The FCEM Notebook: Revision notes and clinical resource for emergency physicians is the essential aid to passing the Fellowship of the College of Emergency Medicine (FCEM) examination. Containing short but challenging clinical questions and scenarios followed by fully reasoned answers and explanations, the book is brimming with a wealth of clinical information, along with hints and tips. Following the College of Emergency Medicine curriculum and uniquely written for emergency doctors, the clinical cases contain only the most recent evidence-based emergency medicine approaches, allowing for rapid revision and instant access to key information.

- Includes challenging clinical cases for practice
- Provides reasoned answers and explanations
- Consolidates knowledge ready to apply in the examination
- Contains hints and tips to excel in the examination

This portable, practical book is an essential revision guide for all those studying for the MCEM and FCEM examinations and is a valued day-to-day clinical resource for doctors in the emergency department.

About the Author

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THE FCEM NOTEBOOK
Revision notes and clinical resource for emergency physicians
THE FCEM NOTEBOOK

Revision notes and clinical resource for emergency physicians

Joanna S Rowlinson MB BS MRCP FCEM
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Queen Alexandra Hospital, Portsmouth
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To my family
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The FCEM Notebook is a focused, concise, portable emergency medicine revision resource for FCEM exam candidates and emergency physicians. The examinations for Fellowship of the College of Emergency Medicine (FCEM) demand knowledge, retention and application of a huge breadth of complex clinical information. Extensive personal reading based on the college curriculum is an essential foundation for the exams and multiple literature resources need to be identified and accessed. This book contains short questions related to the FCEM curriculum and succinct answers to allow candidates to revise and test their knowledge as time allows.

Notes below the answers also contain additional material, current relevant national guidelines references and suggested resources for further personal reading that have been selected for their particular relevance to emergency medicine. Space is also available for readers to add their own notes.

This book does not intend to be a direct reproduction of previous FCEM exam questions and the format aims to aid in the retention of knowledge. The question style commonly encountered in the exam is not as direct. The candidate is often required to interpret a stem, establish a diagnosis and then answer further questions. For example, rather than asking directly for the features of Kawasaki disease, an unwell child with fever for six days and red eyes will be described, or rather than asking directly regarding cyanide poisoning, an unconscious patient in a factory fire will be described. Should the initial diagnosis reached make proceeding with the subsequent questions difficult, reconsider the overall diagnosis. Both adult and paediatric EM topics are included within each subsection.

This book is also of relevance and interest to emergency physicians post FCEM refreshing, or helping colleagues preparing for the exams, and also emergency nurse practitioners, trainee nurse consultants, MCEM candidates, foundation years doctors and medical students working within the ever interesting, challenging and enjoyable world of emergency medicine.

Joanna Rowlinson
ABBREVIATIONS

ACE  angiotensin converting enzyme
AF   atrial fibrillation
AIDS acquired immunodeficiency syndrome
ALS  advanced life support
ALSG Advanced Life Support Group
ANS  autonomic nervous system
ASOT antistreptolysin O titer
BMA  British Medical Association
BMI  body mass index
BNF  British National Formulary
BP   blood pressure
bpm  beats per minute
BTS  British Thoracic Society
CCU  coronary care unit
CEM  College of Emergency Medicine
CK   creatinine kinase
CMV  cytomegalovirus
CNS  central nervous system
CO₂  carbon dioxide
COPD chronic obstructive pulmonary disease
CPR  cardiopulmonary resuscitation
CRP  C-reactive protein
CT   computerized tomography scan
CVA  cerebrovascular accident
CVS  cardiovascular system
CXR  chest X-ray
DIC  disseminated intravascular coagulation
DKA  diabetic ketoacidosis
DVLA Driver and Vehicle Licensing Agency
EBV  Epstein–Barr virus
ECG  electrocardiogram
ED   emergency department
ENT  ear, nose and throat
ESR  erythrocyte sedimentation rate
<table>
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<td>ET</td>
<td>endotracheal</td>
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<tr>
<td>ETA</td>
<td>estimated time of arrival</td>
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<td>FBC</td>
<td>full blood count</td>
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<td>FFP</td>
<td>fresh frozen plasma</td>
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<td>FVC</td>
<td>forced vital capacity</td>
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<td>GCS</td>
<td>Glasgow coma scale</td>
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<td>GI</td>
<td>gastrointestinal</td>
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<td>GMC</td>
<td>General Medical Council</td>
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<td>GP</td>
<td>general practitioner</td>
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<td>G&amp;S</td>
<td>group and save</td>
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<td>GTN</td>
<td>glyceral trinitrate</td>
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<td>HDU</td>
<td>high dependency unit</td>
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<td>HIV</td>
<td>human immunodeficiency virus</td>
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<td>HONK</td>
<td>hyperosmolar non-ketotic coma</td>
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<td>HPA</td>
<td>Health Protection Agency</td>
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<td>Hrs</td>
<td>hours</td>
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<td>HSP</td>
<td>Henoch-Schönlein purpura</td>
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<td>HUS</td>
<td>haemolytic uraemic syndrome</td>
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<td>ICD</td>
<td>implantable cardiac defibrillator</td>
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<td>ITP</td>
<td>idiopathic thrombocytopenia purpura</td>
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<td>ITU</td>
<td>intensive therapy unit</td>
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<td>IV</td>
<td>intravenous</td>
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<td>LBBB</td>
<td>left bundle branch block</td>
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<td>LFT</td>
<td>liver function tests</td>
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<td>LP</td>
<td>lumbar puncture</td>
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<td>LV</td>
<td>left ventricle</td>
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<tr>
<td>mg</td>
<td>milligram</td>
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<td>mg/kg</td>
<td>milligrams per kilogram</td>
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<tr>
<td>MI</td>
<td>myocardial infarction</td>
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<tr>
<td>Mins</td>
<td>minutes</td>
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<td>mL</td>
<td>millilitre</td>
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<tr>
<td>MRI</td>
<td>magnetic resonance imaging</td>
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<td>NG</td>
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<td>NICE</td>
<td>National Institute of Health and Clinical Excellence</td>
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<td>NSAIDs</td>
<td>non-steroidal anti-inflammatory drugs</td>
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<td>OPD</td>
<td>outpatient department</td>
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<td>OSCE</td>
<td>objective structured clinical examination</td>
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<td>PE</td>
<td>pulmonary embolus</td>
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<td>PEFR</td>
<td>peak expiratory flow rate</td>
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<td>PICU</td>
<td>paediatric intensive care unit</td>
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PIPJ  proximal interphalangeal joint
PPE  personal protective equipment
PR  rectal examination
PTH  parathyroid hormone
RTC  road traffic collision
SIADH  syndrome of inappropriate antidiuretic hormone
SIGN  Scottish Intercollegiate Guidelines Network
SLE  systemic lupus erythematos
TB  tuberculosis
TTP  thrombotic thrombocytopenic purpura
U&E  urea and electrolytes
UK  United Kingdom
UTI  urinary tract infection
UV  ultraviolet
VF  ventricular fibrillation
VT  ventricular tachycardia
VTE  venous thromboembolism
XR  X-ray
VENTRICULAR TACHYCARDIAS (VT)
VT is defined as a ventricular rhythm of three or more beats at a rate of over 120 bpm.
Can be sustained or non-sustained.

ECG features seen in VT
- Capture beats – amidst the AV dissociation an atrial impulse is chance conducted through to the ventricles and produces a narrow QRS followed by an upright T wave (the narrow complex seen briefly thus therefore excludes an SVT with LBBB)
- Fusion beats – simultaneous impulses from the atria and ventricles coincide to create a combination complex with a QRS wider than a supraventricular complex but narrower than a ventricular complex
- AV dissociation – p-waves seen within ECG not related to QRS complexes (also seen clinically as cannon waves)
- Concordance of all complexes
What are the associated stigmata of infective endocarditis?

**ASSOCIATED STIGMATA OF INFECTIVE ENDOCARDITIS**

- Osler’s nodes (painful raised red lesions on hands and feet)
- Janeway lesions (small painless flat red lesions on hands and feet)
- Roth’s spots (white-centred retinal haemorrhages)
- Subungual ‘splinter’ haemorrhages
- Petechiae
- Haematuria
- Clubbing (now very rare)

**READING**


How is the QT interval calculated?
List the causes of a long QT

LONG QT

QT interval is from the beginning of the Q-wave to the end of the T-wave and represents the duration of activation and recovery of the ventricular myocardium.
QT interval is dependent on heart rate, therefore formula is used to calculate the QTc (heart rate corrected QT interval).

$$\text{QTc} = \frac{\text{QT length}}{\sqrt{\text{RR interval (in seconds)}}}.$$  
QTc longer than 0.44 seconds are generally considered abnormal (can be up to 0.46 sec in females).

Causes of prolonged QT interval

Inherited long QT conditions (including Jervell and Lange-Nielsen syndrome and Romano-Ward syndrome)
Drug induced (including erythromycin, tricyclic antidepressants, sotalol, amiodarone)
Hypothyroidism
Hypokalemia
Hypomagnesemia
Hypothermia
Myocarditis
Subarachnoid haemorrhage

READING

List the causes of ST elevation

**ST ELEVATION**

- Acute pericarditis
- Aortic dissection
- Benign early repolarization
- Cardiac contusion
- Hyperkalaemia (later)
- LBBB
- Myocardial infarction
- Normal variant
- Paced complexes
- Aneurysmal LV
- Brugada syndrome
- Coronary artery spasm
- Hypertrophic cardiomyopathy
- LV hypertrophy
- Myocarditis
- Pulmonary embolus
- Raised intracranial pressure
- Subarachnoid haemorrhage

**READING**


List the causes of a low-voltage ECG

LOW-VOLTAGE ECG COMPLEXES

**Definition**
QRS amplitude <5 mm in all limb leads and/or
QRS amplitude <10 mm in all chest leads

**Causes**
- Cardiomyopathy (end stage)
- Constrictive pericarditis
- COPD (severe)
- Hypothyroidism
- Myocardial infiltration (e.g. amyloid, sarcoid)
- Obesity
- Pericardial effusion
- Pleural effusion
- Pneumothorax
- Severe global ischaemic heart disease
- Subcutaneous emphysema
  (pseudo – gain incorrectly set)

**NOTES:**
- Electrical alternans
  - Alternating normal and low-voltage ECG complexes
  - Most commonly seen with pericardial effusion

**CAUSES OF PERICARDIAL EFFUSIONS**

- Aneurysm rupture
- Chemotherapy
- Lupus
- Nephrotic syndrome
- Radiotherapy
- Severe hypothyroidism
- Tuberculosis
- Viral pericarditis (including CMV, HIV)
- Congestive heart failure
- Dressler’s syndrome
- Malignancy
- Post cardiac surgery
- Rheumatoid arthritis
- Trauma
- Uraemia
List eight causes of sinus bradycardia

**CAUSES OF SINUS BRADYCARDIA**

- Acute MI (especially inferior)
- Drugs
- Hypothyroidism
- Raised intracranial pressure (Cushing syndrome)
- Sino-atrial disease
- Sleep apnoea

- Athletes
- Hypothermia
- Increased vagal tone
- Pain
- Sleep
- Typhoid

**NOTES:**

Two double OSCE stations focus on a resuscitation scenario. Resuscitation skills may also feature in other areas of the exam. Ensure in-depth, fluent knowledge of current guidelines. Practice scenarios with your hospital’s resuscitation training officers.

**READING**

- http://www.resus.org.uk
- http://www.alsg.org.uk
A well patient with no arrhythmias is experiencing multiple ICD shocks. What is your management?

What does a ‘bleeping’ ICD indicate?

An ICD patient has received no device shocks but is in VT with hypotension. What is your management?

**IMPLANTABLE CARDIOVERTER DEFIBRILLATORS (ICDs)**

*Multiple shocks with no arrhythmia:*
  - Presumed sensing errors
  - Deactivate by placing ring magnet over ICD
  - Urgent cardiology review, continuous cardiac monitoring and admission to CCU required
  - Device requires urgent interrogation

*Patient with ICD experiencing persistent arrhythmias with cardiac compromise:*
  - Presumed ICD failure
  - Manage as per normal ALS algorithms
  - Carry out CPR as normal
  - Externally defibrillate as normal
  - Administer ALS drug as normal
  - Device requires urgent interrogation

*One or two shocks received, now well, no acute ECG abnormalities and asymptomatic:*
  - Presumed appropriate shock
  - Inform patient’s cardiologist to arrange rapid access OPD review and discharge home

*Unwell patient following shock or more than two shocks received:*
  - Possible new onset of increased frequency of arrhythmia or new onset of illness (myocardial ischaemia, electrolyte abnormalities) or coexisting drug treatment no longer therapeutic
  - Full clinical assessment
  - Urgent cardiology review, continuous cardiac monitoring and admission to CCU required

**ICD bleeping**
  - Device warning mechanism
  - Possible imminent device failure, e.g. lead failure or battery failure
Urgent cardiology review, continuous cardiac monitoring and admission to CCU required
Device requires urgent interrogation

NOTES:

Patients carry an ICD identification card. Placing a ring magnet over an ICD should not also turn off a pacing function. Avoid external defibrillations directly over ICDs or magnets but do not delay/withhold treatment over concerns of damaging the ICD. CPR can be safely performed with an ICD in situ. Any shocks delivered by the ICD would not be harmful to the person carrying out cardiac compressions. Remember significant psychological sequelae have been recognized in patients following ICD shocks.

READING


What are the ECG findings in hypokalaemia?
Give six causes of hypokalaemia

HYPOKALAEMIA

ECG changes
- Flat or inverted T-waves
- ST depression
- VT/VF/torsades

Causes
- Diuretics
- Intestinal fistulae
- Liquorice
- Insulin treatment

Symptoms
- Lethargy, weakness, constipation, paralysis, tetany, paraesthesia

NOTES:

Hypercalcaemia ECG findings
- Short QT
- J-waves
- Broad T-waves
- VT/VF when severe
What are the diagnostic features of Kawasaki disease?

KAWASAKI DISEASE

Fever for more than five days  Sudden onset, swinging, above 40°C, poor response to antipyretics or antibiotics

With four of the following five:

Lymphadenopathy  Painful, solitary, greater than 1.5 cm
Oral changes  Cracked lips, erythema of lips, strawberry tongue
Rash  Starts in a few days for around 1 week
Often marked in groin and may peel
Many forms but not vesicular

Conjunctival injections  Bilateral, non-purulent
Extremity change  Oedema, erythema, periungal desquamation, usually 2–3 weeks post onset

(If proven new coronary artery involvement, only three of above needed.)

Following diagnosis, patients are treated with immunoglobulin and aspirin.
HENOCHE SCEONLEIN PURPURA (HSP)

Widespread small vessel vasculitic process with onset commonly following minor bacterial or viral infection. Most common in 3 to 11 yrs old. Child generally appears well.

Classical findings of:

- **Palpable purpura (100%)** Traditionally extensor surfaces and buttocks, but can affect other areas
- **GI symptoms (75%)** Abdominal pain, intussusception, vomiting, haematemesis, PR bleed
- **Arthralgia (up to 60%)** Especially large joints
- **Renal (25–60%)** Micro/macroscopic haematuria and proteinuria
- **Other associations** Testicular pain/swelling/torsion
  - Oedematous hands and feet and periorbital
  - Seizures, encephalopathy, intracranial haemorrhage/infarct

**Admit if**

- Systemically unwell
- Renal failure
- Hypertensive
- Unclear diagnosis
- Increasing proteinuria or haematuria
- Poor pain control/unable to mobilize due to arthralgia

**Investigations**

Clinical diagnosis, investigate to identify complications and exclude other differential diagnoses.

- **Throat swab** Urine dipstick with MC&S
- **FBC, coagulation** Protein, albumin
- **Creatinine** U&E
- **Calcium** Blood cultures
- **Blood pressure** Weight and height
- Consider abdominal USS for intussusception

**Management**

Supportive; most have a good prognosis.

Analgesia and rest – paracetamol, NSAIDs (if no renal involvement).
Monitor hydration and nutrition. Carers can manage at home and with OPD with written advice plan and symptom/urine dip diary if no concerning features. Prednisolone – can be used if progressing renal involvement/oedema, significant GI features/arthralgia or onset of other significant complications. Discuss with duty paediatrician before starting in the ED.
LYME DISEASE
Tick-borne infection caused by *Borrelia burgdorferi*. Most common in southern England in the UK, but also in travellers to Europe and America.

**Presentation**
Three stages

*Stage 1 – Early localized infection*
Erythema chronicum migrans rash, flu-like symptoms

*Stage 2 – Early disseminated infection (days to weeks)*
Cardiac  First degree/complete heart block, myopericarditis
Neurological  Neuroborreliosis, cranial nerve palsies especially unilateral or bilateral 7th, meningitis, encephalitis, lymphocytic meningo radiculoneuritis (Bannwarth syndrome), peripheral mononeuritis
Arthralgia/muscle pain
Ophthalmology  Conjunctivitis, optic neuropathy

*Stage 3 – Late persistent infection*
Lyme arthritis, encephalomyelitis, acrodermatitis chronica atrophicans (blue-red rash)

**Investigations and management**
Discuss with dermatologist, infectious disease specialist or immunologist. Can be diagnosed clinically if history of tick bite and erythema chronicum migrans is present. Alternatively, send serology for *B. burgdorferi* antibodies.
Early localized infection is treated with 14 days oral doxycycline (or oral cefuroxime if pregnant, children under 12, or breastfeeding). Discuss if other manifestations present.

**TICK REMOVAL**
Use fine-toothed tweezers to gently pull tick as close to the place of attachment without twisting or crushing. Clean skin with soap, water and antiseptic, and then wash hands. Do not use lighted cigarette ends, match heads, creams, nail varnish or oils. Advise patient to seek medical advice if redness, rash, flu like symptoms
or neurological symptoms develop. Antibiotic prophylaxis following a tick bite is not currently recommended in the UK (discuss if immunocompromised).

**Erythema chronicum migrans**
Large, painless, circular red, pink or purple rash radiating from tick bite, commonly with central sparing, and well-demarcated edges.

**READING**
Lyme disease guidelines at Health Protection Agency website: http://www.hpa.org.uk
What descriptors are used in dermatology?

Give five differential diagnoses of a bullous rash

Give the associated causes of Stevens Johnson

**DERMATOLOGY DESCRIPTORS**

- **Macule**: Flat, nonpalpable lesion <10 mm diameter
- **Papule**: Elevated, palpable lesion <10 mm diameter
- **Plaque**: Palpable lesion elevated or depressed to skin surface >10 mm diameter
- **Vesicles**: Raised, clear, fluid-filled blister <10 mm diameter
- **Bullae**: Raised, clear fluid-filled blister >10 mm diameter
- **Pustule**: Vesicle containing pus
- **Telangiectasia**: Foci of small, permanently dilated superficial blood vessels
- **Nikolsky sign**: Skin separates when lightly rubbed
- **Köbner phenomenon**: Lesions arise in area of previous skin trauma

**BULLOUS RASH CAUSES**

- **Contact dermatitis**: Dermatitis herpatiformis
- **Drugs, e.g. barbiturates**: Erythema multiforme
- **Friction blisters/burns**: Herpes zoster/shingles
- **Impetigo**: Insect bite
- **Pemphigoid**: Porphyria
- **Staphylococcal scaled skin syndrome**: Toxic epidermal necrolysis

**CAUSES OF STEVENS JOHNSON SYNDROME**

- **Infections**: herpes virus, EBV, mycoplasma pneumoniae, streptococcus
- **Drugs**: NSAIDs, sulphamides, salicylates, penicillins, barbiturates
Give the causes of erythema nodosum

ERYTHEMA NODOSUM
Raised, red, painful lesions (2–8 cm) on shins (sometimes on arms and thighs), often symmetrical. Colour will evolve similar to a bruise over 6–8 weeks.

Causes include
- Behçet disease
- Drugs (barbiturates, codeine, oral contraceptives, penicillin, salicylates, sulphonamides)
- EBV
- Leukaemia
- Mycoplasma
- Rheumatic fever
- Streptococcus
- Tuberculosis
- Crohn disease
- Idiopathic
- Lymphoma
- Pregnancy
- Sarcoidosis
- Syphilis
- Ulcerative colitis

Treat symptomatically with NSAIDs, rest and elevation and investigate for underlying cause (CXR, ESR, CRP, ASOT, throat swab, pregnancy test, FBC and other as per history/examination findings).
ENDOCRINOLOGY
What are biochemical findings in, and precipitants of, an acute Addisonian crisis?

ADDISONIAN CRISIS

**Biochemical findings**

- Hypoglycaemia
- Hyponatraemia
- Hyperkalaemia
- Hypercalcaemia
- Raised eosinophils
- Increased urea
- Low pH

**Precipitants of acute Addisonian crisis**

- Adrenal haemorrhage
- Severe coagulopathy
- Sepsis (Waterhouse-Friderichsen syndrome)
- Abrupt withdrawal of steroid treatment
- Surgery
- Dehydration
- Trauma
- Infection
- Pregnancy
- Burns
- General anaesthetic
- Omission of steroid treatment
- Vomiting
- Non-compliance
- Iatrogenic

**NOTES:**

May co-exist with other endocrinopathies. Addison disease hyperpigmentation can be seen in skin creases, scars, gums and buccal mucosa, skin folds, extensor surfaces.

**READING**

Wass JAH. How to avoid precipitating an acute adrenal crisis. BMJ. 2013;345:e6333.
### ELECTROLYTE ABNORMALITIES CAUSES

#### Hyperchloraemia
- diarrhoea
- acetazolamide
- IV sodium chloride
- renal tubular acidosis

#### Hypochloraemia
- vomiting
- diuretics
- NG suction
- diarrhoea

#### Hypernatraemia
- burns
- vomiting, NG suction
- IV hypertonic saline
- excessive salt ingestion
- nephrogenic diabetes insipidus
- Cushing syndrome
- HONK
- excessive sweating (marathons, heat illness)
- GI fistulae
- sodium bicarbonate administration
- central diabetes insipidus
- Conn syndrome

#### Hyponatraemia
- Hypervolaemic
  - cardiac failure
  - nephrotic syndrome
  - renal failure
  - liver failure
  - inappropriate intravenous therapy

- Normovolaemic
  - SIADH
  - hypothyroidism
  - psychogenic polydipsia and other causes of water intoxication
  - glucocorticoid deficiency
  - excess intravenous hypotonic fluids

- Hypovolaemic
  - diuretic therapy
  - cerebral salt wasting
  - (subarachnoid haemorrhage, head injury)
  - diarrhoea
  - burns
  - pancreatitis
  - salt-losing nephropathy
  - Addison disease
  - vomiting
  - sweating
  - seen with coexisting raised glucose, protein, lipids and mannitol
Give six possible differential diagnoses of hypoglycaemia in an adult

HYPOGLYCAEMIA IN ADULTS

Use the mnemonic EXPLAIN
- Exogenous drugs (alcohol, insulin)
- Pituitary insufficiency
- Liver failure
- Addison disease
- Insulinomas (islet cell tumours), infection (malaria, sepsis)
- Non-pancreatic tumours (as some release insulin like peptides)

NOTES:
Metformin and glitazones do not usually cause hypoglycaemia. Sulphonylureas can cause hypoglycaemia. Inherited metabolic diseases can present for the first time in adulthood with hypoglycaemia. Inherited metabolic diseases should be considered in patients presenting with hypoglycaemia, metabolic acidosis and high measured ammonia. Emergency treatment regimes for adults and children are available at http://www.bimdg.org.uk and also discuss with an expert.

READING
The hospital management of hypoglycaemia in adults with diabetes mellitus.
What are the features of cerebral oedema with DKA in children?

CEREBRAL OEDEMA IN PAEDIATRIC DKA

Features

- Headache, irritability, restless, agitated, incontinence, slowing pulse, rising BP
- Reducing GCS, focal neurological signs, abnormal posturing
- Papilloedema and convulsions and respiratory arrest (late signs)

Initial approach

- Exclude hypoglycaemia
- Call for senior help (consultant paediatrician, PICU)
- Mannitol 1g/kg (5 mL/kg Mannitol 20% over 20 mins) or 3% hypertonic saline (3–5 mL/kg over 20 mins)
- Restrict IV fluids to 2/3 maintenance and replace deficit over 72 rather than 48 hrs
- Once stable CT head (to exclude other causes – haemorrhage, infarct, thrombosis)

NOTES:

- When Managing DKA treat shock with 5–10 mL/kg 0.9% saline boluses (up to max 30 mL/kg – discuss with consultant if felt to need more). The degree of initial dehydration is commonly overestimated. Do not correct for over 10% estimated dehydration. Do not include urine volume losses in fluid replacement calculations. Fluid rehydration should be delivered evenly over 48 hours.
- Antibiotics are not given routinely. Fever is not part of DKA, but DKA can be precipitated by sepsis.
- One hour after starting IV fluids start continuous insulin infusion 0.05–0.1 units/kg/hr (dose depending on local consensus). Initial insulin boluses or insulin sliding scales are not used.
- Children with DKA die from cerebral oedema, hypokalaemia and aspiration pneumonia.

READING

ALTITUDE MEDICINE

Features of acute mountain sickness (AMS)
Recent ascent over 2500 m and headache with any one or more:
- GI upset (nausea, vomiting, reduced appetite)
- insomnia
- fatigue or weakness
- dizziness or lightheadedness
Symptoms scored 0–3 depending on severity, higher score = more severe (moderate = score 4).
Treatment – descend or rest. Ibuprofen for headache. Acetazolamide. Only re-ascend when completely asymptomatic.

Features of high altitude cerebral edema (HACE)
Recent gain in altitude and either:
- change in mental state/conscious level and or ataxia in climber with AMS, or
- present of both mental state change and ataxia in climber without AMS
Treatment – immediate descent. If unable (e.g. weather) temporize with oxygen, hyperbaric bag, dexamethasone.

Features of high altitude pulmonary edema (HAPE)
Occurs with or without AMS.
Diagnosis:
- Symptoms (at least two): breathless at rest, cough (dry or pink frothy sputum), weakness or decreased exercise tolerance, chest tightness (grade mild/moderate/severe)
- Signs (at least two): crackles or wheeze, central cyanosis, tachypnoea, tachycardia
Treat – immediate descent, Nifedipine, oxygen, hyperbaric bag

NOTES:
Ref - See Lake Louise Consensus on the Definition and Quantification of Altitude Illness. Acetazolamide acts as respiratory stimulant to improve oxygenation. Side effects of paraesthesia, change in taste, tinnitus and rarely blurred vision.
What information is initially reported from a major incident scene?

MAJOR INCIDENT

Initial report
Two formats are recognized: METHANE and CHALETS

<table>
<thead>
<tr>
<th>METHANE</th>
<th>CHALETS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Major incident declared/standby</td>
<td>Casualties, number, type and severity</td>
</tr>
<tr>
<td>Exact location</td>
<td>Hazards present and potential</td>
</tr>
<tr>
<td>Type of incident</td>
<td>Access routes</td>
</tr>
<tr>
<td>Hazards at scene, present and potential</td>
<td>Location</td>
</tr>
<tr>
<td>Access routes</td>
<td>Emergency services present and required</td>
</tr>
<tr>
<td>Number of casualties, type and severity</td>
<td>Type of incident</td>
</tr>
<tr>
<td>Emergency services present and required</td>
<td>Safety</td>
</tr>
</tbody>
</table>

Initial approach at major incident scene
CSCATT
  - Command and control
  - Safety
  - Communication
  - Assessment
  - Triage
  - Treatment
  - Transport

NOTES:
Patients will be triaged by medical priority to leave scene. Discriminators used for triage sieve are whether able to walk, respiration rate and heart rate.

READING
Course manuals for the ALSG courses MIMMS (major incident medical management and support) and HMIMMS (hospital MIMMS). http://www.alsg.org
What are the clinical features of heat stroke?

HEAT ILLNESS SPECTRUM AND INITIAL TREATMENT APPROACH

**Heat rash**
Inflamed blocked sweat gland causing limb and trunk itching and red rash. Treatment – dry skin, remove tight clothes and cool. Flucloxacillin for secondary infection.

**Heat oedema**
Temporary swelling of hands, ankles and feet. Treatment – oral rehydration and shelter.

**Heat cramps**
Painful limb spasms due to electrolyte depletion. Treatment – rest, stretch, oral rehydration solutions.

**Heat syncope**
Venous pooling with vasodilatation. Treatment – rest, shelter, oral rehydration solutions.

**Heat exhaustion**
Volume and electrolyte depletion. Headache, lethargy, nausea, vomiting, light headed, dehydration, sweating, tachycardia, tachypnoea, postural hypotension, hypotension, temperature under 40°C. Treatment – rest, shelter, simple cooling, oral or IV fluid and electrolyte replacement. Admit with close observation. Recovery over 18–24 hours.

**Heat stroke**
Failure of thermoregulation. Temperature >41°C, headache, CNS dysfunction (seizures, reduced GCS, muscle rigidity, ataxia), lack of sweating, tachycardia, hypotension, coagulopathy. Treatment – resuscitate, rapid cooling to <39°C (cool IV fluids, icepacks to groin, axillae, neck, scalp), sponge with tepid water and fan, may require cooling with peritoneal and gastric lavage or bypass, lorazepam for seizures, catheterize and monitor urine output. ICU admission. Mortality 10% (multi-organ failure).
**NOTES:**
Avoid paracetamol as antipyretic. Patients with significant heat stroke may still sweat. Significant heat illness can occur in not excessively hot environments.

**Biochemical abnormalities in heat stroke**
- Acute renal failure: Hyperkalaemia
- Metabolic acidosis: Hypocalcaemia
- Raised liver transaminases: Respiratory alkalosis
- Raised CK: Myoglobinuria
- DIC: Hyperglycaemia
What is the differential diagnosis of hyperpyrexia?

DIFFERENTIAL DIAGNOSIS OF HYPERPYREXIA

- Amphetamines
- CVA
- Cocaine
- Ecstasy
- Heat stroke
- Intracranial haemorrhage
- Malaria
- Malignant hyperthermia
- Neuroleptic malignant syndrome
- Sepsis
- Serotonin syndrome
- Thyroid storm
ATMIST mnemonic
A handover tool used by the ambulance service for both trauma and medical presentations.

- Age
- Time of incident/time onset of symptoms
- Mechanism of injury/medical complaint
- Injuries/examination findings
- Vital Signs (pulse rate, BP, saturations, respiration rate, GCS, temperature, BM)
- Treatment given
- ETA and mode of transport
MEDICAL CAUSES OF ABDOMINAL PAIN

Addison’s disease DKA
Henoch Scohlein purpura Hereditary angioedema
Hypercalcaemia Lead poisoning
Lower lobe pneumonia Mesenteric adenitis
Myocardial infarction Opiate withdrawal
Porphyria Sickle cell crisis
Typhoid UTI

ALVARADO SCORE

A clinical scoring system used to aid the diagnosis of appendicitis in adults (max 10 points).

Abdominal pain that migrates to the right iliac fossa (1)
Anorexia (1)
Pain on pressure in the right iliac fossa (2)
Nausea or vomiting (1)
Rebound tenderness (1)
Fever (1)
Leukocytosis (2)
Neutrophilia with left shift (1)

Score

<5 appendicitis unlikely
5–6 possible appendicitis
7–8 probably appendicitis
9–10 very probable acute appendicitis

What are the biochemical findings associated with pyloric stenosis?

PYLORIC STENOSIS
Hypertrophy of the pylorus results in gastric outlet obstruction.

Biochemistry
Hypochloraemic, metabolic alkalosis, hypokalaemic

Presentation
Most commonly between three and six weeks of age
Non-bilious vomiting which progressively worsens and becomes projectile
Initially very hungry then poor feeding and lethargy
Dehydration
Poor weight gain
Visible peristalsis
Palpable hypertrophied pylorus below right costal margin (‘palpable olive’)
On occasion haematemesis

Investigations
FBC, UEs, blood gas, glucose, split bilirubin, abdominal ultrasound scan

Management
Assess dehydration
Fluid resuscitation boluses 10–20 mL/kg 0.9% normal saline, then maintenance fluids
Correct biochemical abnormalities including potassium replacement
Nil by mouth, NG tube
Theatre for pyloromyotomy (fluid resuscitation is overall priority)

READING
List the causes of a metabolic alkalosis and the causes of a respiratory alkalosis

**METABOLIC ALKALOSIS CAUSES**
- Alkaline diuresis for treatment of salicylate poisoning
- Alkali ingestion
- Hyperaldosteronism
- Massive blood transfusion
- Milk-alkali syndrome
- Pyloric stenosis, vomiting, NG suction
- Sodium bicarbonate ingestion
- And other very rare conditions

**RESPIRATORY ALKALOSIS CAUSES**
- Anxiety
- Brain stem lesions
- Hypoxia
- Iatrogenic overventilation
- Liver failure
- Pain
- Psychological
- Salicylate overdose
What is the differential of jaundice in pregnancy?

JAUNDICE IN PREGNANCY

Pregnancy-related differentials

- HELLP
- Hyperemesis gravidarum
- Cholestasis of pregnancy
- Acute fatty liver of pregnancy

Also consider non-pregnancy-related causes including

- Drug-induced hepatitis
- Viral hepatitis
- Gallstone disease
- Autoimmune hepatitis
- Wilson disease
- Gilbert syndrome
GRADING OF HEPATIC ENCEPHALOPATHY

Grade I  Mild behavioural changes and mild reduced level of consciousness
Grade II  Drowsy but rousable, mild confusion, marked behaviour changes
Grade III Marked confusion, intermittent agitation, very drowsy
Grade IV  Unresponsive or only responsive to pain
What are the causes of priapism and the treatment approach?

PRIAPISM

Causes

High flow (non-ischaemic)
  Increased arterial blood flow, most commonly from local trauma, not usually painful
  Refer directly to urology

Low flow (ischaemic)
  Reduced venous drainage, painful

Associated causes
  Sickle cell disease
  Cervical spine trauma
  Drugs (e.g. calcium channel blockers, chlorpromazine, cocaine, marijuana, anti-impotence treatment overuse, anticoagulants)
  Malignancy

Investigations

FBC, blood film, clotting profile, sickle screen
Penile Doppler (to help differentiate between high and low flow priapism if unable with history)
ABG analysis of aspirated cavernosal blood (pH <7.25 suggestive of ischaemic low flow priapism)

Treatment options (low flow)

Ice, gentle walking, opiate analgesia.
Oral terbutaline.
Using local anaesthetic aspirate 20–30 mL cavernosal blood from 10 o’clock or 2 o’clock position.
If not resolved inject phenylephrine following aspiration, liaise with urology for surgical interventions.
Apply dressing to prevent later haematoma.
Simultaneously treat co-existing medical precipitating causes.
Give six causes of bowel obstruction in neonates

NEONATAL BOWEL OBSTRUCTION

*Differential diagnosis includes*

- Colonic atresia
- Congenital diaphragmatic hernia
- Duodenal atresia
- Hirschsprung disease
- Intussusception
- Imperforate anus
- Large or small bowel atresia
- Malrotation
- Meconium ileus
- Meconium plug
- Necrotizing enterocolitis
- Volvulus
- Paralytic ileus associated with sepsis/electrolyte abnormalities
- Pyloric stenosis

*Abdominal XR findings*

- Can be normal despite significant pathology
- Dilated bowel
- Perforation
- Malrotation of gas shadows
- Relatively gasless
- ‘Double bubble’ (seen in duodenal atresia, as air in stomach and proximal duodenum separated by pyloric sphincter)
- Target sign/crescent sign (seen in intussusception as two concentric lines or crescent shaped lucency with a soft tissue mass)

*NOTES:*

Neonatal bilious vomiting or failure to pass meconium in first 24 hours of birth require prompt referral to paediatricians and paediatric surgeons for further investigation. Bilious vomit is green. 98–99% of term neonates should pass meconium within 24 hours of birth and all by 48 hours (can be longer in preterm infants).

Patients with significant pathology can present non-specifically with mild vomiting or with severe shock, sepsis or ischaemic bowel. Depending on location of obstruction and stage of presentation abdominal signs can be very subtle. Examine for features of shock/sepsis, reduced bowel signs, pain/guarding, abdominal wall erythema, abdominal distension, abdominal fullness, PR blood/patency, vomiting with or without bile (ask to view vomit on clothing). Also
consider and examine for other co-existing syndromic abnormalities (including cardiac and dysmorphic features [e.g. Down syndrome]). May give history of maternal polyhydramnios.

**READING**

HAEMATOLOGY
What are the precipitants of an acute painful sickle crisis? List the types of sickle cell crisis

**SICKLE CELL CRISIS**

**Precipitants**
- Acidosis
- Alcohol intoxication
- Dehydration
- Hypoxia
- Pregnancy
- Cold weather
- Emotional stress
- Infection
- Sedative drugs

**Types of sickle cell crisis**
- **Painful crisis**: Acute bone pain/swelling due to bone infarction
- **Chest crisis**: Pain, fever, cough, tachypnoea, pulmonary infarction, hypoxia
- **Priapism**
- **Cerebral crisis**: Focal neurology, seizures, haemorrhagic or ischaemic stroke, TIAs
- **Sequestration**: Sudden splenic enlargement (most common in children, liver sequestration can occur in adults)
- **Haemolytic crisis**
- **Aplastic crisis**: Resulting in severe anaemia, often following parvovirus B19 infection
- **Severe sepsis**: Risk due to reduced splenic function (even when taking prophylaxis)

**Management approach**
- **Analgesia**: Pain score and offer analgesia within 30 minutes to be painfree within 60 minutes, offer IV opiates (avoid pethadine and entonox) with appropriate antiemetics and laxatives
- **Hydration**: With IV fluids and monitor fluid balance
- **Oxygen**: Apply if new hypoxia or saturations <95%
- **Antibiotics**: Treat underlying infections identified
- **Transfusions**: Discuss with haematologist before considering any transfusions
- **Monitor**: BP, oxygen saturations, heart rate, respiratory rate, temperature
- **Investigations**: FBC, blood film, reticulocytes, U&E, LFT, CRP, group and save, MSU
CXR and arterial blood gas if new hypoxia or sats <92%
or chest signs or symptoms
Other radiology imaging as per clinical findings
Give the acute complications of a blood transfusion

TRANSFUSION COMPLICATIONS

Acute complications of blood transfusion

- Acute haemolytic transfusion reaction due to infusion of ABO-incompatible blood
- Infusion of blood contaminated with bacteria
- Anaphylaxis
- Transfusion-related acute lung injury
- Transfusion associated circulatory overload
- Febrile non-haemolytic transfusion reactions
- Urticarial rash
- Itching
- Hypothermia

ED management of acute transfusion reaction

Stop transfusion.
Assess respiratory rate, oxygen saturation, heart rate, blood pressure, temperature.
Assess for features of anaphylaxis, respiratory distress, cardiac and circulatory failure, urticaria and bleeding.
Move to resuscitation area and give oxygen.
Check patient identification label and compare with the details on the blood product being administered and compatibility label.
Give IV saline (inotropes may be required), catheterize and maintain urine output.
Contact ITU and haematology consultant for immediate advice and support.
Treat DIC, allergic/anaphylactic reactions or fluid overload with the usual therapies.
Inform hospital transfusion department and return blood unit and giving set containing remaining products. Do not use other blood products issued and also return to blood bank.
Take blood for repeat group and crossmatch, clotting profile, renal function, arterial blood gas and save serum. Dip and send urinalysis.
Consider and assess for bacterial contamination (blood product pack discoloration or abnormal smell). Take blood cultures from pack. If bacterial contamination suspected, start broad spectrum antibiotics (as per local neutropaenic sepsis guideline).
NOTES:
Symptoms of acute haemolytic transfusion reactions can be non-specific (pyrexia, flushing, urticaria, myalgia/chest/abdominal/bone pain, tachycardia, hypotension, hypertension, sudden collapse, breathlessness, nausea) and recognition is likely to be late in unconscious patients. Transfusion reactions and related adverse events are reported to MHRA SABRE (serious adverse blood reactions and events) and SHOT (serious hazards of transfusion).

READING
www.gov.uk never events
Give the differential diagnoses of fever with petechiae in a four-year-old

PETECHIAE

Causes of petechiae

Disruptions of vascular integrity  Trauma, tourniquets, coughing/vomiting, vasculitis, vitamin C deficiency

Disruptions of haemostasis  Thrombocytopenia (ITP, bone marrow infiltration, aplastic anaemia, HUS, TTP etc.), anticoagulants

Clotting factor disorders  Haemophilia A, B, von Willebrand disease etc.

Petechiae with fever

Bacteria (e.g. Neisseria meningitides, Strep pneumonia, Haemophilus influenza)

Viral (e.g. influenza, enterovirus)

Associated disease process – leukaemia, HSP, Kawasaki, lupus

Unknown

Vomiting/coughing illness resulting in head/neck petechiae (SVC distribution)

NOTES:

Petechiae – pinpoint red/purple marks up to 2 mm appearing under the dermis that do not blanch with pressure

Purpura – red/purple marks more than 2 mm appearing under the dermis that do not blanch with pressure

READING


When is antibiotic prophylaxis indicated following meningococcal meningitis contact?

ANTIBIOTIC PROPHYLAXIS FOLLOWING MENINGOCOCCAL MENINGITIS CONTACT

Chemoprophylaxis indicated
Prolonged close contact with the case in a household type setting during the seven days before onset of illness (e.g. pupils in the same dormitory, boy/girlfriends, or university students sharing a kitchen in a hall of residence)
Transient close contact and directly exposed to large particle droplets/secre-tions from the respiratory tract of a case around the time of admission to hospital (e.g. staff intubating a patient)

Prophylaxis not indicated (unless already identified as close contacts)
Staff and children attending same nursery or crèche
Students/pupils in same school/class/tutor group
Work or school colleagues
Friends
Residents of nursing/residential homes
Kissing on cheek or mouth (would normally bring into close prolonged contact category)
Food or drink sharing or similar low level of salivary contact
Attending the same social function
Travelling in next seat on same plane, train, bus or car
Contact with possible case until investigations make the diagnosis confirmed or probable

Treatment of contacts
Ciprofloxacin one off dose or Rifampacin twice daily for two days, given as soon as possible – following diagnosis of index case
HPA may also advise re vaccinations depending on strains isolated
HPA and public health coordinate contact tracing and treatment

NOTES:
Rifampacin is an enzyme inducer.

READING
Describe your approach to the management following a needlestick injury from a discarded needle in a public toilet

COMMUNITY-ACQUIRED NEEDLESTICK INJURY

Discard any needles brought to ED into yellow sharps bin (do not send to virology)
Clean wound, wash with soap and water, encourage to bleed but do not squeeze
Check tetanus immunisation status (unlikely to be tetanus prone unless sharp contaminated with soil or manure)
Commence accelerated hepatitis B vaccination as per BNF
If considered to be high risk (source is likely to have blood borne virus, visible blood on needle, needle attached to syringe, deep injection causing bleeding) consult HIV on call to consider HIV PEP (post-exposure practice)
Take blood for baseline serum save for HIV, hepatitis B&C. GP to follow up with HIV, hepatitis B&C bloods at three and six months post injury
If appropriate, advise abstinence or barrier contraception until blood testing complete
Reassure negligible risk of HIV and very low risk of contracting hepatitis B or C through community needlestick injuries

NOTES:
HIV patients attending the ED:

- Avoid stopping/delaying HIV medication. Liquid formulae are often available or HIV on-call consultants can advise on potential adaptations to a patient’s regime that can limit the impact, should a temporary gap in treatment appear unavoidable.
- HIV drug interactions can be significant. Refer to the BNF or www.hiv-druginteractions.org.
SEPSIS

SIRS (systemic inflammatory response syndrome) = two or more of:
- Temp >38°C or <36°C
- HR >90
- RR >20 (PaCO₂ <4)
- WCC >12 <4, 10% band formation

Sepsis = SIRS due to infection
Severe sepsis = hypotension and end organ changes – lactic acidosis, oliguria, altered GCS
Septic shock = sepsis and hypotension resistant to fluid resuscitation

EARLY GOAL-DIRECTED THERAPY PROTOCOL

Supplemental oxygen +/- intubation and ventilation
Central venous and arterial catheterization
CVP 8–12 (aggressive fluid boluses)
MAP >65 and <90 (vasopressors or dilators)
Haematocrit >30%, central venous oxygen saturation (ScvO₂) of >70% (red blood cell transfusions and inotropes)

NOTES:
Surviving sepsis campaign care bundles:

Within three hours:
1. Measure lactate level
2. Obtain blood cultures prior to administration of antibiotics
3. Administer broad spectrum antibiotics
4. Administer 30 mL/kg crystalloid for hypotension or lactate ≥4 mmol/L

Within six hours:
5. Apply vasopressors (for hypotension that does not respond to initial fluid resuscitation) to maintain a mean arterial pressure (MAP) ≥65 mm Hg
6. In the event of persistent arterial hypotension despite volume resuscitation (septic shock) or initial lactate ≥4 mmol/L (36 mg/dL):
   • Measure central venous pressure (CVP), target ≥8 mm Hg
   • Measure central venous oxygen saturation (ScvO₂), target ≥70%
7. Remeasure lactate if initial lactate was elevated, target for normalization of lactate
Surviving sepsis campaign: http://www.survivingsepsis.org
State the discharge advice for a well two-year-old presenting with a fever and presumptive diagnosis of a viral URTI

FEVER IN CHILD DISCHARGE ADVICE

Discuss with carer
Seek medical help if child develops any of the following:

- Struggling to breathe
- Pale, mottled or blue colour
- Unable to wake up, drowsy or confused
- Neck stiffness
- Rash that does not disappear with pressure (explain glass test)
- Dark green vomit
- A fit
- High temperature for five days
- Unable to take treatments
- Dehydration (dry mouth, sunken eyes, hasn’t urinated for more than six hours, sunken fontanelle [soft spot on a baby’s head], no tears

General advice

Prevent dehydration
Offer regular drinks (if breast fed, most appropriate fluid is breast milk)

Clothing
Avoid under- or overdressing and adjust clothing if signs of sweating or shivering

Medicines
Not essential to use paracetamol or ibuprofen to treat fever; use if appears distressed (read dose instructions on the bottle)

Sponging
Discourage, as will not reduce fever

Checking
Check during night

School/nursery
Not to attend if still feverish; notify establishment of the illness

If worried

If you think your child is very unwell call 999 for an ambulance.
You can return to your nearest emergency department at any time without an appointment.
You should also be able to see your own GP or an ‘out-of-hours’ doctor.
You can get advice at any time from the NHS telephone 111 service.
Collect and review your local ED’s discharge advice leaflets. Patient advice is common in the exam.

NICE guidelines (CG047), May 2007 and (CG160), May 2013. Feverish illness in children – Assessment and initial management in children younger than five years.
MEDICOLEGAL
When should the coroner be informed following the death of a patient?

INFORMING THE CORONER
After death in the following circumstances (list is not exhaustive):

No doctor has seen the patient within 14 days before death
Death within 24 hours of admission to hospital
Doubt about cause of death for any reason
Identity of deceased is unknown
Cause of the death is unknown
Death was sudden and unexpected
Suspicious death
Violent (homicide, suicide, accidental) or unnatural death
Related to surgery or anaesthetic
In prison or custody
From an industrial disease or occupational disease or accident
In receipt of an industrial or war pension
By suicide, poisoning or drugs
Result of an abortion
From neglect – hospital, care home, family, self, etc.
Related to medications or drugs (including prescriptions and illicit)

READING

Bereavement: http://www.childbereavement.org.uk/Support/Professionals/Reading
and resources


Treatment and care towards the end of life: Good practice in decision making.
What are never events?

NEVER EVENTS
Incidents considered unacceptable and eminently preventable, including the following.

Wrong site surgery
Wrong implant/prosthesis
Retained foreign object post-operation
Wrongly prepared high-risk injectable medication
Maladministration of potassium-containing solutions
Wrong route administration of chemotherapy
Wrong route administration of oral/enteral treatment
Intravenous administration of epidural medication
Maladministration of insulin by health professional resulting in death or severe harm
Overdose of midazolam during conscious sedation following use of high-strength midazolam resulting in death or severe harm
Opioid overdose of an opioid-naïve patient
Inappropriate administration of daily oral methotrexate
Suicide using non-collapsible rails within mental health inpatient premises
Escape of a transferred prisoner from medium or high secure mental health services
Falls from unrestricted windows
Entrapment in bedrails
Transfusion of ABO-incompatible blood components
Transplantation of ABO-incompatible organs as a result of error
Misplaced naso- or orogastric tubes
Wrong gas administered
Failure to monitor and respond to oxygen saturation
Air embolism resulting from intravascular infusion/bolus administration or through haemodialysis circuit
Misidentification of patient, thus administration of the wrong treatment
Severe scalding of patient by water used for washing or bathing
Maternal death due to postpartum haemorrhage after elective caesarean section

READING

What is your understanding of controlled drugs?

CONTROLLED DRUGS (CDs)
The legal requirements and responsibilities regarding the use and handling CDs in the UK include the following.

The Misuse of Drugs Act 1971 controls drugs that are dangerous or harmful. It uses a three-tier system of classification (Classes A, B and C) that correlate with the criminal penalties that will result following illegal activity undertaken or misuse of these drugs.

The Misuse of Drug Regulations 2001 regulates the control and availability of drugs according to therapeutic, legitimate and recognized uses, and potential for misuse. The drugs are classified into five schedules. The regulations were amended following the Shipman Inquiry.

The Controlled Drugs (Supervision of Management and Use) Regulations 2006/2013 details CD handling regulations. Hospitals are responsible for ensuring appropriate systems are in place for CD legislation compliance including stocking, locked storage, storage access, security, ordering and receipt, distribution, prescribing, issuing, destroying, registers, records, accountable officers, monitoring, inspection, auditing, systems for recording, reporting and investigating CD incidents and concerns, maintaining up-to-date standard operating procedures, staff training.

**Classification examples (lists are not exhaustive)**

Schedule 1  Cannabis, LSD, no recognized medicinal use, specific licence to possess (e.g. research)
Schedule 2  Diamorphine, morphine, pethidine, amphetamine, cocaine
Schedule 3  Barbiturates midazolam, temazepam
Schedule 4  Remaining benzodiazepines, androgenic and anabolic steroids
Schedule 5  Certain controlled drugs (e.g. codeine, morphine) when present in medicinal products of low strengths

Class A  Ecstasy, LSD, heroin, cocaine
Class B  Amphetamines, cannabis, codeine
Class C  Gamma hydroxbutyrate (GHB), ketamine

**READING**

Read your hospital’s CD policy and discuss with your department’s pharmacist.

Also read the policy for the management of patients found to be in possession of CDs on arrival to the ED.
What are the clinical findings in necrotizing fasciitis?

**NECROTIZING FASCIITIS**
Rapidly spreading infection of the fascia with necrosis.

**Clinical features**
- Significant disproportionate/unexplained pain
- Pain beyond margins of erythema
- Swelling
- Crepitus
- Erythema, later purple/dusky skin discolouration
- Lethargy
- Pyrexia, hypotension, tachycardia
- Bullae, later become haemorrhagic
- Minor skin changes initially with later rapidly spreading skin changes
- Offensive discharge
- Skin necrosis
- Anaesthesia of affected area
- Lack of bleeding from deep tissues

**Risk factors/associations**
- Diabetes
- Alcohol excess
- Sea swimming
- Immunocompromised
- Insect bites/stings
- Post op surgical wounds/invasive procedure/minor procedures
- Chronic renal failure
- Malignancy
- Chronic liver disease
- IV drug misuse
- Minor skin trauma

**Investigation**
Initially a clinical diagnosis, with surgical exploration required to confirm.
- Blood cultures, blood gas, clotting screen, U&E, albumin, LFT, CRP, ESR, CK, calcium, wound swabs, cross-match blood
- XR and CT may show air in soft tissues and demonstrate extent

**Management**
- Fluid resuscitation
- Antibiotics, liaise with microbiology, e.g. benzylpenicillin, clindamycin and metronidazole
- Analgesia
- Aggressive, prompt extensive surgical debridement
- Admit to intensive care unit
Organisms
There are often mixed anaerobic and aerobic bacteria. Organisms include:

- Group A streptococcus: *Staphylococcus aureus*
- Streptococci: *Clostridium perfringens*
- Coliforms: *Proteus*
- *Pseudomonas*: *Klebsiella*

**NOTES:**
The initial skin wound can be minimal with limited skin findings and the patient appearing well, followed by a rapid deterioration and high mortality rate. Commonly misdiagnosed initially as cellulitis.
Describe the features of Kanavel sign and the significance of this sign
Give the causes of a radial nerve palsy

KANAVEL SIGN

Four components
- Finger is held in slight flexion
- Fusiform swelling of the finger (‘sausage-shaped finger’)
- Tenderness along the course of the flexor tendon sheath
- Pain on passive extension of the finger

Clinical features are found in infection of a flexor tendon sheath in the hand. Infection usually occurs following a bite or a puncture wound. Early recognition is essential to prevent tendon scarring and loss of function. Patients require IV antibiotics, analgesia and referral for urgent incision and drainage of the flexor tendon sheath.

CAUSES OF RADIAL NERVE PALSY
- Compression in axilla ‘crutch palsy’
- Compression of upper medial humerus – ‘Saturday night palsy’
- Humeral fracture
- Elbow dislocation
- Compression at wrist from tight handcuffs or watch strap
- Upper arm injections in infants
What are the features of a tetanus-prone wound?
What is the UK tetanus immunization schedule?
Describe the clinical findings in tetanus

TETANUS

Tetanus-prone wounds
Wounds or burns that require surgical intervention that is delayed for more than six hours
Wounds or burns that show a significant degree of devitalized tissue or a puncture-type injury, particularly where there has been contact with soil or manure
Wounds containing foreign bodies
Compound fractures
Wounds or burns in patients who have systemic sepsis

Higher risk
Injecting drug users (tetanus-contaminated illicit drugs, especially through pre-existing skin abscesses)
Heavy contamination with material likely to contain tetanus spores (manure, soil)
Extensive devitalized tissue

UK TETANUS IMMUNIZATION PROGRAMME
Immunization given at two months, three months, four months, four years, 14 years.

CLINICAL FINDINGS IN TETANUS
Hypertonia
Painful muscular contractions, especially face (risus sardonicus), jaw (lock-jaw), back (opisthotonus), neck
Generalized muscle spasms triggered by minimal stimuli (e.g. noise, light, touch)
Autonomic dysfunction
Dysphagia (pharyngeal muscle spasms)
Airway obstruction (laryngeal spasm)

NOTES:
Five doses of tetanus-containing vaccine at appropriate intervals are considered to give long-term protection. Those born in the UK before 1961 may not have been immunized. Immunosuppressed patients who had been fully immunized should be managed as if incompletely immunized. Tetanus toxin causes failure of inhibition of motor reflex response following infection with Clostridium tetani.
WHAT ARE THE FEATURES OF A TETANUS-PRONE WOUND?

Spores present in soil or manure, most commonly introduced through puncture wounds, burns and minor wounds. Clusters occur in IV drug misuse population. Mortality 10% to 90%. (Neonatal tetanus is due to infection of the umbilical stump. Clinical findings – inability to suck aged 3–10 days, irritability, poor feeding, rigidity, facial grimacing, spasms when touched.)

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READING

COMPARTMENT SYNDROME

Associated causes
- Bone fractures
- Cannula extravasation
- Haemorrhage
- Large vessel injury
- Penetrating trauma
- Seizure
- Tetany
- Burns
- Crush trauma
- Intramuscular or intra-arterial injection
- Over tight casts/dressings
- Prolonged lie on limb
- Snake bites
- Vigorous exercise

Clinical features
- Enhanced pain, pain on passive range of movement, tense swollen limb
- Late signs – pallor, paralysis, paraesthesia, reduced pulses

Initial approach
- FBC, U&E, CK, coagulation screen, urinalysis (myoglobinuria)
- Remove casts/dressings fully
- Urgent orthopaedic referral for fasciotomy
- Keep limb level with body
- Intravenous 0.9% saline
- Analgesia
- Compartment pressures can be measured
Define the following eponyms

**EPONYMS**

<table>
<thead>
<tr>
<th>EPONYM</th>
<th>DESCRIPTION</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bohler angle</td>
<td>Angle formed at the crossing of lines drawn from</td>
</tr>
<tr>
<td></td>
<td>the posterior and anterior aspects of the superior</td>
</tr>
<tr>
<td></td>
<td>calcaneum on lateral radiographs. Angle less</td>
</tr>
<tr>
<td></td>
<td>than 20 degrees is seen in calcaneum fractures</td>
</tr>
<tr>
<td></td>
<td>(but angle can also be normal in a fracture, nor-</td>
</tr>
<tr>
<td></td>
<td>mal range 20–40 degrees).</td>
</tr>
<tr>
<td>Boutonniere deformity</td>
<td>Rupture of central slip of extensor tendon at PIPJ.</td>
</tr>
<tr>
<td>De Quervain</td>
<td>Tenosynovitis of extensor pollicis brevis and</td>
</tr>
<tr>
<td></td>
<td>abductor pollicis longus tendons causing pain</td>
</tr>
<tr>
<td></td>
<td>over radial styloid. Finkelstein test is positive</td>
</tr>
<tr>
<td></td>
<td>in De Quervain tenosynovitis (fist is made over</td>
</tr>
<tr>
<td></td>
<td>thumb and wrist is ulnar-deviated; test is posi-</td>
</tr>
<tr>
<td></td>
<td>tive if causes pain over radial styloid).</td>
</tr>
<tr>
<td>Freiberg disease</td>
<td>Avascular necrosis head of the second metatarsal.</td>
</tr>
<tr>
<td>Keinbock lunate</td>
<td>Avascular necrosis of lunate.</td>
</tr>
<tr>
<td>Kohler disease</td>
<td>Avascular necrosis of navicular.</td>
</tr>
<tr>
<td>Lisfranc injury</td>
<td>Disruption of the tarsometatarsal ligamentous</td>
</tr>
<tr>
<td></td>
<td>joint complex.</td>
</tr>
<tr>
<td>Osgood Schlatter</td>
<td>Pain from tibial attachment of patella tendon.</td>
</tr>
<tr>
<td>Severs disease</td>
<td>Inflammation of the calcaneum apophysis.</td>
</tr>
<tr>
<td>Simmond test</td>
<td>Test for ruptured Achilles tendon. A positive</td>
</tr>
<tr>
<td></td>
<td>test if no movement of foot is seen when the calf</td>
</tr>
<tr>
<td></td>
<td>is squeezed on the affected side (also known as</td>
</tr>
<tr>
<td></td>
<td>Thompson test).</td>
</tr>
<tr>
<td>Terry Thomas sign</td>
<td>Increase in the scapholunate space on AP wrist</td>
</tr>
<tr>
<td></td>
<td>radiograph indicative of scapholunate dissociation.</td>
</tr>
<tr>
<td>Trethowan sign</td>
<td>A line drawn along superior surface of femoral</td>
</tr>
<tr>
<td></td>
<td>neck should pass through femoral head. Positive</td>
</tr>
<tr>
<td></td>
<td>sign, indicative of slipped femoral epiphysis, if</td>
</tr>
<tr>
<td></td>
<td>line is above femoral head.</td>
</tr>
</tbody>
</table>

Musculoskeletal and Injury 105
## EPONYMS

<table>
<thead>
<tr>
<th>Fracture</th>
<th>FINDINGS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bennett fracture</td>
<td>Intra-articular fracture at the base of first metacarpal with dislocation/subluxation</td>
</tr>
<tr>
<td>Chance fracture</td>
<td>Compression of anterior column of vertebra with distraction of posterior portion of vertabra (hyperflexion injury)</td>
</tr>
<tr>
<td>Clay shoveler’s fracture</td>
<td>Fracture of spinous process of C6/C7/T1</td>
</tr>
<tr>
<td>Galeazzi fracture</td>
<td>Fracture between middle and distal thirds of radius with dislocation of the radial ulnar joint at the wrist</td>
</tr>
<tr>
<td>Gamekeeper’s thumb</td>
<td>Ulnar collateral ligament injury at the thumb metacarpophalangeal joint (now more common in falls holding a ski-pole – skier’s thumb)</td>
</tr>
<tr>
<td>Holstein–Lewis fracture</td>
<td>Fracture of distal third of humerus commonly associated with radial nerve injury</td>
</tr>
<tr>
<td>Jefferson fracture</td>
<td>Fracture of C1 anterior and posterior arches (following axial load on occiput of head)</td>
</tr>
<tr>
<td>Jones fracture</td>
<td>Fracture at the fifth metatarsal metaphyseal-diaphyseal junction</td>
</tr>
<tr>
<td>Maisonneuve fracture</td>
<td>Fracture of proximal third of fibula with injury of medial ankle including fracture of medial malleolus, rupture of deltoid ligament/intraosseous membrane/anterior talofibular ligament (external rotation injury)</td>
</tr>
<tr>
<td>Monteggia fracture</td>
<td>Fracture of proximal third of ulna with dislocation of the radial head</td>
</tr>
<tr>
<td>Rolando fracture</td>
<td>Three-part intra-articular comminuted Y-shaped fracture at base of first metacarpal</td>
</tr>
<tr>
<td>Segond fracture</td>
<td>Avulsion fracture seen as a lateral proximal tibia; associated with anterior cruciate tear and menisci injury</td>
</tr>
</tbody>
</table>
Tillaux fracture  Salter Harris III fracture of the distal anterolateral tibial epiphysis (commonly external rotation injury in 12–15 yr-olds)

NOTES:
Eponyms should not be used solely to describe a fracture but it can be useful to consider eponymous fractures when initially viewing radiographs. Remember to document whether right/left limb.

READING

Pain management

Nerve block techniques
**What is the Parkland formula?**

**PARKLAND FORMULA FOR BURNS RESUSCITATION**

Total fluid requirement in 24 hours = \(4 \text{ mL} \times \text{body surface area (\%)} \times \text{body weight (kg)}\)

- 50% given in first 8 hours
- 50% given in next 16 hours

Use for burns over 20%

Time of initial burn/injury (not the time of writing fluid chart) should be used when prescribing, thus the initial 50% of fluids may, for example, in reality need to be given over six hours following a two-hour pre-hospital extrication

**NOTES:**

Rule of Nines to estimate the area of adult medium to large burns:

- Anterior and posterior arm 9%
- Anterior and posterior head 9%
- Anterior and posterior leg 18%
- Posterior trunk 18%
- Anterior trunk 18%
- Genitalia 1%
- Palmar surface including fingers 1%
When are ankle/foot X-rays indicated following trauma?

**OTTAWA RULES**

**Ankle X-rays indicated if**
- Bone tenderness distal 6 cm of the posterior edge of the fibula or tip of the lateral malleolus
- Bone tenderness distal 6 cm of the posterior edge of the tibia or tip of the medial malleolus
- Inability to weight bear both immediately and in the emergency department

**Foot X-rays indicated if**
- Pain in the midfoot and any one of the following
  - Bone tenderness navicular
  - Bone tenderness base of fifth metatarsal
- Inability to weight bear both immediately and in the emergency department


**READING**

Describe the key features to identify on a child’s elbow radiograph

PAEDIATRIC ELBOWS

Specifically look for the following

Anterior humeral line – Drawn along anterior cortex of distal humerus metaphysis and should pass through the middle third of the capitellum
Radiocapitellar line – Drawn through radial neck and should pass through the capitellum
Presence of anterior and/or posterior fat pads

Order elbow epiphyses appear in a child (mnemonic CRITOL)

<table>
<thead>
<tr>
<th>Epiphysis</th>
<th>Age</th>
</tr>
</thead>
<tbody>
<tr>
<td>Capitellum</td>
<td>1 year</td>
</tr>
<tr>
<td>Radial head</td>
<td>3 years</td>
</tr>
<tr>
<td>Internal (medial) epicondyle</td>
<td>5 years</td>
</tr>
<tr>
<td>Trochlea</td>
<td>7 years</td>
</tr>
<tr>
<td>Olecranon</td>
<td>9 years</td>
</tr>
<tr>
<td>Lateral epicondyle</td>
<td>11 years</td>
</tr>
</tbody>
</table>

NOTES:
The exact age of epiphysis development can vary normally between children, though the order of appearance should always follow as above. Thus, bony changes not present in order should be presumed to be due to a fracture.
IO (INTRA-OSSEOUS) NEEDLES

Complications

- Compartment syndrome
- Fracture
- Fat embolus
- Haematoma
- Infection (osteomyelitis, cellulitis)
- Possible growth plate injury

Contraindications

- Inability to locate landmarks
- Extensive pelvic injury (use upper limb site)
- Previous attempts in same limb
- Overlying skin infection
- Fracture of limb
- Vascular injuries on same side
- Osteogenesis imperfect

Insertion sites

- Tibia (one finger breadth below tibial tuberosity)
- Humerus (base of greater tuberosity, palpate for protrusion in humeral head with arm internally rotated and adducted)
- Distal tibia (proximal to medial malleolus)
- Distal femur

Notes:
Indicated in cardiac arrest or when urgent vascular access is required but not immediately available.
To reduce the risk of late recognition of compartment syndrome avoid bandaging/covering the limb. Monitor limb in comparison with other limb for capillary refill distally, swelling, firmness, colour (pink, pale, blue, white) every 15 minutes. Stop using and remove if clinical concerns and discuss with orthopaedic team re fasciotomy. Cease using IO needles once alternative vascular access has been achieved.
What are the presenting features of toxic shock syndrome in children following a burn, and what is the initial management approach?

**TOXIC SHOCK SYNDROME (TSS) FOLLOWING BURNS IN CHILDREN**

*Presenting features of TSS in children*

- Fever >39°C
- Rash
- Diarrhoea +/- vomiting
- Irritability
- Lymphopaenia

*Treatment approach*

- Move to resuscitation area
- Obtain intravenous access
- Send blood and microbiology samples (FBC, U&E, clotting screen, G&S, blood cultures, wound swabs)
- Resuscitate and treat hypoperfusion with fluid boluses, normal saline 10 mL/kg and reassess (may need 40–60 mL/kg)
- Give intravenous antibiotics, anti-staphylococcal and streptococcal (flu-cloxacillin and penicillin)
- Give FFP 10 mL/kg (repeat if necessary) or immunoglobulin to provide passive immunity against staphylococcal toxic shock syndrome toxin 1 (TSST-1)
- Remove dressings, inspect and clean burn wound
- Consider catheterization for fluid balance
- Manage in paediatric HDU
- Review hourly until improving

**NOTES:**

TSS is a toxin-mediated illness that is challenging to diagnose due to the initial non-specific symptoms that mimic other common childhood illnesses. High mortality up to 50% if untreated, thus early recognition is essential. Currently there is no evidence on methods to prevent.

**READING**

**What are the presenting features of botulism?**

**BOTULISM**
Toxin produced by *Clostridium botulinum* prevents acetylcholine transmission across the neuromuscular junction.

**Presentation**
Symmetrical cranial nerve palsies, progressing to symmetrical descending flaccid paralysis, progressing to respiratory arrest
‘The Five Ds’ – diplopia, dysarthria, dysphonia, dysphagia, descending
Other features include blurred vision, ptosis, facial weakness, dry mouth, postural hypotension, nausea, vomiting, constipation, loss of tendon reflexes, diarrhoea (absence of confusion)

**Types**
Foodborne (especially home-canned foods)
Wound infection (intravenous drug misusers)
Inhalation (deliberate toxin release)
Iatrogenic (cosmetic use of concentrated toxin)
Intestinal

**Management**
Antitoxin given early can stop progression of paralysis
Monitor vital capacity and arterial blood gases with intubation as indicated
Inform health protection agency
## List 10 stroke mimics

### DIAGNOSES WHICH CAN MIMIC A STROKE

<table>
<thead>
<tr>
<th>Brain tumour – primary, metastatic</th>
<th>Carbon monoxide poisoning</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cerebral abscess</td>
<td>Cranial nerve palsies</td>
</tr>
<tr>
<td>Drug/alcohol intoxication</td>
<td>Functional</td>
</tr>
<tr>
<td>Hemiplegic migraine</td>
<td>Hepatic encephalopathy</td>
</tr>
<tr>
<td>HONK</td>
<td>Hypertensive encephalopathy</td>
</tr>
<tr>
<td>Hypoglycaemia</td>
<td>Hyponatraemia</td>
</tr>
<tr>
<td>Multiple sclerosis</td>
<td>Myasthenia gravis</td>
</tr>
<tr>
<td>Oculogyric crisis</td>
<td>Parkinsonism</td>
</tr>
<tr>
<td>Peripheral neuropathy</td>
<td>Sepsis (neuro or systemic)</td>
</tr>
<tr>
<td>Spinal cord pathology</td>
<td>Subarachnoid/intracerebral haemorrhage</td>
</tr>
<tr>
<td>Subdural/extradural haemorrhage</td>
<td>Syncope</td>
</tr>
<tr>
<td>Todd paralysis</td>
<td>Transient global amnesia</td>
</tr>
<tr>
<td>Venous thrombosis</td>
<td>Vertigo</td>
</tr>
</tbody>
</table>

### READING


What are the clinical findings in cerebellar lesions?
Give five causes of cerebellar syndrome

CEREBELLAR SIGNS (MNEMONIC DANISH)

Dysdiadochokinesia (inability to perform rapid, alternating movements)
Ataxia
Nystagmus
Intention tremor
Staccato/scanning or slurred speech
Hypotonia and heel–shin test inability

Ipsilateral signs

CEREBELLAR SYNDROME CAUSES

Multiple sclerosis
TIA/CVA
Space occupying lesion (primary or secondary tumours, abscess)
Severe hypothyroidism
Thiamine deficiency (including chronic alcohol misuse)
Phenytoin toxicity
Paraneoplastic
Meningo-encephalitis
Heavy metal poisoning
Friedreich ataxia

NOTES:
Causes of CT brain ring enhancing lesions (MAGICAL DR):

Metastasis
Abscess
Glioblastoma multiforme
Infarct
Contusion
AIDS/HIV – toxoplasmosis, TB
Lymphoma in immunocompromised
Demyelinating disease
Radiation necrosis, resolving haematoma
GUILLAIN–BARRÉ SYNDROME (GBS)
Acute inflammatory demyelinating polyradiculoneuropathy

Presenting features
Progressive onset of bilateral, ascending, symmetrical, proximal and distal, limb weakness
Absent deep tendon reflexes
Respiratory failure
Variable paraesthesia and sensory loss
Autonomic dysfunction (hyper/hypotension, arrhythmias, urine retention) can occur
Cranial nerves can be affected (facial weakness, bulbar palsy, ophthalmoplegia)
Frequently there is a recent history of respiratory or gastrointestinal infection

Infections associated with GBS
Campylobacter jejuni
Cytomegalovirus
Epstein–Barr virus
HIV
Influenza
Mycoplasma

Other GBS associations
Haematological malignancies
Vaccinations
Post-partum

Investigations
Clinical diagnosis
Vital capacity monitoring (blood gases and oxygen saturation monitoring can be normal and falsely reassuring)
Nerve conduction studies
Cerebrospinal fluid analysis (increase protein in GBS)
MRI brain and spine for alternative diagnoses

Management approach
Plasma exchange, IV immunoglobulins (steroids are ineffective)
Supportive, VTE prophylaxis, physiotherapy
GIVE THE PRESENTING FEATURES OF GUILLAIN–BARRÉ SYNDROME

Intubate and ventilate if FVC <15 mL/kg
Mortality up to 10% (from pulmonary emboli, cardiac arrhythmias respiratory failure or sepsis)

NOTES:
Miller–Fisher syndrome is an inflammatory neuropathy causing ophthalmoplegia, ataxia, areflexia but no weakness.
Neurology 129

What can precipitate acute worsening of symptoms in myasthenia gravis?

MYASTHENIA GRAVIS (MG)
An autoimmune process affecting the neuromuscular junction acetylcholine receptors.

Exacerbation
- Pregnancy
- Bacterial/viral infections
- Post-operative
- Emotional stress
- Drugs (e.g. propranolol, gentamycin, magnesium)
- Omission of drug doses (iatrogenic, unable to swallow)

Clinical findings
- Muscle fatigue and weakness on exercise, improves with rest (e.g. unable to sustain upward gaze or complete counting to 50)
- Ocular muscle weakness ptosis, diploplia (most common)
- Head drop (neck muscle weakness)
- Limb weakness (proximal > distal)
- Slurred speech
- Dysphagia
- Respiratory muscle weakness leading to respiratory failure
  (Normal reflexes, tone and sensation, no muscle wasting)

Management approach
- Monitor vital capacity and arterial blood gases.
- Refer to neurology and ITU for consideration of immunoglobulin, plasma-apheresis, immunosuppression.

NOTES:
Myasthenic crisis can be difficult to differentiate between cholinergic crisis (i.e. overuse of anticholinergic medication – pyridostigmine). Cholinergic crisis is associated with sweating, miosis, salivation, fasciculations.
- Eaton–Lambert syndrome – Autoimmune process against calcium channels on motor nerves resulting in lack of acetylcholine. Most commonly a paraneoplastic syndrome associated with small cell lung cancer. Similar presentation to MG but limb weakness is predominant feature. Investigate for underlying malignancy.

READING
What is the treatment approach for a fitting child?

**FITTING CHILD**

Note time and start clock.
Assess and protect airway and give high flow oxygen.
Check blood glucose level.

1. Five minutes after convulsion started
   - Administer benzodiazepine (IV route first choice)
   - Rectal diazepam 0.5 mg/kg (max 20 mg)
   - Buccal Midazolam 0.5 mg/kg (max 10 mg)
   - Lorazepam 0.1 mg/kg IV/IO (max 4 mg) or Diazepam IV

2. Seizure for further 10 mins post step 1
   - Administer second dose of benzodiazepine
   - Call for senior help
   - Start to prepare phenytoin

3. Seizure for further 10 mins post step 2
   - Give phenytoin 20 mg/kg IV/IO over 20 mins
   - If already on phenytoin give phenobarbitone 20 mg/kg IV/IO over 5–10 minutes

4. Seizure for further 20 mins post step 3
   - RSI with thiopentone 4 mg/kg IV/IO

**NOTES:**
Carers may carry a personal emergency status epilepticus treatment plan.
Give the features in history and examination which would be important to elicit in a six-year-old presenting following a first fit

**PAEDIATRIC FIRST FIT**

**Important history findings**
- Seizure lasting more than 10 minutes
- Required more than one dose of benzodiazepine
- Focal seizure
- Drugs, alcohol or toxins
- Head injury
- Pre-existing neurological condition
- Pre-existing syndrome diagnosis
- Developmental delay or regression
- Bleeding disorder
- Immunosuppression

**Important examination findings**
- Febrile
- Appears unwell
- GCS not returned to 15
- Focal neurology
- Signs of meningism
- Signs of raised intracranial pressure
- Abnormal observations
- Abnormal head circumference
- Dysmorphic features
- Abnormal cardiac examination

Fitting child minimum investigations include BM, temperature, ECG, MSU (and pregnancy test in older children). Fully undress and examine.
What is your initial approach on identification of umbilical cord prolapse?
Describe the APGAR score

UMBILICAL CORD PROLAPSED

**ED approach**
Knees to chest.
Insert size 16 Foley catheter into bladder. Fill with 500 mL normal saline (or until presence of distended bladder above pubis). Inflate balloon and clamp catheter.
Urgent obstetric assessment re presence of cord pulsation, fetal heart beat, fetal movements, and transfer to theatre for caesarean section.

**APGAR SCORE**
Numeric evaluation of condition at birth. Score at one minute and five minutes post birth.

<table>
<thead>
<tr>
<th>Colour</th>
<th>Blue/pale (0)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pink body, blue limbs (1)</td>
</tr>
<tr>
<td></td>
<td>Pink (2)</td>
</tr>
<tr>
<td>Heart rate</td>
<td>Absent (0)</td>
</tr>
<tr>
<td></td>
<td>&lt;100 (1)</td>
</tr>
<tr>
<td></td>
<td>&gt;100 (2)</td>
</tr>
<tr>
<td>Reflex irritability</td>
<td>No response to stimulus (0)</td>
</tr>
<tr>
<td></td>
<td>Grimace (1)</td>
</tr>
<tr>
<td></td>
<td>Strong cough/sneeze/cry (2)</td>
</tr>
<tr>
<td>Tone</td>
<td>Limp (0)</td>
</tr>
<tr>
<td></td>
<td>Flexed limbs with minimal movement (1)</td>
</tr>
<tr>
<td></td>
<td>Active movement (2)</td>
</tr>
<tr>
<td>Breathing</td>
<td>Absent (0)</td>
</tr>
<tr>
<td></td>
<td>Irregular gasping/slow (1)</td>
</tr>
<tr>
<td></td>
<td>Normal/crying (2)</td>
</tr>
</tbody>
</table>

Score 7–10 = normal

**Mnemonic APGAR**
Appearance, Pulse, Grimace, Activity, Respiration
Describe the clinical features of pre-eclampsia and the management of an eclamptic seizure

PRE-ECLAMPSIA AND ECLAMPSIA

**Symptoms and signs**

- Headache
- Visual disturbances (flashing, floaters)
- Vomiting
- Epigastric pain
- Nondependent (especially facial) or pulmonary oedema
- Right upper quadrant pain
- Recent hypertension >160/110 with proteinuria >1 g in 24 hr
- Hypereflexia with clonus

**Management approach imminent eclampsia or eclampsia**

**Initial approach**

Left tilt, assess and protect airway, apply oxygen, ventilate as required, assess pulse and BP, IV access

**Call for help**

Senior obstetric anaesthetist, obstetrician, neonatologist, midwife, emergency physician, intensive care, haematologist

**Seizures**

Magnesium IV load 4 g over 10–20 min followed by 1 g/hr infusion

**Ongoing seizures**

Further MgSO$_4$ (2 g under 70 kg, 4 g over 70 kg) over 5–10 min

Diazepam 10 mL IV or IV thiopentone 3–5 mg/kg paralyze, intubate and ventilate

**Hypertension**

Treat if systolic >170 or diastolic >110 or MAP >125

Hydralazine, labetolol

**Deliver**

Once mother stabilized, deliver baby; fetal heart rate and CTG monitoring

**Investigations**

FBC, platelets, U&E, LFT, urate, clotting, group and save, urine

**Fluid balance**

Avoid iatrogenic fluid overload
NOTES:

**Hypermagnasaemia**
Review magnesium levels and stop infusion if:
- Urine output <100 mL in 4 hr
- Patella reflexes absent
- Respiratory rate <16/min
- Oxygen saturations <90%

**Symptoms**
- Feeling warm
- Flushing
- Double vision
- Slurred speech
- Loss of tendon reflexes
- Respiratory depression then arrest
- Cardiac arrest

**Management**
- 10% calcium gluconate 10 mL over 10 min

**HELLP**
Can occur with severe pre-eclampsia:
- Haemolysis
- Elevated Liver enzymes
- Low Platelets

**READING**
What is your approach on identifying shoulder dystocia? 
List the causes of post-partum haemorrhage

SHOULDER DYSTOCIA
The following mnemonic can help in shoulder dystocia. The order of manoeuvres is not essential. Mnemonic HELPERR:

- Call for Help (obstetric consultant, obstetric anaesthetist, midwife, neonatology).
- Evaluate for episiotomy (may not help resolve the shoulder dystocia in itself but allows more space to perform internal manoeuvres and reduces significant vaginal lacerations).
- Legs into McRobert’s manoeuvre. Flex, abduct and rotate both thighs outwards (straightens sacrum and causes cephalic rotation of the pelvis to help free an impacted shoulder).
- Pressure (suprapubic).
- Enter for rotatory manoeuvres.
- Remove the posterior arm.
- Roll (Gaskin manoeuvre; move to an all-fours position with the back arched).

Most cases of shoulder dystocia are unpredictable.
Complications of shoulder dystocia include cerebral hypoxia, cerebral palsy, fracture of clavicle, fracture of humerus, brachial plexus injury, post-partum haemorrhage, vaginal and perianal lacerations, uterine rupture.

CAUSES OF POST-PARTUM HAEMORRHAGE

- Tone Reduced uterine tone
- Tissue Retained placenta
  Retained products
  Placenta praevia
  Uterine inversion
- Tear Genital tract trauma
  Uterine rupture
- Thrombin Coagulopathy

READING

MOET course manual ALSG (Managing Obstetric Emergencies and Trauma).
What are the findings in retrobulbar haemorrhage?

RETROBULBAR HAEMORRHAGE

Clinical Findings
Reduced visual acuity
Proptosis
Reduced eye range of movement
Subconjunctival haemorrhage
Raised intraocular pressure
Afferent pupillary defect
Painful eye/movement
Pale retina

Causes
Trauma
Spontaneous
Post-operative
Coagulopathy

Management
Lateral canthotomy
Medical options include mannitol, acetazolamide, dexamethasone but lateral canthotomy should be first line
Sight threatening emergency

NOTES:
YouTube has several examples of how to perform a lateral canthotomy.
List the differential diagnosis of papilloedema

PAPILLOEDEMA

Differential diagnosis

Raised intracranial pressure (tumour, hydrocephalus, abscess, subdural haematoma, etc.)
Venous thrombosis
Benign (idiopathic) intracranial hypertension
Central retinal vein thrombosis
Meningitis
Grade 4 hypertensive retinopathy
Drug toxicity (e.g. tetracycline, lead, vitamin A)
Hypoparathyroidism
Hypercapnoea
List the causes of, and clinical findings in, central retinal artery occlusion

CENTRAL RETINAL ARTERY OCCLUSION

Clinical findings
Sudden, painless, striking, unilateral vision loss (finger counting acuity occurring in seconds)
Pale retina with cherry red spot
Afferent pupillary defect

Causes
Hypertension
Diabetes
Atherosclerosis
Sickle cell disease
Atrial fibrillation
Embolism
Endocarditis
Patent foramen ovale
Intravenous drug misuse
Arteritis/vasculitidies
Migraine
Hypercoagulopathies
Polycythaemia
Arterial spasm
Dissection

NOTES:
Avoid giving answers that are already inferred in the question (e.g. if the stem has stated the patient has atrial fibrillation and asks for associations with retinal artery occlusion, points would not be awarded for writing atrial fibrillation).

READING
What are the fundoscopy findings in central retinal vein occlusion?
Which conditions are associated with central retinal vein occlusion?

**RETINAL VEIN OCCLUSION**

Sudden unilateral painless blurring/reduced/loss of vision
Branch (more common) or central retinal vein occlusion

**Fundoscopy (‘stormy sunset’)**

Widespread flame haemorrhages
Optic disc swelling
Cotton-wool spots
Dilated retinal veins
Afferent pupillary defect

**Associations**

- Diabetes
- Hypertension
- Hyperlipidaemia
- Sarcoid
- Thrombophilias

- Glaucoma
- Hyperviscosity syndromes (myeloma, leukaemias)
- Obesity
- Smoking
- Vasculitis

**Investigations**

- Visual acuity
- ECG
- Cholesterol and lipids
- ESR

- BP
- BM
- FBC and blood film

Others as indicated by history (plasma protein electrophoresis, thrombophilia screening, etc.)

**NOTES:**

History points for loss of vision. Enquire whether sudden, one or both eyes, painful, redness, transient or persistent, trauma, headache, temporal pain, other neurological signs, medical history (AF, TIA, etc.), all of vision versus hemianopia/quantrantanopia/central, distorted vision, flashers, floaters, toxins (methanol).
**What is Seidel's test?**

**EYE TRAUMA**

Seidel’s test: uses fluorescein to identify a corneal wound causing an aqueous humour leak. Fluorescein will appear to dilute in a stream across the surface of the cornea (a negative test does not exclude a corneal wound).

Following blunt or penetrating trauma document and examination for:

- Bone pain
- Diplopia
- Eye movements
- Globe intact
- Intra-ocular haemorrhage
- Iris irregularity
- Periorbital bruising
- Pupillar abnormalities – reactive/irregular/shape
- Retinal haemorrhage/detachment
- Subconjunctival haemorrhage
- Visual acuity and fields

- Corneal abrasions/clouding
- Eye emphysema
- Eyelid wounds
- Hyphaema
- Infra orbital paraesthesia
- Lens dislocation
- Proptosis
- Red reflex
- Soft tissue swelling
- Traumatic mydriasis

**NOTES:**

The ‘black eyebrow sign’ is commonly missed on facial X-rays. A lucent (blacker) area is seen by the superior orbital rim on the affected sign which mimics an eyebrow. Indicates free air/intraorbital emphysema following an orbital blow-out fracture.
A patient presents with painful eyes following a day walking in snow. What is the likely diagnosis?

PHOTOKERATITIS

Eye exposure to ultraviolet light damages the corneal epithelium. History of UV exposure without wearing appropriate eye protection – examples:
- Welding
- Sun reflection during snow/ice/water activities
- Sunbed use
- Viewing direct sunlight

Symptoms onset 4 to 12 hours after exposure:
- Pain
- Watering eyes
- Foreign body sensation
- Photophobia
- Reduced visual acuity (mild)
- Eye erythema
- Periorbital skin redness

Both eyes should be symptomatic (consider alternative diagnosis if unilateral). Fluorescein staining shows diffuse punctuate uptake.

Management
- Oral analgesia (NSAIDs), sunglasses, advise to not use contact lenses
- Reassure symptoms should improve over 1–2 days
- Re-present if symptoms worsen or do not resolve
- Give advice regarding UV light and appropriate eye protection
## Differential Diagnosis of Psychosis

### Recreational drugs (acute use, chronic use or abrupt withdrawal)
- Cocaine
- Alcohol
- Hallucinogenics
- Amphetamines
- Cannabis

### Drugs
- Corticosteroids
- Propranolol
- Mefloquine
- Levodopa

### Electrolyte disturbance
- Hyponatraemia
- Hyper/hypocalaemia
- Hypoglycemia

### Endocrine
- Hyper/hypothyroidism
- Cushing disease

### Infective
- Sepsis
- HIV
- Cerebral malaria
- Encephalitis

### Metabolic
- Hepatic encephalopathy
- Wilson disease
- Thiamine deficiency
- Acute intermittent porphyria
- Niacin deficiency
- B12 deficiency

### Neurological
- Dementia
- Acute/chronic head injury
- Cerebral tumours
- Multiple sclerosis
- CVA
- Migraine
- Huntington disease
- Following seizure (post ictal)

### Other
- Post-partum
- SLE
- Heavy metal poisoning
- Hypoxia
- Paraneoplastic

### Notes:
The most common cause is drug-induced from recreational, prescription or over-the-counter drugs. An organic cause should be considered in the ED prior to attributing to a primary psychotic disorder. An organic cause of psychosis can co-exist with a psychiatric diagnosis.
How should capacity be assessed?

ASSESSING CAPACITY

*Lack of capacity requires demonstration of both*

Impairment of or disturbance in functioning of mind or brain, and
Inability to do any of the following (mnemonic CURB):

- Communicate decision
- Understand information given
- Retain the information to use for a decision
- Balance: Weigh up the treatment options/information

*Mental Capacity Act principles*

Assume capacity
Take all practical steps to enable a person to make own decisions
Help people who have capacity
An unwise decision does not equal a lack of capacity
Decisions for people without capacity should be in their best interests
Decisions should be least restrictive as possible

NOTES:
Mental capacity assessments are decision specific.

READING

The Mental Capacity Act: http://www.legislation.gov.uk
Capacity assessment in children – GMC guidance 0–18 years: guidance for all doctors.
MENTAL HEALTH ACT

Section 2  Admission for assessment up to 28 days
Section 3  Admission for treatment up to 6 months
Section 5(2)  Compulsory detention of patient already receiving inpatient treatment for up to 72 hours by a doctor
Section 136  Police officer detains in a public place to a place of safety
Describe the clinical features seen with SVCO (superior vena cava obstruction)

SVCO (SUPERIOR VENA CAVA OBSTRUCTION)
Reduction of venous return through the SVC to the right atrium due to tumour invasion of vessel wall, blood clot obstructing the lumen or external pressure.

Clinical features
- Fixed dilated neck, anterior chest wall and arm veins
- Face, neck, upper chest, arm oedema
- Plethoric or cyanosed face
- Conjunctival injection
- Hoarseness, stridor
- Dysphagia
- Headaches, heady fullness
- Breathlessness
- Chest pain
- Facial flushing, congestion and stridor may worsen on raising both arms
  (Pemberton sign)

Causes
- Primary lung cancer (most common cause)
- Retrosternal tumours (including lymphoma, thymoma)
- SVC catheter complications (thrombosis)
- Retrosternal goitre
- Mediastinal lymphadenopathy (lymphoma)
- Aortic aneurysm
- Mediastinitis

NOTES:
An oncological/surgical emergency. Image for underlying cause with CXR, CT/ MRI, venogram. Nurse sitting up.
Depending on underlying cause treatment includes dexamethasone, chemotherapy, radiotherapy, antiocoagulation, stents, surgical resection.
Give six causes of pneumomediastinum

PNEUMOMEDIASTINUM

Causes

Asthma
Boerhaave syndrome (oesophageal rupture following vomiting)
Blunt/penetrating chest trauma
Diving
Iatrogenic following endobronchial or oesophageal procedure
Illicit drug inhalation
Mechanical ventilation
Perforated bowel
Pnemothorax
Spontaneous

(Most common listed; literature lists many other case associations)

Hamman sign can be found with pneumomediastinum – crunching sound heard synchronous with the heartbeat

NOTES:
Causes of life threatening chest injuries (ATOM FC):

Airway obstruction
Tension pneumothorax
Open pneumothorax
Massive haemothorax
Flail chest
Cardiac tamponad

READING

How can a difficult airway be predicted?

**PREDICTING DIFFICULT AIRWAYS**

**LEMON – predicts difficult laryngoscopy and intubation if score 5 or more**

Look  
Face trauma, big incisors, beard, moustache, big tongue (1 point each)

Evaluate (3-3-2 rule)  
Inter-incisor distance <3 finger breadths  
Hyoid–mental distance <3 finger breadths  
Thyroid–floor of mouth <2 finger breadths (1 point each)

Mallampati  
Score 3 or more (1 point)

Obstruction  
Tumours, epiglottitis etc.

Neck mobility  
Limited, e.g. rheumatoid arthritis

**MOANS – predicts difficult bag-valve-mask**

Mask seal  
Likely inadequate, e.g. trauma, beard

Obesity  
BMI >26

Age  
55 years old +

No teeth

Stiff ventilation  
Late pregnancy, ARDS, COPD, asthma

**SHORT – predicts difficult cricothyroidotomy**

Surgery  
Prior neck surgery

Haematoma  
Significant neck haematoma

Obesity  
BMI >26

Radiation  
Prior neck radiation

Tumour  
Neck tumours
Give the causes of the following capnography traces in an intubated patient: sudden loss of trace, gradually increasing trace, slanted expiratory trace, gradually falling size

### CAPNOGRAPHY TRACES

#### Straight line/sudden loss of trace
- Cardiac arrest
- Capnograph sampling tubing blockage
- Disconnected capnograph
- Dislodged ET tube
- Lung blockage (e.g. severe bronchospasm)
- Respiratory arrest/no breaths bagged
- Obstruction of airway (e.g. foreign body, tracheal tube obstruction)
- Ventilator faulty

#### Gradually increasing size
- Inadequate ventilation (hypoventilation)
- Increased CO₂ production (e.g. malignant hyperpyrexia)
- Pain
- Respiratory depressant drugs
- Shivering

#### Slanted expiratory trace
- Incomplete exhalation
- Partial obstruction of airway (e.g. tracheal tube secretions, kinking)
- Partial obstruction of lungs (e.g. bronchospasm, COPD, mucous plugging)
- Poor sampling technique

#### Gradually falling size
- Cardiopulmonary arrest
- Dead space ventilation
- Hyperventilating
- Hypothermia
- Massive blood loss
- PE
- Sudden hypotension
**Other traces**

Saw-like mini waves/ripple cardiac oscillations
Stepwise gradually rising baseline re-breathing
Small dips along plateau phase relaxation notches

**NOTES:**
Oesophageal intubation can cause:
- Flat line
- Abnormally shaped capnograms with a gradually falling trace
- Small irregular humps

**DOPES mnemonic – differential for loss of capnography/desaturation in an intubated patient**

<table>
<thead>
<tr>
<th>Category</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Displacement</td>
<td>Tube migration above cord, oesophageal intubation, right main bronchus intubation</td>
</tr>
<tr>
<td>Obstruction</td>
<td>Blocked tube (mechanical kink/bitten, secretions, bronchospasm)</td>
</tr>
<tr>
<td>Pneumothorax</td>
<td>Decompress if clinical signs</td>
</tr>
<tr>
<td>Equipment</td>
<td>Disconnected from oxygen/ventilator, ventilator failure, oxygen supply failure</td>
</tr>
<tr>
<td>Sensitivity and Stomach</td>
<td>Impending anaphylaxis (e.g. contract media, antibiotic), respiratory compromised due to gastric distension (especially in children)</td>
</tr>
</tbody>
</table>
What are the echocardiography findings in a significant PE?

ECHOCARDIOGRAPHY FINDINGS IN SIGNIFICANT PULMONARY EMBOLISM (PE)

- Right ventricle dilatation
- Right ventricle diffusely hypokinetic
- Right atrial dilatation
- Septal flattening/paradoximal movement
- Pulmonary artery dilatation
- Tricuspid regurgitation
- Dilated non-collapsing inferior vena cava
- Elevated pulmonary pressures

READING

Give the causes of cavitating lung lesions

CAUSES OF CAVITATING LUNG LESIONS

- Abscess
- Fungal infection
- Lung infarction
- Wegener granulomatosis
- Brochogenic carcinoma
- Hydatid cyst
- Tuberculosis

CAUSES OF LUNG ABSCESES

- Actinomycosis
- Aspergillus
- Staph aureus
- Amoebic abscess
- Klebsiella
- Tuberculosis

READING

How can the severity of pneumonia be assessed in adults?

PNEUMONIA
CURB65 score, used to assess severity of community acquired pneumonia. Score 1 point for each.

- New Confusion
- Urea >7 mmol/L
- Respiratory rate ≥30/min
- Blood pressure <90 systolic or ≤60 diastolic
- Age ≥65

0 = Low severity, likely suitable for home treatment
1–2 = Moderate severity
3–4 = High severity and risk of death. ICU involvement recommended

NOTES:
Clinical judgement, the patient’s social circumstances and the presence of other coexisting pathology should be considered along with the CURB65 score when assessing severity and deciding location for treatment.

Reproduced from BTS Guidelines for the Management of Community Acquired Pneumonia in Adults, copyright (2009) with permission from BMJ Publishing Group Ltd.
What is the differential diagnosis of new onset stridor in a three-year-old?

Suggest features to differentiate between causes of stridor

Describe the UK childhood immunization schedule

**PAEDIATRIC STRIDOR DIFFERENTIAL DIAGNOSES**

**Croup**
- Inspiratory stridor, barking cough, mild fever, possible preceding coryza, night presentation

**Inhaled foreign body**
- Sudden onset in previously well child, afebrile, history of choking/gagging

**Epiglottitis**
- Systemically unwell, fever >38.5°C, soft stridor, limited/no cough, drooling, rapid onset, unimmunized

**Allergic angioedema**
- Periorbital oedema, lip swelling, urticarial rash, pruritis, shock

**Bacterial tracheitis**
- Moderate to high fever, drooling

**Abscess (tonsillar or retropharyngeal)**
- High fever, dysphagia/painful swallow, neck hyperextension/painful range of movement/swelling, trismus, smoke/chemical irritation, detailed pre-hospital history, similar symptoms in family

**UK CHILDHOOD IMMUNIZATION SCHEDULE**

**Two months**
- Diphtheria, tetanus, pertussis, polio, haemophilus influenza type b, pneumococcal, rotavirus

**Three months**
- Diphtheria, tetanus, pertussis, polio, haemophilus influenza type b, meningococcal group C, rotavirus

**Four months**
- Diphtheria, tetanus, pertussis, polio, haemophilus influenza type b, pneumococcal

**12 to 13 months**
- Haemophilus influenza type b, meningitis group C, pneumococcal, measles, mumps, rubella

**Three years four months**
- Diphtheria, tetanus, pertussis, polio, measles, mumps, rubella

**12 to 13 years**
- Human papillomavirus (girls)

**14 years**
- Tetanus, diphtheria, polio, meningitis C
How should a child with croup be initially assessed in the ED?

CROUP

Assessing croup severity
Westley croup score (mnemonic SCARE)
- Stridor: 0 = none, 1 = with agitation, 2 = at rest
- Cyanosis: 0 = none, 4 = with agitation, 5 = at rest
- Alertness: 0 = normal, 5 = disorientated
- Recession: 0 = none, 1 = mild, 2 = moderate, 3 = severe
- Entry of air: 0 = normal, 1 = decreased, 3 = markedly decreased

<2 mild, 3–5 moderate, 6+ severe.
Hypoxia and tiring are late signs.

Consider safe for discharge
- Well with no significant respiratory distress at rest or after ED play
- Normal observations
- Playing normally and no agitation
- Suitable home environment
- Carers no longer concerned
- Eating and drinking adequately
- No fever >38°C while in ED
- Written advice card and safety net advice given

Croup management approach
Ensure correct diagnosis and have considered all other causes of stridor in children.
- Avoid distressing child, leave in comfortable position with carer.
- Do not insert tongue depressor, take bloods or X-ray.
- Assess for severity.
- Steroids – Dexamethasone 0.15 mg/kg PO or nebulized 2 mg budesonide.
- Observe.

If features of severe/deterioration
- Nebulized adrenaline 0.5 mL/kg 1:1000 (max 5 mL).
- 100% oxygen as tolerated. Ensure adequate hydration.
- Call senior paediatrician, senior ENT, PICU/ICU.
- Consider intubation if evidence of impending respiratory failure, worsening hypoxia, respiratory rate >60 despite treatment, or falling respiratory rate without improvement, normal or rising CO₂, exhaustion. (Is it definitely croup?)
Which features of a bruise in a child would be concerning?  
Describe concerning burns

BRUIsing in CHILDREN

Features of a bruise to be considered as potentially resulting from maltreatment

- Shape of a hand, ligature, stick, tooth marks, grip or implement imprint
- Clear demarcation
- Bruising or petechiae that are not explained by a medical condition (e.g. coagulopathy)
- Bruising in non-ambulant child (e.g. child that does not roll yet or child with cerebral palsy who cannot move independently)
- Multiple bruises
- Multiple of similar shapes and sizes
- In clusters
- Away from bony prominences
- Face, back, abdominal, arm, buttock, ear and hand bruises
- On the neck that look like attempted strangulation
- On the ankles and wrists that look like ligature marks

Concerning Burns

- Symmetrical hands or feet distribution
- Circumferential distribution on limb
- Absence of splash marks
- Sparing in flexion creases
- Area not expected to be in contact with hot object (back of hand, sole of feet, back, buttocks)
- Cigarette shaped

Notes:

Other concerning history features:

- History inadequate, vague or inconsistent or no suitable explanation given
- History not appropriate for child’s age
- Unwitnessed injury
- Delayed presentation beyond what a ‘reasonable’ parent would do
- Injury in under one year
- Previous similar injuries/multiple attendances
- Concerns about child’s demeanour/parent-child interaction

Reading


Who has parental responsibility?

PARENTAL RESPONSIBILITY UK

A mother automatically has parental responsibility (PR) for her child from birth.

A father has parental responsibility if he:
- is married to the child’s mother at time of birth
- is listed on the birth certificate (from Dec 2003 England and Wales, Apr 2002 Northern Ireland, May 2006 Scotland)
- has jointly adopted a child
- has entered into a PR agreement with the mother
- has been issued a PR order from a court

Local authorities have PR if the child is subject to a care order.
Adoptive parents who jointly adopt a child have PR.
Parents do not lose PR if they divorce.
A guardian who will have PR can be appointed by a court.

READING

GMC 0–18 Years: Guidance for all Doctors. Appendix 2. 2007.
What are the Caldicott principles?

CALDICOTT PRINCIPLES

Caldicott principles when handling confidential patient data

Justify the purpose(s) for using patient data
Don’t use patient-identifiable information unless it is absolutely necessary
Use the minimum necessary patient-identifiable information
Access to patient-identifiable information should be on a strict need-to-know basis
Everyone should be aware of their responsibilities to maintain confidentiality
Understand and comply with the law, in particular the Data Protection Act
The duty to share information can be as important as the duty to protect patient confidentiality

NOTES:
A Caldicott Guardian is a senior NHS person who is responsible for ensuring that his or her organization adheres to the Caldicott principles and ensures patient data is kept secure. It is now a requirement for every NHS organization to have a Caldicott guardian.

READING

Information: To share or not to share? *The Information Governance Review*. 2013.
SPINAL
What are the symptoms, precipitants and treatment approach in autonomic dysreflexia?

**AUTONOMIC DYSREFLEXIA**
Phenomenon occurs in patients with spinal injuries at or above T6. An irritating stimulus occurring below the level of injury causes uncontrolled sympathetic activity, in particular severe hypertension. Can be life threatening, including cerebral haemorrhage.

**Symptoms (only one or several symptoms may be present)**
- Headache pounding
- Feeling of doom
- Nausea
- Sweating above cord lesion level
- Bradycardia
- Hypertension
- Goose bumps above or below cord lesion level
- Vision blur or spots
- Anxiety
- Nasal stuffiness
- Dilated pupils
- Chest tightness
- Flushing/blotching above cord lesion level
- Penile erection
- Cardiac arrhythmias
- Contraction of bladder and bowel

**Precipitants include**
- Bladder: Over-distension, blocked catheter/tubing/full drainage bag, UTI, stones (bladder problems are the most common precipitants)
- Bowel: Constipation, digital evacuation, rectal examination, haemorrhoids, appendicitis
- Skin: Pressure sores, abrasions, abscesses, fractures, ingrown toe nails, burns, blisters
- Other: Tight clothes/shoes, pregnancy, labour, sexual activity, menstruation, DVT

**Treatment approach**
- Keep in sitting position.
- Measure BP.
- Thoroughly examine for and remove/treat precipitant. Start with assessing catheter patency. Perform PR with anaesthetic gel.
- Sublingual nifedipine 10 mg or GTN.
- Diazepam for spasms/fits.
- Paracetamol/codeine for analgesia (avoid NSAIDs).
- Discuss with local spinal injury unit.

**NOTES:**
Tetraplegic patients can be relatively hypotensive as their norm, e.g. systolic of 90. A rise of 20–40 mm Hg can be significant.
What are the concerning features in a patient with back pain?

**BACK PAIN RED FLAGS**

- Thoracic pain
- Onset less than 20 yrs or over 55 yrs of age
- Bowel or bladder incontinence/retention
- Reduced anal tone
- Neurological deficit/signs
- Fever or recent treatment for sepsis
- Saddle anaesthesia
- Previous malignancy
- Abdominal aortic aneurysm
- Weight loss (unexplained)
- Progressive pain not relieved by rest
- Immunosuppressed
- Intravenous drug use
- HIV
- Longstanding steroid use
- Structural spinal abnormality
- Recent trauma
- Night pain

**NOTES:**

Surface anatomy landmarks:

- C7: lowest prominent C spine process
- T3: spinous process of scapula
- T7: inferior tip of scapula
- L4: iliac crests
- Tip coccyx: ischeal tuberosities

Deep tendon reflexes:

- Biceps C5, C6
- Supinator C5, C6
- Triceps C7, C8
- Knee, L3, L4
- Ankle S1

**READING**

What are the clinical findings in anterior cord, Brown-Sequard, central cord and cauda equine syndromes?

SPINAL SYNDROMES

Anterior cord syndrome
Motor paralysis, pain and temperature loss. Vibration, proprioception intact.
Anterior cord infarction due to vascular insufficiency from anterior spinal artery.

Brown-Sequard syndrome
Ipsilateral motor paralysis and loss of proprioception and vibration.
Contralateral loss of temperature and pain.
Hemisection of cord, most commonly due to penetrating trauma.

Central cord syndrome
Motor deficit worse in upper limbs than lower. Varying sensory deficit.
Neck hyperextension injuries.

Cauda equine syndrome
Unilateral or bilateral perineal (saddle) sensory changes with motor and sensory deficits in legs. Bladder, bowel and sexual dysfunction. Lower back pain.
Compression of nerve roots below the conus medullaris.

READING
Guideline on the management of alert, adult patients with potential cervical spine injury in the Emergency Department. CEM 2010.
What are the contraindications for the use of activated charcoal?
What are the indications for multidose activated charcoal?

ACTIVATED CHARCOAL
A porous form of carbon that has a large surface area.

Contraindications and cautions
Risk of pulmonary aspiration (causes severe inhalation injury)
Bowel obstruction (cannot ultimately be excreted)
Ingestion of an acid, an alkali or a petroleum product (impedes ability to physically see GI tract damage during endoscopy)
Unpalatable and causes vomiting
Substances not bound to charcoal

MULTIPLE-DOSE ACTIVATED CHARCOAL
Repeated doses of charcoal at 4-hour intervals, up to a total of four doses, have been shown to increase elimination for a small number of drugs (including carbamazepine, digoxin, theophylline).

NOTES:
Activated charcoal binds toxin to prevent stomach and intestinal absorption. Toxins therefore stay within the GI tract and are removed in stools. Interrupts enterohepatic and enteroenteric circulation of some drugs/toxins and their metabolites.
Effect decreases with time post ingestion – therefore is used within one hour of ingestion.
Can be given orally or via NG tube.
Dose adults 50 g, children 1 g/kg (can mix with fruit juice to disguise taste).

READING
http://www.toxbase.org
ACCIDENTAL INJECTION OF EPIPEN (ADRENALINE) INTO THUMB/FINGER

Results in digit pain, paraesthesia, pallor and coolness due to vasoconstriction, occasionally with potential for tissue ischaemia.

Minimal symptoms – Place affected digit into bowl of warm water, observe and arrange follow-up at 24 and 48 hr.

Significant features – Local injection of phentolamine +/- local injection of lignocaine.

NOTES:

Differential diagnosis of angioedema: allergic, hereditary, ACE inhibitors, idiopathic.

Potential anaphylaxis triggers include

- Stings (wasp, bee)
- Food (nuts, eggs)
- Drugs (antibiotics, anaesthetics, vaccines)
- Contrast media
- Latex

Mast cell tryptase levels

Useful for follow-up of suspected anaphylactic reactions.

Send samples after resuscitation has started, at 1–2 hr, at 24 hr/in OPD clinic.

READING


What are the features of Wernicke encephalopathy?

WERNICKE KORSAKOFF

Wernicke encephalopathy
Acute, reversible triad of acute confusion, ataxia, and ophthalmoplegia due to thiamine deficiency.

Causes
- Chronic alcohol excess
- Malnutrition
- Malignancy
- Eating disorders
- Hyperemesis gravidarum
- HIV
- Dialysis

Investigations (to also exclude other differential diagnoses)
- Blood test – FBC, UE, LFT, glucose, arterial blood gas, cholesterol, thiamine levels
- LP
- CT/MRI brain

Management – IV thiamine as Pabrinex (two pairs of vials 1 and 2 diluted in 100 mL of crystalloid IV over 30 min acutely in the ED and continued tds for two days if admitted)

Korsakoff syndrome
Irreversible confabulation, retrograde amnesia and memory loss due to thiamine deficiency

NOTES:
Patients with a history of chronic alcohol ingestion or other risk factors for thiamine deficiency who receive IV dextrose (e.g. to treat hypoglycaemia) should also immediately receive IV Pabrinex.

READING

NICE guidelines (CG100). Alcohol-use disorders: Diagnosis and clinical management of alcohol-related physical complications. June 2010.

METHAEMOGLOBINAEMIA
Methaemoglobinemia results from elevated levels of methaemoglobin in the blood (normal levels <1%). The haemoglobin molecule ferrous (Fe\textsuperscript{2+}), which is oxygen carrying, is oxidized to the ferric form (Fe\textsuperscript{3+}), which reduces oxygen release to tissues.

**Presentation**

Classical presentation
- Chocolate brown-coloured blood
- Cyanosis unresponsive to oxygenation
- pO\textsubscript{2} is normal but measured oxygen saturations are low

Symptoms relate to concentration:
- 10–30% blue-grey discolouration, lethargy, headaches
- >50% seizures, reduced GCS, respiratory depression, arrhythmias
- >70% potentially fatal

Patients with pre-existing cardiac and respiratory disease, sepsis or sickle cell are likely to experience more significant symptoms at lower levels.

**Treatment approach**

- High flow oxygen
- Methylen blue (methylthioninium). Refer to toxbase and discuss with national poisons information service. Reduces ferric back to normal ferrous iron.
- Remove cause/prevent further absorption

**Causes**

- Congenital (autosomal recessive)
- Acquired, including poppers (nitrites), nitrates, trimethoprim, local anaesthetics (prilocaine, lignocaine)

**READING**

http://www.toxbase.org
ORGANOPHOSPHATE POISONING

**Presentation**

Agitation  Bradycardia and tachyarrhythmias  
Bronchial hypersecretion  Confusion  
Diarrhoea  Lacrimation  
Hypotension  Miosis  
Muscle fasciculations  Respiratory failure  
Rhinorrhoea  Salivation  
Sweating  Urinary retention  
Weakness/exhaustion  

Organophosphates are found in pesticides and nerve gases. They inhibit acetylcholinesterase, resulting in the lack of degradation of acetylcholine causing excessive muscarinic and nicotinic receptor stimulation in the ANS and CNS. Toxicity can result from inhalation, ingestion, skin or eye exposure, with symptoms occurring at exposure or up to 12 hours later. Toxicity is a clinical diagnosis based on the presenting toxidrome. Cholinesterase levels can be measured in the blood (send EDTA sample). Avoid self-contamination with PPE and decontamination. Antidotes are atropine (antimuscarinic) and pralidoxime (reactivates AChE by binding to organophosphates).

**NOTES:**

Other antidotes:

- Beta blockers  Glucagon  
- Cyanide  Dicobalt edentate, sodium thiosulphate, hydroxocobalamin  
- Ethylene glycol  Fomepizole, alcohol  
- Extrapyramidal side effects  Procyclidine  
- Hydrofluoric acid  Calcium gluconate gel  
- Iron  Desferrioxamine  
- Methaemoglobinaemia  Methylene blue  
- Opiates  Naloxone  
- Paracetamol  N-acetylcysteine  

**READING**

http://www.toxbase.org includes drug doses of antidotes.
How can the anion gap be applied?

**ANION GAP**
Calculating the anion gap can help differentiate the cause of a metabolic acidosis.
Formula:

$$(\text{Na}^+ + K^+) – (\text{Cl}^- + \text{HCO}_3^-)$$

Normal range = 8–12

**High anion gap metabolic acidosis causes (mnemonic MUD PILES)**
- Methanol
- Uraemia
- DKA
- Paraldehyde poisoning
- Iron poisoning
- Lactic acidosis
- Ethylene glycol poisoning
- Salicylate poisoning

**Normal anion gap metabolic acidosis causes (mnemonic FUSED CAR)**
- Fistulae
- Uretogastric conduits
- Saline administration
- Endocrine – Addison, hyperparathyroidism
- Diarrhoea
- Carbonic anhydrase inhibitors
- Ammonium chloride
- Renal tubular acidosis

**Low anion gap metabolic acidosis causes**
- Hyperparaproteinaemia
- Hypoalbuminaemia
How is the osmolar gap calculated?

**OSMOLAR GAP**

Calculated osmolality = 2 × (Na) + urea + glucose

Normal range 285–295 mOsm/kg

Osmolar gap = (measured osmolality) – (calculated osmolality)

Normal range less than 10

**Causes of high osmolar gap**

- Methanol
- Ethylene glycol
- Ethanol
- Mannitol
- Sorbitol
- Acetone
- DKA
- Propylene glycol (found in IV lorazepam)
- Alcoholic ketoacidosis
- Myeloma (increased plasma proteins)
- Hyperlipidaemia
What are the features associated with a smoke inhalation injury?

INHALATION INJURY

Associated clinical features

- Facial, neck or lip burns
- Breathlessness
- Carbonaceous sputum
- Wheezing
- Blistering/oedema of mouth
- Cough
- Soot visible mouth or nose
- Stridor
- Singed facial/forehead hair
- Hoarse voice
- Nausea/vomiting
- Hypoxia
- Dizziness
- Confusion
- Headache
- Bronchoscopy or laryngeal evidence of contamination

NOTES:
Smoke inhalation causes a combination of a thermal injury, systemic toxicity (e.g. carbon monoxide and hydrogen cyanide poisoning) and lung injury from particle deposits and pulmonary irritants. Consider also associated trauma (falls from height/stairs escaping a blast) and hypothermia (extended cold water first aid). Significant inhalation injuries can be relatively asymptomatic initially, with clinical deterioration at 12 to 24 hrs+. A history of loss of consciousness or entrapment at the scene is significant. Assess for corneal burns with fluorescein and measure carboxyhaemoglobin levels.
What are the indications for starting renal replacement therapy?

INDICATIONS FOR STARTING RENAL REPLACEMENT THERAPY

Hyperkalaemia
Potassium persistently >6.5
Severe acidaemia pH<7.1
Significant uraemia >30 mmol/L
Uraemia complications – pericarditis, myopathy, encephalopathy, neuropathy
Oliguria <200 mL in 12 hours
Significant hyperthermia
Significant hypothermia
Significant volume overload
Persistent severe hypercalcaemia >4.5
Persistent severe hypermagnesaemia
Persistent severe acute hyponatraemia <115 or hypernatraemia >160
Drug overdose, including lithium, aminoglycosides, salicylates, methanol

NOTES:
Not all drugs are removed by haemodialysis.
How much 1% lignocaine can be used for a seven-year-old?  
What are the features of local anaesthetic toxicity?

LOCAL ANAESTHETIC (LA) TOXICITY

Local anaesthetic calculation examples
Maximum dose of 1% lignocaine for a seven-year-old
   Maximum safe dose lignocaine is 3 mg/kg, seven-year-old weight 22 kg, therefore = 66 mg
   1% lignocaine solution contains 10 mg/mL, therefore maximum volume is 6.6 mL
Maximum dose of 0.5% bupivacaine for a five-year-old
   Max safe dose bupivacaine is 2 mg/kg, five-year-old weight 18 kg, therefore = 36 mg
   0.5% bupivacaine solution contains 5 mg/mL, therefore maximum volume is 7.2 mL

Volumes calculated are the maximum safe volume, not the volume aiming to be used.

Presentation of LA toxicity
Neurological symptoms (CNS excitation followed by CNS depression) then cardiovascular symptoms
   Perioral and tongue paraesthesia, light-headedness, visual disturbances, headaches, restlessness
   Dysarthria, tinnitus, metallic taste
   Muscle twitching, drowsiness
   Seizures, reduced GCS
   Respiratory arrest
   Tachycardia, hypertension then hypotension
   Cardiovascular depression and arrhythmias

NOTES:
LA toxicity occurs following an excessive dose or accidental intravascular administration.
Early features can be subtle in children or sedated patients, with first symptom as CVS collapse. Large bolus injections can cause simultaneous CNS and CVS features. Toxicity can also occur late at 10–25 mins post injection.
Drug concentration is expressed as a percentage (e.g. bupivacaine 0.25%, lignocaine 1%).
Percentage is measured in grams per 100 mL (i.e. 1% is 1 g/100 mL).
Therefore 1 mL of 1% lignocaine contains 10 mg of lignocaine.
WHAT ARE THE FEATURES OF LOCAL ANAESTHETIC TOXICITY?
The FCEM Notebook: Revision notes and clinical resource for emergency physicians is the essential aid to passing the Fellowship of the College of Emergency Medicine (FCEM) examination. Containing short but challenging clinical questions and scenarios followed by fully reasoned answers and explanations, the book is brimming with a wealth of clinical information, along with hints and tips.

Following the College of Emergency Medicine curriculum and uniquely written for emergency doctors, the clinical cases contain only the most recent evidence-based emergency medicine approaches, allowing for rapid revision and instant access to key information.

- Includes challenging clinical cases for practice
- Provides reasoned answers and explanations
- Consolidates knowledge ready to apply in the examination
- Contains hints and tips to excel in the examination

This portable, practical book is an essential revision guide for all those studying for the MCEM and FCEM examinations and is a valued day-to-day clinical resource for doctors in the emergency department.

About the Author

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