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Carole Foot and Liz Hickson

ESSENTIALS The first step in the clinical approach to a patient who is very ill is the recognition of this fact. While experienced clinicians will intuitively recognize a seriously ill or deteriorating patient, a large body of data has demonstrated that warning signs are often missed or not acted upon, resulting in preventable harm to patients. This has led to the development and adoption of 'track and trigger' systems. Track and trigger systems aim to ensure that hospitalized patients undergo regular review and objective observation, with abnormal observation or staff concerns being triggers to escalate care. Escalated care may be urgent review by the treating team, or the calling of a 'rapid response team' to attend to a more critically ill patient. While calling criteria and team composition varies from country to country, the principle of rapidly taking skilled care to critically ill patients remains the unifying underlying principle. A critically ill patient needs a rapid structured clinical assessment (relevant history and examination) with simultaneous treatment of life-threatening problems. A structured approach to the ABCs (airway, breathing, circulation) by an individual, or by a trained team with each individual having a defined role, modelled on the approach first developed to assess and treat multiply injured patients, offers patients the best outcome. Recognizing the critically ill patient The ability of an experienced clinician to stand and observe a patient at the bedside and determine if they are very ill, the so-called 'end of bed test', remains an important clinical skill (see Fig. 17.1.1). However, such intuitive determinations are not sensitive enough to form the basis for a safe system of recognizing critically ill and deteriorating patients. The apparent tacit ability of skilled individuals to identify patients who are very ill has been dissected. Specific features which can reliably indicate concern have been codified into broadly relevant systematic approaches to patient assessment. The outcomes of such efforts are modern systems that seek to ensure that hospitalized patients are continuously screened for signs of deterioration. When concerns are identified, patients are assessed and managed using logical and thorough approaches by healthcare providers specifically trained for the task. The specific

nature of how such systems operate is highly variable inter- nationally. Despite this, the shared focus is the use of a systematic approach that attempts to differentiate patients who are ‘well, ill, very ill, or nearly dead’, the goals of ‘track and trigger systems’ (see Fig. 17.1.2 for an example). Responding to the needs of a very ill patient is time-critical, and consequently history taking, physical examination, and ordering of investigations should be performed simultaneously, usually with more than one team member attending to each of these elements. For example, a patient may have a peripheral venous cannula in- serted and have blood tests collected while being questioned re- garding key symptoms as they receive a nebulized bronchodilator. Useful mnemonic acronyms can facilitate efficient focused history taking, some of which may be rapidly obtained from the patient’s records and prescription charts for fluids and medications (see Box 17.1.1).

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Carole Foot and Liz Hickson Fig. 17.1.1 This patient has several worrying features suggesting he may be critically unwell. Note that his eyes are closed, suggesting a possible reduced level of consciousness. He is propped up on pillows suggesting that he may be experiencing difficulty breathing supine. His neck muscles are prominent possibly due to accessory muscle use and/or cachexia. He seems unable to hold on his own mask, which may be related to weakness, somnolence, and/or confusion. The nurse appears worried. Photograph courtesy of Matt Tinker Photography.

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Fig. 17.1.2 In a track and trigger system, nursing staff review their patients regularly and in the absence of an immediate need to trigger an emergency response, they measure patients’ vital signs. These are recorded on a chart with designated ranges; observations outside the acceptable range trigger intervention or escalation. The standard observation chart shown is from New South Wales, Australia, and the system is called ‘between the flags’. This is in keeping with a Surf Life Saving programme to prevent drowning where swimmers are maintained in safe waters designated by coloured yellow and red flags. On the observation chart pictured, ill patients are designated as those entering a ‘yellow zone’ and very ill as those in a ‘red zone’.

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Similarly, a structured approach to physical examination is es- sential to ensure that key signs are identified in order of priority. These signs, in conjunction with relevant history and simple bedside investigations (e.g. finger prick blood glucose sampling, arterial blood gas, electrocardiogram (ECG), dipstick urine ana- lysis), may be linked to immediate, life-saving therapeutic inter- ventions (see Table 17.1.1). The most commonly encountered triggers for escalation of care will vary between centres and with case mix. In some contexts ser- ious nurse concern, and more recently serious unresolved concern of a patient or their loved one have been recognized as important stimuli to help clinicians recognize critically ill patients, with some supporting evidence, particularly in the paediatric population. Useful observations such as subtle changes in behaviour, cognition, or the nature of pain may be missed with screening systems that are based exclusively on physiological data. Valuable information heralding or helping define the specific diagnosis and/or severity of serious illness may also be obtained from routine blood tests. Classical data sets or constellations of abnormalities when found together may be diagnostic or highly suggestive of pathology not obvious from clinical assessment alone (see Table 17.1.2 for some examples). Causes of acute clinical deterioration

In the era of track and trigger systems, the differential diagnosis for commonly encountered causes of acute deterioration can be con- sidered in terms of the principal acute physiologic derangement that prompts escalation of care. In Boxes 17.1.2–17.1.11, important causes of abnormal systolic blood

pressure and heart rate, oxygen desaturation, and altered respiratory rate, abnormal urine output, and altered level of consciousness are summarized. General principles of management The mainstay of management of critically ill patients is meticulous supportive care that aims to correct life-threatening physiologic derangements and optimize organ system function while preventing complications. Well Vital signs within specified normal parameters & no other concerns Ill 1 or more vital signs outside specified range but not to a marked degree e.g. RR 5–10/min, SpO₂ <90–95%, SBP 90–100 or 180–200 mm Hg, HR 40–50 or 120–140/min, temp >38.5 or <35.5°C, no response to voice on AVPU score Very ill 1 or more vital signs outside specified range to a marked degree e.g. RR<5 or

30/min, SpO₂ <90%, SBP<90 or >200 mm Hg, HR <40 or 140/min, temp 38.5 or <35.5°C, uncontrolled severe pain Nearly dead Cardiopulmonary arrest or an acute crisis detected (e.g. threatened airway, severe respiratory distress, sudden loss of consciousness) Continue current management plan Simple interventions (e.g. apply oxygen) and/or review by treating team (e.g. within 30 minutes) Simple interventions (e.g. apply oxygen) and rapid review by treating team plus a critical care clinician (e.g. within 15 minutes) Initiate Basic and Advanced Life Support algorithms. Immediate response by treating team and critical care clinicians Regular nursing review Mandatory minimum frequency of vital sign monitoring with escalation triggered by detected abnormalities commensurate with their severity Fig. 17.1.2 Continued Box 17.1.1 SAMPLE history mnemonic Symptoms Allergies Medications Pertinent medical history Last oral intake Events preceding the deterioration

Section 17 Critical care medicine 3832 The simultaneous clarification of specific diagnoses and institution of appropriate targeted therapies is the other pillar of care. Specific knowledge for managing diverse issues that cross specialties (e.g. from intoxications to neurologic emergencies to traumatic injuries) may also be required. Increasingly patients also require simultaneous management of chronic medical comorbidities. Respiratory support Hypoxia is a major threat to life. It may be classified based on the mechanism leading to the tissues receiving an insufficient oxygen supply for essential metabolic processes (see Box 17.1.10). Oxygen therapy is the mainstay when managing all types of hypoxia, however, specific treatment of the underlying cause is also essential. For example, blood transfusion may be required for severe acute anaemia, restoration of circulation for ischaemic hypoxia, or methylene blue for histotoxic anaemia due to severe methaemoglobinaemia. The most common type of hypoxia is hypoxaemic. Causes of this are shown in Box 17.1.11 and further classified as type 1 or 2 respiratory failure based on the accompanying carbon dioxide (CO₂) level. The emphasis on oxygen therapy during initial resuscitation is to defend against tissue hypoxia that will injure cells rapidly. The negative effects of hypercarbia, including reduced consciousness, peripheral vasodilation, and pulmonary vasoconstriction, are secondary considerations. There are a few caveats to consider. The most recognized is in a subset of patients with chronic obstructive pulmonary disease (COPD) who have chronic hypercarbia and are dependent on hypoxic respiratory drive. In these individuals uncontrolled oxygen therapy may cause or exacerbate hypercarbia due to loss of central hypoxic respiratory centre drive and/or worsening V/Q mismatch from overcoming hypoxic vasoconstriction.

tion. Caution is also warranted in a tiny subset of patients who are primed for oxygen-exacerbated pneumocyte toxicity (e.g. bleomycin chemotherapy or paraquat toxicity). Prolonged high FiO₂ is also associated with lung toxicity, atelectasis from nitrogen washout with loss of the nitrogen 'splinting effect' in the alveoli. A recent systematic review and meta-analysis of 25 randomized controlled trials of conservative versus liberal oxygen therapy in over 16 000 acutely ill adults with a variety of conditions showed that mortality was higher with liberal oxygen (relative risk of death in hospital, 1.21; at 30 days 1.14; at longest follow up 1.10), hence targeting an SpO₂ range above 94–96% would seem to be unwise. In patients where there are concerns regarding excess oxygen, aiming for a PaO₂ greater than 60 mm Hg or SpO₂ of 88–92% is appropriate. The rationale is that this is the area of the sigmoid shaped Hb-Oxygen dissociation curve close to the shoulder of the curve. Small increases in SpO₂ above this point will result in disproportionately large increases in PaO₂, which may be avoided by aiming for this range of oxygen saturation.

Table 17.1.1 An ABCDEFG approach

Element Important things to determine about the patient Possible interventions

A—airway Is there audible stridor or wheeze? Can the patient talk? (If patient can then the airway must be patent) • Sit up and lean the alert patient forward in the 'sniffing position' • If obtunded open the airway with chin lift and jaw thrust and consider suctioning the oropharynx and sweeping with a gloved finger for a foreign body • Nebulise adrenaline for stridor • Nebulise salbutamol for bronchospasm

B—breathing Is there central cyanosis? What is the work of breathing? Is there accessory muscle use? Is chest wall movement symmetrical? • Commence oxygen therapy • Consider needle thoracocentesis to decompress a tension pneumothorax • Intravenous furosemide for acute cardiogenic pulmonary oedema

C—circulation Are the peripheries cold and vasoconstricted, or warm and vasodilated? Is capillary refill time prolonged? Is the patient diaphoretic? Is there skin mottling or tissue oedema? Is there any evidence of external or occult bleeding? Feel for pulse abnormalities—is it irregular, bounding, or thready? • Establish venous access • Commence an intravenous fluid bolus for hypovolaemia • Obtain an ECG to characterize a suspected dysrhythmia and look for signs of ischaemia • Rapid assessment by cardiac echo (RACE) to assess ventricular size and function, and exclude pericardial effusion

D—disability What is the level of consciousness and has this changed? Do an AVPU score (are they Alert, responding only to Voice or to Pain or are they Unresponsive?) Is there any suspicion of seizure activity? • Place the patient on his/her side in the recovery position until definitive airway protection is obtained with tracheal intubation • Administer intravenous benzodiazepine (e.g. diazepam) to terminate a tonic-clonic seizure

E—exposure Are there wounds including pressure areas, rashes, intravenous access devices, AV fistulas, surgical drains? Is there suspicion of a limb DVT? Is there central hypothermia or hyperthermia? • Commence cooling or warming strategies • Ensure intravenous cannulae are patent and attach a bag of fluid

F—fluids What fluids have been administered? What has the urine output and fluid balance been? Does the patient look obviously fluid overloaded and oedematous or dehydrated with reduced skin turgor? • Flush the indwelling urinary catheter to exclude blockage in an oligo-anuric patient

G—glucose Measure blood glucose concentration—is it high or low? • Administer 50 ml of 50% glucose intravenously if patient hypoglycaemic and obtunded

AV, atrioventricular; ECG, electrocardiogram. This is based on material from the NSW Health DETECT programme. DETECT is an acronym for key principles relevant to caring for hospital patients. It stands for Detecting deterioration, making a skilled Evaluation, instituting appropriate Treatment, Escalating concerns to ensure timely medical interventions and the importance of excellent, structured Communication in Teams.

17.1 The seriously ill or deteriorating patient 3833 There are several methods for delivering oxygen noninvasively. Key points related to these are summarized in Table 17.1.3. When oxygen is required for anything other than a short period, the issue of humidification becomes increasingly important. This is the process of making inhaled gases warmed and moist in order to maintain normal cilia function and mucus composition. There is usually progressive heating and humidification of gas as it travels down the airways until the isothermic boundary is reached just below the carina where gases are 100% saturated at 37°C. A lack of humidification results in increased mucus viscosity, reduced mucociliary clearance, cytological injury to the tracheobronchial epithelium with acute inflammation, microatelectasis secondary to obstruction, and mucus plugging with airway obstruction.

Table 17.1.2 Some laboratory data sets

- Diabetic ketoacidosis • Increased anion gap (ketoacids, lactate) and normal anion gap (HCO₃ wasting)
- metabolic acidosis • Variable serum potassium (but total body depletion) • Increased urea and creatinine • Elevated glucose with increased osmolality and hyponatraemia (correct for glucose; serum Na decreases 1 mmol/litre for every 3 mmol/litre increase in glucose concentration)
- Hypoadrenalism • Hypo-osmolar hyponatraemia • Hyperkalaemia • Hypoglycaemia • Mildly increased urea • Mild metabolic acidosis • Hypercalcaemia • Eosinophilia
- Syndrome of inappropriate ADH secretion (SIADH) • Hypo-osmolar hyponatraemia • Increased urinary Na (>20 mmol/litre) with hypertonic urine relative to serum in the setting of normal renal, thyroid, pituitary and cardiac function in the absence of stimulating drugs or physiologic stimuli for ADH secretion
- Tumour lysis syndrome • Hyperkalaemia (real or pseudohyperkalaemia from potassium release from tumour cells in vitro) • Increased urea and creatinine (with increased urea:creatinine ratio) • Hyperuricaemia • Metabolic acidosis
- Rhabdomyolysis • Hyperkalaemia • Hyperphosphataemia • Hypocalcaemia • Increased urea and creatinine (with reduced urea:creatinine ratio) • Increased serum CK, AST, LDH • Myoglobinuria • Metabolic acidosis
- Acute pancreatitis • Hypocalcaemia • Hypophosphataemia • Hyperglycaemia • Increased urea and variable creatinine • Increased serum amylase and lipase
- Thrombotic thrombocytopenic purpura (TTP) The classic pentad of fever, thrombocytopenia, microangiopathic haemolytic anaemia, renal and neurologic dysfunction manifests as: • Low platelets • Reduced Hb with polychromasia, schistocytes, and spherocytes • Increased reticulocytes • Reduced haptoglobin • Increased lactate dehydrogenase • Unconjugated hyperbilirubinaemia with urinary urobilinogen • Variable neutrophilia • Increased urea and creatinine

ADH, antidiuretic hormone; AST, aspartate aminotransferase; CK, creatine kinase; LDH, lactate dehydrogenase; TTP, thrombotic thrombocytopenic purpura.

Box 17.1.2 Causes of hypotension Consider the categories of shock; note shock is frequently multifactorial • Hypovolaemic (e.g. blood loss, dehydration) • Distributive (e.g. sepsis, drugs, anaphylaxis, hypoadrenalism, acute high spinal cord injury) • Cardiogenic (e.g. dysrhythmia, pump failure) • Obstructive (e.g. massive pulmonary embolism, pericardial tamponade, tension pneumothorax)

Box 17.1.3 Causes of hypertension

Acute hypertension • Anxiety/fear • Pain • Urinary retention • Cushing's response to increased intracranial pressure • Tracheal intubation • Drug effect (e.g. vasoconstrictors, amphetamines)

Chronic hypertension—primary (essential) or secondary Hypertensive crises—urgency or emergency

Section 17 Critical care medicine 3834 Additional strategies to improve oxygenation and/or for CO₂ management (ventilation) include noninvasive ventilation with nose or face masks (e.g. continuous/bilevel positive airway pressure—CPAP or BiPAP), tracheal intubation, and ventilation and when this fails, consideration of extracorporeal membrane oxygenation (ECMO). Principal indications for tracheal intubation are summarized in Table 17.1.4. Only those practitioners with

the necessary knowledge and skill should perform tracheal intubation and manage such patients. In contrast, bag-valve mask ventilation is an important life-saving technique and part of the essential skill set for providers of basic and advanced life support. Haemodynamic optimization

Important aspects of haemodynamic support include administration of fluids, electrolytes, blood and blood products, and the use of vasoactive drugs. These therapies necessitate intravenous access. A central venous catheter allowing simultaneous central venous pressure monitoring and vasoactive drug administration and an arterial line for blood pressure monitoring are commonly used. The strategy for venous access should reflect the needs of the patient with considerations such as site (peripheral versus central vein), size (catheter diameter and length), and stability of the patient. For example, patients with life-threatening bleeding require rapid administration of warmed fluids and blood products with flow best achieved through short, wide bore peripheral or central venous cannulae. An intraosseous needle (e.g. EZ-IOTM) inserted into the upper humerus or tibia may be life-saving when a peripheral cannula cannot be inserted and essential drugs and fluids for resuscitation are required. A central line is a priority when stabilizing a patient with complex shock requiring inotropes and vasopressors. Although some advocate the use of algorithms, at the outset it is not possible to judge precisely how much fluid will be needed to resuscitate a patient. The only way to determine this is by frequent clinical examination as fluid is given. In the patient who is very unwell and clearly volume depleted, standard practice is to give 500 ml of blood, plasma expander, or 0.9% saline (as appropriate and as available) as fast as the access will allow (applying pressure to the bag by manual or mechanical compression if the patient is in extremis).

Box 17.1.4 Causes of bradycardia

- Cardiac • sick sinus syndrome, junctional rhythm, AV block
- Noncardiac • Drugs (e.g. β -adrenergic or calcium channel blockers, digoxin, clonidine, narcotics) • Vagal stimulation (e.g. pain, vomiting, coughing) • Athletes/young fit patients • Hypothermia • Neurologic (e.g. increased intracranial pressure, spinal cord injury) • Severe hypoxia • Severe hyperkalaemia • Reflex response to severe hypertension

Box 17.1.5 Causes of tachycardia

- Narrow complex—sinus tachycardia, atrial fibrillation, atrial flutter, AV nodal re-entrant, accessory pathway, atrial tachycardia, junctional tachycardia
- Broad complex—narrow complex tachycardia with conduction system defect or accessory pathway, ventricular tachycardia, pacemaker-mediated tachycardia
- Precipitating factors — Increased sympathetic nervous system activity (e.g. fever, pain, anxiety, drugs with β -adrenergic activity, hyperthyroidism) — Physiologic reflex response (e.g. hypovolaemia, vasodilatation) — Myocardial ischaemia — Acute pulmonary embolism — Sepsis

Box 17.1.6 Causes of arterial desaturation/hypoxia

- Brain (e.g. coma, decreased respiratory drive)
- Spinal cord (e.g. trauma, myelitis)
- Peripheral nervous system (e.g. Guillain-Barre syndrome)
- Airways — Large airways (e.g. tumour, angioedema) — Small airways (e.g. asthma, COPD)
- Lungs — Alveolar (e.g. fluid, infection, haemorrhage, tumour) — Interstitium (e.g. fibrosis)
- Pleural space (e.g. effusion, pneumothorax)
- Chest wall (e.g. flail chest, circumferential burn)
- Diaphragm (e.g. phrenic nerve palsy)
- Abdominal (e.g. abdominal distension and/or pain)

Box 17.1.7 Causes of oliguria

- Prerenal (e.g. hypovolaemia, shock, vascular occlusion)
- Renal (e.g. acute tubular necrosis, contrast nephropathy, glomerulonephritis, acute interstitial nephritis)
- Postrenal (e.g. bladder outlet obstruction, blocked indwelling catheter)

Box 17.1.8 Causes of polyuria

- Physiologic (e.g. mobilization after excess fluid loading, cold diuresis, diuretics, induced hypertension)
- Pathologic (e.g. diabetes mellitus or insipidus, post relief of urinary obstruction, recovery phase of acute kidney injury)

Box 17.1.9 Causes of altered level of consciousness

Think 'AEIOU TIPS'

- A—alcohol
- acidosis
- E—epilepsy, endocrine emergency, electrolytes
- I—infection
- O—overdose, oxygen/carbon dioxide problem
- U—uraemia and other organ failures
- T—trauma to brain
- I—insulin and glucose

disorders P—psychosis and other psychiatric problems S—stroke and intracranial bleeding

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inflating versions can allow ventilation even if oxygen flow ceases – Requires operator skill High-flow nasal cannulae – High and set FiO₂ – High flow rate reduces need for room air entrainment, has effects similar to continuous positive airway pressure (CPAP) system—reduced work of breathing and improved compliance and CO₂ clearance – Gases are humidified and warmed – Comfortable/less claustrophobic than masks

Table 17.1.4 Indications for endotracheal intubation

Indication Examples Obtain an airway Airway obstruction of any cause Protect an airway Coma with risk of obstruction and aspiration Oxygenation Hypoxia refractory to noninvasive oxygen therapy Ventilation Hypercarbia with reduced level of consciousness

Box 17.1.12 Common physiologic parameters

$BP = CO \times TPR$ $CO = HR \times SV$ SV determined by: • Preload • Afterload • Contractility

$MAP = [(2 \times DBP) + SBP]/3$ BP, blood pressure; CO, cardiac output; TPR, total peripheral resistance; HR, heart rate; SV, stroke volume; MAP, mean arterial pressure; DBP, diastolic blood pressure; SBP, systolic blood pressure.

Section 17 Critical care medicine 3836 See Table 17.1.6 for examples of how these principles are employed in developing a strategy for managing shocked patients in contrasting circumstances.

Renal considerations Acute kidney injury is another important consideration in any unstable patient. Determining and treating the cause of oliguria is important in order to prevent progressive dysfunction. When renal failure is progressive, the goals of therapy are to manage life-threatening complications. This may require medical interventions such as intravenous loop diuretics to treat volume overload and/or drugs that target hyperkalaemia (e.g. calcium gluconate/chloride for myocardial stabilization, insulin/glucose to facilitate intracellular shift of potassium ions, resonium to improve potassium removal). Common indications for renal replacement therapy (RRT) are shown in Box 7.1.13. For further discussion see Chapter 21.5. Continuous RRT is commonly performed in an acute setting, particularly in haemodynamically unstable patients, via a venovenous technique using a centrally placed large bore dialysis catheter. The underlying physiologic principles include dialysis (solute movement via diffusion) and/or haemofiltration (water and solute movement via hydrostatic pressure) across a semipermeable filter utilizing an extracorporeal circuit and machine.

Diagnosis of specific conditions The initial management of patients who are desperately ill does not depend on making a precise diagnosis of the cause of their predicament. However, as soon as resuscitation is underway, attention must turn towards making a diagnosis. Although the naive might think that the more severe the illness, the more obvious the cause should be, the opposite is often the case. When dead, all patients look identical, and the same is true just before they die. Patients who are in extremis, whether due to profound hypoxia or with next to no blood pressure, are not lucid historians, and it may be that the only question that they can usefully answer is: ‘Do you have any pain?’ If they indicate their chest or their abdomen, this might be a helpful clue. The pragmatic approach to making a diagnosis in the patient with cardiorespiratory collapse is to use a ‘surgical sieve’ technique, looking systematically for features on examination and investigation to diagnose conditions that can kill (Table 17.1.7). Details of the management of the many specific disorders listed in Table 17.1.7 can be found in the relevant sections of this book, but one general point is extremely important: if initial investigations do not give any clear diagnostic lead, then treatment must be started ‘on suspicion’, especially for disorders that cannot reliably be diagnosed or excluded by clinical examination or by tests that are rapidly available. In particular, pulmonary embolism and sepsis should always be considered. If the clinical context makes pulmonary embolism likely, for instance the patient has collapsed after an operation a week or so ago, then—in the absence of other explanation for the problem—it is sensible to start

Table 17.1.5 Vasoactive drugs Principal effect Examples Vasodilators—widening of blood vessels

from wall relaxation Glyceryl trinitrate, sodium nitroprusside, dobutamine, milrinone, levosimendan Vasopressors—narrowing of blood vessels from wall contraction Noradrenaline, metaraminol phenylephrine vasopressin Chronotrope—increases heart rate Isoprenaline, adrenaline, dobutamine Inotrope—increases myocardial force of contraction Adrenaline, noradrenaline, dobutamine, milrinone levosimendan Lusitrope—facilitates myocardial diastolic relaxation Dobutamine, milrinone a These agents also commonly have dromotropic effects (increase rate of atrioventricular conduction). Table 17.1.6 Examples of haemodynamic strategies to optimize the circulation

Condition	Heart rate (HR)	Preload	Afterload	Contractility
Massive blood loss	Restore circulating volume with fluids and blood and blood products			
Sepsis	Treat arrhythmias such as AF	Fluid load aiming for CVP 8–12	Vasopressors to overcome pathological vasodilatation	Inotropes if sepsis-related myocardial depression
Decompensated heart failure	Treat arrhythmias; avoid bradycardia with ventricular overdistention from prolonged filling time	Avoid fluid overload with left ventricular overdistention; use diuretics to correct intravascular volume overload	Lower afterload with vasodilators	Inotropes to improve contractility
Complete heart block related to a right ventricular (RV) myocardial infarct	Chronotropes and pacing	Fluid load but avoid RV overdistention	Reduce RV afterload with pulmonary vasodilators	Avoid low diastolic pressure to ensure maximal perfusing coronary blood flow; inotropes to assist RV contractility
Severe aortic stenosis with left ventricular hypertrophy	Slow HR 60–80 ideal to allow time for ventricular filling	Maintain sinus rhythm or use atrial pacing to maintain atrial contraction and ventricular filling	Fluid load for CVP >10	Normal or increased afterload to improve diastolic coronary flow to hypertrophied ventricle
	Inotropes not required; drugs with lusitropic properties may improve diastolic dysfunction but caution needed to avoid vasodilatation	AF, atrial fibrillation; BP, blood pressure; CVP, central venous pressure; RV, right ventricular.		

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Box 17.1.13 Indications for renal replacement therapy (RRT)

- Fluid overload unresponsive to diuretics
- Severe hyperkalaemia despite medical therapies
- Severe metabolic acidosis
- Symptomatic uraemia
- Removal of select toxins in life-threatening intoxications (e.g. lithium)

Table 17.1.7 Diagnosis of specific conditions in the patient with cardiorespiratory collapse

Diagnosis	Key finding on examination	Key initial investigation	Definitive investigations
Cardiovascular Myocardial infarction	No specific findings likely	ECG	ECG, cardiac enzymes
Arrhythmia	Pulse rate and rhythm	ECG	ECG
Aortic dissection	Absence or reduction in one or more peripheral pulses, especially left radial. Blood pressure lower in left arm than right	Chest radiograph showing widened mediastinum	Imaging of aorta, usually by CT scan or transoesophageal echocardiography
Cardiac tamponade	Raised jugular venous pressure. Pulsus		

paradoxus (pulse becomes impalpable on inspiration in extreme cases) Chest radiograph may show globular heart. ECG may show low-voltage complexes or electrical alternans Echocardiography Cardiorespiratory Pulmonary embolus Raised jugular venous pressure. Right ventricular heave. Loud P2. Right ventricular gallop rhythm. Signs of deep vein thrombosis in leg ECG may show features of acute right heart strain Echocardiogram—impaired and dilated right ventricle and underfilled left ventricle Ventilation/perfusion scan. Imaging of pulmonary vessels by CT scan or pulmonary angiography CT or digital subtraction pulmonary angiogram Pulmonary oedema Gallop rhythm. Fine inspiratory crepitations Chest radiograph ECG, echocardiography, cardiac enzymes Respiratory Tension pneumothorax Tracheal deviation. Hyperexpansion of one side of chest. Mediastinal shift. Absent breath sounds on one side of chest Chest radiograph—but should be treated by needle decompression on basis of clinical diagnosis Chest radiograph— but should be treated by needle decompression on basis of clinical diagnosis Pneumonia May have high fever. Signs of consolidation or pleurisy Chest radiograph Chest radiograph. Blood culture. Serological tests Asthma Wheezes, but beware of silent chest Response to treatment (β -agonist), but chest radiograph excludes other respiratory diagnoses Peak flow measurements before and after β -agonist Exacerbation of chronic obstructive pulmonary disease Features of chronic obstructive pulmonary disease A clinical diagnosis, but chest radiograph excludes other respiratory diagnoses See Chapter 18.8 Abdominal Gastrointestinal haemorrhage Usually obvious, but do not forget rectal examination for blood/melaena in the patient with unexplained hypotension A clinical diagnosis Endoscopy Perforated viscus Peritonism Erect abdominal radiograph to look for free air CT scan or laparotomy, depending on clinical situation Pancreatitis Peritonism. Bruising in flanks Serum amylase and lipase Imaging of pancreas, usually by CT scan Ruptured abdominal aortic aneurysm Peritonism. Palpable aneurysm. Bruising in flanks A clinical diagnosis CT scan or laparotomy, depending on clinical situation (continued)

Section 17 Critical care medicine 3838 (GI) tract, and hospital-acquired pneumonia. Ensuring adequate nu- trition (using enteral or parenteral nutrition), glucose control, and ad- dressing issues such as pain, anxiety, and delirium are also important. Communication with the patient and/or their family and rec- ognizing when a patient is dying and managing their end-of-life needs are vital. The substantial personal costs to survivors of crit- ical illness are increasingly being recognized. Strategies to improve the quality of life in survivors of critical illness may require diverse interventions ranging from physical rehabilitation of significant deconditioning to treatment of post-traumatic stress disorder. Finally, while ongoing certification to ensure responders to the very ill have vital competency in areas such as basic and advanced life support, there is a progressive recognition that the curriculum needs to extend beyond medical knowledge and skills. Education on topics such as team crisis resource management, stress management, improving self-awareness, and interprofessional communication are becoming routine. FURTHER READING Chu DK, et al. (2018). Mortality and morbidity in acutely ill adults treated with liberal versus conservative oxygen therapy (IOTA): a systematic review and meta-analysis. *Lancet*, 391, 1693–705. Clinical Excellence Commission (2013). Between the Flags Program. NSW Health, Sydney. <http://www.cec.health.nsw.gov.au/patient-safety-programs/adult-patient-safety/between-the-flags> Dickinson E, et al. (2008). *Emergency care*, 11th edition. Prentice Hall, Englewood Cliffs, NJ, p. 242. Foot C, et al. (2011). *Examination intensive care medicine*, 2nd edition. Churchill Livingstone, Australia, pp. 179–80. Gerdik C, et al. (2010). Successful implementation of a family and pa- tient activated rapid response team in an adult level 1 trauma center. *Resuscitation*, 81, 1676–81. Grunau B, et al. (2018). Extracorporeal cardiopulmonary resuscita- tion for refractory out-of-hospital cardiac arrest: the state of the evidence and framework for application. *Canadian J*

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