnancy or a ruptured AAA (see Chapter 22).

Post-shock complications

After a patient has previously been resuscitated from a hypotensive episode, watch out during the next few days for:

- Multiorgan failure—a combination of lung, renal, cardiac, coagulation, bowel and other dysfunction requiring ICU care.
  - Brain—thrombotic stroke or watershed cortical infarction with sudden blindness.
  - Lung—hypoxia with diffuse lung infiltrates causing ARDS.
  - Heart—diffuse myocardial injury with elevated troponin.
  - Kidney—acute tubular necrosis with oliguria, and rising urea and creatinine.
  - Centrilobular hepatic necrosis with jaundice and elevated liver enzymes.
  - Bowel ischaemia or infarction with the onset of bloody diarrhoea and deteriorating metabolic acidosis.