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REVISION NOTES FOR FRCEM INTERMEDIATE SAQ PAPER

Second Edition

Ashis Banerjee | Clara Oliver

Revision Notes for FRCEM Intermediate SAQ Paper

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SECOND EDITION

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Preface

Successful completion of the Fellowship of the Royal College of Emergency Medicine (FRCEM) examination is a requisite for obtaining a Certificate of Completion of Specialist Training in Emergency Medicine in the United Kingdom. A new examination structure has been introduced as of August 2016.

The FRCEM Primary replaces the MRCEM Part A, and comprises a paper of 180 single-best answer questions lasting three hours. The FRCEM Intermediate replaces the MRCEM Part B with a 60 three-mark short-answer question (SAQ) paper lasting three hours. The FRCEM Intermediate also will include a Situational Judgement Paper, replacing MRCEM Part C and being phased in from autumn 2017.

This book provides candidates with a comprehensive revision tool for the FRCEM Intermediate SAQ paper, the curriculum being the same as the erstwhile MRCEM Part B, which is being continued overseas.

The chapters are divided into systems and the content is based on the Royal College of Emergency Medicine Curriculum for 2015. The format for each topic follows a similar layout incorporating pathophysiology (when relevant), clinical features, investigations, scoring systems (where they exist), ED management, and definitive management (when applicable). National clinical guidelines are often used as the basis for questions and, where relevant, these are summarized. Within each chapter there are 'exam tips' boxes and 'key points' boxes. The exam tips provide advice for the exam and the key points summarize the main learning points from a section. Where, to my knowledge, previous subjects have appeared in a SAQ paper, they will be highlighted. We would like to thank Geraldine Jeffers and Rachel Goldsworthy of the Oxford University Press for guiding this project through to its conclusion and for their meticulous attention to detail. At the end of each chapter are sample SAQs and suggested answers, to enable the candidate to practice questions and reinforce key facts from the chapter.

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Contents

Abbreviations

AAA	abdominal aortic aneurysm
AAGBI	Association of Anaesthetists of Great Britain and Ireland
ABG	arterial blood gases
ACCS	acute care common stem
ACEI	angiotensin converting enzyme inhibitors
ACS	acute coronary syndrome
ADH	antidiuretic hormone
AF	atrial fibrillation
AKI	acute kidney injury
ALS	advanced life support
ALTE	apparent life-threatening event
APLS	advanced paediatric life support
ARB	angiotensin receptor blockers
ARDS	adult respiratory distress syndrome
ASA	American Society of Anaesthesiologists
ASW	approved social worker
ATLS	advanced trauma life support
ATN	acute tubular necrosis
BASH	British Association for the Study of Headache
BiPAP	bi-level positive airways pressure
BLS	basic life support
BPPV	benign paroxysmal positional vertigo
BSA	body surface area
BSPED	British Society of Paediatric Endocrinology and Diabetes
BTS	British Thoracic Society
CAFCASS	Children and Family Court Advisory and Support Service
CAP	community-acquired pneumonia
CCDC	Consultant in Communicable Disease Control
CES	cauda equina syndrome
CK	creatine kinase
CNS	central nervous system
CO	cardiac output
COPD	chronic obstructive pulmonary disease
CPAP	continuous positive airways pressure
CPP	cerebral perfusion pressure
CPR	cardiopulmonary resuscitation
CRAO	central retinal artery occlusion
CRF	corticotrophin-releasing factor
CRP	C-reactive protein
CRRT	continuous renal replacement therapy
CRVO	central retinal vein occlusion
CSF	cerebrospinal fluid
CVP	central venous pressure
CXR	chest X-ray
DI	diabetes insipidus
DIC	disseminated intravascular coagulation
DIPJ	distal interphalangeal joint
DKA	diabetic ketoacidosis
DNAR	Do Not Attempt Resuscitation
DVLA	Driver and Vehicle Licensing Agency
DVT	deep vein thrombosis
ECG	electrocardiogram
ED	emergency department
EGDT	early goal-directed therapy
EM	erythema multiforme
ENT	ear, nose, and throat
EPAP	expiratory positive airway pressure
EPLS	European Paediatric Life Support
ESR	erythrocyte sedimentation rate
ETT	endotracheal tube
FAST	face arm speech test
FAST	focused assessment with sonography in trauma
FBC	full bood count
FDP	Flexor digitorum profundus
FDS	Flexor digitorum superficialis
FRC	functional residual capacity
FRCEM	Fellowship of the Royal College of Emergency Medicine
FVC	forced vital capacity
GABA	gamma-aminobutyric acid
GCS	Glasgow coma scale
GFR	glomerular filtration rate

GMC General Medical Council
GRACE Global Registry of Acute Cardiac Events
HELLP haemolysis, elevated liver enzymes, and low platelets
HHS hyperosmolar hyperglycaemic state
HSV herpes simplex virus
IBD Inflammatory bowel disease
ICHD International Classification of Headache Disorders
ICP intracranial pressure
ICU intensive care unit
IMCA independent medical capacity advocates
IO intraosseous
IPAP inspiratory positive airway pressure
IPJ interphalangeal joint
ITP idiopathic thrombocytopenic purpura
IVC intravenous
IVC inferior vena cava
LACS lacunar stroke
LAT lidocaine, adrenaline, tetracaine
LMA laryngeal mask airway
LMN lower motor neuron
LMWH low molecular weight heparin
LPA Lasting Power of Attorney
MAP mean arterial pressure
MRCEM Membership of the Royal College of Emergency Medicine
MHRT Mental Health Review Tribunal
MI myocardial infarction
MSCC metastatic spinal cord compression
NAI non-accidental injury
NCEPOD National Confidential Enquiry into Patient Outcomes and Death
NEXUS National Emergency X-Radiography Utilization Study
NG nasogastric
NICE National Institute for Health and Care Excellence
NIHSS National Institute for Health Stroke Score
NIV non-invasive ventilation
Nok next of kin
NR nearest relative
NSAID non-steroidal anti-inflammatory drug
NSTEMI non-ST-segment elevation myocardial infarction
NYHA New York Heart Association
OSCE objective structured clinical examination
PACS partial anterior circulation stroke
PCI percutaneous coronary intervention
PCR polymerase chain reaction
PD peritoneal dialysis
PE pulmonary embolism
PEA pulseless electrical activity
PEF peak expiratory flow
PEFR peak expiratory flow rate
PEP post-exposure prophylaxis
PID pelvic inflammatory disease
POCS posterior circulation stroke
PVC premature ventricular contractions
RCEM Royal College of Emergency Medicine
RCOG Royal College of Obstetricians and Gynaecologists
RIPL Rare and Imported Pathogens Laboratory
RMO responsible medical officer
ROSC return of spontaneous circulation
ROSIER recognition of stroke in the emergency room
RRT renal replacement therapy
RSI rapid sequence induction
RTC road traffic collisions
SAQ short-answer question
SBP spontaneous bacterial peritonitis
SCD sudden cardiac death
SCT supervised community treatment
SIGN Scottish Intercollegiate Guidelines Network
SIRS systemic inflammatory response syndrome
SJS Steven-Johnson Syndrome
SSC Surviving Sepsis Campaign
SSRI selective serotonin reuptake inhibitors
SSSS Staphylococcal scalded skin syndrome
STEMI ST-elevation myocardial infarction
TACS total anterior circulation stroke
TCA tricyclic antidepressant
TEN toxic epidermal necrolysis
TIA transient ischaemic attack
TIMI thrombolysis in myocardial infarction
TIPS transjugular intrahepatic portosystemic shunting
TLoC transient loss of consciousness
TLS tumour lysis syndrome
TNF tumour necrosis factor
TRH thyrotropin-releasing hormone
TSH thyroid stimulating hormone
UMN upper motor neuron
UTI urinary tract infection
VF ventricular fibrillation
VHF viral haemorrhagic fevers
VT ventricular tachycardia
WCC white cell count
WFNS World Federation of Neurological Surgeons

CHAPTER 1

Exam tips

CONTENTS

1.1 Structure of the exam

The Fellowship of the Royal College of Emergency Medicine (FRCEM) exam is a pre-requisite for obtaining the Certificate of Completion of Specialist Training in the United Kingdom. It consists of three sections, Primary, Intermediate, and Final FRCEM, as from August 2016.

The FRCEM Primary consists of a single best-answer question paper of 180 questions in three hours, and replaces the MRCEM Part A. A maximum of six attempts is allowed to pass the examination. Previous attempts at the MRCEM Part A examination will not be included in the number of available attempts for the FRCEM Primary examination.

The FRCEM Intermediate replaces MRCEM Parts B and C for Emergency Medicine trainees in the United Kingdom. It comprises a short-answer question (SAQ) paper consisting of 60 three-mark questions in three hours, and a situational judgement paper consisting of 120 single best-answer questions in two hours.

The regulations relating to the FRCEM examinations can be found on the Royal College of Emergency Medicine website (https://www.rcem.ac.uk).

This book is aimed at candidates sitting the FRCEM Intermediate SAQ paper.

1.2 Marking system

The FRCEM Intermediate consists of 60 SAQs in three hours. The questions are typically divided into three or four parts and are carefully constructed so that failure to answer the first part of the question does not preclude answering other parts. The available marks for each part of the question are displayed so that candidates can judge how much detail to write.

The exam is scored cumulatively and there is a pass mark, not a rate. The pass mark is determined before the exam, depending on the difficulty index of the exam. Examiners meet before the exam to determine the pass mark and ensure the questions are clear, unambiguous, and fair. The pass mark is roughly in the range 60–68%. There are no critical response or 'sudden death' questions.

It is often easier to gain the first 5 marks of a question than the last 5. Therefore, if you are struggling with a question, it is worth moving on. Questions will frequently indicate the number of

responses expected. Examiners will mark the answers in the order written, therefore ensure your first answer is your 'best answer' and do not give more answers than requested. If the question asks for four answers and you write five, only the first four will be marked, even if the fifth is correct. Marks will not be gained for repeating an answer in the same question or reiterating information given in the stem of the question.

It is very important to read the stem of the question carefully. Had this scenario been that the patient had recently fractured his or her femur and asked for four other risk factors, then 'recent lower limb trauma' would not gain a mark.

Answers must be legible and any corrections made clearly so the examiner knows which answer is to be considered. The exam is written in pencil, which sometimes surprises candidates, so it is worth practising writing in pencil beforehand.

EXAM TIP

Example question:

Give four risk factors for a pulmonary embolism (4 marks)

Recent immobilization	Correct (1 mark)
Recent lower limb trauma	Correct (1 mark)
Diabetes	Incorrect
Clinical deep vein thrombosis	Correct (1 mark)
Malignancy	(not marked because fifth answer)

This answer would score 3 out of a 4 possible marks because, despite having four correct answers, only three of the first four are correct.

1.3 Content

Emergency Medicine encompasses many specialties, and national guidance and evidence-based medicine protocols often appear in the examination. This is done to ensure that the exam is up-to-date, but also to encourage candidates to keep up-to-date with recently published scientific evidence. If there are multiple guidelines, then allowance is made for this in the marking scheme.

The basic sciences are primarily examined in the FRCEM Primary but may also be assessed in FRCEM Intermediate. There is not a separate basic sciences chapter in this book but, where relevant, information on basic sciences is included.

Some topics appear more commonly in exams than 'real-life' practice (e.g. dermatology and ophthalmology). This is particularly true in the FRCEM Intermediate examination, because questions based around visual images are relatively easy to design for a SAQ, but more difficult to include in an objective structured clinical examination (OSCE).

1.4 Curriculum

The College of Emergency Medicine published a new curriculum in August 2015, replacing the June 2010 curriculum.

The curriculum is divided into major and acute presentations. Due to this design, certain conditions are applicable to several different presentations; for example, pulmonary embolism is relevant to the major presentations of cardiorespiratory arrest and shock, and the acute presentations of breathlessness, chest pain, blood gas interpretation, and cyanosis. Therefore, to avoid unnecessary repetition, this book's chapters are written by system and not by presenting complaint. To

enable the candidate to use this book in conjunction with the new curriculum, the following tables list the presentations and corresponding chapters that trainees are expected to cover in their first three years of UK Emergency Medicine training (i.e. the curriculum that is used for the FRCEM examination).

The paediatric curriculum is largely covered in Chapter 20 with cross-referencing to other chapters, where necessary.

1.4.1 Major adult presentations

Table 1.1 lists the chapters covering the major adult presentations.

Table 1.1 Chapters covering the major adult presentations

CMP1—Anaphylaxis	Chapter 2
CMP2—Cardiorespiratory arrest	Chapter 2
CMP3—Major trauma	Chapter 4
CMP4—Septic patient	Chapter 15
CMP4—Shocked patient	Chapter 2
CMP5—Unconscious patient	Chapters 4 and 11

1.4.2 Acute adult presentations

Table 1.2 lists the chapters covering the acute adult presentations.

Table 1.2 Chapters covering the acute adult presentations

CAP1—Abdominal pain, including loin pain	Chapter 6
CAP2—Abdominal swelling, mass, constipation	Chapter 6
CAP3—Acute back pain	Chapter 5
CAP4—Aggressive/disturbed behaviour	Chapters 18 and 21
CAP5—Blackout/collapse	Chapters 9 and 11
CAP6—Breathlessness	Chapter 10
CAP7—Chest pain	Chapter 9
CAP8—Confusion/acute delirium	Chapters 11 and 18
CAP9—Cough	Chapter 10
CAP10—Cyanosis	Chapter 10
CAP11—Diarrhoea	Chapter 13
CAP12—Dizziness and vertigo	Chapters 7 and 11
CAP13—Falls	Chapter 11
CAP14—Fever	Chapter 15
CAP15—Fits and seizures	Chapter 11
CAP16—Haematemesis and melaena	Chapter 13
CAP17—Headache	Chapter 11

(*continued*)

Table 1.2 Continued

CAP18—Head injury	Chapter 4
CAP19—Jaundice	Chapter 13
CAP20—Limb pain and swelling (atraumatic)	Chapter 5
CAP21—Neck pain	Chapter 4
CAP22—Oliguric	Chapter 6
CAP23—Pain management	Chapter 3
CAP24—Painful ears	Chapter 7
CAP25—Palpitations	Chapter 9
CAP26—Pelvic pain	Chapter 8
CAP27—Poisoning	Chapters 17 and 18
CAP28—Rash	Chapter 16
CAP29—Red eye	Chapter 7
CAP30—Mental health	Chapter 18
CAP31—Sore throat	Chapter 7
CAP32—Syncope and pre-syncope	Chapter 9
CAP33—Traumatic limb and joint injuries	Chapters 4 and 5
CAP34—Vaginal bleeding	Chapter 8
CAP35—Ventilatory support	Chapters 3 and 10
CAP36—Vomiting and nausea	Chapter 13
CAP37—Weakness and paralysis	Chapters 4 and 11
CAP38—Wound assessment and management	Chapter 5

1.4.3 Major paediatric presentations

Table 1.3 lists the chapters covering the major paediatric presentations.

Table 1.3 Chapters covering the major paediatric presentations

PMP1—Anaphylaxis	Chapter 19
PMP2—Apnoea, stridor, and airway obstruction	Chapter 19
PMP3—Cardiorespiratory arrest	Chapter 19
PMP4—Major trauma	Chapter 19
PMP5—The shocked child	Chapter 19
PMP6—The unconscious child	Chapter 19

1.4.4 Acute paediatric presentations

Table 1.4 lists the chapters covering the acute paediatric presentations.

Table 1.4 Chapters covering the acute paediatric presentations

PAP1—Abdominal pain	Chapters 6 and 19
PAP2—Accidental poisoning, poisoning, and self-harm	Chapters 17 and 18
PAP3—Apparent life-threatening events (ATLE)	Chapter 19
PAP4—Blood disorders	Chapters 19 and 20
PAP5—Breathing difficulties	Chapter 19
PAP6—Concerning presentations	Chapter 19
PAP7—Dehydration secondary to diarrhoea and vomiting	Chapter 19
PAP8—Ears, nose, and throat (ENT)	Chapter 7
PAP9—Fever in all age groups	Chapters 15 and 19
PAP10—Floppy child	Chapter 19
PAP11—Gastrointestinal bleeding	Chapter 19
PAP12—Headache	Chapters 11 and 19
PAP13—Neonatal presentations	Chapter 19
PAP14—Ophthalmology	Chapter 7
PAP15—Pain in children	Chapter 3
PAP16—Painful limbs in children—atraumatic	Chapter 19
PAP17—Painful limbs in children—traumatic	Chapters 5 and 19
PAP18—Rashes in children	Chapter 16
PAP19—Sore throat	Chapter 7

1.5 Glossary

The questions for the FRCEM examinations are put through a rigorous refinement process. Every word of the SAQ clinical scenario is carefully considered. Candidates are provided with a glossary of terms used in the exam, which can be downloaded from the College website (https://www.rcem.ac.uk) and is worth looking through before the exam.

The terms commonly used in exam questions are summarized.

- **Abnormality:** any feature in an examination or investigation that is outside the standard deviation of the population being studied. A clinical abnormality relates to a pathologically relevant abnormality (e.g. the presence of facial burns and not the presence of an endotracheal tube).
- **Assessment:** history-taking, physical examination, and use of investigations.
- **Clinical findings:** these may include symptoms, signs, and vital signs. It is information gleaned from the clinical evaluation, but not the results of investigations, even bedside ones (e.g. blood glucose or urine dipstick).
- **Criteria:** refer to the fact that there is a formal international/national guideline or scoring system that allows you to define the seriousness of a condition (e.g. CURB-65 score for pneumonia).

- **Definitive management:** this may include things you would do in the emergency department but usually requires you to list the operation or procedure that will cure or contain the condition.
- **Emergency department (ED) management:** this requires you to list actions that are life or limb-saving or that might improve the course of the condition if done within the ED. It is not definitive management. This may, however, include analgesia, referral to specialty team, and so on.
- **Essential:** this indicates life-saving treatments/management steps that are the priority, and would not normally include things like analgesia, communication, and so on.
- **Features:** in the context of a medical history, may be either a symptom or a sign. If asked for key features, you should give the symptoms or signs that are definitive for that condition, rather than general abnormalities that might be present. When asked for in the context of an electrocardiogram (ECG) or chest X-ray (CXR), it might be a pathological abnormality or might simply be the presence of an endotracheal tube (ETT) or central line.
- **Investigations:** specific tests undertaken to make a diagnosis or monitor the patient's condition. They may include bedside tests such as urine dipstick or blood glucose, unless otherwise specified.
- **Management:** aspects of care, including treatment, supportive care, and disposition. This does not include investigations.
- **Pathophysiological sequence of events:** this requires you to list in chronological order, the events that happen on a cellular or hormonal level, leading to the current condition. For example, if a lactate is high in the presence of sepsis, you could suggest:
  - hypotension;
  - poor organ perfusion;
  - tissue hypoxia;
  - anaerobic metabolism;
  - glycolysis and lactate build-up.
- **Treatment:** measures undertaken to cure or stabilize the patient's condition. This includes oxygen, fluids, drugs, and may also mean surgery. It does not include investigations.

Throughout this book, terminology has been used in keeping with the College glossary to help you become familiar with how the terms are used.

1.6 Making the most of your knowledge

Having reached this stage in your medical career, you are already well-versed in sitting exams. However, it may have been several years since your last written exam. A consistent theme of feedback from examiners is that candidates let themselves down through their exam technique. Practising questions before the exam is crucial to achieve the best result possible.

Each chapter of this book has practice SAQs, which will help refine your technique. It is also useful to sit mock exams to get used to performing under exam conditions. Such mocks are available via revision courses or frequently from regional Emergency Medicine training schemes.

When answering questions, think carefully about what is being asked. Long lists of investigations or differential diagnoses are unlikely to be acceptable. If asked to give investigations, try to use ones that will help differentiate between causes or be useful in the risk assessment of the disease. Often questions will ask you to justify why you want a certain investigation; for example, measuring urea in pneumonia so that a CURB 65 score can be calculated. Similarly, when answering questions on treatments, the mechanism of action may be asked for (e.g. salbutamol is a beta-agonist and used in asthma as a bronchodilator).

Knowledge of drug doses often worries candidates because they are uncertain which ones they need to remember. As a general rule, drugs doses that are used in the emergency resuscitation setting should be known because in practice there is often not enough time to look these up. Similarly, commonly used drugs (e.g. analgesics, antibiotics) should be known. Other questions may ask for the drug plus the dose and route. Candidates often lose marks on these questions because they write the drug name, but don't write the dose or route because they are uncertain of the dose. Remember there is no negative marking, so it is worth a guess and even if you are uncertain of the dose, the route is usually known, so don't forget to include it in your answer.

No matter how much preparation is done before the exam, there will always be unexpected questions. It is important to remember that these will be unexpected for the majority of candidates and not to panic. There is usually something that can be remembered about the topic and it is worth having a guess because there is no negative marking.

1.7 How to use this book

The chapters are divided into systems and the content is based on the Royal College of Emergency Medicine Curriculum 2015. Each chapter has a contents page listing the main topic headings, which are numbered numerically to enable easy reference. Section 1.4 in this chapter, 'Curriculum', provides a cross-reference between each book chapter and presenting complaint, as laid out in the new curriculum.

The format for each condition within a chapter follows a similar layout incorporating pathophysiology (when relevant), clinical features, investigations, scoring systems (where they exist), ED management, and definitive management (when applicable). The exception to this are conditions that require a more practical knowledge (e.g. resuscitation, airway management, and so on).

Increasingly, the College is using national guidelines as the basis for questions. Where relevant, this book summarizes established UK guidelines (e.g. National Institute for Health and Care Excellence (NICE), Scottish Intercollegiate Guidelines Network (SIGN), British Thoracic Society (BTS), and so on). If UK guidance does not exist, then European guidelines may be used. The guidelines used are clearly identified and website addresses provided so that the candidate can check for any updates prior to the exam.

Within each chapter there are 'exam tips' boxes and 'key points' boxes. The exam tips provide advice for the exam and the key points summarize the main learning points from a section.

EXAM TIP

Always ensure you read the question carefully, remain calm, and write clear, concise answers.

Previous subjects known to have appeared in previous examinations will be highlighted.

Previous MRCEM question

At the end of each chapter are sample SAQs and suggested answers. The SAQs can be used for exam practice and will reinforce key facts from the chapter.

Further reading

Royal College of Emergency Medicine. Available at: www.rcem.ac.uk [Online].

KEY POINTS

The FRCEM exam has three sections, Primary, Intermediate, and Final, which have to be passed in succession to be awarded Fellowship of the Royal College of Emergency Medicine.

The FRCEM Intermediate focuses on data interpretation skills and is a short-answer question (SAQ) paper. The paper consists of 60, 3 -mark questions. The exam lasts three hours.

There is no negative marking.

There are no critical response or 'sudden death' questions.

Examiners will mark the answers in the order written, therefore ensure your first answer is your 'best answer' and do not give more answers than requested.

The Royal College of Emergency Medicine published a new curriculum in August 2015.

Section 1.4 in this chapter, 'Curriculum', provides a cross-reference between book chapter and presenting complaint as laid out in the new curriculum.

The questions for the FRCEM examinations use specific terms which are defined in a glossary that the candidate is provided with in the exam and is available on the College website. Look through the glossary before the exam so that you are familiar with the terms.

CHAPTER 2

Resuscitation

CONTENTS

2.1 Advanced life support

2.1.1 Introduction

Completion of an advanced life support (ALS) course is a mandatory requirement of Acute Care Common Stem (ACCS) training and all candidates would be expected to be ALS providers prior to sitting the FRCEM Intermediate examination.

This section is based on the 2015 Resuscitation Council Guidelines. It includes the ALS algorithms and focuses on cardiac arrest in special circumstances. The aim is to highlight the main features of, and changes in, the 2015 guidance and focus on areas that could be questioned in an short-answer question (SAQ) paper.

EXAM TIP

The Resuscitation Council manuals are a good resource for revision. The latest guidance is freely available on their website https://www.resus.org.uk/.

ALS is most likely to be examined in the OSCEs of the Final FRCEM but knowledge of the guidance could appear in a SAQ.

2.1.2 ALS algorithm

Figure 2.1 shows the adult ALS algorithm and the main features of the 2015 guidelines are presented in Table 2.1.

2.1.3 Shockable rhythms (VF/VT)

The first monitored rhythm in approximately 28–35% of cardiac arrests out of hospital is ventricular fibrillation (VF) or pulseless ventricular tachycardia (VT) with approximately 21% surviving to hospital discharge. In hospital, VF/VT accounts for approximately 18% of cardiac arrests, with approximately 44% surviving to discharge.

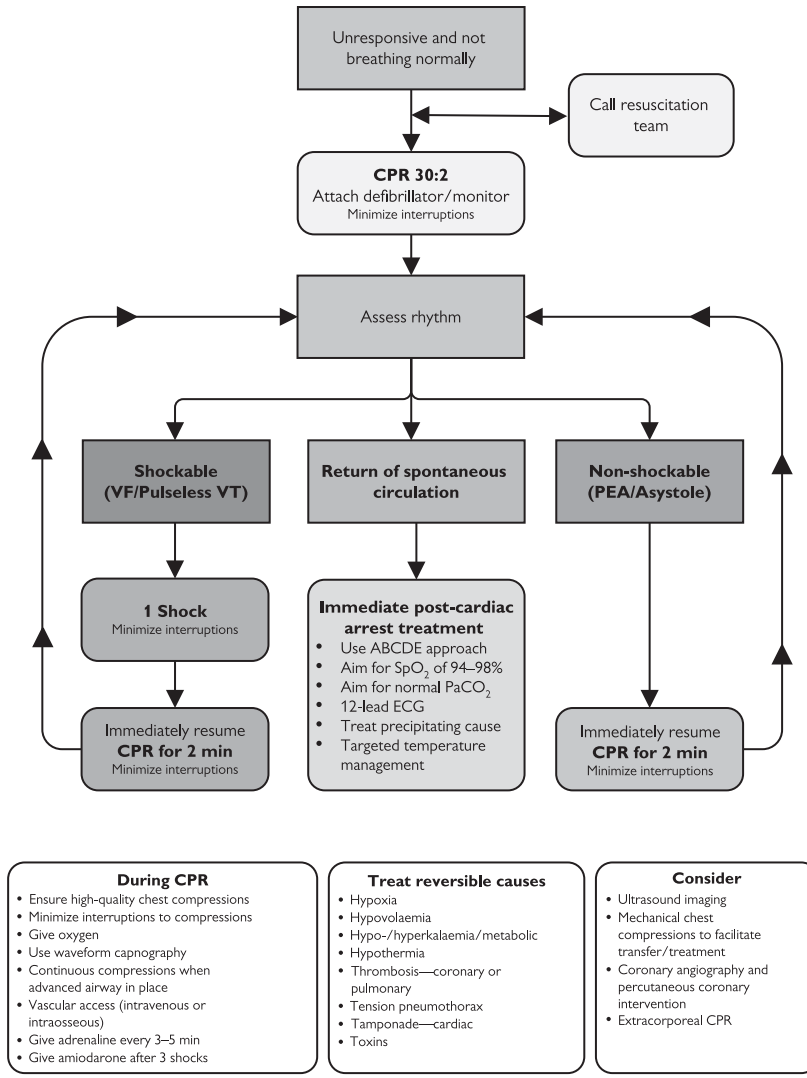

Figure 2.1 Adult advanced life support (ALS) algorithm.

Reproduced with the kind permission of the Resuscitation Council (UK).

Defibrillation strategy

- The 2005 guideline first introduced the single-shock strategy instead of the previously used three-stacked shocks protocol. The 2015 guidelines continue to recommend a single-shock strategy in order to minimize interruptions to chest compressions.
- In a few very specific circumstances, three initial stacked shocks have been reintroduced: patients who have just had cardiac surgery; patients in the cardiac catheter laboratory; and patients who have a witnessed monitored arrest and are already connected to a manual defibrillator. If given, the three initial stacked shocks should be considered as the first shock in the ALS algorithm, and both adrenaline and amiodarone should be given after a further two defibrillation attempts (i.e. delayed until after the fifth shock).

Table 2.1 Main features of the 2015 Resuscitation Council Adult Guidelines

BLS	● Minimally interrupted high-quality chest compressions remain essential to improving outcomes and should be to a depth of 5–6 cm and at a rate of 100–120 per minute ('push hard and fast'). After each compression, the chest should be allowed to recoil completely. When providing rescue breaths/ventilations, about 1 s should be spent inflating the chest with sufficient volume to ensure visible chest wall rise. The ratio of chest compressions to ventilations remains 30:2. Chest compressions should not be interrupted for more than 10 s to provide ventilations. ● The routine use of mechanical chest compression devices is not recommended, however they may be useful in certain situations, where sustained high-quality manual chest compressions are impractical or compromise patient safety.
Defibrillation	● Interruptions to chest compressions should be kept to a minimum and pauses should only occur briefly to enable specific planned interventions (e.g. defibrillation, intubation). ● Chest compressions should continue during charging of the defibrillator to reduce the pre-shock pause to less than 5 s. ● Chest compressions should be continued immediately after delivering a shock, as it is very rare for a pulse to be palpable immediately after defibrillation, and as the duration of asystole can be longer than 2 minutes in as many as 25% of successful shocks. ● The focus on using self-adhesive pads for defibrillation continues, although it is recognized that defibrillator paddles are used in some settings.
Drugs	● Peripheral venous access is quicker, easier to perform, and safer. If intravenous (IV) access is not achievable, intraosseous (IO) access should be used. ● In the shockable ventricular fibrillation/ventricular tachycardia (VF/VT) algorithm, adrenaline 1 mg is given after the third shock, once chest compressions have resumed. Adrenaline is then given every 3–5 min (alternate cycles). ● Amiodarone 300 mg is given after the third shock in VF/VT cardiac arrests. Consider a further dose of amiodarone 150 mg after a total of five shocks. Lidocaine 1 mg/kg may be used as an alternative if amiodarone is not available but should not be given if amiodarone has been given already. ● There is greater equipoise (uncertainty regarding the therapeutic benefits) concerning the role of drugs in improving outcomes from cardiac arrest. Drugs are of secondary importance to high-quality uninterrupted chest compressions and early defibrillation.
Airway	● A stepwise approach to airway management based on patient factors, the phase of the resuscitation attempt and the skills of the rescuer is recommended. This includes compression-only CPR, compression-only CPR with the airway held open, mouth-to-mouth breaths, mouth-to-mask, bag-mask ventilations with simple airway adjuncts, supraglottic airways, and tracheal intubation. ● The use of waveform capnography to monitor end-tidal CO_2 is recommended to confirm and continually monitor tracheal tube placement, quality of chest compressions during CPR, and to provide an early indication of return of spontaneous circulation (ROSC). ROSC can be detected without pausing chest compressions and bolus adrenaline injection can be avoided as a result.
Ultrasound	● The potential role of ultrasound in identifying reversible causes of cardiac arrest continues to be emphasized (e.g. cardiac tamponade; pulmonary embolism (PE); myocardial infarction (regional wall motion abnormality); aortic dissection; hypovolaemia; and pneumothorax).

(*continued*)

Table 2.1 Continued

Post-resuscitation care	• Hyperoxia may be harmful after ROSC, therefore oxygen should be titrated to maintain saturations of 94–98%. • There is a greater emphasis on urgent coronary catheterization and primary percutaneous coronary intervention (PCI) in patients with ROSC following out-of-hospital cardiac arrest of likely cardiac cause. • Blood glucose levels >10 mmol/L should be treated, but hypoglycaemia should be avoided. • Targeted temperature management or temperature control should be considered in comatose survivors of cardiac arrest associated with non-shockable, as well as shockable, rhythms. There is an option to target a temperature of 36 C instead of the previously recommended 32–34 C. • Extracorporeal life support techniques may be used as a rescue therapy in selected patients where standard ALS measures are not successful, or to facilitate specific interventions (e.g. coronary angiography, PCI, or pulmonary thrombectomy).

- The recommended initial biphasic shock should be at least 150 J. Recommendations for subsequent shocks depend on the manufacturer of the machine and may be fixed at 150 J or escalate. When using a monophasic machine, all shocks should be at 360 J.

Fine ventricular fibrillation

Fine VF that is difficult to distinguish from asystole should be treated as asystole with good-quality closed chest compression. This may improve the amplitude and frequency of the VF and improve the chances of successful defibrillation. Repeated defibrillation of fine VF is very unlikely to restore a perfusing rhythm and will increase myocardial injury, both directly by the electrical current and indirectly by interruptions in chest compressions.

2.1.4 Non-shockable rhythms (pulseless electrical activity and asystole)

Pulseless electrical activity (PEA) and asystole are the commonest initial cardiac arrest rhythms and have a less favourable outcome than shockable rhythms (8% of out-of-hospital patients and 7% of in-hospital patients surviving to discharge). Unless a reversible cause can be found and treated, survival is unlikely.

2.1.5 Reversible causes

Potential reversible causes should be considered and treated in both shockable and non-shockable cardiac arrests.

- **Hypoxia**—should be corrected by delivering high-concentration oxygen to the patient via a bag-valve-mask, supraglottic airway device, or endotracheal tube. Effective ventilation should be confirmed by chest wall movement and auscultation.
- **Hypovolaemia**—is usually due to severe haemorrhage (e.g. trauma, GI bleeding, aortic aneurysm rupture). Intravenous fluids should be infused rapidly if hypovolaemia is suspected. Crystalloid (0.9% sodium chloride or Hartmann's) is appropriate because there is no clear advantage for colloid.
- **Hyper/hypokalaemia, hypocalcaemia, and other metabolic disorders**—are usually detected on arterial blood gas analysis, recent blood results, or suggested by the patient's history. The management of electrolyte disturbances is discussed in the special circumstances section (section 2.2).

- **Hypothermia**—should be checked for with a low-reading thermometer. If the patient is hypothermic, they should be rewarmed to 36°C.
- **Tension pneumothorax**—can be detected clinically or via ultrasound. Needle thoracocentesis should be performed to decompress the pneumothorax and followed up with a chest drain.
- **Cardiac tamponade**—can be difficult to detect clinically in an arrested patient. There should be a high index of suspicion for patients suffering penetrating thoracic trauma. Echocardiography may be used to confirm the diagnosis. If present, resuscitative thoracotomy should be performed.
- **Toxins**—the history is important in determining whether therapeutic or toxic substances could have precipitated the arrest. If an appropriate antidote (e.g. naloxone for opiates) exists, it should be administered.
- **Thromboembolic**—a massive pulmonary embolism (PE) can cause circulatory obstruction and cardiac arrest. The diagnosis is difficult and may be based purely on the patient's history prior to the arrest. If rapid echocardiography is available, this may support the diagnosis (dilated right heart). The use of thrombolysis in cardiac arrest is decided on a case-by-case basis. If thrombolysis is given, it may take up to 90 minutes to be effective and should only be administered if it is appropriate to continue cardiopulmonary resuscitation (CPR) for this length of time.

2.1.6 Drugs used in cardiac arrest

Table 2.2 shows the drugs used in cardiac arrest.

Table 2.2 Drugs used in cardiac arrest

Drug	Actions	Dose/Indications
Adrenaline	• Direct-acting sympathomimetic amine. • Stimulates α and β adrenergic receptors. • Stimulation of α receptors produces vasoconstriction, increasing systemic vascular resistance, and improving cerebral and coronary perfusion during CPR. • Stimulation of β_1 receptors increases heart rate and the force of contraction (in a beating heart).	• Adrenaline dose 1 mg. • Give as soon as possible in PEA/asystole. • Give after the third shock in VF/VT. • Repeat every 3–5 min (alternate cycles).
Amiodarone	• Membrane-stabilizing anti-arrhythmic drug. • Increases the duration of the action potential and refractory period in atrial and ventricular myocardium, and slows AV conduction. • Slight negative inotropic action and can cause peripheral vasodilatation.	• Amiodarone dose 300 mg bolus after the third shock in VF/VT. • A further 150 mg may be given for refractory VF/VT followed by a 900 mg infusion over 24 h.
Magnesium	• Constituent of many enzyme systems, especially those involved with generating adenosine triphosphate (ATP) in muscle. • Decreases acetylcholine release and reduces the sensitivity of the motor end plate. • Improved contractility of the stunned myocardium and limits infarct size (mechanism unknown). • Bronchodilator.	• Magnesium dose 2 g. Indications: • Shock refractory VF/VT if hypomagnesaemia suspected. • Ventricular tachyarrhythmias if hypomagnesaemia suspected. • Torsades de pointes. • Digoxin toxicity.

(*continued*)

Table 2.2 Continued

Drug	Actions	Dose/Indications
Sodium bicarbonate	• Alkalinization to correct pH. • Bicarbonate administration generates carbon dioxide, which diffuses rapidly into cells. Effects include: • Exacerbation of intracellular acidosis. • Negative inotropic effect on ischaemic myocardium. • Large, osmotically active, sodium load to an already compromised circulation and brain. • Shifts the oxygen dissociation curve to the left inhibiting the release of oxygen to the tissues.	• Bicarbonate dose 50 ml of 8.4% (50 mmol). Indications: • Hyperkalaemia. • Tricyclic acid overdose.
Calcium	• Involved in the cellular mechanisms underlying myocardial contraction.	• Calcium chloride dose 10 ml of 10%. • Can be repeated PRN. • Indications: • Hyperkalaemia. • Hypocalcaemia. • Overdose of calcium channel blocking drugs.

KEY POINTS

Pauses in chest compressions should be kept to a minimum.

When defibrillating, the pre-shock pause should be kept to a minimum (<5 s) by planning ahead, continuing chest compressions during charging, and using a very brief safety check.

Adrenaline 1 mg should be given as soon as IV/IO access is gained in asystole/PEA and after the third shock in VF/VT.

Amiodarone 300 mg IV is given after the third shock in VF/VT.

Waveform capnography should be used, if available, in an intubated patient to confirm and continually monitor tube placement, quality of CPR, and to provide an early recognition of return of spontaneous circulation (ROSC).

If thrombolysis is given for a massive PE in cardiac arrest, CPR should be continued for 60–90 min.

Targeted temperature management remains important with a target of 33–36 C depending on local policy.

2.2 Cardiac arrest in special circumstances

2.2.1 Introduction

Good-quality CPR is paramount, regardless of the cause of cardiac arrest; however, certain circumstances require additions and modifications to the resuscitation algorithm. These 'special circumstances' could well appear in an SAQ.

2.2.2 Electrolyte disorders

Hyperkalaemia

Hyperkalaemia is defined as a K^+ >5.5 mmol/L. The management of hyperkalaemia in the non-arrested patient is discussed in Chapter 12, section 12.4. The modifications for ALS in the arrested hyperkalaemic patient are:

- Calcium chloride: 10 ml of 10% given as a rapid bolus
- Sodium bicarbonate: 50 ml of 8.4% given as a rapid injection
- Insulin/glucose: 10 units of short-acting insulin and 50 ml of 50% glucose given as a rapid infusion
- Haemodialysis: considered in resistant hyperkalaemia

Hypokalaemia

Hypokalaemia is defined as a K^+ <3.5 mmol/L and severe hypokalaemia as a K^+ <2.5 mmol/L.

Causes of hypokalaemia include:

- Gastrointestinal losses (e.g. diarrhoea)
- Drugs (e.g. diuretics, laxatives, steroids, adrenaline, and so on)
- Renal losses (e.g. renal tubular acidosis, diabetes insipidus, dialysis)
- Endocrine disorders (e.g. Cushing's syndrome, hyperaldosteronism)
- Metabolic alkalosis
- Hypomagnesaemia

Clinical features of hypokalaemia include:

- Fatigue
- Weakness
- Cramps
- Constipation
- Rhabdomyolysis
- Ascending paralysis

Electrocardiogram (ECG) features of hypokalaemia are:

- U waves
- T-wave flattening
- ST segment elevation
- Arrhythmias
- VF/VT, asystole, PEA

Management of hypokalaemia includes:

- Stopping any causative agent(s).
- K^+ supplementation—the speed of replacement depends on the K^+ level, the patient's symptoms, and presence of ECG changes. The maximum recommended IV infusion rate is 20 mmol/h. More rapid infusions can be given via a central line in a critical care setting. Continuous ECG monitoring is essential during the infusion.
- In a cardiac arrest, K^+ can be given more rapidly (2 mmol/min for 10 minutes, followed by 10 mmol over 5–10 minutes).
- Patients may also be deficient in magnesium. Repletion of magnesium stores will facilitate more rapid correction of hypokalaemia and is recommended in severe cases (magnesium sulfate 2 g IV).

Hyper/hypocalcaemia

The management of calcium disorders is discussed in Chapter 14, section 14.10.

In the event of cardiac arrest secondary to hypocalcaemia or a calcium channel blocker overdose, a bolus of calcium chloride 10 ml of 10% should be given. Hypocalcaemia is difficult to correct if hypomagnesaemia is not corrected, therefore patients should also receive magnesium sulfate 2 g IV.

2.2.3 Poisoning

Poisoning is a leading cause of cardiac arrest in patients younger than 40 years old. Toxicology is discussed in more detail in Chapter 17.

Opiates

Opiate poisoning causes progressive depression of the central nervous system leading to coma, respiratory depression, and eventually respiratory arrest. Cardiac arrest is the consequence of hypoxia. The pupils are usually equal and pin-point. The antagonist to opiates is naloxone, which can be given IV, intraosseous (IO), intramuscularly (IM), intranasally (IN), or via an endotracheal tube.

Management of opiate poisoning:

- The priority is airway management, oxygenation, and ventilation.
- The initial dose of naloxone is 400 mcg IV (800 mcg IM and 2 mg IN). It should be repeated if there is no response within two minutes. Naloxone is a competitive antagonist and large doses (4 mg) may be required in the seriously poisoned patient. The empirical administration of naloxone to all unresponsive victims of possible opiate-associated life-threatening emergency may be reasonable.
- The plasma high-life of naloxone is shorter than all opiate analgesics and an intravenous infusion of naloxone may be required. This involves adding 2 mg of naloxone to 500 ml of normal saline or 5% glucose, giving a final concentration of 4 mcg/ml. The usual infusion rate is 25–100 ml/hour (100–400 mcg/hour).
- In the event of cardiac arrest, standard resuscitation guidelines should be followed.

Tricyclic antidepressants

The management of tricyclic antidepressant (TCA) overdose is discussed in Chapter 17, section 17.6. In the event of cardiac arrest secondary to a TCA overdose sodium bicarbonate (50 ml of 8.4%) should be given and prolonged resuscitation attempts of up to one hour may be required.

Cocaine toxicity

Cocaine is a sympathomimetic, which may cause agitation, tachycardia, hypertension, hyperthermia, and myocardial ischaemia.

Management of cocaine toxicity:

- Intravenous benzodiazepines (diazepam or lorazepam) are the first-line agents to manage agitation, convulsions, hypertension, and chest pain.
- Persisting hypertension can be treated with IV nitrates. Calcium channel blockers (e.g. nifedipine, diltiazem, and verapamil) are second-line agents. Beta-blockers should be avoided due to the risk of paradoxical hypertension and vasoconstriction from unopposed alpha-adrenergic effects. Phentolamine is an alternative in patients with hypertension but it can cause a precipitous fall in blood pressure and should be avoided in those with a history of cardiac ischaemia.
- Cocaine causes coronary artery vasospasm and chest pain should be treated with aspirin, diazepam, and nitrates. If chest pain continues despite these treatments and the ECG shows

changes suggestive of myocardial infarction, conventional reperfusion management should be followed (e.g. thrombolysis or primary angioplasty).
- In the event of cardiac arrest, standard resuscitation guidelines apply.

2.2.4 Hypothermia

Hypothermia exists when the core body temperature is less than 35°C. It can be classified as:

- Mild 32–35°C
- Moderate 30–32°C
- Severe <30°C

A low-reading thermometer is needed to measure the core temperature. Hypothermia may develop rapidly (e.g. sudden immersion in cold water) or gradually due to immobility or impaired conscious level secondary to illness, alcohol, or drugs. The elderly and very young are more suitable to hypothermia due to impaired thermoregulation. Hypothermia in cardiac arrest may be the primary cause, or it may be secondary to a normothermic cardiac arrest followed by cooling due to the environment. Hypothermia can exert a protective effect on the brain after cardiac arrest. Confirmation of death should not be made until the patient has been rewarmed, or attempts to rewarm the patient have failed.

Modifications to the ALS algorithm in a hypothermic cardiac arrest:

- Palpate a major artery for a pulse and look for signs of life for up to one minute before concluding that there is no cardiac output. If possible, use echocardiography or Doppler ultrasound to confirm the presence or absence of cardiac output. If there is doubt about the presence of a pulse, begin CPR.
- The drug regime should be modified in a hypothermic cardiac arrest because the heart may be unresponsive to cardioactive drugs and drug metabolism is slower, leading to potentially toxic plasma concentrations of drugs. Withhold cardioactive drugs until the core temperature is >30°C. Once 30°C has been reached, double the interval between doses until the temperature returns to normal (>35°C).
- If VF/VT is detected, the patient should be defibrillated. If VF/VT persists beyond three shocks, further attempts at defibrillation should be postponed until the temperature is >30°C.
- Interventions such as central line placement and intubation should be performed by an expert to avoid excessive movement of patient and the risk of precipitating VF.
- Rewarm the patient to 32–34°C, a target temperature of 36°C is an alternative. A period of therapeutic hypothermia may be beneficial post-arrest.
- Patients may require large volumes of IV fluids as they rewarm and vasodilate.

Hypothermia in patients with a pulse

As the core temperature falls, cerebral and cardiovascular function deteriorates. Patients become ataxic, dysarthric, and their conscious level falls, progressing to coma. The blood pressure falls and arrhythmias develop; typically sinus bradycardia becomes atrial fibrillation, followed by VF and asystole.

Investigations in hypothermia:

- Renal function—risk of rhabdomyolysis
- FBC and clotting studies—hypothermia may precipitate a coagulopathy
- Toxicology screen
- Blood glucose—risk of hypoglycaemia
- Amylase—hypothermia may precipitate pancreatitis
- ABG—to determine the level of oxygenation, effectiveness of ventilation, and presence of metabolic acidosis

- ECG—prolonged PQRST complex, J waves (delayed repolarization), and arrhythmias may develop
- CXR—to look for evidence of aspiration, pneumonia, or pulmonary oedema
- CT head—if a head injury or cerebrovascular accident (CVA) is suspected

Management of hypothermia:

- Patients should be rewarmed. This may be a combination of active and passive rewarming techniques, depending on the severity and duration of hypothermia, and available facilities. Aim for a rate of 0.5–2°C per hour. Rapid rewarming may precipitate pulmonary and cerebral oedema, especially in the elderly (Table 2.3).

Table 2.3 Rewarming techniques

Passive	● Remove cold or wet clothing. ● Dry the patient. ● Cover with blankets/hot air blanket. ● Warm room (overhead heaters, if available). ● Forced-air warming system (e.g. Bair Hugger).
Active	● Warmed, humidified oxygen. ● Warmed IV fluids. ● Gastric, peritoneal, pleural, or bladder lavage with warmed fluids. ● Cardiopulmonary bypass.

- ECG monitoring is necessary to detect the development of arrhythmias. Most arrhythmias other than VF tend to revert spontaneously as the core temperature increases and do not usually require immediate treatment.
- Regular blood glucose levels should be checked to monitor for hypoglycaemia. If present, it should be corrected with 50% glucose.
- IV fluids may be required as the patient rewarms. If the blood pressure falls, 300–500 ml of warmed 0.9% sodium chloride should be given. There is a risk of pulmonary oedema and unstable patients should be monitored with a central venous pressure (CVP) line and urinary catheter.

2.2.5 Hyperthermia

Hyperthermia develops when the body's ability to thermoregulate fails and the core temperature exceeds that normally maintained by homeostatic mechanisms (controlled by the hypothalamus).

The pathophysiology of fever and hyperthermia are discussed in Chapter 15, section 15.2.

Causes of hyperthermia

Exogenous:

- Environmental conditions—high temperatures/humidity.

Endogenous:

- Prolonged muscular activity—seizures, marathon running, excessive dancing secondary to recreational drugs (e.g. ecstasy).
- Drugs—ecstasy, cocaine, amphetamines, and so on.
- Neuroleptic malignant syndrome—idiosyncratic drug reaction to antipsychotics. (Discussed further in Chapter 18, section 18.5.)

- Malignant hyperthermia—rare autosomal dominant condition related to the use of suxamethonium or inhaled anaesthetic agents. (Discussed further in Chapter 3, section 3.3.5).

Severity of hyperthermia

Heat cramps

The core temperature is 37–39°C. Heat cramps are typically seen during exercise where insensible fluid losses (sweating) are replaced with hypotonic fluids. Sodium levels fall and the patient develops muscle cramps. Management is with oral rehydration fluids.

Heat exhaustion

The core temperature is <40°C. Patients have both sodium and water depletion. Symptoms include weakness, fatigue, headache, dizziness, nausea, vomiting, and syncope. The patient's homeostatic mechanisms still function but are overwhelmed.

Patients should be removed from the heat. Treatment ranges from oral rehydration fluids in mild cases to IV rehydration in more severe cases.

Heat stroke

The core temperature is >40.6°C. Early symptoms are similar to heat exhaustion but progress to confusion, seizures, coma, hypotension, and arrhythmias. All thermoregulatory control is lost and ultimately multiorgan damage occurs.

Complications of heat stroke include:

- CNS—cerebral oedema and petechial haemorrhages
- Renal—acute kidney injury due to hypovolaemia and rhabdomyolysis
- Liver—raised liver enzymes and jaundice after 24 hours
- Haematological—thrombocytopenia and disseminated intravascular coagulation (DIC)
- Metabolic—hyper/hypokalaemia, metabolic acidosis, hypoglycaemia
- Muscle—rhabdomyolysis

Management of hyperthermia

- Cooling: remove the patient from the hot environment and take off any clothing. Evaporative cooling should be employed; spray the patient with tepid water and blow air over them with fans. Ice packs can be used in the neck, axillae, and groins. Aim to rapidly reduce the core temperature to 39°C. Advanced cooling techniques can be used as for therapeutic hypothermia after cardiac arrest: cold IV fluids, intravascular cooling catheters, surface cooling devices, and extracorporeal circuits, if available.
- Monitor for hypoglycaemia and treat accordingly.
- IV fluids should be given cautiously due to the risks of pulmonary/cerebral oedema. If the blood pressure remains low despite a reduction in temperature, titrate 0.9% sodium chloride cautiously (consider the use of a CVP line and urinary catheterization). IV fluids will need to be given more rapidly if there is evidence of rhabdomyolysis.
- There is no evidence for the use of antipyretics (paracetamol, NSAIDSs) in heat stroke.
- If the hyperthermia is due to neuroleptic malignant syndrome or malignant hyperthermia, dantrolene can be given.

2.2.6 Trauma

The ERC 2015 guidelines highlight the fact that traumatic cardiac arrest carries a high mortality but survival with a good neurological outcome is more likely with ROSC.

Causes of cardiac arrest in trauma

- Severe traumatic brain injury
- Hypovolaemia from massive haemorrhage
- Hypoxia from respiratory arrest
- Direct injury to major organs or vessels
- Tension pneumothorax
- Cardiac tamponade
- Commotio cordis (blunt chest wall trauma leading to VF)

Modifications to the ALS algorithm in trauma

- In the presence of a history of trauma, it is important to determine whether this is a primary traumatic arrest or whether there a medical event that has led to secondary trauma.
- In primary traumatic cardiac arrest, the emphasis is on correction of reversible causes, which takes priority over chest compressions, which are not likely to work in the presence of hypovolaemia, cardiac tamponade, or tension pneumothorax.
- Hypoxia—early tracheal intubation should be undertaken, if possible.
- Hypovolaemia—external haemorrhage should be controlled (e.g. direct pressure, elevation, splinting, tourniquets, and haemostatic agents). In cardiac arrest, aggressive IV fluid resuscitation should be given. If the patient has a pulse, fluids should be given more cautiously (NICE guidance on pre-hospital fluid replacement in trauma recommends 250 ml boluses of crystalloid until a radial pulse is achieved). The emphasis is on rapid surgical control of bleeding. Tranexamic acid loading 1 G IV followed by a 1 G infusion over eight hours should be considered.
- Tension pneumothorax—can be difficult to diagnose in a cardiac arrest. Decompression should be performed by lateral or anterior finger thoracostomy (incision in the chest wall through to the pleural cavity) in the fourth intercostal space. A thoracostomy is likely to be more effective than needle decompression and quicker than inserting a chest drain.
- Cardiac tamponade—consider resuscitative thoracotomy.

Resuscitation Council guidance on when to consider an emergency department thoracotomy:

- Penetrating cardiac injuries who arrive, after a short pre-hospital time, with witnessed signs of life or ECG activity.
- Penetrating non-cardiac thoracic injuries.
- Exsanguinating abdominal vascular injury to enable cross-clamping of the descending aorta.
- Only in blunt trauma when there are vital signs on arrival and a witnessed cardiac arrest.

2.2.7 Asthma

Asthma still causes many deaths in young adults. Asthma management is discussed further in Chapter 10, section 10.1.

Causes of cardiac arrest in asthma

- Hypoxia—severe bronchospasm and mucous plugging leading to asphyxia.
- Cardiac arrhythmias—due to hypoxia, stimulant drugs (e.g. salbutamol, aminophylline), or electrolyte abnormalities.
- Dynamic hyperinflation (gas trapping)—can occur in ventilated asthmatic patients. The increased intrathoracic pressure decreases cardiac output.
- Tension pneumothorax (may be bilateral).

Modifications to the advanced life support algorithm in asthma

- Standard basic life support (BLS) should be performed although ventilation may be hindered by increased airway resistance.
- Patients should be intubated early to reduce the risk of gastric inflation and hypoventilation.
- If dynamic hyperinflation is suspected, compression of the chest wall and/or a period of apnoea (disconnection of tracheal tube) may relive gas trapping.
- Dynamic hyperinflation increases transthoracic impedance. In VF/VT, higher shock energies for defibrillation may be necessary.
- Pleural ultrasound may allow rapid bedside detection of tension pneumothorax. Tension pneumothorax should be treated with needle thoracocentesis (performed in the second intercostal space, mid-clavicular line). A chest drain will then be required. In a ventilated patient a thoracostomy may be quicker and more effective.

2.2.8 Pregnancy

Cardiac arrest is fortunately rare in pregnant patients.

Causes of cardiac arrest in pregnancy

- Pre-existing cardiac disease
- Thromboembolism
- Suicide
- Eclampsia
- Sepsis
- Ectopic pregnancy
- Uteroplacental haemorrhage
- Amniotic fluid embolism

Modifications to the advanced life support algorithm in pregnancy

- Supine positioning will result in aortocaval compression. Aortocaval decompression is achieved by manual displacement of the uterus to the left or by using a 'Cardiff wedge' to support the pelvis and thorax, to achieve the left lateral position, if the fundal height is at or above the level of the umbilicus.
- The hand positioning for chest compression may have to be slightly higher than normal to adjust for elevation of the diaphragm and abdominal contents caused by the gravid uterus.
- Patients should be intubated early due to the risk of pulmonary aspiration of gastric contents.
- Defibrillator pad position may have to be adjusted due to the left lateral tilt and large breasts.
- Consider hypovolaemia as a cause and give a large fluid challenge.
- Consider giving magnesium (4 g IV) in the event of cardiac arrest in eclampsia.
- Emergency delivery of the foetus (>20 weeks gestation) via caesarean section should occur within five minutes of cardiac arrest, when initial resuscitation attempts fail. The rationale is to decompress the inferior vena cava (IVC) for the mother and remove the foetus from the hypoxic environment, providing it with the best chance for survival. The team should not wait for surgical equipment if not immediately available, as only a scalpel is required to start the procedure. Emergency caesarean for gestational age <20 weeks is not necessary because a gravid uterus of this size is unlikely to compromise maternal cardiac output.

KEY POINTS

Good-quality CPR is paramount, regardless of the cause of cardiac arrest, and the ALS algorithm should be followed.

Consideration and treatment of the reversible causes is the mainstay of treatment.

Here is a summary of the main additions and modifications to the resuscitation algorithm for cardiac arrest in special circumstances:

- Hyperkalaemia—calcium chloride 10 ml of 10%, sodium bicarbonate 50 ml of 8.4%, and insulin/glucose infusion (10 units of insulin and 50 ml of 50% glucose).
- Hypokalaemia—K^+ 2 mmol/min for 10 minutes, followed by 10 mmol over 5–10 minutes. Plus magnesium sulfate 2 g IV, if concurrent hypomagnesaemia suspected.
- Hypocalcaemia—calcium chloride 10 ml of 10%, plus magnesium sulfate 2 g IV, if concurrent hypomagnesaemia suspected.
- Opiates—naloxone 400 mcg IV. Repeated doses up to a total of 4 mg may be required.
- Tricyclic antidepressants—sodium bicarbonate 50 ml of 8.4%.
- Local anaesthetic toxicity—20% lipid emulsion IV bolus (1.5 ml/kg). Further details can be found in Chapter 3, section 3.7.5.
- Hypothermia—palpate a pulse/look for signs of life for up to 1 minute (if possible use Doppler). Withhold drugs until temperature >30°C and then double the dosing interval until >35°C. If VF/VT persists beyond three shocks, postpone further attempts at defibrillation until the temperature >30°C. Rewarm to 32–34°C.
- Hyperthermia—active cooling (cold IV fluids, intravascular cooling catheters, surface cooling devices, and extracorporeal circuits). Dantrolene if hyperthermia is due to neuroleptic malignant syndrome or malignant hyperthermia.
- Trauma—intubate early. Manage hypovolaemia with haemorrhage control and fluids. Emergency department (ED) thoracotomy is only indicated in very specific circumstances.
- Asthma—intubate early. Consider the possibility of tension pneumothorax (possibly bilateral) and treat accordingly.
- Pregnancy—place the patient in the left lateral position. Emergency delivery of the foetus (>20 weeks gestation) via caesarean section should occur within five minutes of cardiac arrest.

2.3 Anaphylaxis

2.3.1 Introduction

Anaphylaxis is a severe, life-threatening, systemic, type 1 hypersensitivity reaction. Anaphylaxis is triggered when an antigen binds to immunoglobulin E (IgE) antibodies on mast cells, causing degranulation and the release of inflammatory mediators.

An anaphylactic reaction is likely when all of the following three criteria are met:

- Sudden onset and rapid progression of symptoms.
- Life-threatening airway and/or breathing and/or circulation problems.
- Skin and/or mucosal changes (flushing, urticaria, angioedema).

The diagnosis is supported by exposure to a known allergen for the patient. Anaphylaxis can be triggered by a broad range of substances, but those most commonly identified are food, drugs, and venom.

- Food—most commonly nuts
- Drugs—antibiotics, radiological contrast media, muscle relaxants, NSAIDs
- Venom—wasp or bee stings

The speed of onset will depend upon the trigger. Intravenous medications will cause a more rapid onset than stings, which, in turn, tend to cause a more rapid onset than orally ingested triggers.

2.3.2 Clinical features of anaphylaxis

- Airway—swelling resulting in pharyngeal and laryngeal oedema. This can cause difficulty in breathing and swallowing, a hoarse voice, and stridor.
- Breathing—dyspnoea, wheeze, cyanosis, and ultimately respiratory arrest.
- Circulation—tachycardia, hypotension, dizziness, collapse, reduced level of consciousness, and ultimately cardiac arrest.
- Disability—A, B, and C problems can lead to decreased cerebral perfusion resulting in confusion, agitation, and loss of consciousness.
- Exposure—skin and mucosal changes (urticaria and angioedema) occur in 80% of anaphylactic reactions but may be subtle or absent. Skin changes without life-threatening A, B, or C problems do not signify an anaphylactic reaction.
- Other features—patients may have gastrointestinal symptoms of vomiting, abdominal pain, and diarrhoea.

2.3.3 Management of anaphylaxis

An ABCDE approach should be followed and life-threatening problems treated as they are identified. Initial treatments should not be delayed by the lack of a complete history or definite diagnosis. If possible, the trigger should be removed by stopping any drug or fluid infusions, or removing the sting.

The anaphylaxis algorithm (Figure 2.2 and Table 2.4) details the Resuscitation Council guidance.

Fluids—large volumes of fluid may be required due to vasodilatation and capillary leak. Fluid should be given as 500 ml boluses and repeated as necessary. There is no evidence for colloid over crystalloid in this setting. If the patient is receiving colloid at the time of developing anaphylaxis, consider it as a possible cause and stop the infusion.

Glucagon—adrenaline may fail to reverse the clinical manifestations of an anaphylactic reaction in patients taking β-blockers. Glucagon (1–2 mg IV) can be useful in this situation.

Cardiac arrest—if cardiac arrest occurs, the usual ALS algorithm should be followed and adrenaline should no longer be given IM but IV at a dose of 1mg.

2.3.4 Investigations in anaphylaxis

Anaphylaxis is a clinical diagnosis. The specific investigation which may help confirm the diagnosis retrospectively is measurement of the mast cell tryptase.

Mast cell tryptase

Tryptase is the major protein component of mast cell secretory granules. In anaphylaxis, mast cell degranulation leads to increased blood levels of tryptase. The investigation is useful for follow-up in suspected cases of anaphylaxis but will not alter ED management. However, the timing of the levels is very important.

Tryptase levels may not increase significantly until more than 30 minutes after the onset of symptoms and peak one to two hours later. The half-life of tryptase is short (approximately two hours) and levels will return to normal within six to eight hours. A sample for serum tryptase testing should be obtained as soon as possible after emergency treatment, with a second sample ideally within one to two hours (no later than four hours) from the onset of symptoms. Ideally, a third sample can be taken at 24 hours or in convalescence (e.g. in a follow-up allergy clinic) to establish baseline tryptase levels. Not all patients will have a documented tryptase rise and the absence of a rise does not reliably exclude anaphylaxis.

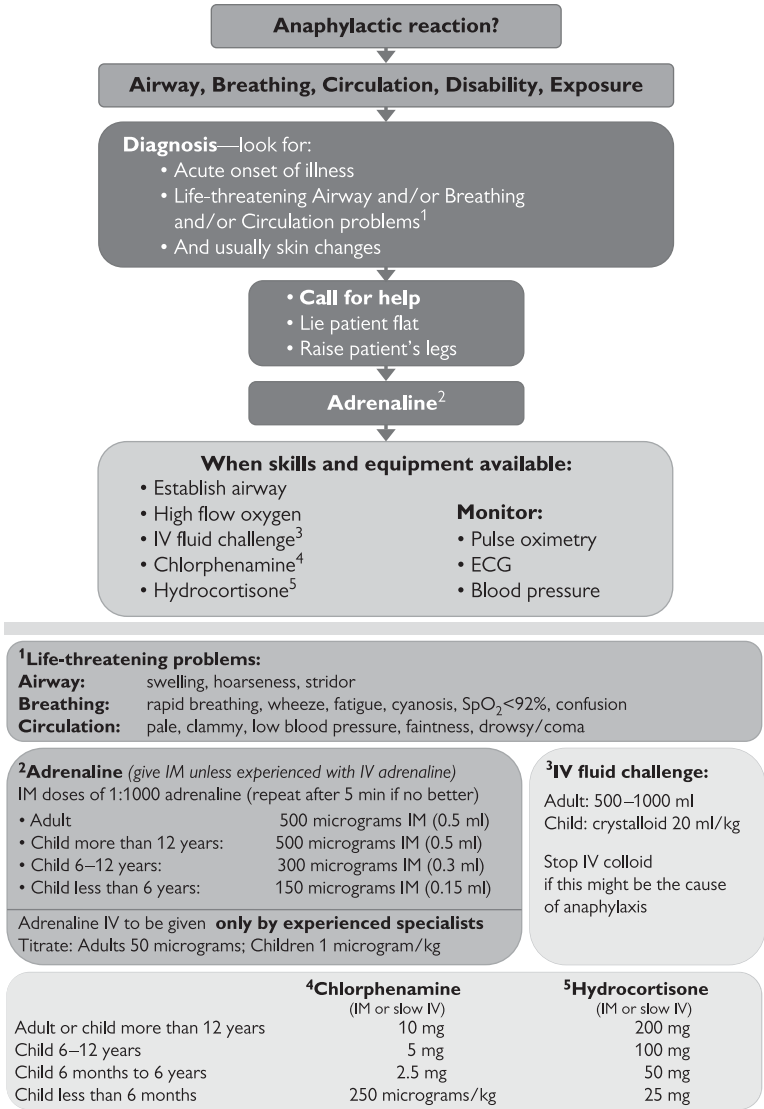

Figure 2.2 Anaphylaxis algorithm.

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2.3.5 Discharge and follow-up after anaphylaxis

Patients recovering from anaphylactic reactions may be cared for by the ED on a clinical decision unit or equivalent. It is therefore possible that a SAQ will include elements of the discharge advice and follow-up care.

- Patients who have had a suspected anaphylactic reaction should be observed for at least 6–12 hours.

Table 2.4 Drugs used in the treatment of anaphylaxis

Drug	Actions	Indications	Dose
Adrenaline	α-agonist—reverses peripheral vasodilatation and reduces oedema. β-agonist—bronchodilation, positive inotrope, suppresses histamine and leukotriene release. β_2 actions also inhibit the activation of mast cells, attenuating the severity of IgE-mediated allergic reactions.	Give as soon as the diagnosis of anaphylaxis is made.	0.5 mg IM (the best site is the anterolateral aspect of the middle-third of the thigh). Repeat after 5 minutes if no improvement. IV adrenaline (50 mcg boluses) should only be used by those experienced in the use and titration of vasopressors.
Antihistamines	H_1-antihistamine may help counter histamine-mediated vasodilatation and bronchoconstriction. H_2-antihistamine (e.g. ranitidine) there is little evidence to support the use in the initial treatment of anaphylaxis.	Second-line treatment.	10 mg chlorpheniramine IV or IM.
Corticosteroids	May prevent or shorten protracted reactions.	For use after the initial resuscitation.	200 mg hydrocortisone IV or IM.
Bronchodilators	β-agonist (salbutamol) and/or antimuscarinic (ipratropium bromide).	If evidence of wheeze on auscultation.	Salbutamol 5 mg and ipratropium bromide 500 mcg nebulizer.

- Patients should be warned of the risk of recurrence, being informed about anaphylaxis, including the signs and symptoms of a biphasic reaction in the next 24 hours. The risk of a biphasic reaction is greater in the following groups:
  - Severe reactions with slow onset caused by an unknown trigger.
  - Reactions in patients with asthma.
  - Reactions with possible continuing absorption of the allergen.
  - Reactions in a patient with a history of biphasic reactions.
- Patients should be reviewed by a senior clinician prior to discharge and if at risk of a biphasic reaction observed for longer (e.g. 24 hours).
- Patients may be discharged with a three day prescription of oral steroid and antihistamines.
- All patients who have had an anaphylactic reaction should be prescribed two adrenaline autoinjector devices as an interim measure before their specialist allergy appointment.
- Patients should be educated on the likely trigger, how to avoid it, and what to do in the event of a future attack.
- All patients should be referred on to an age-appropriate specialist allergy service for follow-up.

KEY POINTS

Patients who have an anaphylactic reaction have life-threatening airway and/or breathing and/or circulation problems usually associated with skin and mucosal changes.

Early recognition of anaphylaxis and treatment with adrenaline 0.5 mg IM is paramount.

The most important investigation in suspected anaphylaxis is the collection of timed serum samples for mast cell tryptase.

Patients should be observed for a minimum of 6–12 h after an anaphylactic reaction.

Patients suspected of having an anaphylactic should be referred on to an allergy specialist.

Urticaria and angioedema can be caused by more than just anaphylaxis. Angioedema can be classified as allergic, hereditary, acquired, drug-induced, or idiopathic. Further details on the different types of angioedema can be found in Chapter 16, section 16.2.5.

2.4 Post-resuscitation care

ROSC is only the first step in recovery from a cardiac arrest. Patients may develop post-cardiac-arrest syndrome, the severity of which varies according to the duration and cause of cardiac arrest.

The post-cardiac-arrest syndrome comprises brain injury, myocardial dysfunction, systemic ischaemia/reperfusion response, and persistence of the precipitating pathology.

- Post-cardiac-arrest brain injury—manifests as coma, seizures, myoclonus, varying degrees of neurocognitive dysfunction, and brain death. It can be exacerbated by microcirculatory failure, impaired autoregulation, hyperoxia, hypoxia, hypercarbia, pyrexia, hyperglycaemia, and seizures.
- Post-cardiac-arrest myocardial dysfunction—manifests as hypotension, arrhythmias, cardiogenic shock, and potentially further cardiac arrest. It usually recovers after two to three days.
- Systemic ischaemia/reperfusion—results in activation of the immunological and coagulation pathways contributing to multiorgan failure and increasing the risk of secondary infection.

The 2015 Resuscitation Council Guidelines continue to emphasize the treatment of the post-cardiac-arrest syndrome. The ABCDE approach to management is recommended (Table 2.5).

Table 2.5 Post-resuscitation care

	Recommendation	Rationale
Airway and breathing	Advanced airway. Waveform capnography. Aim for normocarbia. Aim for normoxia (Sp02 94–98%). Consider sedation, intubation, and ventilation in any obtunded patient.	Hypoxia and hypercarbia increase the likelihood of further cardiac arrest and may contribute to secondary brain injury. Hyperoxia may cause oxidative stress and harm post-ischaemic neurons. Controlled oxygenation and ventilation enable normocarbia and normoxia to be achieved.

Table 2.5 Continued

	Recommendation	Rationale
Circulation	12 lead ECG. Aim for SBP > 100 mm Hg. Crystalloid to restore normovolaemia. Intra-arterial blood pressure monitoring. Consider vasopressor/inotrope to maintain SBP. Patients with ST-elevation should undergo early reperfusion therapy, ideally PCI. Patients without ST-elevation, who are suspected of having coronary artery disease as the cause of their arrest, should be considered for early PCI. Myocardial dysfunction should be managed with IV fluids and inotropes. If this is insufficient, an intra-aortic balloon pump should be considered.	Primary PCI is the preferred treatment in ST-elevation myocardial infarction (STEMI) if it can be performed in a timely manner (within 90 min of first medical contact). If primary PCI is not feasible, thrombolysis should be given (CPR is not a contraindication). Chest pain and ST-elevation are relatively poor indicators of acute coronary occlusion post-cardiac-arrest. Therefore, a low threshold for early PCI is warranted.
Disability	Seizures should be controlled with benzodiazepines, phenytoin, sodium valproate, propofol, or a barbiturate.	Seizures increase cerebral metabolism up to 3-fold and may cause cerebral injury.
Glucose control	Blood glucose should be maintained ≤10 mmol/L. Hypoglycaemia should be avoided.	Hyperglycaemia post-cardiac arrest is associated with a poorer neurological outcome. Intensive glucose control (4.5–6.0 mmol/L) has not been shown to be superior to conventional glucose control and increases the risk of hypoglycaemia, which is associated with increased mortality in the critically ill.
Temperature control	Hyperthermia should be avoided with antipyretics and active cooling. Control shivering. Targeted temperature management or control (32–36°C) is recommended for comatose survivors of both non-shockable and shockable cardiac arrests. Targeted temperature management should be initiated in the ED. Techniques include ice packs and/or wet towels, cooling blankets or pads, water- or air-circulating gel-coated pads, transnasal evaporative cooling, intravascular heat exchanger (via femoral or subclavian vein), and extracorporeal circulation (cardiopulmonary bypass, ECMO).	There is an association between post-cardiac-arrest pyrexia and poor outcome. Mild hypothermia is neuroprotective and improves outcome after a period of global cerebral hypoxia. Cooling suppresses apoptosis and reduces the cerebral metabolic rate (~6% for each 1°C reduction). Hypothermia may reduce the release of excitatory amino acids and free radicals. Animal data indicate that earlier cooling after ROSC produces better outcome.

2.5 Peri-arrest arrhythmia management

2.5.1 Introduction

Arrhythmia management is a subject that many candidates find difficult. The 2015 Resuscitation Council Guidelines have tried to simplify the management as much as possible. Knowledge of this guidance should be sufficient for the MRCEM examination.

2.5.2 Generic management of peri-arrest arrhythmias

- All patients should be assessed using the ABCDE approach. Patients should be given oxygen, have an IV line inserted, and assessed for adverse features.
- If time allows, patients should have a 12-lead ECG.
- Any electrolyte abnormalities (e.g. K^+, Ca^{2+}, Mg^{2+}) should be corrected.
- If there is an underlying condition (e.g. MI, sepsis), this must also be treated.
- The two main questions to answer when managing an arrhythmia are:
  - Is the patient stable?
  - What is nature of the arrhythmia?

Adverse features in peri-arrest arrhythmias

There is now a single set of adverse features for tachy- and bradyarrhythmias. If adverse features are present, it suggests that the patient is potentially unstable from their arrhythmia.

The adverse features are:

- Shock—hypotension (systolic BP <90 mmHg), pallor, sweating, cold extremities, clammy, confusion, or impaired level of consciousness.
- Syncope—transient loss of consciousness due to a reduction in cerebral perfusion.
- Myocardial ischaemia—ischaemic chest pain and/or evidence of ischaemia on the ECG.
- Heart failure—pulmonary oedema and/or raised jugular venous pressure (with or without peripheral oedema and liver enlargement).

If the patient is unstable, the treatment of choice is electricity: cardioversion for tachyarrhythmias and pacing for bradyarrhythmias (see Figure 2.3). Table 2.6 details the drugs used in peri-arrest arrhythmias.

2.6 Peri-arrest tachycardias

2.6.1 Stable versus unstable tachyarrhythmias

If the patient is unstable, due to their tachyarrhythmia, synchronized cardioversion is the treatment of choice. Cardioversion should be carried out under general anaesthesia or conscious sedation. The defibrillator should be set to synchronized mode. The recommended energies vary slightly depending on the type of tachyarrhythmia, but 120 J biphasic would be an appropriate initial energy for any tachyarrhythmia. If this fails, a further two shocks at increasing increments should be tried.

If electrical cardioversion fails to restore sinus rhythm, and the patient remains unstable, amiodarone 300 mg IV should be given over 10–20 minutes. Electrical cardioversion can then be repeated. The loading dose of amiodarone can be followed by an infusion of 900 mg over 24 hours.

If the patient is stable and has no adverse features, then there are several possible treatment options depending on the nature of the arrhythmia. The ECG assessment should include whether the QRS is broad or narrow, and whether it is regular or irregular.

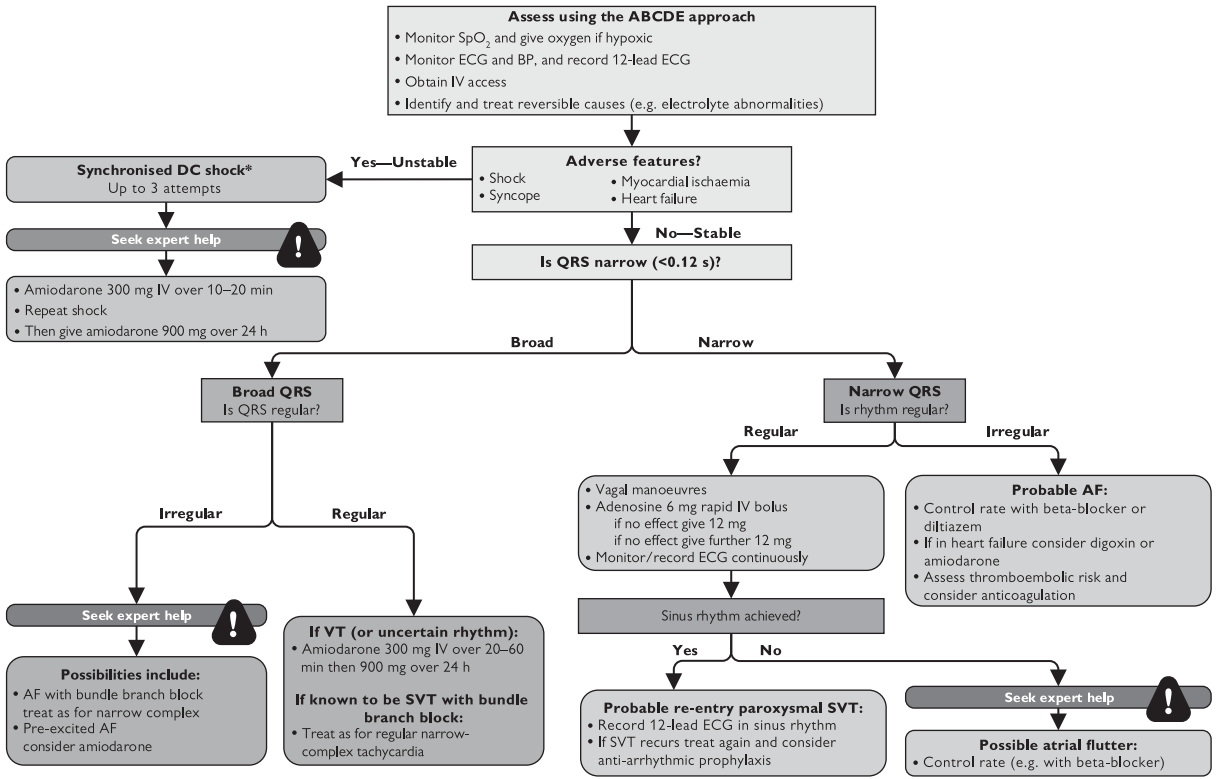

Figure 2.3 Adult tachycardia (with pulse) algorithm.

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Table 2.6 Drugs used in peri-arrest arrhythmias

Drug	Actions	Indications/Dose
Adrenaline	• As detailed in Table 2.2.	• Treatment of cardiogenic shock. Dose 0.05–0.1 mcg/kg/min • Bradycardia if there is a delay for pacing. Dose 2–10 mcg/min IV.
Amiodarone	• As detailed in Table 2.2	• Stable VT and wide-complex tachycardias of unknown origin. • To control a rapid ventricular rate in pre-excited AF. • Following unsuccessful electrical cardioversion in unstable arrhythmias. Dose: 300 mg IV over 10–60 min. Followed by 900 mg over 24 h.
Adenosine	• Blocks transmission across the AV node. • Very short half-life (10–15 s). Must be given as a rapid bolus into a large cannula, in a large vein with a 20 ml saline flush.	• Paroxysmal SVT. Dose 6 mg IV bolus. If unsuccessful a further two doses at 12 mg IV may be tried.
Atropine	• Antagonizes the action of the parasympathetic neurotransmitter acetylcholine at muscarinic receptors. • Blocks the action of the vagus nerve at the SA and AV nodes, increasing sinus automaticity, and facilitating AV node conduction.	• Unstable bradycardia. Dose 500 mcg IV bolus. Repeated up to a maximum of 3 mg.
Magnesium	• As detailed in Table 2.2	• Polymorphic VT (Torsades de pointes). • Digoxin toxicity. • Ventricular tachyarrhythmias if hypomagnesaemia suspected. Dose 2 g IV over 10 min.

2.6.2 Broad-complex tachycardia (QRS >0.12 seconds)

Broad-complex tachycardias are usually ventricular in origin but may also be caused by supraventricular rhythms (SVT) with aberrant conduction (bundle branch block). There are several features on the ECG that can help distinguish VT from SVT with aberrant conduction (Figure 2.4 and Table 2.7). If there is doubt about the source of the arrhythmia and the patient is unstable, assume the rhythm is ventricular in origin and perform electrical cardioversion.

Treatment options for regular broad-complex tachycardias in stable patients

- If a regular broad-complex is diagnosed as SVT with aberrant conduction, it can be treated with adenosine.
- If VT is diagnosed, the treatment is amiodarone 300 mg IV over 20–60 minutes, followed by an infusion of 900 mg for 24 hours.
- In a stable patient with a regular broad-complex tachycardia of uncertain origin, adenosine can be tried.

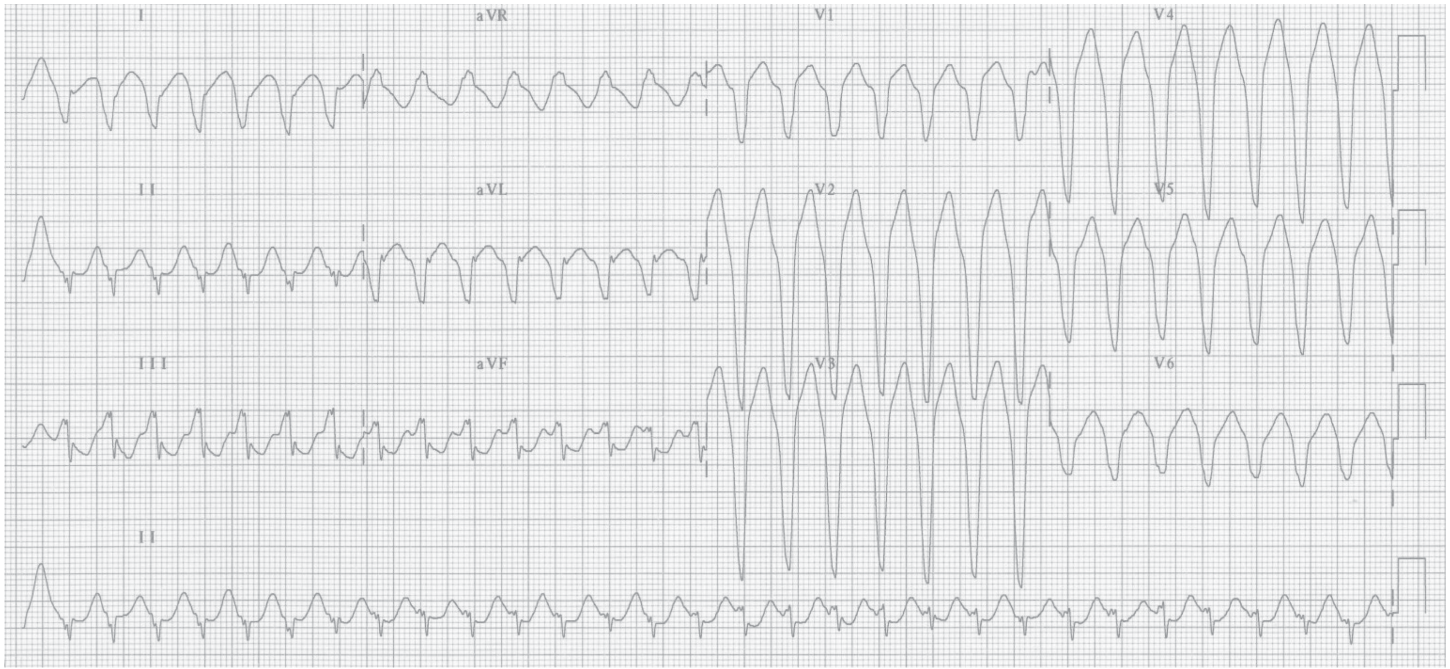

Figure 2.4 A broad-complex tachycardia with a ventricular rate of 180 bpm. AV dissociation is seen with buried P waves in V6. There is concordance across the chest leads.

Reproduced from Saul G. Myerson, Robin P. Choudhury, and Andrew R. J. Mitchell, *Emergencies in Cardiology*, 2010, Figure 21.35, p. 421, with permission from Oxford University Press.

Table 2.7 Distinguishing VT from SVT with aberrant conduction

Regularity	● Monomorphic VT originates from one area in the ventricle and therefore is regular. ● SVT may be irregular if the underlying rhythm is atrial fibrillation, atrial flutter with variable block, or multifocal atrial tachycardia.
QRS width	● A broader QRS (>0.14 s) favours VT.
QRS concordance	● In monomorphic VT, all the precordial leads (V1–V6) point in the same direction. ● SVT may have mixture of positive and negative QRS deflections in the precordial leads.
Beat-to-beat variability	● In VT there is often beat-to-beat variability of the QRS morphology.
Electrical axis	● VT normally has an axis in the extreme right quadrant. ● SVT will have right axis deviation in right bundle branch block and left axis deviation in left bundle branch block.
AV dissociation	● In VT, the atria and ventricles are depolarizing independently and therefore the P waves (if seen) have no relationship to the QRS. ● In SVT, if P waves are seen they correlate with the QRS.
Capture beats	● In VT, the AV dissociation is often intermittent and therefore occasionally the atria are able to conduct a beat through the AV node resulting in a normal QRS ('capture beat'). ● In SVT, the AV node is already conducting maximally, so there is no opportunity for a capture beat.
Fusion beats	● In a similar mechanism to capture beats, the AV node conducts an atrial impulse that combines with the ventricular depolarization wave giving a fused complex on the ECG ('fusion beat').

Treatment options for irregular broad-complex tachycardias in stable patients

- Irregular broad-complex tachycardias are most likely to be atrial fibrillation (AF) with bundle branch block and should be treated as AF (if uncertain, amiodarone 300 mg IV over 20–60 minutes is usually a reasonable choice).
- Torsades de pointe (polymorphic VT) should be treated by stopping all drugs known to prolong the QT interval, correction of electrolyte abnormalities, and magnesium sulphate 2 g IV over 10 minutes. Overdrive pacing may be indicated to prevent a relapse once the arrhythmia has been corrected.

2.6.3 Regular narrow-complex tachycardias

The ECG should be examined to determine if the rhythm is regular or irregular. Causes of a regular narrow-complex tachycardia include:

- Sinus tachycardia—a physiological response to a stimulus. Treatment is of the underlying cause (e.g. sepsis, hypovolaemia, anaemia, and so on).
- Atrioventricular (AV) nodal re-entry tachycardia—the commonest type of regular narrow-complex tachycardia. Occurs due to a re-entry circuit within or just next to the AV node. The circuit usually involves two pathways: a slow pathway and a fast pathway. The slow pathway is usually the anterograde limb of the circuit and the fast pathway, the retrograde limb. The circuit is triggered by an atrial premature complex, which is able to conduct down the slow pathway due to a short refractory period, but not the fast pathway due to a longer refractory period. Once the impulse reaches the ventricle, the fast pathway has recovered from the

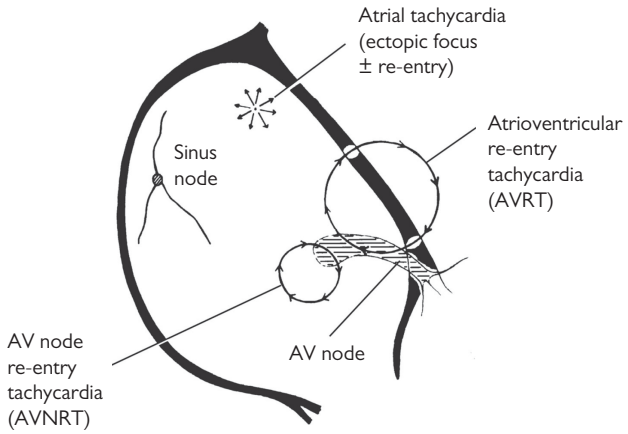

Figure 2.5 Types of supraventricular tachycardia.

Reproduced from Punit Ramrakha, Kevin Moore, and Amir Sam, *Oxford Handbook of Acute Medicine,* 2010, Figure 1.9, p. 69, with permission from Oxford University Press.

refractory period and is able to conduct the impulse retrogradely to the atrium and the atrial end of the slow pathway, perpetuating the circuit (Figure 2.5).

- AV re-entry tachycardia—usually due to Wolff–Parkinson–White (WPW) syndrome. An accessory pathway exists between the atria and ventricles. It is separate from the AV node and in conjunction with the AV node can set up a re-entry circuit.
- Atrial flutter with regular AV conduction (usually 2:1)—typically atrial flutter has a rate of 300, so atrial flutter with 2:1 conduction produces a tachycardia of 150 bpm. It may be indistinguishable from AVRT and AVNRT initially.

Treatment options for regular narrow-complex tachycardias depend on whether the patient is stable or not (Table 2.8).

Adenosine

Adenosine slows transmission across the AV node and therefore is highly effective at terminating AVNRT and AVRT. It has a very short half-life (10–15 seconds) and needs to be given as a rapid bolus into a large cannula, in a large vein (e.g. antecubital fossa) with a 20 ml saline flush.

Table 2.8 Treatment options in regular narrow-complex tachycardias

Unstable patient	If the patient is unstable, with adverse features, treat with synchronized electrical cardioversion. While preparations are being made to perform synchronized cardioversion, it is reasonable to try adenosine, provided it does not delay electrical cardioversion should it fail to restore sinus rhythm.
Stable patient	Step wise progression until sinus rhythm restored: ● Vagal manoeuvres—Valsalva and/or carotid sinus massage. ● Adenosine 6 mg as a rapid IV bolus. ● Repeat adenosine at 12 mg and again at 12 mg if no response. ● Consider verapamil 2.5–5 mg IV over 2 min (if adenosine is contraindicated).

Patients should be warned of transient unpleasant side effects including nausea, flushing, and chest tightness.

Cautions when using adenosine:

- Asthma due to the risk of bronchospasm.
- Patients with an accessory pathway (e.g. WPW) and AF due to the risk of promoting conduction down the accessory pathway and inducing VF.
- Patients with denervated hearts (e.g. heart transplant) who have a markedly exaggerated response to adenosine.
- Patients taking theophyllines because it blocks the effects of adenosine.
- Patients taking dipyridamole or carbamazepine due to a dangerously exaggerated response to adenosine.

If adenosine is contraindicated and the arrhythmia is known to be of supraventricular origin, verapamil (2.5–5 mg IV) can be used. Flecanide (2 mg/kg IV) is another alternative, but should not be used in patients with structural heart disease due to the risk of fatal arrhythmias.

Wolff–Parkinson–White (WPW) syndrome

WPW syndrome is due to an accessory pathway (bundle of Kent) between the atria and ventricles (Figure 2.6). It can often be diagnosed incidentally on the ECG of an asymptomatic patient, due to a short PR interval and a widened QRS complex due to the slurred upstroke (delta wave).

Patients may remain asymptomatic their entire life but they are at risk of tachyarrhythmias. Medications that block the AV node (e.g. adenosine, diltiazem, verapamil, and digoxin) should be avoided in patients with an accessory pathway who develop AF or atrial flutter because they can lead to a relative increase in pre-excitation. Treatment options for such situations include amiodarone, electrical cardioversion, or flecanide (if the heart is known to be structurally normal).

2.6.4 Irregular narrow-complex tachycardias

An irregular narrow-complex tachycardia is likely to be AF or, less commonly, atrial flutter with a variable AV block. If the patient is unstable, with adverse features caused by the arrhythmia, they should be treated with synchronized electrical cardioversion.

In stable patients there are several treatment options:

- Rate control by drug therapy (e.g. beta-blocker or rate-limiting calcium antagonist)
- Rhythm control by drug therapy (e.g. amiodarone or flecanide)
- Rhythm control by electrical cardioversion
- Anticoagulation to prevent thromboembolism (should be considered in all patients)

The management of AF is discussed in more depth in Chapter 9, section 9.5.

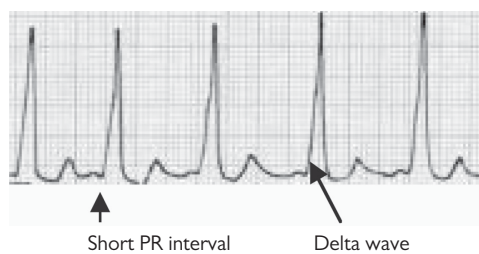

Figure 2.6 Wolff–Parkinson–White syndrome.

Reproduced from Nick Dunn, Hazel Everitt, and Chantal Simon, *Cardiovascular Problems*, 2007, Figure 3.9, p. 91, with permission from Oxford University Press.

2.7 Peri-arrest bradycardias

2.7.1 Introduction

Patients with bradycardia (heart rate <60 beats per minute) should be assessed using the ABCDE approach. Bradycardia may be:

- Physiological
- Cardiac in origin (e.g. AV block, sinus node disease, post-MI)
- Non-cardiac in origin (e.g. vasovagal, hypothermia, hypothyroidism, raised intracranial pressure)
- Drug-induced (e.g. β-blockade, calcium channel blockers (diltiazem), digoxin, amiodarone)

See Table 2.9 and Figures 2.7 and 2.8.

2.7.2 Unstable bradycardias

The same adverse features are used for bradyarrhythmias as for tachyarrhythmias:

- Shock
- Syncope
- Myocardial ischaemia
- Heart failure

If adverse features are present, treatment should be started immediately.

Table 2.9 Types of bradyarrhythmias

First-degree heart block	Delayed conduction between the atria and ventricles resulting in a prolonged PR interval (>0.2 s).
Second-degree heart block	Only a proportion of the P waves are conducted to the ventricles. There are two main types: ● Mobitz type I (Wenckebach)—the PR interval gets progressively longer until a P wave fails to conduct. ● Mobitz type II—constant PR but occasionally a P wave fails to conduct. This may be in a regular pattern (e.g. 2:1 or 3:1 block) or irregular.
Third-degree heart block (complete heart block)	Atrial activity is not conducted to the ventricles. The atria and ventricles work independently of each other (P waves and QRS complexes are not related to each other). The rate and breadth of the QRS complexes depends upon level of the block. With a proximal block (e.g. at the AV node) the escape rhythm will arise from the AV node or bundle of His resulting in a narrower QRS complex and a rate of approximately 50 bpm. With a more distal block, the escape rhythm will produce broader QRS complexes at a slower rate (approximately 30 bpm).
Sick sinus syndrome	Due to ischaemia or fibrosis/degeneration of the SA node. Resulting in sinus pauses (>2 s) or sinus arrest. Junctional or other escape rhythms (e.g. AF) may emerge, often known as 'tachy-brady' syndrome. Patients ultimately need a pacemaker to manage the bradyarrhythmias and medical therapy (e.g. β-blockers) to manage the tachyarrhythmias.

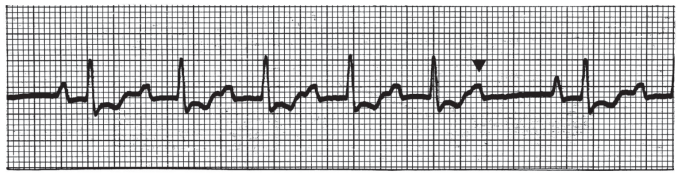

Figure 2.7 Second-degree heart block, type I (Wenckebach). The PR interval progressively prolongs until there is a failure of conduction following a P wave (arrow).

Reproduced from David A. Warrell, Timothy M. Cox, and John D. Firth, *Oxford Textbook of Medicine*, 2010, Figure 16.4.8, p. 2693, with permission from Oxford University Press.

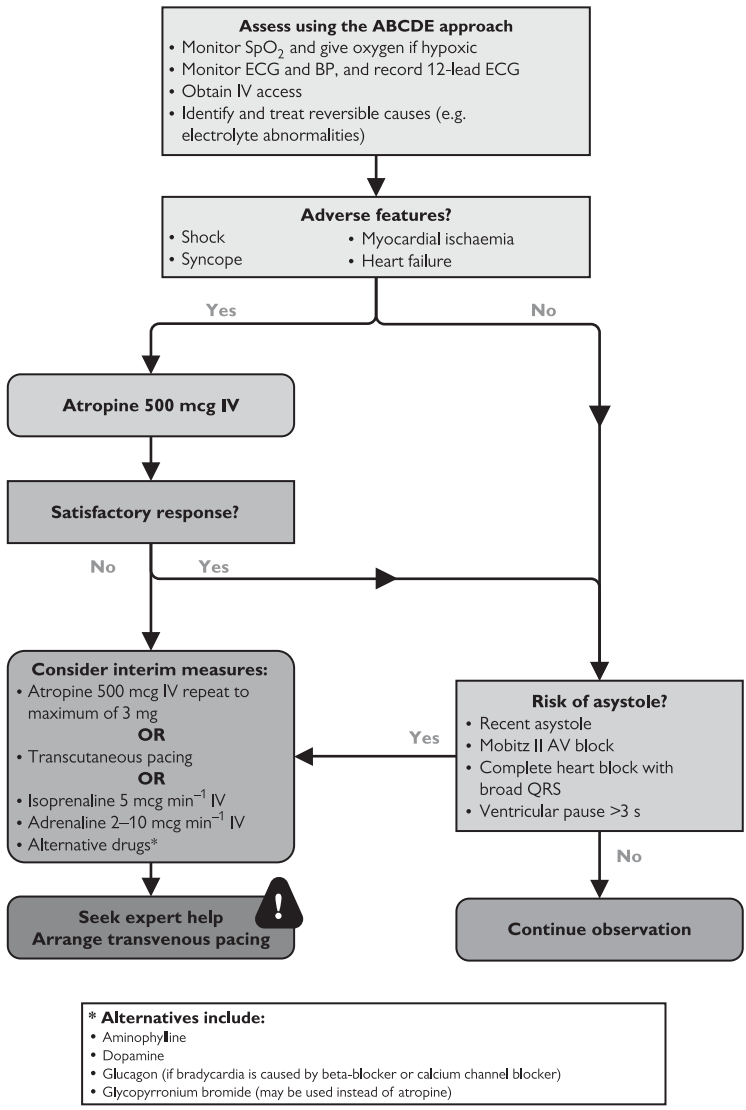

Figure 2.8 Adult bradycardia algorithm.

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Treatment of unstable bradycardia

- **Atropine**—is the first-line agent in bradycardia (500 mcg IV boluses up to a total dose of 3 mg). Atropine antagonizes the action of the parasympathetic neurotransmitter acetylcholine at muscarinic receptors. It blocks the action of the vagus nerve at the SA and AV nodes, increasing sinus automaticity and facilitating AV node conduction. Atropine should be used cautiously in those with acute myocardial ischaemia or infarction because the increased heart rate may worsen ischaemia or increase infarction size. Atropine should not be given to patients with heart transplants because their hearts are denervated and will not respond to the vagal blockade by atropine; paradoxically they may develop sinus arrest or high-grade AV block.
- **Pacing**—should be started if bradycardia with adverse signs persists despite atropine. Transcutaneous pacing can be painful and the patient should be sedated. For pacing to be successful, there must be electrical capture (i.e. a QRS complex after each pacing stimulus) and mechanical capture (i.e. a palpable pulse corresponding to the QRS complexes). Transcutaneous pacing is a temporary intervention and arrangements should be made for placement of a transvenous pacing wire.
- **Other drugs**—may be appropriate if there is a delay for transcutaneous pacing or in certain clinical situations. If there is a delay for pacing, adrenaline 2–10 mcg per minute IV can be given. If the likely cause is β-blockers or calcium channel blockers, then IV glucagon (1–2 mg) can be tried. If the bradycardia is due to digoxin toxicity, then digoxin-specific antibody can be considered. Theophylline (100–200 mg slow IV) can be given for bradycardia complicating acute inferior wall MI, spinal cord injury, or cardiac transplantation.

2.7.3 Stable bradycardias

Patients without adverse features should be assessed for the risk of asystole. The risk of asystole is greater in patients with the following ECG findings:

- Ventricular pause >3 seconds
- Mobitz type II AV block
- Complete heart block
- Recent asystole

These patients should be considered for temporary transvenous pacing.

KEY POINTS

There is a single set of adverse features for all peri-arrest arrhythmias: shock, syncope, myocardial ischaemia, and heart failure.
If a patient is unstable from their arrhythmia, the treatment of choice is electricity: synchronized cardioversion in tachyarrhythmias and pacing in bradyarrhythmias.
Broad-complex tachycardias are usually ventricular in origin but may be SVT with aberrant conduction. Know the ECG features that help distinguish them.
Stable VT is treated with amiodarone 300 mg IV over 20–60 min.
Torsades de pointe is treated with magnesium 2 g IV over 10 min.
SVT is treated with vagal manoeuvres, followed by adenosine.
AF can be rate-controlled with β-blockers or calcium channel blockers.
Bradycardia is treated with atropine 500 mcg IV to a maximum dose of 3 mg.

2.8 Shock

Shock is an abnormality of the circulatory system resulting in inadequate tissue perfusion and oxygenation.

2.8.1 Physiology of cardiac output

Provided the haemoglobin level and oxygen saturation are adequate, the main determinant of oxygen delivery to the tissues is cardiac output. In order to understand shock, it is necessary to understand the physiology of the cardiac output.

Cardiac output

The cardiac output (CO) is the volume of blood pumped out by the heart each minute:

$$CO = \text{Heart Rate (HR)} \times \text{Stroke Volume (SV)}.$$

Stroke volume

The stroke volume is the amount of blood pumped out per cardiac contraction. Stroke volume is determined by the following:

- Preload
- Myocardial contractility
- Afterload

Preload

Preload is the volume of venous return to the heart and is defined as the ventricular wall tension at the end of diastole. It is determined by venous capacitance, volume status, and the difference between venous systemic pressure and right atrial pressure.

The volume of venous blood returned to the heart determines the degree of ventricular filling and the length of myocardial muscle fibres. Muscle fibre length is related to the contractile properties of the myocardium (Frank–Starling law). Increased muscle fibre length results in a greater SV. However, above a certain point, the ventricle becomes overstretched and further filling results in a fall in SV.

Previous MRCEM question

Knowledge of the Frank–Starling curve (Figure 2.9) has appeared in previous SAQs.

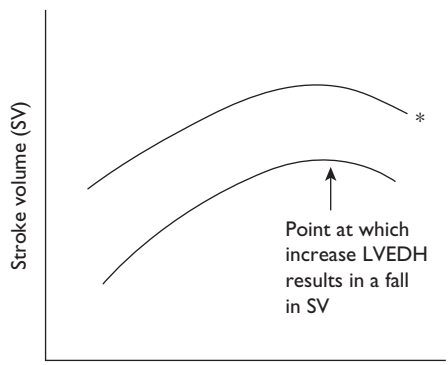

Figure 2.9 Frank–Starling curve.
Data from Frank 1918, and Starling 1919

Myocardial contractility

Myocardial contractility is the ability of the heart to work independent of the preload and afterload. Contractility can be increased by inotropes resulting in an increased SV for the same preload (Figure 2.9). Decreased myocardial contractility may result from intrinsic heart disease or from myocardial depression (e.g. caused by acidosis, hypoxia, sepsis, drugs, and so on).

Afterload

Afterload is defined as the ventricular wall tension at the end of systole. It is the resistance to forward blood flow.

2.8.2 Pathophysiology of shock

Shock can be the result of numerous pathophysiological processes and can be broadly divided into two groups: those that impair cardiac output and those that impair systemic vascular resistance (Table 2.10).

Irrespective of the cause of shock, inadequate delivery of oxygen to the tissues results in a failure of aerobic metabolism. Anaerobic respiration ensues, which is less efficient than aerobic, and results in the production of lactic acid. Eventually cell metabolism ceases, leading to cell death, and end organ dysfunction.

2.8.3 Clinical features of shock

Patients should be assessed and managed using the ABCDE approach. Any physiological derangements should be noted and corrected during the assessment. The underlying cause should be sought and treated.

The body has a range of compensatory mechanisms to cope with a reduction in oxygen delivery. These mechanisms account for some of the initial clinical features seen in a patient developing shock. Later features are those of organ dysfunction, as compensatory mechanisms fail (Table 2.11).

Shock due to acute blood loss

Shock secondary to acute blood loss can be classified into four groups depending on the estimated percentage blood loss (Table 2.12). The classification system is useful for demonstrating the clinical features of shock as the body compensates, and how late a fall in blood pressure can occur.

2.8.4 Investigations and monitoring in shock

Investigations should be guided by the suspected underlying cause (e.g. ECG in MI, FAST/CT scan in trauma, echocardiography in massive PE, septic screen in sepsis).

Certain investigations are generic and applicable to all causes of shock. This enables the severity of shock to be determined and monitoring of the response to treatment.

Table 2.10 Causes of shock

Cardiac output falls	Systemic vascular resistance falls
• Hypovolaemic (reduced preload), for example haemorrhage, diarrhoea and vomiting, burns. • Cardiogenic (reduced contractility), for example MI, myocardial contusion, myocarditis, late sepsis, overdose (e.g. β-blockers), complete heart block. Obstructive (increased afterload), for example PE, cardiac tamponade, tension pneumothorax.	• Sepsis. • Anaphylaxis. • Neurogenic—loss of sympathetic tone due to a spinal cord injury.

Table 2.11 Clinical features of shock

Clinical features	Compensatory response	Caveats
↑ Heart rate	Reduced CO results in sympathetic activation via arterial baroreceptors. This results in an increased heart rate to try and restore the CO.	Not all patients with a reduced CO develop a tachycardia. Patients taking certain medications (e.g. β-blockers) will not develop a tachycardia. Patients with a cardiogenic cause for their shock (e.g. complete heart block, β-blocker overdose) may not develop a tachycardia.
↑ Respiratory rate	Hypoxia may increase the respiratory rate (e.g. in pneumonia). Respiratory compensation for metabolic acidosis will raise the respiratory rate.	The respiratory rate may be normal or low in patients who are tiring and compensatory mechanisms are failing.
↓ Blood pressure	Blood pressure may not fall until 30–40% of the blood volume is lost in hypovolaemia (see Table 2.12). Blood pressure is often maintained by the release of catecholamines, which increases the systemic vascular resistance.	Before the BP falls, the pulse pressure may narrow due to a diastolic increase in response to vasoconstriction.
Cool peripheries, sweating	Vasoconstriction of cutaneous, muscle, and visceral circulation preserves blood flow to the heart, kidneys, and brain. Consequently the skin is pale and cool. Catecholamine release can result in sweating/clamminess.	In distributive shock (early sepsis, anaphylaxis, neurogenic) there is usually peripheral vasodilatation.
↓ Conscious level	Conscious level may be reduced due to decreased cerebral perfusion.	Do not assume a reduced conscious level is related purely due to shock. Ensure other causes are excluded (e.g. hypoglycaemia, intracranial bleed).
↓ Urine output	Urine output is typically reduced and patients should be catheterized early.	Urine output is of limited use in the initial assessment of patients in the ED.

Table 2.12 Classification of shock due to acute blood loss

Class of shock	Class I	Class II	Class III	Class IV
Volume of blood loss (ml)	Up to 750	750–1500	1500–2000	>2000
Volume of blood loss (%)	0–15%	15–30%	30–40%	>40%
Heart rate	<100	>100	>120	>140
Blood pressure	Normal	Normal	Decreased	Decreased
Pulse pressure	Normal or increased	Decreased	Decreased	Decreased
Respiratory rate	14–20	20–30	30–40	>35
Mental state	Slightly anxious	Mildly anxious	Anxious, confused	Confused, lethargic

- **Lactate**—is produced by anaerobic respiration. It is a useful marker of the severity of shock. Elevated lactate levels (>4 mmol/L) are associated with increased intensive care unit admissions and mortality in sepsis. The normalization of lactate in trauma and post-cardiac arrest patients correlates with improved survival.
- **Central venous pressure monitoring**—enables measurement of right heart filling pressures and guides fluid resuscitation. A CVP line also allows delivery of inotropic drugs and the measurement of central venous oxygen saturations.
- **Central venous oxygen saturations ($ScvO_2$)**—allows a measure of the oxygen content of blood returning to the heart. As oxygen is extracted from the blood, the oxygen saturation falls and the level can give an indication of total body oxygen extraction. The oxygen concentration in the mixed venous blood of the pulmonary artery (SvO_2) is usually 70–75%. If the SvO_2 is lower than this, it indicates that oxygen extraction has increased and in shocked states this is usually because oxygen delivery has become inadequate (demand > supply).

In the ED it is impractical to sample blood form the pulmonary artery (SvO_2) and consequently the central venous oxygen saturation ($ScvO_2$) is used. The $ScvO_2$ is about 5–7% higher than the SvO_2. Early goal-directed therapy in sepsis has incorporated the $ScvO_2$ as a guide for blood transfusion and inotropic support. Sepsis management is discussed in more detail in Chapter 15, section 15.1.

- **Invasive arterial pressure monitoring**—allows beat-to-beat measurement of the arterial blood pressure and easy serial blood gas analysis. Significant variation in the amplitude of the arterial pressure wave ('respiratory swing') is characteristic of hypovolaemia.

2.8.5 Management of shock

The management of shock should be directed at treating the underlying cause and correction of the physiological derangement. Specific treatments for the various causes of shock are dealt with in other chapters (e.g. Chapter 4, Chapter 9, Chapter 10, and Chapter 15).

Resuscitation to correct the physiological deficit is a dynamic process guided by the patient's clinical features, investigations, and monitoring. Resuscitation should occur in conjunction with the ABCDE assessment.

Airway and breathing

- Patients should receive high-flow oxygen to ensure oxygen delivery to the tissues is maximal.
- Intubation and ventilation should be considered early in the management of shocked patients. Positive pressure ventilation can dramatically reduce the work of breathing and therefore oxygen consumption. Patients requiring large volumes of fluid resuscitation are at risk of pulmonary oedema due to increased capillary permeability, especially in sepsis, and may require intubation.

Circulation

- Adequate fluid resuscitation is critical in shock. Patients should be given fluid boluses of 250 ml IV and reassessed after each. There is no evidence that colloid is superior to crystalloid (0.9% sodium chloride or Hartmann's is appropriate).
- In certain circumstances (e.g. penetrating chest trauma), cautious fluid resuscitation is appropriate until haemostasis is achieved.
- Haemoglobin is critical in determining adequate oxygen delivery to tissues. As a general rule, transfusing to haemoglobin of 7–9 g/dl is a reasonable target in otherwise healthy patients.
- Inotropes and vasopressors may be required if hypotension persists despite adequate intravenous fluid resuscitation.

Table 2.13 Effects of agonists on vasoactive receptors

Receptor	Effect
α_1	Vasoconstriction
β_1	Inotropic (increased force of contraction) and chronotropic (increased heart rate)
β_2	Vasodilatation (and bronchodilation)
Dopamine	Splanchnic and renal vasodilatation

2.8.6 Inotropes and vasopressors

Vasoactive drugs can be classified as:

- inotropes—which increase cardiac contractility, or
- vasopressors—which increase systemic vascular resistance.

Combinations of vasoactive drugs are often used to optimize cardiac output and perfusion pressure (Table 2.13).

The ACCS curriculum includes knowledge of vasoactive drugs used in shocked patients (Tables 2.14 and 2.15). The most commonly used inotrope in the ED is adrenaline, because it is the most useful in hypotensive states when the overall haemodynamic status is unclear.

Table 2.14 Actions of inotropic agents

Inotrope	Actions
Adrenaline (epinephrine)	Stimulates α and β receptors. At low doses, the β effects predominate—increasing CO and vasodilatation. At higher doses, α_1 effects predominate—increasing systemic vascular resistance. It also causes splanchnic vasoconstriction, hyperglycaemia (↑ glycogenolysis and gluconeogenesis), increases myocardial oxygen consumption, and increases lactate.
Dobutamine	Stimulates β_1 and β_2 receptors. β_1 actions increase the heart rate and force of contraction. β_2 actions cause peripheral vasodilatation. It is useful in low CO states (e.g. post-MI) when vasomotor tone is reasonably maintained. It is frequently used in combination with noradrenaline, which provides the peripheral vasoconstriction to maintain systemic vascular resistance.
Dopamine	Stimulates dopamine, α_1 and β_1 receptors. It also releases noradrenaline from adrenergic nerves. At low doses, it acts predominantly on dopamine receptors resulting in increased splanchnic and renal perfusion. At higher doses, it acts on α_1 and β_1 receptors leading to vasoconstriction and increased CO.

Table 2.15 Actions of vasopressor agents

Vasopressor	Actions
Noradrenaline (norepinephrine)	Stimulates α_1 receptors. Causes peripheral vasoconstriction. Excessive use can increase afterload and reduce CO, reduce renal blood flow, reduce splanchnic blood flow, and impair peripheral perfusion.
Phenylephrine	Stimulates α_1 receptors. Causes peripheral vasoconstriction.
Metaraminol	Stimulates α_1 receptors. Causes peripheral vasoconstriction. Can be given peripherally. Often used in cardiovascularly unstable patients during induction of anaesthesia.

KEY POINTS

Shock is an abnormality of the circulatory system, resulting in inadequate tissue perfusion and oxygenation.

Blood pressure = Cardiac output (CO) × Systemic vascular resistance (SVR).

CO = Heart rate × Stroke volume.

Stroke volume is determined by the preload, myocardial contractility, and afterload.

Global oxygen delivery is determined by CO and arterial oxygen content, but perfusion of individual organs depends on many other factors.

Shock can be classified by causes that reduce the CO and causes that reduce the SVR:

- CO reduced—hypovolaemia, cardiogenic, obstructive
- SVR reduced—sepsis, anaphylaxis, neurogenic

Initial compensatory responses can conceal the development of shock. Do not be falsely reassured by a normal heart rate and blood pressure.

Lactate, $ScvO_2$, and urine output are useful adjuncts to the initial clinical assessment and to guide ongoing resuscitation.

Inotropes and vasopressors should not be used as a substitution for adequate fluid resuscitation. In a shocked patient of unknown aetiology, adrenaline is the inotrope of choice.

2.9 SAQs

2.9.1 Tachycardia

A 34-year-old lady attends, having developed palpitations three hours ago while walking the dog. She has never experienced these symptoms before. Figure 2.10 shows her ECG.

a) What is the rhythm on the ECG (Figure 2.10)? (1 mark)
b) What four adverse features should you assess her for according to the Resuscitation Council Guidelines? (2 marks)
c) (i) She has no adverse features and vagal manoeuvres are unsuccessful. What is the recommended first-line drug to try and cardiovert the patient? What is the initial dose of this drug and the two subsequent doses, if this is unsuccessful? (1 mark for drug, 2 marks for doses)
   (ii) List four relative contraindications for the drug in answer c(i). (2 marks)
d) Name two other drugs, and the doses, you might use to try and achieve cardioversion in this patient. (2 marks)

Suggested answer

a) What is the rhythm on the ECG? (1 mark)
   Supraventricular tachycardia or narrow-complex tachycardia
b) What four adverse features should you assess her for according to the Resuscitation Council guidelines? (2 marks)
   Shock
   Syncope
   Myocardial ischaemia
   Heart failure

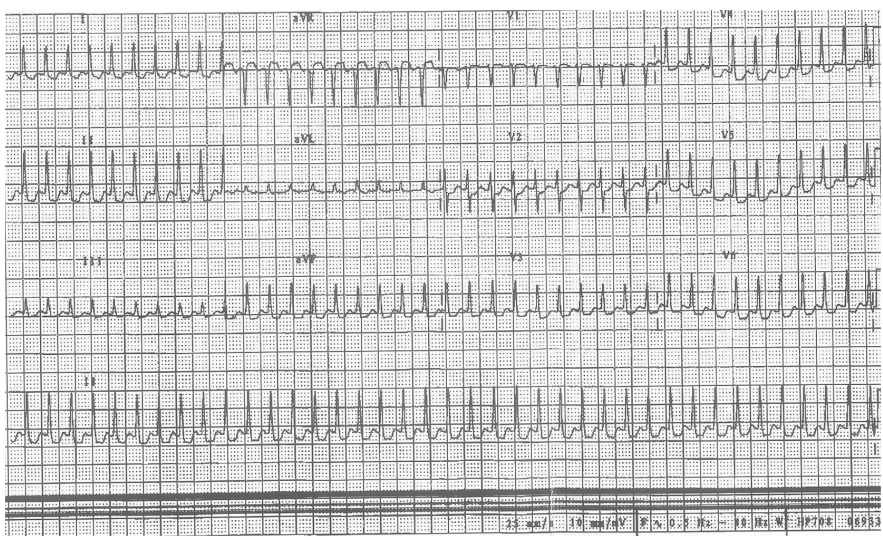

Figure 2.10 Tachycardia in a 34-year-old woman.

c) (i) **She has no adverse features and vagal manoeuvres are unsuccessful. What is the recommended first-line drug to try and cardiovert the patient? What is the initial dose of this drug and the two subsequent doses if this is unsuccessful?**

Adenosine
Doses: 6 mg, 12 mg, 12 mg
(1 mark for drug; 1 mark for 6 mg; 1 mark for both 12 mg doses)

(ii) **List four relative contraindications for the drug in answer c (i). (2 marks)**

Asthma
Heart transplant (denervated heart)
Sick sinus syndrome
AF/flutter with accessory pathway or WPW
Dipyridamole use
Carbamazepine use
Theophylline use
Second- or third-degree heart block

d) **Name two other drugs, and the doses, you might try to achieve cardioversion in this patient. (2 marks)**

Amiodarone 300 mg IV over 20–60 minutes
Flecainide 2 mg/kg IV over 10 min or 100–200 mg PO
Verapamil 5 mg IV over two minutes, repeat after five minutes
Beta-blockers, for example sotalol 20–60 mg IV or 80–160 mg PO; or metoprolol 5 mg IV repeat after five minutes or 50 mg PO.

2.9.2 Cardiac arrest in special circumstances

A 55-year-old man is brought to the ED in cardiac arrest. He was seen to fall through the ice on a lake and took 10 minutes to be rescued. Figure 2.11 shows his ECG following a ROSC.

(a) **List four modifications to the ALS algorithm for patients in a hypothermic cardiac arrest. (2 marks)**

(b) **Name two methods of passive rewarming and two methods of active rewarming. (2 marks)**

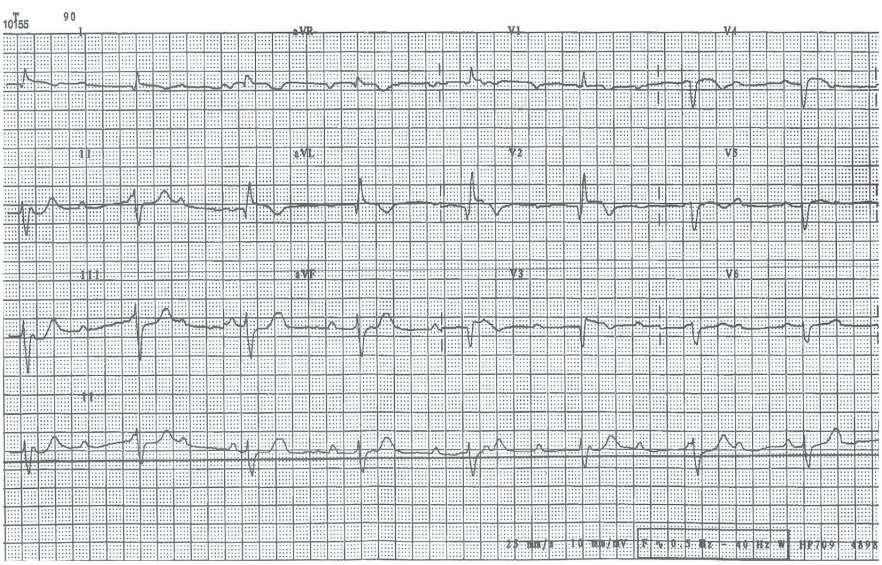

Figure 2.11 Electrocardiogram (ECG) in a hypothermic patient.

(c) He has a ROSC and his temperature is 31°C. What temperature should he be rewarmed too and why? (1 mark for temperature, 1 mark for reason)

(d) (i) What is the rhythm on his ECG? (1 mark)

(ii) What are your three main treatment options for managing this arrhythmia in this patient? (3 marks)

Suggested answer

a) List four modifications to the ALS algorithm for patients in a hypothermic cardiac arrest. (2 marks)

Pulse check/look for signs of life for up to one minute.
Consider Doppler/echocardiography to confirm the presence or absence of cardiac output.
Withhold cardioactive drugs until the core temperature is >30°C.
>30°C double the interval between doses of cardioactive drugs (until >35°C).
If VF/VT persists beyond three shocks, postpone further shocks until the temperature is >30°C.
Patients may require large volumes of IV fluids as they rewarm and vasodilate.
Give drugs via a central or large proximal vein if possible.
Monitor glucose regularly due to the risk of hypoglycaemia.
Confirm the temperature with a low-reading thermometer.
Warm the patient actively and passively.
Perform interventions (e.g. CVP insertion or intubation) carefully to avoid excessive movement and precipitating VF.

b) Name two methods of passive rewarming and two methods of active rewarming. (2 marks)

Passive: Remove cold or wet clothing; dry the patient; cover with blankets/hot air blanket; warm room (e.g. overhead heater); forced-air warming system (Bair Hugger).
Active: Warmed humidified oxygen; warmed IV fluids; gastric, peritoneal, pleural, or bladder lavage with warmed fluids; cardiopulmonary bypass.

c) He has a ROSC and his temperature is 31°C. What temperature should he be rewarmed too and why? (1 mark for temperature, 1 mark for reason)

Between 32–34°C. In comatose survivors, a period of therapeutic hypothermia may be beneficial.

d) (i) What is the rhythm on his ECG? (1 mark)

Complete (third-degree) heart block.

(ii) What are your three main treatment options for managing this arrhythmia in this patient? (3 marks)

Rewarm the patient—most arrhythmias revert spontaneously with rewarming.
Drugs—atropine 500 mcg IV boluses, to a total of 3 mg (only if rewarming fails) (adrenaline IV if atropine fails and pacing is delayed).
Pacing—only indicated if the bradyarrhythmia persists after rewarming.

Further reading

National Institute for Health and Care Excellence, December 2011. NICE clinical guideline 134. Anaphylaxis: assessment and referral after emergency treatment. Available at: https://www.nice.org.uk/guidance/CG134 [Online].

Resuscitation Council (UK), 2015. Resuscitation Guidelines. Available at: https://www.resus.org.uk [Online].

CHAPTER 3

Anaesthetics and pain management

CONTENTS

3.1 Emergency airway care

3.1.1 Introduction

Effective airway management is a key skill of an emergency physician. Good airway care is paramount in the care of critically ill and injured patients. The theoretical aspects of acute airway assessment and management are likely to feature in the Intermediate FRCEM examination.

3.1.2 Indications for intubation

The decision to intubate or not is often the first key decision in the management of a critically ill patient. This does not detract from the need to provide supplemental oxygen and good basic airway management.

The advantages of intubation include:

- A secured airway protected from obstruction by swelling, blood, or vomit.
- Lungs protected from aspiration.
- Optimized oxygenation.
- Improved ventilation, which can assist in the correction of respiratory or metabolic acidosis.
- Removal of the work of breathing, reducing metabolic demands.
- Safe sedation of the patient without the risk of respiratory compromise.
- Minimization of the risk of gastric insufflation.
- Ventilation without interrupting chest compressions in cardiac arrest.

The risks of intubation include:

- Failed intubation.
- Inability to intubate or ventilate the patient following induction of anaesthesia.
- Misplaced endotracheal tube. This may be during the intubation itself or subsequently dislodged. This is catastrophic if not identified.
- Cardiovascular instability following the use of induction agents.
- Raised intracranial pressure.
- Precipitating laryngospasm.

- Trauma to the lips, teeth, oropharynx, larynx, and trachea.
- Interruptions to chest compressions while intubating in cardiac arrest.

There are four main situations when intubation may be indicated:

- Apnoeic patient in respiratory arrest.
- Patient with a partially or completely obstructed airway, where basic airway management is ineffective (e.g. burns, facial trauma, actively vomiting, or bleeding).
- Patient requiring invasive respiratory support for oxygenation or ventilatory failure (e.g. severe pneumonia, chest trauma, overdose).
- Patient whose predicted clinical course includes a high probability of airway obstruction, aspiration, or ventilatory failure (e.g. epiglottis, early burns); or, a patient requiring transport where airway management will be difficult (e.g. CT scanner, ambulance transfer).

The urgency of the intubation may vary depend on the clinical situation:

- Immediate intubation—patient is deteriorating rapidly and definitive airway care is required with a minimum of delay (e.g. a hypoxic patient with partial airway obstruction secondary to burns).
- Urgent intubation—basic techniques are maintaining adequate oxygenation and ventilation at present but will only do so for a short time (e.g. a head-injured patient with a Glasgow coma scale (GCS) <8 whose airway obstruction can be relieved with a jaw thrust).

Intubation is indicated when the risks of continuing basic airway support are greater than the risks of intubation. The difficulty of intubation must be factored into the decision, and a patient with an anticipated 'difficult airway' may need to be postponed (if possible) until more senior help arrives and a difficult airway cart is available.

Reversible causes

In some patients there may be reversible causes for airway obstruction, respiratory compromise, or reduced conscious level. If a reversible cause is identified, it should be treated, which may negate the need for intubation (Table 3.1).

Table 3.1 Potential reversible causes of airway obstruction, respiratory failure, and reduced conscious level

Reversible cause	Treatment
Opiate overdose	Naloxone
Hypoglycaemia	10% or 50% glucose
Seizures	Benzodiazepines, phenytoin
Hypercapnia (e.g. in COPD)	Reduce inspired oxygen concentration ± non-invasive ventilation
Hypovolaemia	IV fluids, inotropes
Arrhythmia	Electrical cardioversion
Anaphylaxis	IM adrenaline
Asthma	Nebulizers (salbutamol, ipratropium bromide), magnesium, aminophylline, steroids
Heart failure	Diuretics, nitrates, non-invasive ventilation
Agitation (with impaired conscious level)	Analgesia, if in pain
	IV fluids if hypotensive
	Glucose if hypoglycaemic
	Ensure bladder empty

KEY POINTS

Intubation is always preceded by basic airway management and oxygenation.

Intubation is indicated when the risks of continuing basic airway support are greater than the risks of intubation.

The four main indications for intubation are:

- Apnoeic patient in respiratory arrest
- Patient with a partially or completely obstructed airway, where basic airway management is ineffective
- Patient requiring invasive respiratory support for oxygenation or ventilatory failure
- Patient whose predicted clinical course includes a high probability of airway obstruction, aspiration, or ventilatory failure

Reversible causes for airway obstruction, respiratory compromise, and reduced conscious level should be considered and treated, which may negate the need for intubation.

3.2 Identifying the difficult airway

Difficulties with the airway should be expected in all emergency patients. Patients requiring intubation in the ED are a different cohort to those patients undergoing elective intubation and the risks are greater. ED patients may have deranged physiology, poor cardiorespiratory reserve, and an unstable cervical spine.

Airway difficulties are more than just difficulties intubating and can be categorized according to the different components of airway management:

- Difficult basic airway manoeuvres; for example, neck immobility limiting the ability to perform a head tilt; facial fractures limiting a jaw thrust; trismus preventing insertion of an oropharyngeal airway; and nasal deformity preventing insertion of a nasopharyngeal airway.
- Difficult mask ventilation—gaining an adequate seal with a facemask may be prevented by facial hair, lack of teeth, cachexia, obesity, or trauma. Ventilation may be difficult in those with diaphragmatic splinting, abdominal distension, or increased airway resistance (e.g. asthma).
- Difficult laryngoscopy—classified by Cormack and Lehane (Figure 3.1). In grade 3 and 4 views, the vocal cords are not visible and intubation may be impossible without specialist equipment.
  - Grade 1—the vocal cords are visible
  - Grade 2—the vocal cords are only partially visible
  - Grade 3—only the epiglottis is seen
  - Grade 4—the epiglottis is not visible*

Figure 3.1 Cormack and Lehane classification of glottic visualization.

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- Difficult intubation—defined as occurring when an experienced laryngoscopist, using direct laryngoscopy, requires more than two attempts with the same blade; or a change of blade; or an adjunct (bougie); or an alternative device.
- Difficult cricothyroidotomy—an assessment of the patient's neck should be made prior to induction of anaesthesia to determine how difficult a surgical airway would be to perform if required.

3.2.1 Airway assessment

There are several tests described to predict difficult intubation. Unfortunately the sensitivity and specificity of them is fairly poor. A patient may have a positive test and not have a difficult airway, and, conversely, a patient with a negative test may have difficulties. However, they do provide a guide to identifying those with a potentially difficult airway and knowledge of such tests is included in the curriculum.

To help remember predictors of a difficult airway there are several different mnemonics. It is not essential to use a mnemonic but some candidates find it useful. A commonly used mnemonic is LEMON (Table 3.2).

Table 3.2 LEMON assessment of airway

Look— for characteristics that are known to cause difficult intubation or ventilation	• Obesity • Facial trauma • Excessive facial hair • Facial deformity • Sunken cheeks • Edentulous • Prominent upper incisors • High arched palate • Receding mandible • Short neck • Thick neck circumference (> 45 cm) • Narrow mouth • Macroglossia • Prognathia (inability to move lower teeth in front of upper teeth) • History of snoring (obstructive sleep apnoea)
Evaluate—mouth opening and thyromental distance (the 3–3–2 rule)	• 3 fingers breadth between incisors • 3 fingers between hyoid bone and the chin • 2 fingers between thyroid notch and floor of the mouth
Mallampati score (Figure 3.2)—used to assess how much of the hypopharynx is visible with the patient sitting, mouth fully open, and tongue protruding	• Class 1—faucial pillars, soft palate, and uvula visible • Class 2—faucial pillars and soft palate visible; tip of uvula masked by base of tongue • Class 3—only soft palate visible • Class 4—soft palate not visible
Obstruction—pathology within or surrounding the upper airway	• Peri-tonsillar abscess • Epiglottis • Retro-pharyngeal abscess • Trauma • Burns • Tumour

Table 3.2 Continued

Neck mobility—assessed by asking the patient to place their chin on their chest and extend their neck to look to the ceiling	• In-line stabilization • Rheumatoid arthritis • Osteoarthritis • Ankylosing spondylitis • Previous neck injuries or surgery • Neck extension undesirable: unstable cervical spine fracture; severe cervical stenosis; vertebral artery insufficiency; Chiari malformation

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3.2.2 Pre-anaesthetic assessment

In most emergency situations there is time to perform a pre-anaesthetic assessment. This is a more focused assessment than that used in elective anaesthesia and forms an important part of the preparation for rapid sequence induction.

The pre-anaesthetic assessment should include a relevant history and examination (Table 3.3).

KEY POINTS

Airway difficulties may be encountered during basic airway manoeuvres, bag-valve-mask ventilation, laryngoscopy, intubation, and/or cricothyroidotomy.

The LEMON mnemonic can be used to assess the airway and identify those with potential difficulties:

- **L**ook—for characteristics that are known to cause difficult intubation or ventilation.
- **E**valuate—mouth opening and thyromental distance (the 3–3–2 rule).
- **M**allampati—grades 1–4.
- **O**bstruction—is there evidence of pathology within or surrounding the upper airway.
- **N**eck mobility.

A pre-anaesthetic assessment should be performed, focusing on the relevant history and examination.

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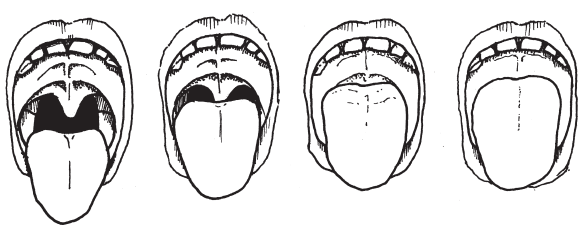

Figure 3.2 Mallampati test (Samsoon and Young modification).

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Table 3.3 Emergency pre-anaesthetic assessment

History	Examination
• History of events and current condition • Past medical and surgical history • Current medications • Allergies • Previous anaesthetics and any complications • Last oral intake	• Cardiorespiratory status • Assessment of face and neck • Assessment for pneumothorax • GCS • Focal neurological signs • Evidence of pathology in the chest, abdomen, and pelvis

3.3 Emergency airway drugs

Anaesthesia requires three components—'the triad of anaesthesia':

- Hypnosis
- Analgesia
- Muscle relaxation

3.3.1 Induction agents (hypnosis)

There are four main induction agents used in emergency rapid sequence induction (RSI) in the United Kingdom (Table 3.4). All of the agents have limitations and the drug used depends on the condition of the patient and the familiarity of the practitioner with the drug.

3.3.2 Muscle relaxation

Muscle relaxation is usually achieved with suxamethonium. However, occasionally a non-depolarizing agent, such as rocuronium, may be used.

Suxamethonium

Suxamethonium is a depolarizing muscle relaxant and the only one in clinical use today. Its rapid onset and short duration of action make it the drug of choice for muscle relaxation in emergency RSI (Table 3.5).

Non-depolarizing muscle relaxants

Occasionally a non-depolarizing muscle relaxant may be used instead of suxamethonium (e.g. if suxamethonium is contraindicated) (Table 3.6). Of the non-depolarizing muscle relaxants, rocuronium is the agent most likely to be used for a modified RSI. Other non-depolarizing muscle relaxants may be used for maintenance of paralysis following recovery from suxamethonium.

One of the main concerns about using a non-depolarizing muscle relaxant is the prolonged duration of action compared to suxamethonium. The short duration of suxamethonium is advantageous if a difficult airway is encountered and intubation and/or ventilation are not possible. However, in 2008, a reversal agent for rocuronium was licensed, sugammadex—the first selective relaxant binding agent. This has resulted in rocuronium being a much more feasible first-line agent for RSI. Reversal of non-depolarizing muscle relaxants prior to the introduction of sugammadex was with neostigmine, an anticholinesterase. Neostigmine is still in use but causes autonomic instability and bradycardia, so has to be given concurrently with an anti-muscarinic (e.g. atropine or glycopyrronium bromide). Sugammadex is associated with much greater cardiovascular and autonomic stability, and therefore is better suited to cardiovascularly compromised patients in the ED.

Table 3.4 Induction agents used in emergency RSI

	Indications	Dose	Onset/Recovery	Physiological effects
Thiopental	Most useful in cardiovascularly stable patients with an isolated head injury or seizures.	4–5 mg/kg IV (1.5–2 mg/kg if cardiovascularly unstable)	Onset: 5–15 s Recovery: 5–15 min	Cerebroprotective: ● Decreases cerebral metabolic oxygen consumption. ● Decrease cerebral blood flow. ● Decreases intracranial pressure (ICP). ● Maintains cerebral perfusion pressure. ● Anticonvulsant properties. ● Cardiovascular depression: ● Venodilatation. ● Myocardial depression.
Propofol	Most commonly used induction agent in elective anaesthesia. Can be used for maintenance of anaesthesia. Used for procedural sedation.	1.5-2.5mg/kg IV	Onset: 20–40 s Recovery: 5–10 min	Marked hypotension in patients with cardiovascular compromise (due to vasodilatation and myocardial depression). Apnoea. Pain on injection. Anticonvulsant properties (although patient may have some involuntary movements on induction).
Etomidate	Useful in haemodynamically compromised patients.	0.3 mg/kg IV	Onset: 5–15 s Recovery: 5–15 min	Relative haemodynamic stability. Adrenocortical suppression. Attenuates increase in ICP on laryngoscopy. Reduces cerebral blood flow. Reduces cerebral oxygen demand.
Ketamine	Severe bronchospasm. Cardiovascularly unstable patients. Burns.	1–2 mg/kg IV 5–10 mg/kg IM	Onset: 15–30 s (IV) Recovery: 15–30 min	Analgesia. Sedation. Dissociative state. Amnesia. Sympathetic stimulation: ↑HR, ↑BP. Bronchodilation. Enhanced laryngeal reflexes (potential for laryngospasm). Increased respiratory secretions. Emergence phenomena.

Table 3.5 Summary of the properties of suxamethonium

Indication	Muscle paralysis for RSI
Dose	1–2 mg/kg IV
Induction characteristics and recovery	5–15 s: fasciculations 45–60 s: paralysis 3–5 min: first return of respiratory activity 5–10 minutes: return of effective spontaneous ventilation
Physiological effects	Depolarizing muscle paralysis Metabolized by pseudocholinesterase
Adverse effects	• Hyperkalaemia (plasma concentration increased by up to 0.5 mmol/L) • Bradycardia (children are at the greatest risk) • Fasiculations resulting in muscle pain • Histamine release causing potential anaphylaxis • Malignant hyperthermia • Suxamethonium apnoea (patient has low or abnormal pseudocholinesterase activity and paralysis may last for several hours)
Contraindications	Hyperkalaemia—known blood result or ECG suggestive Risk of hyperkalaemia: • Severe trauma or infection • Desquamating skin conditions • Burns (risk greatest after 2 days) • Peripheral neuropathy (risk greatest after 5 days) • Spinal cord injury (risk greatest after 5 days) • Upper motor neurone lesions or structural brain disease • Muscular dystrophy

EXAM TIP

It is easy to become overwhelmed by the pharmacology of anaesthetic drugs. It is important to know the general pros and cons of the induction agents and muscles relaxants used in the resuscitation room. However, the exam is not an anaesthetic exam. In-depth pharmacological questions or knowledge of doses are unlikely to be asked and, if they were, they would only be worth 1 or 2 marks.

Table 3.6 Non-depolarizing muscle relaxants

	Dose	Onset/Duration	Metabolism
Rocuronium	Loading dose 0.6–1 mg/kg Maintenance 0.1–0.5 mg/kg/h	Onset 60 s Duration 30–60 min	Hepatic/renal
Atracurium	Loading dose 0.3–0.5 mg/kg Maintenance 5–10 mcg/kg/min	Onset 3–5 min Duration 20–35 min	Hoffman elimination
Vecuronium	Loading dose 0.05–0.1 mg/kg Maintenance 1–2 mcg/kg/min	Onset 3–5 min Duration 20–35 min	Hepatic/biliary

3.3.3 Analgesia in rapid sequence induction

Analgesia is part of the 'triad of anaesthesia' but is not part of the classic RSI technique. However, some practitioners will use opiates to attenuate the cardiovascular response to laryngoscopy and intubation. This is particularly advantageous in patients with raised intracranial pressure (ICP), severe hypertension, or ischaemic heart disease.

The commonly used opiates with RSI are fentanyl or alfentanil. They have a faster onset of action than morphine (alfentanil 1 minute; fentanyl 3 minutes; morphine 5 minutes). However, opiates cause respiratory depression, which may persist beyond the recovery from neuromuscular blockade, requiring reversal with naloxone, if intubation fails.

Analgesics are discussed in more detail in section 3.6.

3.3.4 Maintenance of anaesthesia

Following successful intubation, anaesthesia must be maintained. The commonly used infusions for this are propofol or midazolam and morphine. In the ED, paralysis is usually maintained with a non-depolarizing muscle relaxant.

3.3.5 Complications of anaesthetic drugs

There are a few well-recognized complications of anaesthetic drugs, which could appear in a short-answer question (SAQ).

- Malignant hyperthermia—is a rare autosomal dominant condition related to general anaesthesia (e.g. suxamethonium or gaseous agents). It results in uncontrolled skeletal muscle oxidative metabolism. Masseter muscle spasm may be an early sign of malignant hyperthermia, later effects include rhabdomyolysis, tachyarrhythmias, and pyrexia. Treatment involves stopping the precipitant, supportive management, and dantrolene.
- Suxamethonium apnoea—occurs when the patient has low or abnormal pseudocholinesterase activity. Muscle paralysis may last for several hours after a dose of suxamethonium. Treatment is continued ventilation and sedation until normal neuromuscular activity returns.
- Anaphylaxis—may occur with any anaesthetic agent. Management should be as per advanced life support (ALS) guidance detailed in Chapter 2, section 2.3.

KEY POINTS

Anaesthesia requires three components—'the triad of anaesthesia': hypnosis, analgesia, and muscle relaxation.

RSI involves giving an anaesthetic induction agent to achieve hypnosis, rapidly followed by a muscle relaxant to produce complete paralysis.

The four main induction agents used in emergency RSI in the United Kingdom are thiopental, propofol, etomidate, and ketamine.

Muscle relaxation is usually achieved with suxamethonium. However, occasionally a non-depolarizing agent, such as rocuronium, may be used.

3.4 Rapid sequence induction

RSI involves giving an anaesthetic induction agent to achieve hypnosis, rapidly followed by a muscle relaxant to produce complete paralysis. The drugs are given in quick succession to minimize time from loss of consciousness to intubation because the stomach is assumed to be full. Cricoid pressure is applied as the induction agent is given, to protect the airway from gastric regurgitation. To prevent inflation of the stomach, the lungs are not usually ventilated between induction and intubation.

3.4.1 Preparation for a rapid sequence induction

Prior to performing an RSI, preparations must be made to maximize the chance of success. Intubation rarely has to be achieved so urgently that basic preparatory steps cannot be taken (Table 3.7 and Figure 3.3).

Table 3.7 Preparation for RSI

Monitoring The minimum recommended standards are defined by The Association of Anaesthetists of Great Britain and Ireland.	• Inspired oxygen concentration (FiO_2). • Capnography. • Pulse oximetry. • Non-invasive blood pressure. • 3-lead ECG.
Equipment The airway equipment in the resuscitation room should be checked daily. The practitioner performing the RSI should prepare and check the equipment prior to induction.	Basic airway equipment: • Oxygen. • Oxygen masks and tubing. • Tilting trolley. • Suction. • Airway adjuncts (nasopharyngeal and oropharyngeal). • IV access. • Monitoring. Advance airway equipment: • System to pre-oxygenate the patient and provide ventilation (e.g. bag-valve-mask or Mapleson C anaesthetic circuit/ Waters circuit). • Laryngoscope handles and blades (usually a size 3 and 4 Macintosh). • Magill's forceps. • Intubating stylet and bougie. • Tracheal tubes in a range of sizes (women, 7–7.5 mm; men, 8 mm); tubes should be uncut if there is a risk of facial swelling (e.g. burns). • 20-ml syringe to inflate the cuff and lubricating gel for the tracheal tube. • Tie and/or adhesive tape. • Ventilator. • Drugs (labelled). Failed intubation equipment: • Supraglottic airway device (e.g. laryngeal mask airway sizes 3, 4, and 5). • Surgical cricothyroidotomy set. • Needle cricothyroidotomy set.
Positioning Alignment of the oral, pharyngeal, and laryngeal axes during laryngoscopy provides the best view of the laryngeal inlet.	'Sniffing the morning air' (neck flexed on the torso and head extended on the neck) is the best position in an adult. This position can be achieved by placing a pillow under the patient's head (flexes the neck) and then extending the patient's head. In obese patients, standard neck positioning may flex the head forwards forcing the chin onto the chest. The key to correct positioning in such a patient is to make sure the chin is higher than the highest point on the chest or abdomen (i.e. place a pillow under the shoulders and further pillows under the head to raise it further). If a cervical spine injury is suspected the neck must be maintained in a neutral position. In this situation manipulation of the larynx may improve the view, for example the 'BURP' manoeuvre (backwards, upwards, rightwards laryngeal pressure).

Table 3.7 Continued

Checks Prior to RSI a few final checks should be made to ensure the patient is optimized.	Briefly review the history to obtain relevant clinical information. The AMPLE mnemonic is a useful reminder for this: ● Allergies. ● Medications. ● Past medical history (including previous anaesthetics). ● Last ate/drank. ● Events. *Resuscitation*—ensure the patient is optimized as best as possible. *Final examination*—if time allows record the patients GCS, note any focal neurological signs, assess the abdomen, pelvis, and long bones for any evidence of pathology. *IV access*—ensure there are two functioning IV lines.
Staffing A RSI requires at least 3–4 practitioners to perform safely.	The roles required in a RSI include: ● An airway practitioner. ● An assistant to the airway practitioner who has knowledge of the equipment, techniques, and failed intubation drill. ● Cricoid pressure (this may be the airway assistant). ● Drug delivery (induction agent, muscle relaxant, and fluids/inotropes if required). ● Manual in-line stabilization, if a cervical spine injury is suspected. ● A practitioner to maintain responsibility for overall care of the patient. An appropriately experienced airway practitioner should also be available in case expert help is required.

3.4.2 Performing the rapid sequence induction

The skill of performing a RSI is most likely to be tested in the part C examination. The following section summaries the main stages of a rapid sequence induction.

Pre-oxygenation

The purpose of pre-oxygenation is to maximize the time before desaturation occurs, following the onset of apnoea. Effective pre-oxygenation replaces nitrogen in the alveoli with oxygen, which increase the oxygen reserve in the lungs. Ideally 100% oxygen should be given for three minutes before induction of anaesthesia.

In a breathing patient, this is best delivered via a Mapleson C or Waters circuit. A bag-valve-mask with a good seal can also be used, but the resistance to inspiratory and expiratory flow is high, making it less than ideal.

If the patient is agitated and will not tolerate oxygenation via a bag-valve-mask or Mapleson C circuit, an oxygen mask with a non-rebreathing bag can be used, at flow rates of 15 L, with a well-fitting mask—this provides approximately 90% inspired oxygen concentration.

If the patient is not spontaneously breathing, or breathing inadequately, then assisted ventilation will be required to achieve pre-oxygenation. The time to desaturation is related to the effectiveness of the pre-oxygenation and also the age, weight, and physiological status of the patient. In a healthy adult, the time taken to desaturate to 90% may be up to eight minutes. In a critically ill patient, this time is significantly reduced. Due to the shape of the oxyhaemoglobin dissociation curve (Figure 3.4), once the saturations reach 92%, the rate of desaturation accelerates. Therefore, when saturations of 92% are reached, corrective action is required, and the patient should be reoxygenated immediately.

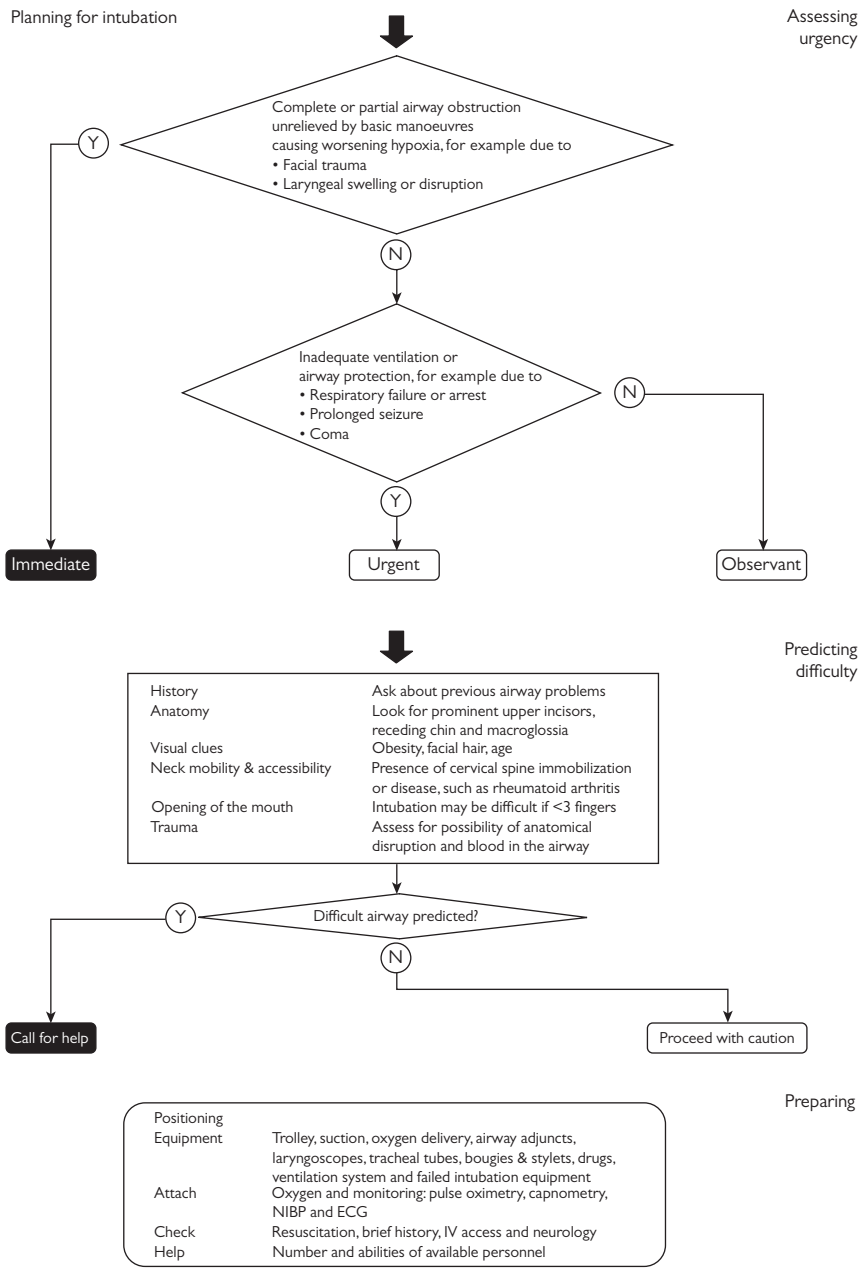

Figure 3.3 Patient assessment and preparation for intubation.

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Drugs

The induction agent and muscle relaxant are given in quick succession in pre-calculated doses. The choice of agents depends on the clinical status of the patient. The emergency airway drugs that are commonly used are discussed in section 3.3.

Cricoid pressure

Cricoid pressure is applied as the induction agent is given. The aim is to compress the upper oesophagus between the cricoid and the cervical vertebrae posteriorly. Cricoid pressure aims to stop passive regurgitation of gastric contents but should not be applied during active vomiting because of the risk of oesophageal rupture.

Technique of cricoid pressure:

- The cricoid is located below the thyroid cartilage and cricothyroid membrane.
- The cricoid ring is stabilized between the thumb and index finger.
- Firm pressure is applied directly backwards as the induction agent is given and consciousness lost.
- The correct pressure is 30–40 Newtons.
- Cricoid pressure is removed only on the instruction of the intubating clinician, once correct tube placement has been confirmed.

Laryngoscopy and intubation

The technique of laryngoscopy is best learnt as a practical skill and details of the technique are unlikely to be asked in an SAQ.

In order to gain the best view at laryngoscopy, the following techniques may be required:

- Suction.
- Adjustment of the cricoid pressure and/or the BURP manoeuvre (backwards, upwards, rightwards laryngeal pressure) may be required.
- Good laryngoscopy technique—clear identification of the epiglottis and insertion of the tip of the laryngoscope into the vallecula, followed by elevation of the laryngoscope in the line of the handle to bring the laryngeal inlet into view.
- Different laryngoscope—if the blade is too short the view can be poor, so generally a size 4 Macintosh is used from the start. If the practitioner is practised with alternative blades (e.g. McCoy blade or straight blade), these may be tried.

In order to increase the likelihood of successful intubation the following techniques can be used:

- Intubating stylet—used to stiffen and pre-shape the tracheal tube. The stylet should never protrude beyond the distal end of tracheal tube and should be withdrawn after the tip of the tube has passed through the vocal cords. This technique minimizes the risk of damaging the trachea.
- Intubating bougie—may be used routinely or in those with poor views (grade 3 or a difficult grade 2). The bougie is bent at one end (shaped like a hockey stick) to enable it to be passed behind the epiglottis and into the trachea. Confirmation of passage of the bougie into the trachea is provided by the detection of clicks as the bougie slides over the tracheal rings and hold-up of the bougie in the distal airways. Once the bougie is within the trachea, the tracheal tube is railroaded over the bougie with the help of an assistant, who stabilizes the bougie at the top end. The tracheal tube is then advanced into the trachea under direct vision and the bougie removed.

Confirmation of tube placement

Carbon dioxide detection is the gold standard for confirming tracheal tube placement. Ideally this should be by continual waveform monitoring of end tidal CO_2. If this is not immediately available, a disposable colourimetric CO_2 detector can be used.

Additional checks of tube placement include inspection and auscultation of the chest to confirm bilateral air entry.

Once tube position is confirmed, cricoid pressure can be removed and the tube secured. In a patient with raised ICP, adhesive tape is better than a tie to avoid impairing venous drainage and increasing intracranial pressure further. The position of the tube at the teeth should be noted for future reference (usually 22–24 cm for a woman and 24–26 cm for a man). An oropharyngeal airway can be used as a 'bite block'.

3.4.3 Failed airway management

Failure to place a tracheal tube correctly after an RSI is not a disaster, but failure to recognize an incorrectly placed tube, or persistent attempts to secure an airway resulting in patient harm, is. The old adage 'if in doubt, take it out' should be followed. The most important factor is to continue oxygenation of the patient (see Figure 3.5).

If the first attempt at intubation fails, there are a series of fundamental questions that should be asked to determine the most appropriate course of action (Table 3.8):

- Is the patient's arterial blood oxygenated enough to enable further attempts at intubation safely? If not, can it be improved?
- Were the intubating conditions ideal?

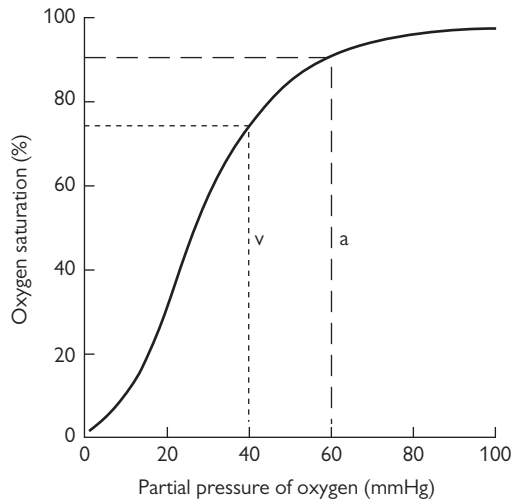

Figure 3.4 The haemoglobin–oxygen dissociation curve. (a) Partial pressure of oxygen of 8 kPa (60 mmHg), which is the definition of arterial hypoxia. (v) partial pressure of oxygen of 5.3 kPa (40 mmHg), which is typical of mixed venous blood. Note that once the PaO_2 falls below 8 kPa, small further falls dramatically decrease the arterial oxygen saturation.