Intern manual
4th edition

Immediate management
of surgical emergencies

Victorian Surgical Consultative Council
www.health.vic.gov.au/vscc

Contents

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Introduction

Purpose of this manual

Welcome to our updated 'survival guide' for Interns, now in its fourth edition. These notes have proved popular with junior medical staff in the initial treatment of urgent surgical conditions. Many of these problems are life-threatening and stressful, and here you will find a safe, practical approach. What you do while waiting for experienced help will save lives.

Background

The Victorian Surgical Consultative Council's (VSCC) specialists have all experienced and responded to such emergencies. We see case reports whose outcome would have been better for involving more senior staff at an early stage, and in your training you should take the opportunity to benefit from their experience.

The intern is usually the first to arrive, and sometimes is the only doctor available. It is in your hands to recognise a serious problem and begin treatment while summoning help or arranging transfer. In some hospitals the immediate management will soon be taken over by MET, ICU or senior staff.

This pocket guide was developed in 2006 by Shepparton-based VSCC member and general surgeon Mr Tony Heinz with the help of his medical students, interns and registrars. The VSCC acknowledges the continuing roles of Mr Heinz, Professor John Royle and the Department of Health, which funds the publication, and the Postgraduate Medical Council.

The content is also found on the VSCC's website at <www.health.vic.gov.au/vscc>. Undergraduates and HMOs are also finding this a useful reference.

Good luck with the challenges ahead! Be alert to clinical deterioration or unusual features in your patients, and remember to call for help early.

Mr Peter L Field, FRACS, Vascular Surgeon,
Chairman, Victorian Surgical Consultative Council
November 2013

Acute external haemorrhage – vascular surgery, penetrating trauma

Theory

Penetrating trauma requires urgent resuscitation, stabilisation and surgical exploration.

If the patient remains unstable, life-threatening haemorrhage may need to be managed surgically, simultaneously with ongoing resuscitation.

Haemorrhage following vascular surgery can be controlled with focal pressure over the bleeding point until definitive help arrives.

Immediate intern management

Attend patient and make rapid assessment.

If large volume haemorrhage, call MET code.

Airway

1. Secure.

Breathing

1. Give oxygen by mask.
2. Ensure no pneumothorax (if penetrating trauma).
3. If pneumothorax present, patient needs urgent chest tube (call code MET while organising chest tube setup).

Circulation

1. Elevate bleeding site.
2. Put pressure focally over site of bleeding.
3. Obtain IV access (X–Match lost blood volume + extra two units), (FBE, U&E, LFT, INT).
4. Fluid resuscitate – 500 ml Gelofusine stat, followed by N. Saline 1 L stat.
5. Reverse reversible clotting abnormality.

Assessment

1. Clinical history.

Other

1. Call surgical registrar and unit registrar.
2. Nil orally.
3. Notify ICU about patient.

Clinical features (obtain rapidly)

History

- Reason for bleeding
  - Trauma/stabbing
  - Post-surgery (nature of surgery)
- Site and estimate of blood loss
- Penetrating trauma – site/implement/direction/force
- General symptoms related to possible organs injured
- History of reversible clotting abnormality (for example, Warfarin therapy or other blood thinning agents)

Examination

- Haemodynamic status
- Site of blood loss
- Relationship to major neurovascular structures
- Site and path of penetrating trauma
- ? Depth (difficult to determine)
- Status of possible organs injured – lung/heart/liver/spleen/kidney

Other (obtain later)

Neurological features distal to injuries

Further definitive management

Airway/breathing

Circulation

- Pressure over bleeding point
  - Bandage (+/- pressure dressing)
- IV access and resuscitation
  - Gelofusine
  - Crystalloid
  - Blood
- X-Match and book theatre

Assessment of injury, mechanism and possible injuries

Surgical exploration

- Arterial tourniquet for limb bleeding
- Extend wounds and assess injured/devitalised structures
- Proximal and distal control for bleeding major vessels
- Conservative debridement devitalised tissues
- Second look and exploration 24–48 hours
- Reconstruction
  - Primary
  - Secondary

Other

- Rehabilitation
- Counselling for trauma

Techniques for resuscitation

1. Multiple large bore IV cannulas (>16 G)
2. Increase height of IV pole
3. Infusion pumps/Imed pumps
4. Rapid volume infuser
5. Use crystalloid, colloid or blood once available (especially after 1.5 L of fluid resuscitation)

- For large volume resuscitation, consider warming fluids

Acute upper airway obstruction

Theory

This is the most serious of all emergency situations and needs immediate assessment and management.

If a patient has no patent airway they will arrest and die rapidly.

Establishing a patent airway is the first step in the management of any patient, especially in the emergency or trauma situation.

Patients on the ward are susceptible to acute airway obstruction, especially if they have an altered conscious state due to opiate analgesia, confusion or the residual effects of anaesthetic agents.

This is the most common cause of acute airway obstruction in hospital patients and is managed by simple airway manoeuvres – chin lift, jaw thrust and head tilt.

Management focuses on relieving the obstruction and establishing a patent airway.

Immediate intern management

Attend patient and make rapid assessment.

If patient unconscious/severe respiratory distress/ respiratory arrest

1. Tell nursing staff to call Code Blue then move to head end of bed.
2. Perform triple manoeuvre – chin lift, jaw thrust, head tilt.
3. Clear mouth – suction secretions, sweep out foreign body from pharynx.

If it is an acute obstruction and the above fail, then a definitive airway is required.

If mechanically obstructed

Surgical airway

- Cricothyroidotomy
- Tracheostomy

If acute neck haematoma

Open neck wound down to and including the deep fascial sutures.

If still obstructed and trachea on view attempt to incise and insert endotracheal tube.

If not mechanical

Bag and mask patient with oxygen using Guedel airway.

These manoeuvres can maintain an airway until help arrives.

Once more experienced staff are available the patient requires intubation and insertion of an Endotracheal tube.

If unable to intubate, can try to insert laryngeal mask but if this does not secure airway:

Surgical airway

- Cricothyroidotomy
- Tracheostomy

Clinical features (obtain rapidly)

History

- Basic information about patient
- Events resulting in obstruction
- Recent drug administration/operation

Examination

- Patient in extremis
- Stridor
- Respiratory distress

- Cyanosis
- Drooling
- Swelling face/tongue

Causes

- Acute mechanical obstruction
- Sputum plug
- Altered conscious state
- Laryngospasm
- Angio-oedema
- Disruption ETT/tracheostomy
- Neck haematoma post neck surgery
- Trauma
- Burns
- Tumours

Investigations

No Investigation should delay treatment.

1. ABG
2. Basic blood tests
3. CXR
4. ECG

Cricothyroidotomy

- Feel for the prominence of the thyroid cartilage
- Incise horizontally in space inferior to thyroid cartilage, (this is cricothyroid membrane)
- Insert handle of scalpel into incision and twist to open incision and allow insertion of endotracheal tube

Points for consideration – call for help early

- An airway is required by any means possible
- Choice depends on the cause as illustrated in 'immediate intern management'

- Suction and simple airway manoeuvres may be enough to establish an airway especially in a patient with secretions or a tracheostomy
- Ideally intubation and advanced airway management should be performed by highly experienced staff
- If simple airway measures are unsuccessful then a definitive airway is required
- Intubation can be attempted but in mechanical obstruction is unlikely to be successful
- A surgical airway is definitive
- First line is cricothyroidotomy
- A tracheostomy may be performed if there is sufficient time and adequate staff are readily available
- Don't confuse stridor (fixed airway noise on inspiration) with wheeze (fixed airway noise on expiration)

Air embolism – central line disruption

Theory

Subclinical air embolism is common. Symptomatic air embolism post CVC line insertion <2%.

Small amount air – subclinical.

Intermediate amounts:

- Collect in pulmonary circulation
- Leads to pulmonary vascular injury: pulmonary vasoconstriction, pulmonary hypertension, endothelial injury and pulmonary oedema

Large amount (3–8 ml/kg) – acute right ventricular outflow obstruction, cardiogenic shock and circulatory collapse.

In setting of ASD/VSD/patent foramen ovale, even small amounts of air can cause peripheral embolisation to territory supplied by blood vessel.

Immediate intern management

Attend patient and make rapid assessment.

If patient unconscious/severe respiratory distress/arrest

1. Tell nursing staff to call Code Blue then move to head end of bed.
2. Triple manoeuvre – chin lift, jaw thrust, head tilt.
3. Bag and mask patient with oxygen.
4. Start CPR if arrested.

Patient conscious/mild distress

1. Stop central line infusion/clamp line.
2. Give 100% oxygen by mask and place patient in trendelenburg position (head down).
3. If patient increasingly distressed:
   Rotate to left hand side with right side facing upwards (left lateral position). (Traps air at apex of ventricle).
   Ask nursing staff to call MET code.
4. Get crash trolley, obtain IV access and send off blood tests.
5. Notify unit registrar, ICU registrar and anaesthetist on call.

Clinical features (obtain rapidly)

History

- Incident following CVC line manipulation
- Chest pain
- SOB
- Palpitations
- Neurosensory symptoms
- Events preceding related to CVC line
- Reason for CVC line
- Cause for hospitalisation

Examination

- Airway
- Breathing; ? respiratory distress/failure
- Assessment haemodynamics
- Cardiorespiratory examination
- Cyanosis/mill wheel murmur
- Agitation and altered conscious state

Other (obtain later)

- CVC line
  - Type, position, ? last CXR for check position
  - When last used

Investigations

1. ABG
   - Low PaO_2
   - High $PaCO_2$
   - Metabolic acidosis
2. ECG
   - Tachycardia
   - R axis deviation
   - RV strain
   - ST depression
3. CXR
   - APO
   - Air in pulmonary tree
4. Basic blood tests

Further definitive management

Immediate cardiorespiratory support and resuscitation

- Check CVC line while CVC clamped:
  - ? Moved/dislodged
  - Attempt to aspirate air through CVC (never inject)
  - (The catheter may have to be advanced to achieve this. Catheter advancement should only be performed in a monitored environment and using sterile technique)

- In cardiovascular collapse:
  External cardiac compression may expel air from the pulmonary outflow tract into the pulmonary circulation re-establishing pulmonary flow.
  Support the right heart with IV fluids and beta-adrenergic agents.
- Admit patient to ICU
- Consider hyperbaric oxygen therapy (liaise with appropriate facility)

Remember there are more common causes for acute SOB in a patient with a central line: for example, pneumothorax, pulmonary embolus, acute pulmonary oedema, sputum retention and anaphylaxis. Initial assessment should be aimed at ruling out these other causes and then, if they are not present, considering the possibility of air embolus.

Anaphylaxis

Theory

The syndrome of anaphylaxis is caused by generalised mast cell degranulation with subsequent release of histamine in varying amounts causing a systemic response.

A number of mechanisms produce mast cell degranulation but the principal mechanism involves a complement fixing reaction between an antigenic stimulus and immunoglobulin E (IgE).

The combination, the rate of progression and the severity of signs and symptoms are variable; therefore, all patients with anaphylaxis who are still deteriorating are potentially at risk of death and require prompt and appropriate treatment.

Appropriate management of anaphylaxis will depend on the severity of the clinical features. A number of graded scales of severity have been proposed to allow appropriate titrated therapy. It is important to commence treatment, if required, with an appropriate initial dose of adrenaline and fluid bolus. The most current schedule of treatment guidelines for anaphylaxis is to be found on the Australian and New Zealand Anaesthetic Allergy Group (**ANZAAG**) website.

The ABC mnemonic will help in remembering some of the main clinical features

Airway Angio-oedema of the larynx, potentially leading to a progressive and rapid airway obstruction with respiratory arrest.

Breathing Bronchospasm, causing lower airway obstruction and potentially severe hypoxia.

Circulation Vasodilation and increased vascular permeability, resulting in hypotension, tachycardia (but occasionally bradycardia) and cardiovascular collapse (shock).

Immediate intern management

Attend the patient and make a rapid assessment. The treatment required will depend on the patient's clinical findings. A suggested protocol is indicated below.

Patient unconscious

1. Tell the nursing staff to call Code Blue then move to the head of the bed.
2. Perform the triple manoeuvre – chin lift, jaw thrust and head tilt – and assess for respiration.
3. Bag and mask the patient with oxygen. Insert oral airway if needed. (Expert assistance will be needed if the patient needs intubation.)
4. Commence CPR.
5. Obtain IV access. (Save some blood for tests as indicated including tryptase level.)
6. Give IV adrenaline 0.5–1 mg and fluid bolus of 20 m/kg of N saline. (N saline and 4% albumen are appropriate in anaphylaxis.)
7. Set up an adrenaline infusion, 10–15 mcg/min and adjust up or down.
8. For further management see **ANZAAG** guidelines.

Patient conscious

1. Tell the nursing staff to call code MET.
2. Give oxygen by mask.
3. Call for a crash trolley, obtain IV access. (Save some blood for tests as indicated including tryptase levels.)
4. Administer adrenaline as per ANZAAG guidelines.
5. Alternatively, as a guide, if rash is the only feature of anaphylaxis and the patient has normal cardiovascular parameters no adrenaline will be required.
6. If the patient is hypotensive commence IV adrenaline at 25–50 mcg. Repeat as necessary and commence an adrenaline infusion.

7. Bronchospasm can be treated with salbutamol, but if refractory may require adrenaline.
8. If IV access cannot be obtained the second best option is IM adrenaline (10 mcg/kg).

Clinical features

The clinical features can be variable in severity and nature.

History

- Rapid onset of dyspnoea consistent with acute bronchospasm or airway oedema
- Collapse
- Known allergies

The above clinical features are especially evident if associated with recent patient exposure to known allergens, a patient with known multiple allergies or recent drug administration.

Examination

- Stridor
- Respiratory distress
- Hypotension, tachycardia, arrhythmia
- Collapse
- Rash (general or focal, erythematous or urticarial)

Suspect the diagnosis when there are two or more of:

- itch, urticaria
- angio-oedema
- upper airway swelling
- hypotension
- bronchospasm
- abdominal features, which can include acute diarrhoea.

Further definitive management

Airway

- Airway
- Patient may require intubation if cardiovascular collapse or if angio-oedema is progressing to airway compromise. Will require expert management.
- Sometimes nebulised adrenaline 5 mg may avert the need for airway intervention.
- Early intervention will usually avoid the need for a surgical airway.

Breathing

- Oxygen by mask

Circulation

- IV access may include central vein cannulation to assess ongoing fluid requirements.
- Routine treatment for more severe reactions usually involves an adrenaline infusion. Range can be 1 mcg/min up to several hundred mcg/min.
- Occasionally other drugs are included to assist with circulatory support such as noradrenaline and vasopressin, but these lack clinically validated data.
- Complex management may include transthoracic echocardiography, mechanical cardiac support and mechanical respiratory support in the form of extracorporeal oxygenation (ECMO).

Other

- Severe reactions usually require a period of ICU observation and/or management.
- Intubated patients remain intubated until airway oedema subsides.
- Glucocorticoids are often used but have no place in initial management and lack level one evidence for efficacy.
- Allergy testing must be offered to the patient to determine the triggering agent.

- Appropriate documentation must be provided for the patient prior to discharge from hospital.
- Adequate patient education must be provided including the use of an EpiPen if thought appropriate.

Aspiration

Theory

Aspiration of gastric contents into the pulmonary alveolar spaces leads to a severe chemical pneumonitis involving gram negatives and gram positives including staph aureus and possibly MRSA.

There is a spectrum of severity from severe pneumonia to ARDS and cardiopulmonary collapse.

Patients who are weak, unwell, debilitated, elderly or who have an altered conscious state are predisposed to aspiration.

Anatomical predisposition to the apical segment in right lower lobe due to its anatomical position.

Immediate intern management

Attend patient and make rapid assessment.

If patient unconscious/severe respiratory distress/ respiratory arrest

1. Tell nursing staff to call Code Blue then move to head end of bed.
2. Perform triple manoeuvre – chin lift, jaw thrust, head tilt.
3. Clear mouth – suction secretions, vomitus.
4. Insert Guedel airway/nasopharyngeal airway and administer oxygen.
5. Bag and mask patient until help arrives.
6. Intubate and insert cuffed ETT.

If patient conscious

1. Clear airway of secretions with suction or by turning patient on their side.

2. Perform basic airway manoeuvres to assist patient in obtaining a clear airway.
3. Administer oxygen by mask to maintain oxygen saturations.
4. Perform continuous pulse oximetry.
5. Carry out rapid clinical assessment.
6. Insert IV line and take set routine blood tests, including ABGs.
7. Inform unit registrar of events.

Other options to be considered

- Insert nasopharyngeal airway.
- Attempt to suction lungs.
- Endotracheal intubation/flexible bronchoscopy.
- Direct tracheal suction.

Predisposing conditions

- Altered conscious state
  - Alcoholism/drug OD
  - Seizures/CVA
  - GA/endoscopy/bronchoscopy
  - Trauma
- Mechanical conditions
  - Bowel obstruction
  - ETT/NGT
  - Tracheostomy
  - Oesophageal disorders (stricture/fistulae/GORD)
  - Impaired swallow (post CVA)
- Neurological conditions
  - Multiple Sclerosis
  - Myasthenia
  - Parkinson's
  - Dementia

Clinical features (obtain rapidly)

History

- Basic information about patient – reason for admission, medical problems
- Events surrounding incident
  - Sudden SOB/cough
  - Vomiting
- Recent drug administration/operation

Examination

- Signs respiratory distress
- Low oxygen saturations and tachypnoea
- Fever (late sign)
- Decreased air entry at bases especially right

Underlying predisposition

- Generally unwell/debilitated
- Altered conscious state
- Neurological conditions
- Bowel obstruction

Investigations

1. ABG
2. CXR
3. Basic blood tests
4. Sputum and blood cultures

Further definitive management

- Consider ICU admission if high oxygen demands or if unstable
- IV antibiotics
  - Ceftriaxone and metronidazole
  - Imipenem and vancomycin
- NGT if has bowel obstruction
- Respiratory support
  - BiPAP, CPAP, intubation and ventilation

In setting of ARDS:

- There is a role for steroids – IV hydrocortisone (would be decided in conjunction with ICU and medical staff)

Cardiac arrest

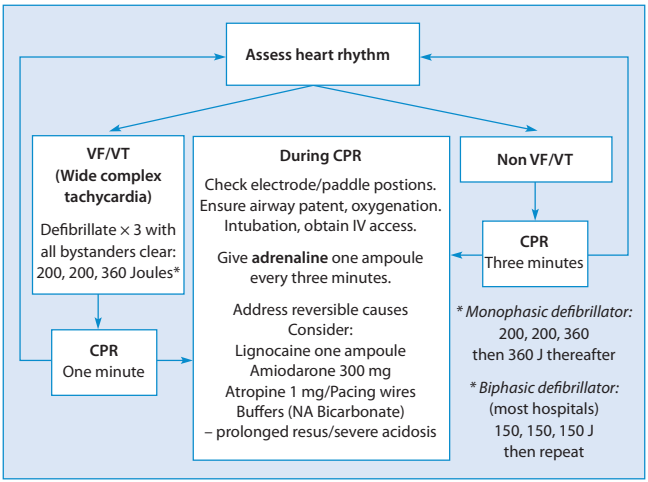
Theory

Abrupt cessation of cardiac function which, unless promptly re-established, will lead to death.

The brain will suffer irreparable damage after four to five minutes without oxygen so cardiopulmonary resuscitation should be started as soon as possible to maintain oxygen delivery to the tissues, while attempts at restarting pump function occur.

Immediate intern management

1. Attend patient and make rapid assessment.
2. Tell nursing staff to call Code Blue then move to head end of bed after removing headboard.
3. Perform triple manoeuvre – chin lift, jaw thrust, head tilt.
4. Clear mouth – suction secretions, vomitus using Yanker Sucker.
5. Insert Guedel airway/nasopharyngeal airway and administer oxygen via bag and mask (found in crash trolley).
6 Check for pulse – if absent begin cardiopulmonary resuscitation. (Single or multiple operators: [two breaths for every 15 compressions]; two or more operators [one breath for every five compressions])
7. Simultaneously place contact paddles on patient from defibrillator and cardiac monitor.



Precipitating factors

1. AMI
   (Reduced coronary artery flow)
2. Low cardiac output states
   Heart failure
   Hypovolaemia
   Cardiac tamponade
3. Metabolic abnormalities
   Hypoxia (PE/pneumothorax)
   Acidosis
   Electrolytes (low or high potassium)
4. Toxins
   Drugs (proarrhythmics)
   Cardiac toxins (cocaine, digoxin)

Reversible causes

- Hypoxia
- Hypovolaemia
- Hyperkalaemia/hypokalaemia
- Other metabolic disturbances
- Hypothermia
- Tension pneumothorax
- Tamponade (cardiac)
- Toxic disturbance
- Thromboembolic/mechanical obstruction

Asystole

- DCR – no role
- Assess reversible causes during CPR
- Adrenaline one ampoule every three minutes

EMD/PEA (pulseless electrical activity)

- DCR – no role
- Assess reversible causes during CPR
- Adrenaline one ampoule every three minutes
- Atropine 1 mg IV every three minutes

Usually intern role in resuscitation is to call a Code Blue and perform basic airway manoeuvres until help arrives. Once help arrives, the senior running the arrest will direct you. Things to do in an arrest as an intern include obtaining a second large bore IV line, sending off basic blood tests and obtaining an ABG, and assisting in CPR.

Compartment syndrome

Theory

Trauma can lead to muscle ischaemia. Oedema occurs from release of free radicals increasing vascular permeability as blood flow is re-established. This results in muscle swelling.

In rigid compartments this can lead to compartment syndrome.

Muscle swelling leads to venous outflow obstruction, decreased arterial flow, further muscle ischaemia and ultimately infarction and necrosis.

The classic site is the calf, or the shin, due to its inexpansible fascial compartments; however, it can occur in the forearm.

Immediate intern management

Attend patient and assess.

Suspect the diagnosis in any patient complaining of increasing pain in a limb post injury or surgery.

1. Split bandage or plaster down to skin end to end.
2. Elevate the limb well above heart level.
3. IV access and basic blood tests.
4. Nil orally.
5. Call surgical registrar and unit registrar.

If in a well supported clinical environment, you may wish to discuss the situation before splitting a plaster or removing a bandage.

Clinical features

History

- Basic patient information
- Recent operation/trauma
- Burning/throbbing pain progressing rather than improving
- Pain at rest

Examination

- Swollen limb
- Hardness on palpation
- Pain on passive stretching of muscle in compartment (most sensitive sign) (Dorsi/plantar flexion)
- Pulses may be present or absent
- Nerve paralysis in compartment

Causes

- Trauma (tibia and fibula)
- Intensive muscle use
    - Tetany
    - Seizures
    - Intense exercise
- Haemorrhage
- Plaster casts, bandages
- Envenomation

Tonometer

A tonometer can be created if one is not available by connecting an arterial pressure transducer to a primed arterial line tube and a spinal needle. Once correctly zeroed, the spinal needle can be inserted into a tissue compartment to record the pressure in the compartment.

Measure rules of thumb:

- <30 mmHg = normal compartment pressure
- 30–40 mmHg = compartment pressure elevated
- >40 mmHg = fasciotomy indicated
- Compartment pressure <40 mmHg below diastolic BP = fasciotomy indicated

Further definitive management

If diagnosis is obvious (uncommon):

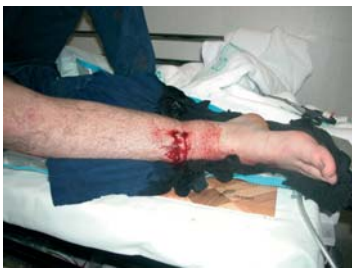
- Patient requires urgent fasciotomy

If diagnosis is unclear:

- Measure compartment pressures if equipment available.

There are four compartments in the calf: peroneal, anterior, posterior superficial and posterior deep. The pressures in each compartment can be measured using a tonometer.

If doubt remains or diagnosis has been made → urgent four-compartment fasciotomy.



Metabolic disturbances – 1. hypocalcaemia, 2. hypoglycaemia, 3. hyperglycaemia

1. Hypocalcaemia

Immediate intern management

Suspect diagnosis.

1. Obtain IV access and basic blood tests (including serum calcium).
2. Administer 20 ml of 10% calcium gluconate.
3. May need to administer magnesium together with calcium.
4. Recheck serum calcium levels in four hours or if symptoms return.

Clinical features

History

- Recent neck surgery, causes listed below
- Tingling around hands, feet, mouth

Examination

- Trousseau's sign

  Carpal spasm within three minutes by BP cuff > systolic blood pressure around arm
- Chovsteks' sign

  Twitching of facial muscles by tapping over branches of facial nerve

Causes

- Acute pancreatitis
- Hypoparathyroidism (post thyroidectomy)
- Hypoalbuminaemia
- Vitamin D deficiency
- Chronic renal failure

Other

- Causes increasing nervous excitability leading to tetany

Further management

Investigations

- Check renal function
- PTH level
- Amylase +/- Ranson's criteria if in setting of pancreatitis, notify surgical registrar as could be a sign of worsening pancreatitis

Management

- Notify appropriate medical staff (registrar)
- Consider ICU review if pancreatitis
- Medical referral if in setting of renal failure or non-surgical cause

2. Hypoglycaemia

Common in diabetic patients

Common causes:

- Fasting status
- Incorrect insulin dosage

Immediate intern management

If conscious

1. Check BSL – finger prick.
2. If alert and not fasting, administer oral glucose solution and Lucozade.

If unconscious

1. Airway/breathing/circulation (ABC).
2. Check BSL – finger prick.
3. Obtain IV access and send off basic blood tests.
4. Administer 25 g of 50% Dextrose. Patient should wake up on end of needle.
5. Consider continuing with 10 per cent Dextrose infusion.

Check BSLs frequently (30 minutely, then hourly if stabilising).

Clinical features

History

- Diabetic, insulinoma
- Recent change to insulin regimen
- Fasting
- Anxiety, tremor
- Palpitations
- Fatigue

Examination

- Tachycardia
- Sweaty
- Confused
- Pallor
- Seizure
- Loss of consciousness

Further management

- Endocrine referral
- Check HbA1c
- Organise diabetic educator
- Liaise with anaesthetist if fasting for surgery

3. Hyperglycaemia

Hyperglycaemia can be divided into:

i) Diabetic ketoacidosis (DKA)

ii) Hyperosmolar non-ketotic-coma (HONKC)

i) DKA

- Occurs in Type I diabetes mellitus
- Results in insulin deficiency with absolute or relative increase in glucagon

Immediate intern management – DKA

If conscious:

1. Check BSL – finger prick.
2. Obtain IV access and send off basic blood tests
   (U&E, glucose, ketones, FBE).
3. ABG
   Metabolic acidosis
   Anion gap (HCO3- <10 mmol/L).
4. Dipstick urine
   Ketonuria.
5. IV fluids
   1 L N saline over 30 minutes
   1 L N. saline over one hour
   Then reassess fluid status.

6. Strict fluid balance chart
   +/- IDC (catheter) to monitor urine output.

Once the diagnosis of DKA has been established, seek advice from senior medical staff, including endocrinology team.

7. Start insulin infusion
   (Actrapid – short acting)
   100 units Insulin in 100 mls N. Saline (one unit = 1 ml).

Infusion protocol

BSL	
<7.0	Cease infusion and recheck BSL in one hour
7.1–9.0	1 ml/hour
9.1–11.0	2 ml/hour
11.1–13.0	3 ml/hour
13.1–15.0	4 ml/hour
15.1–17.0	5 ml/hour
17.1–20.0	6 ml/hour
>20.1	Call help

8. Once BSL <15.0 start 5% dextrose IV at 10/24 rate.
9. Potassium replacement.

Potassium	
K<4.5	30 mmol into IV fluid flask

10. Repeat U&E or venous gases to assess potassium.

Further replacement may be needed.

If unconscious:

1. Do above plus ABC.
2. Notify ICU.
3. Management may include bicarbonate replacement in ICU for severe acidosis.

Clinical features – DKA

History

- Diabetic, past history, recent stress
- Compliance, last insulin dose
- Polyuria, polydipsia, LOW
- Nausea, vomiting
- Blurred vision, cramps
- Abdominal pain

Examination

- Dehydration
- Acetone breath
- Kussmaul breathing
- Hypothermia
- Confusion, comatose

Precipitating factors include:

- Cessation of insulin
- Infection
- Surgery
- Alcohol
- Emotional stress

Issues are

- Metabolic acidosis
- Absolute potassium deficiency
- Dehydration
- Osmotic shifts

Further management

- Cease insulin infusion when:
  - pH normal
  - BSL normal, <7.0
  - Normal bicarbonate
  - Conscious and able to resume normal diet and insulin (Ketones may still be present in urine for 48 hours)
- Find precipitating factors
- Diabetic education
- Refer to appropriate medical staff – endocrinology unit

ii) HONKC

- Occurs in elderly Type II diabetics
- Causes sustained osmotic diuresis causing profound dehydration when patients are unable to drink sufficient water to replace urinary losses

Immediate intern management – HONKC

If conscious

1. Check BSL – finger prick.
2. Obtain IV access and send off basic blood tests (U&E, glucose, FBE).

3. IV fluids need to be administered with care due to age and concurrent cardiac illness.

   Aim for 2–3 L's in first two hours if able to tolerate volume.

Inform medical team and ICU early because patient may need invasive monitoring.

4. Strict fluid balance chart +/- IDC (catheter) to monitor urine output.
5. Start insulin infusion

   (Actrapid – short acting)

   100 units Insulin in 100 mls N. Saline (one unit = 1 ml).

If unconscious:

1. Do above plus ABC.
2. Notify ICU.

Common causes

- Intercurrent illness; for example, stroke or infection which worsens hyperglycaemia and prevents oral intake
- Tube feeding high protein diets
- Peritoneal dialysis
- High carbohydrate intake
- Osmotic agents (mannitol, urea)

Mortality is higher than DKA due to elderly population and co-morbidities (>50%)

Issues are

- Profound dehydration
- Mild acidosis may be present
- Plasma glucose is very high >50 mmol/L
- Serum osmolality is high

Pericardial tamponade

Theory

This is an emergency situation requiring immediate assessment and management.

Management focuses on urgent decompression of the pericardial cavity. Failure to do this leads to rising intra-cardiac pressure, reduced diastolic ventricular filling, decreased cardiac output, and ultimately cardiac arrest.

Immediate intern management

Attend patient and make rapid assessment.

If patient unconscious/arrest

1. Tell nursing staff to call Code Blue then move to head end of bed.
2. Perform triple manoeuvre – chin lift, jaw thrust, head tilt.
3. Clear mouth – suction secretions.
4. Insert Guedel airway and bag and mask.
5. Check pulse and if arrested start CPR.

If conscious/profoundly hypotensive/help arrives

Assess patient – continous pulse oximetry, vital signs

1. Tachycardia, hypotension
2. Raised JVP/ distended neck veins
3. Muffled/ absent heart sounds

} = Pericardial tamponade

Pericardial tamponade (If patient unstable and deteriorating – usually this would be done by senior staff after echo confirmatory evidence)

1. Call Code Blue.
2. Organise for needle pericardiocentesis:
   Obtain the longest and largest IV needle available.
   Obtain sterile set up.
3. Obtain crash trolley.
4. Obtain IV access and IV fluid resuscitate.
5. Nil orally.

By now help should have arrived. Proceed to definitive management.

Clinical features (obtain rapidly)

History

- Basic information about patient
- Recent AMI/underlying medical problems

Examination

- Pt in extremis
- Hypotension, tachycardia
- Raised JVP (central venous hypertension)
- Muffled heart sounds

Other signs

- Absent apex beat
- Kussmaul's sign (JVP rising with inspiration)

Causes

- Post transmural AMI and ventricle rupture
- Trauma
- Thoracic aortic dissection

- Cancer
- Post-op cardiac surgery – notify the surgeon

Pericardiocentesis

1. Use sterile technique
2. Insert large bore needle (may need to use lumbar puncture needle) connected to syringe immediately inferior to xiphisternum directed towards left shoulder tip
3. Continuously aspirate from syringe
4. When in space, a rush of blood should indicate access to pericardial space
5. Improvement in BP should accompany successful decompression of the pericardial cavity

Investigations

1. CXR – enlarged heart
2. ECG – low voltage QRS throughout
3. Echocardiogram

Further definitive management

- There may be time to establish the diagnosis with an echocardiogram prior to needle pericardiocentesis. This decision should be made by more senior staff
- Pericardiocentesis performed as described previously
- Definitive management requires exploratory thoracotomy/ sternotomy to identify and control the cause of bleeding
- This may include creation of a pericardial window to definitively drain a pericardial effusion. This can be done using a variety of approaches (subxiphoid, transcostal, or via thoracoscopic approaches)
- These procedures should be done in theatre

Post-operative neck haemorrhage from thyroid/carotid/neuro/ear nose throat surgery

Theory

This is a dramatic and sudden threat to life.

Problems stem from the need to establish a patent airway and the effect neck haemorrhage has on the patient's airway.

Although an expanding haematoma in the neck can directly compress the trachea, obstruction to the airway usually occurs due to venous congestion, which causes laryngeal oedema and tracheal obstruction.

A neck haematoma need not be very large to cause this.

Pressure applied to frank bleeding from the neck can compromise a patient's airway.

Immediate intern management

Attend patient and make rapid assessment.

If patient unconscious/severe respiratory distress/ STRIDOR/respiratory arrest

1. Tell nursing staff to call Code Blue then move to head end of bed.
2. Perform triple manoeuvre – chin lift, jaw thrust, head tilt.
3. Clear mouth – suction secretions.

If the airway is compromised then the priority is the establishment of a patent airway by either intubation or creation of a surgical airway. Control of haemorrhage is a secondary priority once an airway is established.

As a matter of urgency, in the ward, remove any skin staples and cut the sutures from the wound down to and including the deeper fascial sutures.

Scoop out any blood clot.

This should release the pressure against the trachea and relieve the upper respiratory tract obstruction.

If still obstructed and trachea on view, attempt to incise and insert endotrachael tube.

Neck haemorrhage without airway obstruction

1. Call Code Blue.
2. Assess airway.
3. Assess breathing.

If stable put direct pressure over the bleeding point.

This may compromise the patient's airway.

4. Give oxygen by mask and sit patient up.
5. Transfer to theatre for control of haemorrhage and resuture of wound. Escort the patient to the theatre urgently.

Clinical features (obtain rapidly)

History

- Basic information about patient
  - Nature of past surgery
  - History of event
- Neck swelling or pain
- SOB

Examination

- Pt in extremis
- Stridor (take this VERY seriously)
- Respiratory distress

- Neck swelling
- Frank haemorrhage
- ? Drain tube

Causes

- Primary haemorrhage
  - Failure of surgical technique
  - Slipped tie
  - Unrecognised bleeding
- Secondary haemorrhage
  - Infection
  - Slipped tie
- Cancer
- Erosion of foreign material (graft, tracheostomy)

Investigations

1. ABG
2. CXR = less useful
3. Basic blood tests

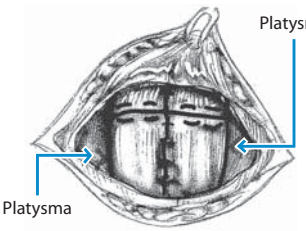
An ECG can be done at a later time.

Notes

The patient will not need analgesia when a fresh surgical wound is opened on the ward.

This is a lifesaving manoeuvre and should be performed by the first doctor at the scene of a patient with signs of upper respiratory tract obstruction post neck surgery.

Removal of sutures is done in the ward. There is insufficient time to transfer patient to theatre. See over the page for illustrations.



After thyroid surgery, sutures need to be removed.

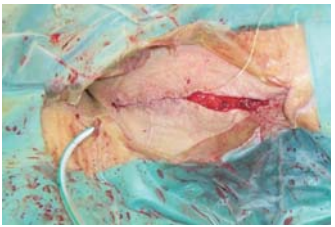
1. Skin
2. Platysma
3. Strap muscles

L Hemithyroidectomy showing sutures in strap muscles

After carotid surgery, sutures need to be removed.

1. Skin
2. Platysma

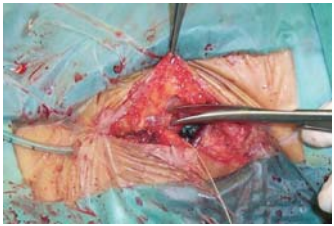
Skin may be closed by metal clips, skin sutures, or a subcuticular suture.



This photo shows a subcuticular skin closure of a left neck carotid endarterectomy incision. This stitch needs to be removed or cut the length of the incision. Note the ear lobe to the right and the drain tube to the left. The sternal notch is beneath the drapes in the top left corner. Drain tubes cannot be relied upon to prevent a neck haematoma developing.

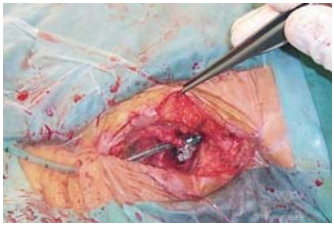
Carotid skin closure

After removal of the skin sutures an underlying haematoma may still not be visible.



After removal of skin sutures, the platysma sutures must be removed. This photo shows the platysma sutures partly removed in the upper part of the wound (the right side of the photo).

The haematoma is visible in the upper part of the wound where the platysma layer has been opened.



Opening the platysma

The wound is now completely open and the haematoma evacuated. Note the drain tube is now completely visible (a haematoma can develop even though a drain tube is in place).

Definitive management

- Patient should be urgently transferred to theatre
- Intubate patient to achieve patent airway
- Explore or re-explore wound
- Identify and ligate bleeding points
- ? Need for drain tube

Pulmonary embolus

Theory

Thrombosis in deep veins of calf, larger veins of leg, or clot in right atrium breaks off and embolises through the right heart into the pulmonary vasculature.

This can be subclinical, result in increased pulmonary vascular resistance and acute right heart failure, give rise to acute symptoms or cause sudden death.

Immediate intern management

Attend patient and make rapid assessment.

If patient unconscious/arrested or in severe respiratory distress

1. Tell nursing staff to call Code Blue then move to head end of bed.
2. Perform triple manoeuvre – chin lift, jaw thrust, head tilt.
3. Clear mouth – suction secretions.
4. Insert Guedel airway and bag and mask.
5. Check pulse and if arrested start CPR.

If patient conscious

1. Administer oxygen by mask to maintain oxygen saturations.
2. Perform continuous pulse oximetry.
3. Obtain IV access and send off basic blood tests.
4. ABG.
5. Organise urgent CXR.
6. Liaise with senior medical staff – unit registrar and/or medical registrar.

Risk factors

- Surgery, especially pelvic/orthopaedic
- Immobility
- Malignancy
- OCP/pregnancy/HRT
- Recent AMI/CVA
- Previous thromboembolism, inherited thrombophilia

Clinical features (obtain rapidly)

History

- Basic information about patient
- SOB
- Pleuritic chest pain
- Haemoptysis

Examination

- Tachycardia
- Oxygen saturations
- Sweaty, anxious
- Pleural rub
- If massive embolus:
    - Pale and sweaty
    - Tachycardia, tachypnoea
    - Central cyanosis
    - Elevated JVP
    - RV heave
    - Gallop rhythm (right heart failure)

Investigations

1. CXR
   Ensure no other reason for symptoms
2. ABG
   Hypoxia, hypercapnia, hypocapnia
3. ECG
   S (I), Q (III), T (III) wave changes
   Right heart strain
4. V/Q scan
   Gives you a probability based on the degree of ventilation and perfusion mismatch
5. CT pulmonary angiogram
   Replacing conventional angiography
6. Pulmonary angiogram
   Gold standard
   Largely replaced by CTPA in many centres

Definitive management

- Definitive investigations to confirm diagnosis
- Anticoagulation. Choices are:
  Heparin infusion
  Therapeutic dose Clexane
- If massive PE with signs of right heart strain, consider:
  Thrombolytics to dissolve clot
  Urgent sternotomy, cardiopulmonary bypass and surgical embolectomy

Other considerations

- Consider use of IVC filter in setting of iliofemoral thrombosis to prevent clot propagation
- Prevention is better than cure
- All at-risk surgical patients should be treated with anti-embolic stockings and prophylactic Clexane 20 mg or 40 mg sc daily
- Encourage early mobilisation and discharge from hospital once patient is well

Pulmonary oedema

Theory

Pulmonary oedema is effusion of serous fluid into the alveoli

and interstitial tissue of the lungs.

When hydrostatic pressure of pulmonary capillaries exceeds the osmotic pressure of plasma, fluid moves from the capillaries into the alveoli. This results in an impaired ability to oxygenate the blood and, ultimately, cardiorespiratory arrest.

Treatment is aimed at combating the increasing fluid in the lung spaces and maintaining oxygen delivery to the tissues.

Immediate intern management

Attend patient and make rapid assessment.

If patient unconscious/arrest

1. Tell nursing staff to call Code Blue then move to head end of bed.
2. Perform triple manoeuvre – chin lift, jaw thrust, head tilt.
3. Clear mouth – suction secretions.
4. Insert Guedel airway and bag and mask.
5. Check pulse and if arrested start CPR.

If patient conscious/pre-arrest

1. Call Code Blue.
2. Oxygen by mask, continuous pulse oximetry.
3. Sit patient up.
4. Obtain IV trolley and insert IV line while taking blood.
5. Give IV frusemide 40 mg (if patient on regular dose of frusemide then double it; if frusemide naive you can give 20 mg).

6. Apply glyceryl trinitrate topically. Use a 5 mg patch for naive patients but a higher dose for patients already receiving a patch.
7. Give IV morphine in 0.5–1 mg, 5 mg aliquots waiting 5 minutes between each aliquot.
8. ECG – look for any ECG changes of ischaemia.
9. Notify senior medical staff if not present already (medical registrar/ICU registrar).
10. Consider non-invasive ventilation (CPAP).

Clinical features (obtain rapidly)

History

- Basic information about patient
- Shortness of breath
- SOBOE
- Chest pain
- Symptoms of CCF (PND, SOA, orthopnoea)
- History of IHD, past AMI
- Fluid balance

Examination

- Vital signs
- Oxygen saturations
- Sweaty, anxious
- Raised JVP
- Crackles throughout lung fields or wheeze
- Cardiac murmur or added heart sounds S3, S4
- Swelling of ankles, peripheral oedema

Causes

- Cardiogenic
  - AMI
  - Acute valvular disorder
- Volume overload
  - Iatrogenic fluid administration (especially post-op in the elderly)
  - Renal failure
- Other
  - ARDS
  - PE
  - Altitude
  - Eclampsia
  - Neurogenic (post convulsions/seizures)
  - Post OD
  - Non-compliance with therapy

Investigations

Emergency:

1. ECG
   - Signs of AMI
2. CXR
   - Upper lobe venous diversion
   - Prominent pulmonary vasculature
   - Kerly B Lines
3. ABG
   - Hypoxia
4. Basic blood tests including cardiac enzymes

Secondary:

1. Echocardiography – ? valvular disease
2. Stress tests

Definitive management – depends on cause

- Above management plus further morphine/frusemide as required to produce a diuresis
- Nitrates (50 mg – 25 mg GTN patch)
  - As long as no history of aortic stenosis, and patient has good BP >110 systolic
- Maintain oxygen delivery by maintaining oxygen saturations:
  - By mask
  - Non-invasive ventilation (CPAP)
  - Intubation and ventilation
- If patient fails to make a prompt response then should be transferred to ICU for invasive monitoring:
  - Central venous line
  - Arterial line
- Insert indwelling catheter for accurate measurement of fluid balance
- Commence fluid balance chart +/- daily patient weights
- Identify and treat underlying cause

Tension pneumothorax

Theory

This is an emergency situation requiring immediate assessment and management.

Management focuses on urgent chest decompression with wide bore needle followed by a chest tube.

It occurs when air leaks from a pulmonary laceration or tear into the pleural cavity, but is sealed in the space by the parietal pleura. The rising tension collapses the affected lung and displaces the mediastinal structures to the other side. This causes:

1. Respiratory compromise from lung collapse.
2. Kinking of the IVC by mediastinal displacement reducing venous return, preload, and cardiac output leading to cardiopulmonary arrest.

Immediate intern management

Attend patient and make rapid assessment.

If patient unconscious/arrest

1. Tell nursing staff to call Code Blue then move to end of bed.
2. Perform triple manoeuvre – chin lift, jaw thrust, head tilt.
3. Clear mouth – suction secretions.
4. Insert Guedel airway and bag and mask.
5. Check pulse and if arrested start CPR.

If conscious/severe respiratory distress

Call code blue

Assess patient – oxygen saturations, PR, BP

1. Tracheal deviation
2. Reduced chest movement
3. Hyperresonance to percussion
4. Decreased breath sounds

} = tension pneumothorax

Tension pneumothorax (TP)

1. Needle pleurocentesis

   Insert wide bore IV needle into second IC space in mid-clavicular line.

   The tension will be relieved instantly but the patient needs an urgent chest tube.
2. Oxygen by mask.
3. Notify surgical registrar/ICU registrar urgently regarding need for chest tube.
4. Organise chest tube and chest tube tray as a matter of priority with nursing/medical staff (but do not leave patient unless help has arrived).
5. If patient deteriorates again (saturations begin to fall) then insert second IV needle into second IC space.
6. Insert chest tube and place on 10 cm continuous underwater suction.
7. Obtain post insertion CXR.

Causes

- Often spontaneous (rupture subpleural bullae)
- Asthma/COAD
- Ventilated patients
- Trauma

- Pleura entered during attempted placement of CVC line
- Carcinoma lung
- Pneumonia, TB, lung abscess, CF
- Connective tissue disorder

Clinical features (obtain rapidly)

History

- Basic information about patient
- Sudden onset sharp pleuritic chest pain with SOB

Examination

- Pt in extremis
- Respiratory distress
- Trachea deviated away from side of TP
- Reduced chest expansion on side of TP
- Hyper-resonant to percussion
- Absent breath sounds on side of TP
- Displaced apex beat

Insertion of chest tube

1. Local anaesthetic. Sterile technique
2. Incision fifth IC space ant. Axillary line or second IC space mid-clavicular line
3. Enter 'above the rib below'
4. Dissect down to muscles
5. Split muscles using artery forceps
6. Blunt dissect through to parietal pleura (air rush)
7. Remove trocar from chest tube. Never use the trocar
8. Insert chest tube with long artery forceps directing upwards
9. Connect to underwater suction system with 10 cm continuous suction. Check CXR for position

Investigations

(Do not wait for these tests before insertion of test tube)

1. Basic/CXR blood tests
2. ABG

Further management

- Insertion of a chest tube as described above
- Continued air leak through pneumothorax may require thoracoscopy/thoracotomy

Acute bowel obstruction

Theory

Small bowel obstruction (SBO) is a common condition with a number of causes. The majority of patients settle with conservative treatment.

Large bowel obstruction (LBO) is less common and more sinister.

A true large bowel obstruction is a surgical emergency and often requires major surgery.

Bowel dilatation results in regional hypoperfusion to the wall of the bowel causing regional ischaemia and inhibiting peristalsis. Management is focused on resuscitation and decompression, which allows an improvement in regional wall blood flow and may result in resolution of the obstruction.

Immediate intern management

Targeted history and examination

1. Obtain IV access and commence IV fluid resuscitation.
   Aim to replace estimated fluid lost and maintenance fluids.
2. Basic set bloods (FBE, U&E, INR, G&H).
3. Nil orally.
4. Anti-emetics (Maxalon 10–20 mg IV QID / Ondansetron 2–4 mg IV bd).
5. Basic definitive investigations – plain X-rays.
6. Insert NGT if patient vomiting (place on free drainage and four-hourly aspirations).
7. Contact surgical registrar and unit registrar.

Clinical features

History

- General patient features
- Colicky central abdominal pain
- Abdominal distension
- Absolute constipation (flatus and faeces)
- Vomiting
- PHx abdominal surgery

Examination

- Hydration status
- Distended abdomen
- Abdominal tenderness
- High pitched tinkling bowel sounds
- Hernia

Causes SBO

Common

- Adhesions
- Incarcerated hernia
- Ileus

Rare

- IBD
- SB tumour

Causes LBO

Common

- Colorectal carcinoma
- Diverticular disease
- Volvulus (caecal/sigmoid)
- Pseudo-obstruction

Uncommon

- IBD
- Hernia

Investigations

First line

1. AXR (erect and supine), erect CXR
   - Dilated loops of small bowel (central) with air fluid level
   - Dilated large bowel (peripheral) with air fluid levels
   - Air under diaphragm

Second line

1. CT scan
2. Contrast studies
   - Enema/follow through

Definitive management

SBO

Initial treatment – trial conservative management

- NGT
- IV fluid therapy
- Nil orally

If NGT drainage becomes faeculent or ongoing obstruction:

- Surgical exploration and repair

Note: An SBO in the setting of a hernia is a surgical emergency requiring urgent operative repair.

LBO

'Never let the sun set twice on an acute large bowel obstruction.'

- IV fluid therapy
- Nil orally
- First line imaging

The key to management of LBO is to establish a likely diagnosis and then decompress the large bowel

- Second line imaging
- Surgical decompression and bowel resection
- Rigid sigmoidoscopic decompression (if sigmoid volvulus)
- Limited period of observation if suspecting pseudo-obstruction

Acute extradural haematoma

Theory

Unlike an acute subdural haematoma, an acute extradural haematoma is an intracerebral bleed at arterial pressure, meaning that it causes much more significant mass effects.

It occurs following a closed head injury and can affect any age group.

The most common pathology is bleeding from the middle meningeal artery from a fracture to the temporal or parietal bone. This causes bleeding between the dura mater and the bones of the skull.

This causes a rise in intracranial pressure and midline shift.

Death from tentorial herniation and coning can occur quickly, because the cranial cavity is a closed compartment and increasing pressure forces the pliable brain substance down through the foramen magnum.

Immediate intern management

- If trauma situation/following a fall – ABCs and cervical spine immobilisation.
- Targeted history and examination.
- Suspect the diagnosis.

These patients only have a limited amount of time before catastrophic neurological deterioration. Urgent stabilisation and CT scanning is required.

1. Sit patient to 30 degrees head up.
2. Nil orally, oxygen by mask.
3. Obtain IV access and commence gentle IV fluids.
4. Measure and record Glasgow Coma Score (GCS).

If GCS falls to <8, patients can not protect airway and require intubation. Urgently notify senior staff/call Code Blue.

5. Commence regular neurological observations:
   every 15 minutes for two hours
   every 30 minutes for two hours
   every one hour for four hours.
6. Organise urgent CT scan of brain.
7. Notify parent registrar and surgical registrar.

Clinical features

History

- History of head trauma
- Lucid interval
- Fluctuating level of consciousness
- Severe headache
- Vomiting
- Confusion
- Fitting

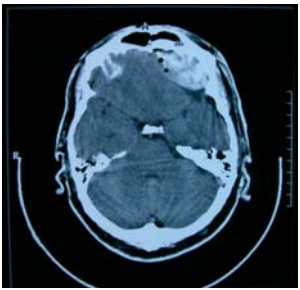
Examination

- GCS
- Neurological examination
  Localising signs/enlarging pupil

Observation

Changing or deterioration in:

- Level of consciousness
- GCS



← **acute extradural haematoma**

Investigations

CT scan brain

Convex haematoma that does not cross suture lines of the skull bones

GCS – Glasgow Coma Score

Best observed ability	Score
Eyes	
Open spontaneously	4
Open to voice	3
Open to pain	2
Closed	1
Verbal	
Normal and appropriate	5
Speaks sentences but confused	4
Speaks words	3
Makes unintelligible sounds	2
Non-verbal	1
Motor	

Best observed ability	Score
Obeys commands	6
Localises to pain	5
Withdraws to pain	4
Flexion to pain response	3
Extends to pain	2
No response	1

Definitive management

Urgent neurosurgical referral is required

Urgent neurosurgical decompression

- Craniotomy over the site of extradural
- Evacuation of clot

Consider:

- Antiepileptic medication
  Phenytoin loading

Acute gastric distension

Theory

Acute gastric distension results in the stomach filling with fluid, which causes nausea and vomiting.

In patients who have an altered conscious state/undergoing general anaesthetic, gastric distension is a significant risk for aspiration.

Decompression relieves symptoms of nausea and vomiting and improves blood flow to the wall of the stomach, which aids in return of peristalsis and resolution of the distension.

Immediate intern management

Targeted history and examination and suspect diagnosis.

1. Nil orally.
2. Obtain IV access and commence IV fluids.
3. Administer anti-emetic medication:
   Maxalon 20 mg IV
   Ondansetron 2–4 mg IV.
4. Simple investigations if diagnosis unclear.
5. Insert NGT
   (Place on free drainage and four-hourly aspirations).
6. Contact surgical registrar and unit registrar.

Clinical features

History

- General patient features
- Nausea
- Colicky upper abdominal pain

- Abdominal distension
- PHx
    - Recent surgery
    - Head injury
    - Last meal
- Vomiting (late symptom)

Examination

- Hydration status
- Distended abdomen
- Succussion splash
- Abdominal tenderness
- High pitched tinkling bowel sounds
- Hernia

Risk factors/reversible causes

- SBO
- Ileus
- Medication
- Head injury
- Altered conscious state
- Metabolic derangement
- Post gastric surgery

Causes

Common

- Post meal
- Enteral feeding
- SBO
- Medications
- Ileus
- Post abdominal surgery

Rare

- Peptic ulceration
- Air swallowing

Investigations

First line

1. AXR (erect and supine), erect CXR
   - Dilated stomach with air fluid level
   - Signs of SBO with multiple air fluid levels
2. Basic blood tests
   - FBE, U&E, CRP

Second line

1. Contrast studies
   - Gastrografin swallow

Definitive management

Identify risk factors and correct reversible factors

- NGT insertion is definitive and allows decompression of the stomach; it also reduces the risks of aspiration from acute gastric distension

Prevention

- NGT insertion for treatment of SBO
- Critical review of medication charts by parent units
- Suspicion in cases of head injury or altered conscious state
- Correct metabolic abnormalities
- Cautious resumption of oral intake post general anaesthetic and abdominal operations
- Ensure adequate period of fasting prior to general anaesthetic

Acute limb ischaemia

Theory

Embolic – will lodge at branch point of artery (aortic bifurcation, common iliac, common femoral, adductor canal).

Thrombotic – a diseased peripheral vessel can thrombose causing acute ischaemia.

Immediate intern management

1. Suspect and make the diagnosis.
2. IV access, basic set bloods (FBE, U&E, INR, GandH).
3. Nil orally, gentle IV fluids.
4. Analgesia.
5. Keep the limb warm (blanket/cotton wool).
6. Nurse limb in dependent position.
7. Inform unit and surgical registrar.
8. Organise appropriate investigations:
   - Arterial US
   - CT angiogram
   - ? Transfer.

Note: In very severe ischaemia, surgery may be required very urgently and investigations (if any) may take place in operating theatre.

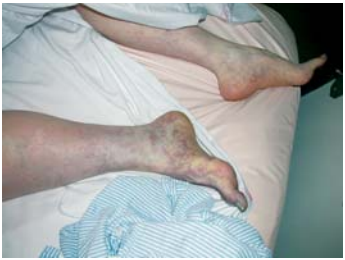
Differential diagnosis

Common

1. Cellulitis
2. Septic arthritis
3. Acute gout
4. DVT

Uncommon

1. Allergic reaction
2. Compartment syndrome
3. Referred pain
4. Acute fracture



Practical tips

- The diagnosis is difficult, but needs to be excluded in a suddenly painful limb
- Clinical features can be absent or subtle
- Duration of ischaemia is critical for limb salvage
- Asymmetrical pulses are a very useful hard sign
- Inform superiors early

Clinical features

History

- Acutely painful limb
- Risk factors: AF, PVD, atrial clot, ventricular aneurysm

Examination

- Pale or mottled appearance
- Swollen
- Cold and tender to touch
- Decreased capillary refill
- Absent or asymmetrical peripheral pulses
- Diminished sensation – indication for urgent surgery
- Diminished muscle movement – indication for urgent surgery

Investigations (urgent) diagnostic:

1. Lower limb arterial ultrasound
   First line test
2. MRA/CT peripheral angiogram
   Second line investigation
3. Peripheral arterial angiography
   Gold standard for acute limb ischaemia

Investigations – underlying factors:

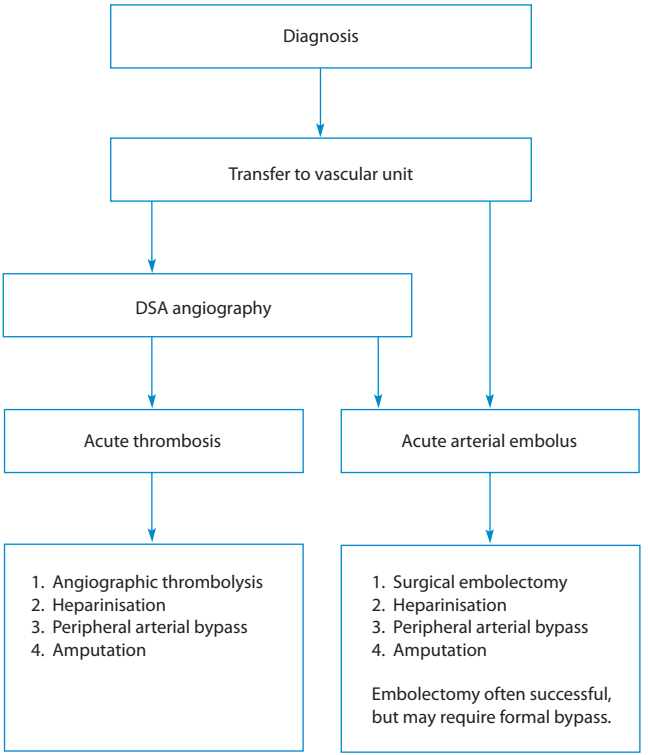
1. ECG ?AF
2. Cardiovascular risk factors
3. Echocardiogram

Ischaemic time

- Time is the most critical determinant of outcome
- Ideally should be less than six hours
- After eight hours, survival of muscle is significantly at risk

Definitive management

Once diagnosis is made, definitive management rests with revascularisation.



Options for definitive management: depend on site and nature of arterial disease

Acute mental confusion and/or fitting

Theory

Acute mental confusion is common. It occurs in 5–15 per cent of all hospitalised patients.

Risk factors include elderly patients, those in ICU/HDU settings or patients with severe or multiple medical problems.

Delirium can be very subtle and you need to suspect the diagnosis.

Many hospitals will have specific guidelines and protocols for the use of drugs in the management of delirium.

Fitting can be caused by epilepsy or a structural/chemical/infectious injury to the brain and is a very serious sign requiring prompt management.

During a fit a patient has a vulnerable airway and is unable to breathe. Management is supportive and aimed at ceasing the fit.

Immediate intern management

Fitting

1. Clear environment from around patient to prevent injury.
2. If possible, patient can be placed in left lateral position.
   (Do not force patient into this position if the fit will not allow).
3. Suction any vomitus from airway.
4. Ask nursing staff to get help of other medical staff/call MET code.
5. Give oxygen by mask.

6. Obtain IV access with nursing assistance, send off basic blood tests and measure at bedside blood sugar level.
   If hypoglycaemic:
   1. Administer 50 ml 50% Dextrose IV.
   2. Continue until patient stops fitting and able to eat.
   3. Give oral glucose.
7. Administer:
   Diazepam 5 mg IV (rectal route can be used if delay in obtaining IV access).
8. If fitting continues, administer further dose:
   Diazepam 5 mg IV.
9. If fitting continues, administer clonazepam 1 mg IV until fitting stops.
10. Load with antiepileptic – phenytoin 300 mg loading dose, followed by further dose 300 mg six hours later.

Delirium

1. Nurse patient in moderately lit, quiet environment with close supervision.
2. Investigate and treat underlying cause.
3. Rationalise medications.
4. Haloperidol 0.5 mg IM/IV can be used for agitation in these patients.

Clinical features

History – delirium

- Consciousness
  Fluctuates throughout the day
  Typically worse in late afternoon/night
  Impaired over hours to days

- Disorientation
- Behaviour disturbance
  - Inactivity/quiet
  - Hyperactivity/agitation
- Thinking
  - Slow and muddled
- Perception
  - Disturbed with delusional features
- Mood disturbance
- Memory impairment

History – fitting

- Epilepsy
- Head trauma
- Headache, vomiting, fever

Examination

- Glasgow Coma Score (GCS)
- Neurological examination
  - Localising signs/enlarging pupil

Observation

- Changing or deterioration in:
  - Level of consciousness
  - GCS

Investigations

Confusion screen:

1. Basic blood tests
   - FBE, U&E, LFT, TFT
   - ABG, cardiac enzymes, blood sugar

2. Cultures
    Blood, urine, sputum
3. Imaging
    CXR
    CT brain

Fitting:

1. Basic blood tests
2. CT brain

Consider:

1. Lumbar puncture
2. MRI

Definitive management

Delirium

- Appropriate investigations
- Treatment of the underlying cause
- Observation over several days
- Most deliriums will resolve with time

Fitting

- Specialist referral for further investigation
- May require commencement of antiepileptic medications (for example, phenytoin) or adjustment to current medications
- Assess compliance in long term epileptics
- Check recent medication changes for drug interactions
- Consider MRI/lumbar puncture
- Give advice about driving and operating heavy machinery

Causes of confusion

- Sepsis
- Drugs – opiates, sedatives
- Hypoxia
- Alcohol withdrawal
- Metabolic
- Trauma
- Raised intracranial pressure
- Epilepsy
- Hypoglycaemia
- CVA
- Meningitis
- Hyper/hypothyroidism
- Nutritional

Acute ruptured abdominal aortic aneurysm

Theory

Seventy-five per cent mortality: 50 per cent prior to reaching hospital; 25 per cent after reaching hospital and undergoing surgery.

Best prognostic indicator is level of consciousness at time of arrival to hospital or at time of diagnosis.

Immediate intern management

Suspect and make the diagnosis by examining the abdomen. It's in your hands.

Sudden onset abdominal and back pain with collapse equals ruptured AAA until proven otherwise. Most have a tender, pulsatile mass to feel.

1. Large bore x2 IV access.
2. Basic set bloods (FBE, U&E, INR, X-Match six units).
3. Nil orally.
4. IV fluids with caution – aim to maintain urine output, but remember hypotension helps the abdominal muscles to tamponade the bleeding.
5. Contact the surgical registrar or consultant urgently.

Organise appropriate definitive investigations (depending on advice of surgical registrar and patient's stability).

CT scan with IV contrast.

If the patient is too haemodynamically unstable for CT scan, has hypotension not responding to IV fluid challenge and persistent tachycardia, an immediate exploratory laparotomy is indicated.

Clinical features

History

- Middle aged to elderly
- Presents with sudden onset severe epigastric pain with collapse
- Pain radiates to back and sometimes into groin
- Vascular risk factors

Examination

- Tachycardic
- Hypotensive
- Pale, sweaty (SHOCK)
- Conscious/moribund
- Tender expansile pulsatile mass in epigastrium
- May feel tense or may be hard to feel
- Weak or absent peripheral pulses
- Cool peripheries

Differential diagnosis

1. Perforated viscus
2. Acute pancreatitis
3. Aortic dissection
4. Enlarging aortic aneurysm
5. Ruptured splenic artery aneurysm

Investigations (urgent) diagnostic

1. Abdominal CT scan with IV contrast

The patient may not be stable enough following rupture to undergo CT scan. Persistent hypotension and tachycardia not responding to fluid challenge is an indication for urgent surgical exploration.

2. X-Match six units
3. Routine set blood tests

Definitive management

Once diagnosis is made, definitive management rests with emergency laparotomy and clamping aorta above site of rupture.

The patient may be stable enough for transfer to a specialist vascular unit. Otherwise surgical management focuses on:

- Transfer urgently to operating theatre
- Organise invasive monitoring: pulse oximetry, arterial line, IDC, central venous line (some of these may be done in theatre)
- Prep skin and theatre team ready
- Crash induction and intubation while surgical team performs laparotomy, confirm diagnosis and clamp aorta proximal to site of rupture

Patients being transferred require the above invasive monitoring

Practical tips

- The diagnosis rests with a history of sudden onset abdominal and back pain with collapse
- The abdominal findings are your chance to save time and life by clinching the diagnosis clinically. Distension or tense muscles may mask the tender pulsatile mass.
- If the patient is too unstable for a CT scan then they need an urgent exploratory laparotomy
- Inform superiors early

Acute spinal compression

Theory

Either acute or chronic compression on the spine results in neurological disturbances to the upper limbs, lower limbs, saddle area and also results in bowel and bladder dysfunction.

Management focuses on recognising the condition and adequate treatment, which is nearly always urgent surgical decompression.

Immediate intern management

Suspect the diagnosis. Any patient complaining of upper or lower limb neurological symptoms needs to have acute spinal cord compression ruled out.

Thorough history and examination, including full neurological examination

1. Obtain IV access and commence gentle IV fluids.
2. Send off basic blood tests.
3. Analgesia for back pain.
4. Strict rest in bed and spinal precautions.

Notify unit registrar and surgical registrar and discuss findings.

6. Organise urgent MRI (CT) scan.

Clinical features

History

- Back pain
- Neurological symptoms
  - Weakness arms, legs
  - Numbness arms/buttocks/legs
- Bladder and bowel dysfunction
  - Incontinence
  - Urinary retention
- Trauma
- Known cancer/myeloma
- Recent spinal/epidural

Examination

- Neurological examination
  - UMN signs in limbs clonus, increased tone, weakness, hyperreflexia, upgoing plantars
  - Sensory change
  - Saddle anaesthesia
- PR
  - Decreased anal tone

Causes

Acute

- Disc prolapse
- Trauma
- Fractures
- Epidural haematoma/abscess

Acute or chronic

- Bony metastases
- Myeloma
- Abscess
- Spinal cord tumour
- Spondylolisthesis
- Tuberculosis
- Pregnancy
- Degenerative spinal disease

Investigations

MRI is the gold standard but may not be available at all centres.

Acute spinal cord compression is an absolute indication for an urgent MRI.

Imaging

1. CT scan spine
2. MRI spine

Associated tests

1. Basic blood tests
2. X-Match
3. CXR
4. ECG

Screening tests

1. ESR
2. PSA
3. Tumour markers
4. Bone scan (tumour hot spots)
5. Inflammatory markers

Definitive management

Urgent neurosurgical evaluation is required

- Urgent spinal decompression – laminectomy
- Resection of tumour or disc prolapse
- Corticosteroids (dexamethasone) have an urgent role in limiting cord oedema (use following discussion with neurosurgery)

Other considerations

- Consider radiotherapy for known metastasis to the spine
- Treatment of myeloma/TB
- Pressure care if sensory disturbance
- IDC for urinary retention
- Specimen cultures if evidence fever/sepsis. A septic patient developing segmental nerve or cord features has spinal abscess with no time to lose.
- Cauda equine syndrome will give LMN signs in the lower limbs.

Acute ureteric obstruction

Theory

Commonest cause is ureteric calculus. Other causes include extrinsic retroperitoneal pathology including abscess, haematoma, fibrosis, malignancy and iatrogenic (surgical injury).

Usually present with unilateral flank pain associated at times with haematuria and/or fever

Examination

Unilateral abdominal and flank tenderness.

Investigations

- FBE
- U&E
- MSU microscopy & culture
- Urinalysis
- Blood cultures (if febrile)
- Serum calcium, uric acid
- KUB plain X-ray
- Non-contrast CT urinary tract
- NOT ultrasound

Management

- IV fluids
- Analgesia
- Contact urologist

Urological Intervention

Obstructed kidney and sepsis – urgent nephrostomy or ureteric stenting.

Ureteric calculus with obstruction

- Large >5mm – ureteric stent/ESWL or ureteroscopy
- Smaller <5mm – conservative, tamsulosin. Intervene as above if ongoing pain or failure to progress.

Urinary stone analysis

'Pyelonephritis' is a clinical syndrome of flank pain and fever. Should never be accepted as a diagnosis without first excluding an obstructed kidney by imaging as above. Ultrasound will be normal in early obstruction and should NOT be used in place of KUB and CT.

Acute subdural haematoma

Theory

Acute subdural haematoma occurs generally in elderly patients following a closed head injury.

Age related atrophy of the cerebral cortex occurs, widening the space between the dura mater and the arachnoid mater.

Following a head injury, bleeding can occur in the bridging veins between the cortex and the venous sinuses in the brain. These sit in the arachnoid space.

Bleeding here develops quickly into a haematoma between the dura and the arachnoid mater. This causes a rise in intracranial pressure and can result in midline shift and possibly death from tentorial herniation and coning, although generally the pressure is not high enough in the venous system to allow this to occur.

Immediate intern management

If trauma/following a fall – ABCs and cervical spine immobilisation.

Targeted history and examination.

Suspect the diagnosis.

1. Sit patient to 30 degrees head up.
2. Nil orally, oxygen by mask.
3. Obtain IV access and commence gentle IV fluids.
4. Measure and record Glasgow Coma Score (GCS).

If GCS falls to <8, patients can not protect airway and require intubation. Urgently notify senior staff/call Code Blue.

5. Commence regular neurological observations:
   every 15 minutes for two hours
   every 30 minutes for two hours
   every one hour for four hours.
6. Organise urgent CT scan of brain.
7. Notify parent registrar and surgical registrar.

Clinical features

History

- Closed head trauma/fall
- Asymptomatic
- Confusion
- Altered/fluctuating conscious state
- Physical and intellectual slowing
- Sleepiness
- Headache
- Change in personality
- Unsteadiness

Examination

- GCS
- Neurological examination
  Focal deficits/enlarging pupil

Observation

- Changing/deteriorating level of consciousness or GCS

Investigations

CT scan brain

Concave haematoma that does not cross midline

- Hyperintense <10 days
- Isointense = 10–12 days
- Hypointense >12 days

GCS – Glasgow Coma Score

Best observed ability	Score
Eyes	
Open spontaneously	4
Open to voice	3
Open to pain	2
Closed	1
Verbal	
Normal and appropriate	5
Speaks sentences but confused	4
Speaks words	3
Makes unintelligible sounds	2
Non-verbal	1
Motor	
Obeys commands	6
Localises to pain	5
Withdraws to pain	4
Flexion to pain response	3
Extends to pain	2
No response	1

Risk factors

- Elderly
- Alcoholics
- Patients on anticoagulants
- Patients prone to falls (can occur after a fall in hospital)
- Sedatives/opiates
- Patients with head trauma
- Acute delirium
- Altered conscious state

Definitive management

Urgent neurosurgical referral is required

Urgent neurosurgical decompression

- Acute <10–12 days
  Requires craniotomy and evacuation of haematoma
- Chronic >12 days
  Can undergo burr hole drainage of liquefied clot

Globe rupture – including penetrating eye injury and intraocular foreign body

Theory

Globe rupture occurs when the integrity of the outer coat of the eye (cornea and/or sclera) is disrupted by blunt or penetrating trauma. The globe is vulnerable to blunt injury by objects smaller than the protective orbital rim e.g. golf ball, squash ball. Blunt trauma causes anterior-posterior compression of the globe, raised intra-ocular pressure and rupture of the globe at sites of relative scleral thinning (limbus, sites of extra-ocular muscle insertion, insertion of the optic nerve). A posterior globe rupture may be difficult to diagnose in the emergency department and a high degree of suspicion is needed. Sharp objects or those travelling at high velocity may perforate the globe directly. Small foreign bodies may penetrate the eye and remain within the globe.

Early recognition and surgical repair is critical to maximising visual outcome. Poor prognostic factors include blunt injuries, poor initial visual acuity, presence of a relative afferent pupil defect or retinal detachment, absence of a red reflex, associated lid trauma and posterior wound location.

Post-traumatic endophthalmitis is a rare complication, but has poor visual prognosis. The most common organisms are Streptococcus species, coagulase-negative Straphylococcus, and Bacillus cereus.

Appropriate ophthalmic surgical treatment is thought to minimise the risk of sympathetic ophthalmia, although evidence for this is weak.

Principles of intern management are to prevent vomiting, pain, infection and further injury prior to transfer to ophthalmic care.

Globe rupture is an emergency and needs repair before attending to other non-life threatening injuries e.g. associated facial lacerations.

Immediate intern management prior to ophthalmic care

Suspect the diagnosis in all cases of blunt and penetrating orbital trauma and all cases involving high-speed projectiles with potential for ocular penetration. The majority of cases are in young men. You do not need to be certain the globe is ruptured – refer all patients in whom the diagnosis is suspected. Signs can be subtle: small lid lacerations may conceal vision-threatening globe perforations. Good visual acuity and absence of pain does not rule out globe rupture.

- Avoid all pressure on or around the injured eye to prevent extrusion of intraocular contents. Protect the eye with a shield. Do not apply eye drops, ointment or patches.
- Leave impaled foreign bodies in situ.
- Administer antiemetics e.g. metoclopramide hydrochloride 10 mg or prochlorperazine 12.5 mg to prevent Valsalva manoeuvres.
- Administer analgesics as indicated.
- Administer antibiotics to prevent endophthalmitis if significant delay is expected

  ciprofloxacin 750 mg (child 20 mg/kg up to 750 mg) orally as a single dose

  PLUS

  vancomycin 25 mg/kg up to 1.5 g (child <12 years 30 mg/kg up to 1.5 g). Give IV as a single dose. Slow infusion required, maximum 5 mg/min

  Alternate prophylaxis

  gentamicin 5 mg/kg IV as a single dose

  PLUS

  cephazolin 2 g (child 50 mg/kg up to 2 g) IV as a single dose
- Document tetanus immune status and update as indicated.

- Ascertain what time was the last meal. The patient should be kept NPO.
- Refer for ophthalmic care. If transfer is needed, transfer by road ambulance or air ambulance with the cabin altitude at sea level or as low as safe.

Clinical features

History

- Decreased vision
- Pain
- History of trauma e.g. golf ball, squash ball
- History of high velocity injury e.g. grinding, hammering metal on metal

Other (obtain later)

- Time of injury
- Tetanus status
- Allergies

Examination

- When globe rupture is suspected, minimise examination to avoid loss of ocular contents. Pressure on eyelids in attempted examination is dangerous
- Measure visual acuity if possible
- Ruptured globe: severe subconjunctival haemorrhage or swelling (chemosis), deep or shallow anterior chamber compared with the other eye, blood in the anterior chamber (hyphaema), pupil abnormalities (dilated, distorted, or unreactive), lids sunken (enophthalmos), limitation of extra-ocular motility, intra-ocular contents outside globe

- Penetrating injury: full-thickness scleral or corneal laceration accompanying signs of ruptured globe
- Consider other injuries. An obvious globe rupture may present with other life-threatening injuries that can be overlooked e.g. frontal lobe injury in patient with a penetrating eye injury through the upper eyelid.

Investigations

Imaging

Perform orbital CT scan only if available and does not cause a delay in transfer.

Gynaecological emergencies

1. Ruptured tubal (ectopic) pregnancy

When ruptured, a tubal ectopic pregnancy may present with lower abdominal pain, vaginal bleeding, signs of collapse and shock, and extreme tenderness in pelvis. Shoulder tip pain may be present. Treatment includes rapid resuscitation and surgery – salpingectomy (laparotomy or laparoscopy) to stop bleeding. The differential diagnosis includes appendicitis and complication of an ovarian cyst. Investigations include pelvic ultrasound, betaHCG and full blood count.

2. Cervical shock from retained products of conception (POC)

An incomplete abortion (miscarriage) may result in POC distending the cervix. The woman may present in a collapsed or shocked state. Speculum examination should be performed and any POC removed either digitally or with sponge forceps. Resuscitation and evacuation of the uterus under GA should follow.

3. Complication of ovarian cyst/mass

The most common complications of ovarian cysts resulting in an emergency presentation include rupture, torsion and haemorrhage. Symptoms include pelvic pain which may be of sudden onset. Cramping pain and nausea often associated with torsion of the ovary. Investigations include pelvic ultrasound, betaHCG and full blood count. Surgery may be necessary.

Shock

Theory

Shock is a colloquial term for cardio-circulatory collapse.

The pathology is that of inadequate tissue perfusion that does not allow adequate maintenance of the body's basic cellular functions.

There is a range of causes that lead to inadequate tissue perfusion.

Management aims at **rapid restoration** of the circulation and then identification and treatment of the underlying cause.

Patients have a range of tolerances to a lack of adequate tissue perfusion, with young fit patients coping better than the elderly. This means that the signs of shock in these patients are more subtle and can easily be underestimated until the state is advanced.

Immediate intern management

Attend patient and make rapid assessment.

Airway

1. Secure.

Breathing

1. Oxygen by mask.

Circulation (PR, BP, periphery – warm/cool)

1. Put pressure focally over site of bleeding.
2. Lie the patient down and elevate the foot of the bed.
3. Stop any epidural infusions.

4. Obtain IV access (X-Match lost blood volume + extra two units), FBE, U&E, LFT, INR).
5. Fluid resuscitate – 500 ml Gelofusine stat, followed by N. Saline 1 L stat.

Assessment

1. Clinical history.
2. ? Underlying cause of shock.

Reassessment

1. BP, PR, periphery, consciousness.
2. ? Responding to treatment.
3. Call surgical registrar and unit registrar.
4. Nil orally.
5. Notify ICU about patient.

Practical tips

Blood pressure = cardiac output x total peripheral resistance

Cardiac output = heart rate x stroke volume

BP = CO x TPR

CO = HR x SV

These equations allow you to think about the likely causes of shock and allow you to address each one of the factors that could be responsible for it and to decide on the best method of correcting the condition.

Shock is an imbalance of these variables, which leads to a loss of blood pressure and inadequate tissue perfusion.

Clinical features (obtain rapidly)

History

- Disorientation
- Dizziness
- Cold
- History of trauma
- Obvious blood loss (quantify)

Examination

- Haemodynamic status
- Pallor
- Cool mottled periphery
- Decreased capillary return
- Oliguria/anuria
- Air hunger
- JVP (high or Low)
- Crackles in lung bases

Watch for

- Warm and swollen periphery
- Wheeze, urticarial rash, oedema
  (Anaphylaxis)
- Neurological abnormalities
  (Neurogenic)

Causes

- Hypovolaemia
  Most common cause post-op/trauma
- Cardiogenic
  Primary cardiac cause
- Sepsis
  Peripheral dilatation and hypotension

- Anaphylaxis
  Peripheral dilatation and hypotension
- Neurogenic
  Primary disturbance of BP, PR homeostasis
- Iatrogenic (drugs/epidural)
  Loss of BP/PR regulation

Further definitive management

Depends on the cause. See relevant sections in intern manual (cardiogenic, anaphylaxis)

Hypovolaemia

- IV fluids therapy
- Blood if blood loss
- Correct cause of hypovolaemia

Sepsis

- IV fluid therapy
- Cultures – blood, urine, sputum
- Commence broad spectrum antibiotics
- Identify cause of sepsis

Neurogenic

- IV fluid therapy
- Neurological assessment and investigation
- Specialist referral
- Maintain circulating volume

Iatrogenic

- Cease epidural/drugs
- IV fluid therapy
- Correct and treat underlying cause

Stridor

Theory

Stridor is a harsh, high-pitched inspiratory upper airway noise. This is one of the most serious signs a patient can have and needs immediate assessment and management.

It is a sign of impending airway loss and as such is a sudden and significant threat to life.

Patients on the ward are susceptible to acute airway obstruction, especially if they have an altered conscious state due to opiate analgesia, confusion or the residual effects of anaesthetic agents.

These non-mechanical causes of stridor can be more easily treated than mechanical causes (haematoma, foreign body), which are more difficult.

Management focuses on relieving or preventing airway obstruction and ensuring a patent airway.

Immediate intern management

Attend patient and make rapid assessment.

If patient unconscious/severe respiratory distress/ respiratory arrest

1. Tell nursing staff to call Code Blue then move to head end of bed.
2. Perform triple manoeuvre – chin lift, jaw thrust, head tilt.
3. Clear mouth – suction secretions, vomitus.
4. Insert Guedel airway/nasopharyngeal airway and administer oxygen.
5. Bag and mask patient until help arrives.
6. Intubate and insert cuffed ETT.

If stridor and early signs of respiratory distress

1. Sit patient up and administer oxygen by mask.
2. Call MET Code or ask senior staff for *urgent* assistance.
3. Perform basic airway manoeuvres to assist patient in obtaining a clear airway.
4. Perform continuous pulse oximetry.
5. Carry out rapid clinical assessment for causes.
6. Insert IV line and take set routine blood tests, including ABGs.
7. Definitive treatment of underlying cause once help arrives or if patient deteriorating and these conditions exist:

Acute mechanical obstruction

Clear airway

- Clear mouth.
- Suction secretions.
- Sweep out foreign body from pharynx.

Surgical airway

- Cricothyroidotomy
- Tracheostomy

Post-operative neck haematoma

Open neck wound down to and including the deep fascial sutures.

If still obstructed and trachea on view, attempt to incise and insert endotracheal tube.

Clinical features (obtain rapidly)

History

- Basic information about patient
- Events resulting in development of stridor
- Recent drug administration/operation

Examination

- Pt in extremis
- Stridor
- Respiratory distress
  - Sitting forward, intercostal recession, tachypnoeic
- Cyanosis
- Drooling
- Swelling face/tongue

Causes

- Acute mechanical obstruction
- Sputum plug
- Altered conscious state
- Laryngospasm
- Acute asthma attack
- Angio-oedema
- Disruption ETT/tracheostomy
- Neck haematoma post neck surgery
- Trauma
- Burns
- Tumours

Stridor is a harsh, high-pitched inspiratory upper airway noise

Investigations

1. Cardiac monitor
2. Basic blood tests
3. ABG – less useful
4. CXR

Cricothyroidotomy

- Feel for the prominence of the thyroid cartilage
- Incise horizontally in space **inferior** to thyroid cartilage (This is cricothyroid membrane)
- Insert handle of scalpel into incision and twist to open incision and allow insertion of endotracheal tube

Definitive treatment

Call for help early. Stridor = acute threat to life.

Stridor is a sign of impending airway loss. Treatment is aimed at identifying and treating the underlying cause.

- Acute mechanical obstruction – relieve obstruction/obtain secure airway
- Sputum plug – suction airway and relieve obstruction
- Altered conscious state – simple airway manoeuvre +/- intubation
- Laryngospasm – adrenaline, antihistamines and secure airway
- Acute asthma – Ventolin nebulisers x3 +/- IV steroids + atrovent
- Angio-oedema – adrenaline, antihistamines and secure airway
- Disruption ETT/tracheostomy – rapidly secure airway
- Neck haematoma – evacuate haematoma and secure airway
- Trauma – basic airway manoeuvres and secure airway
- Burns – secure airway (ETT/surgical airway)
- Tumours – secure airway (ETT/surgical airway)

Urinary retention

Theory

Acute urinary retention is the painful distension of the bladder with the inability to void.

It is commonly secondary to underlying bladder outlet obstruction and may occur associated with pre-existing symptoms (decreased urine flow, hesitancy, nocturia, etc.).

Acute urinary retention may be triggered in patients with or without bladder neck obstruction by other factors common in the hospital inpatient.

In the surgical patient, immobility, pain, analgesics, sedatives, regional anaesthetic, constipation.

In the medical patient, drugs such as diuretics, anticholinergics, bed confinement, stroke, confusion, constipation.

Chronic urinary retention is painless and usually detected after presentation with overflow incontinence and a large volume palpable bladder. These patients often have distension also of the upper urinary tract and associated renal impairment.

Immediate intern management

1. Acute retention

Insert a Foley catheter.

Give parenteral narcotic if there is delay in arranging a catheter setup or if the patient is restless with severe pain (otherwise the fastest pain relief is achieved by catheterisation).

Explain clearly the procedure to the patient.

Sterile procedure – gown, gloves, mask, prep and sterile drape.

Urethral lignocaine gel instill slowly and gently.

Use a size 14 or 16 Fr Foley catheter (5–10 ml balloon). Insert while putting the penile urethra on gentle stretch. Advance the catheter gently without force. There may be a temporary holdup at the level of the pelvic floor (external sphincter). If so, stop and ask the patient to slowly breathe in and out through the mouth. Advance the catheter again as the patient is exhaling. If the catheter has passed up to the hilt and there is urine flow, then inflate the Foley balloon. Sometimes suction using the lignocaine gel syringe and nozzle is needed to initiate urine flow. Never inflate the balloon until the catheter tip is definitely in the bladder. Attach the catheter to the sterile catheter bag. Ensure the bag is emptied after 5 minutes and the volume is recorded on the fluid balance chart. It is very important that this volume (volume of urine in the bladder obtained by catheterisation) is known to help with deciding the appropriate further management.

If catheterisation is unsuccessful:

Give parenteral narcotic (if not already given). Try a smaller size 12Fr catheter. If still unsuccessful, call the surgical registrar (or if available, urology registrar).

The likely causes of catheter failure include urethral stricture and bladder neck contracture (following previous transurethral surgery). Success may be achieved after urethral dilatation. Otherwise a suprapubic catheter is the best solution, but this and urethral dilatation should only be attempted by an experienced registrar.

2. Chronic retention

Catheterise as with acute retention.

In the presence of significant renal impairment, watch for post-obstructive diuresis. Measure urine output hourly. Usually in an alert and cooperative patient, excess fluid loss can be replaced with oral fluids, otherwise IV fluids may be necessary. Rate of IV fluids is determined by the hourly urine output. Usually replace ½ urine output with IV fluid. The most common mistake is to over-replace with IV fluids.

NEVER clamp and release a urinary catheter.

3. Clot retention

Retention may occur due to heavy haematuria of whatever cause. Clot formation in the bladder obstructs the passage of urine or blocks an indwelling catheter.

First evacuate the clots from the bladder with a large catheter and a Toomey syringe. This is best done with the catheter balloon deflated. The catheter is flushed and aspirated with N saline using the Toomey syringe until ALL the clots are out. This step can be done using a 3-way catheter (at least 22Fr).

Remember that the size (French) relates to the outside diameter not the size of the drainage channel. Because a 3-way catheter also incorporates an irrigation channel, the drainage channel of an 18Fr 2-way is similar to a 22Fr 3-way catheter.

Next commence continuous bladder irrigation via a 3-way catheter with the aim of washing out the bloodstained urine to PREVENT further clot formation. The irrigation will not wash out clot so DO NOT irrigate unless the clots have been first evacuated manually with a Toomey syringe.

Wound disruption

Theory

Wound disruption or dehiscence can occur within a few days or weeks post surgery. There are a number of factors that contribute to it.

Disruption exposes the underlying structures and therefore nearly always requires surgical correction.

Immediate management is aimed at stabilising the patient and notifying senior staff.

Immediate intern management

Attend patient and assess.

1. ABC.
2. Clinical assessment.
3. Inspect wound.
4. IV access IV fluids and basic blood tests.
5. Nil orally.
6. Reassure patient.
7. Call surgical registrar and unit registrar.

If vital structures/bowel exposed

Cover exposed structure:

- Bowel – saline soaked sterile towel.
- Vital structure – saline soaked gauze.

Clinical features

History

- Basic patient information
- Nature of recent operation
- Events leading to wound disruption
- Past history
- ? Last meal

Examination

- Basic patient parameters
- Wound
    - Nature and site
    - Underlying/exposed structures
    - Any discharge (colour, smell, and in particular blood stained fluid or fresh blood)
- Surrounding tissues
    - Erythema, swelling, purpura

Abdominal wound dehiscence classically occurs from days five to ten and is heralded by serous wound discharge.

Predisposing factors

1. Elderly
2. Multiple medical problems
3. Diabetes
4. Cancer
5. Malnourishment
6. Immunosuppressants
7. Smoker
8. Poor technique

Causes

- Increasing tension deep to sutures – abdominal compartment syndrome
- Infection
- Wound necrosis
- Haematoma
- Sudden strains across wound eg. coughing, vomiting

Further definitive management

- Surgical consultation and review required
- Decision for re-intervention depends on exposed structures and cause for wound disruption
- Exposed bowel/vital structure
  - Surgical intervention and repair
  - Consider using tension sutures in abdominal wound dehiscence
- Gross infection wound/abscess
  - May elect to leave wound exposed
  - Pack and dress to allow healing by secondary intention
- Haematoma
  - Evacuate
  - Stop bleeding – direct/pack wound

Other considerations

- Try and correct predisposing factors:
  - Optimise diabetic control
  - ? Need immunosuppressants/steroids
  - Pre-operative nutritional support/supplementation
  - Smoking cessation
- Seriously consider indications for surgery
- A serous discharge from an abdominal wound should alert to the possibility of impending wound disruption

The Australian Curriculum Framework for Junior Doctors

(Confederation of Postgraduate Medical Education Councils)

The Australian Curriculum Framework for Junior Doctors (the 'Curriculum Framework') outlines the knowledge, skills and behaviours required of prevocational doctors (PGY1, PGY2 and above) in order to work safely in Australian hospitals and other healthcare settings. As such, it provides a bridge between undergraduate curricula and the curricula that underpin college training programs. The Curriculum Framework provides junior doctors with an educational template that clearly identifies the core competencies and capabilities that are required to provide quality health care.

Visit the Australian Curriculum Framework for Junior Doctors website at: http://curriculum.cpmec.org.au/

Glossary of acronyms

Acronym	Definition
AAA	Abdominal Aortic Aneurysm
ABC	Airway/Breathing/Circulation
ABG	Arterial Blood Gas
AF	Atrial Fibrillation
AMI	Acute Myocardial Infarct
APO	Acute Pulmonary Oedema
AXR	Abdominal X-Ray
ARDS	Acute Respiratory Distress Syndrome
ASD	Atrial Septal Defect
BetaHCG	Beta-Human Chorionic Gonadotropin (ß-hCG)
BiPAP	Bilevel Positive Airways Pressure
BP	Blood Pressure
BSL	Blood Sugar Level
CCF	Congestive Cardiac Failure
CF	Cardiac Failure
CO	Cardiac Output
COAD	Chronic Obstructive Airways Disease
CPAP	Continuous Positive Airways Pressure
CPR	Cardiopulmonary Resuscitation
CRP	C Reactive Protein
CT	Computerised Tomogram
CTPA	Computerised Tomographic Pulmonary Angiogram

Acronym	Definition
CXR	Chest X-Ray
CVA	Cerebro-Vascular Accident
CVC	Central Venous Catheter
DCR	Direct Cardioversion
DKA	Diabetic Ketoacidosis
DSA	Digital Subtraction Angiography
DVT	Deep Vein Thrombosis
ECG	Electrocardiogram
EMD	Electromechanical Dissociation
ESR	Erythrocyte Sedimentation Rate
ETT	Endotracheal Tube
FBE	Full Blood Examination
GA	General Anaesthetic
G&H	Group & Hold
GCS	Glasgow Coma Score
GORD	Gastro Oesophageal Reflux Disease
GTN	Glyceryl Tri Nitrate
HDU	High Dependency Unit
HONKC	Hyperosmolar Non-Ketotic-Coma
HR	Heart Rate
HRT	Hormone Replacement Therapy
IBD	Inflammatory Bowel Disease
IC	Intercostal Catheter
ICU	Intensive Care Unit
ID	Infectious Diseases
IDC	Indwelling Catheter

Acronym	Definition
IHD	Ischaemic Heart Disease
IM	Intramuscular
INR	International Normalised Ratio
IV	Intravenous
IVC	Intravenous Catheter
JVP	Jugular Venous Pressure
KUB	Kidney, Ureters and Bladder X-ray
LBO	Large Bowel Obstruction
LFT	Liver Function Test
LMN	Lower Motor Neurone
LOW	Loss Of Weight
MET	Medical Emergency Team
MRA	Magnetic Resonance Angiogram
MRI	Magnetic Resonance Imaging
MRSA	Methicillin Resistant Staphylococcus Aureus
NGT	Nasogastric Tube
OCP	Oral Contraceptive Pill
OD	Overdose
PE	Pulmonary Embolus
PHx	Past History
PND	Paroxysmal Nocturnal Dyspnoea
POC	Products of Conception
PR	Per Rectum
PSA	Prostate Specific Antigen
PTH	Parathyroid Hormone
PVD	Peripheral Vascular Disease

Acronym	Definition
QID	4 times a day
QRS	QRS complex on ECG
RV	Right Ventricle
SBP	Systolic Blood Pressure
Sc	Subcutaneous
S/C	Subcutaneous
SBO	Small Bowel Obstruction
SOA	Swelling Of Ankles
SOB	Shortness Of Breath
SOBOE	Shortness Of Breath On Exertion
SV	Stroke Volume
ST	Segment On ECG
TB	Tuberculosis
TFT	Thyroid Function Test
TP	Tension Pneumothorax
TPR	Total Peripheral Resistance
UMN	Upper Motor Neurone
U&E	Urea & Electrolytes
US	Ultrasound
VF	Ventricular Fibrillation
V/Q	Ventilation Perfusion Quotient
VSD	Ventricular Septal Defect
VT	Ventricular Tachycardia

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