Addisonian Crisis

Causes

- Sepsis or surgery causing an acute exacerbation of chronic insufficiency (Addison's, Hypopituitarism)
- Adrenal haemorrhage e.g. Waterhouse-Friderichsen syndrome (fulminant meningococcemia)
- Steroid withdrawal

Management

- Hydrocortisone **100mg** IM or IV
- 1 litre normal saline infused over 30-60 min or with dextrose if hypoglycaemic
- Continue hydrocortisone 6 hourly until the patient is stable. No fludrocortisone is required because high cortisol exerts weak mineralocorticoid action
- Oral replacement may begin after 24 hours and be reduced to maintenance over 3-4 days

Anaphylactic Shock

Suspect if there has been exposure to an allergen

Management

- Remove allergen
- ABCD
- Drugs:
    - **Adrenaline** 1:1000 0.5ml INTRAMUSCULARLY (not IV). Repeat after 5 min if no response.
    - Then **Chlorpheniramine** 10mg IV
    - Then **Hydrocortisone** 100-200mg IV

Compartment Syndrome

- This is a particular complication that may occur following fractures (or following ischaemia re-perfusion injury in vascular patients). It is characterised by raised pressure within a closed anatomical space.
- The raised pressure within the compartment will eventually compromise tissue perfusion resulting in necrosis. The two main fractures carrying this complication include supracondylar fractures and tibial shaft injuries.

Symptoms and signs

- Pain, especially on movement (even **passive**)
- **Parasthesiae**
- Pallor may be present
- Arterial pulsation may still be felt as the necrosis occurs as a result of microvascular compromise
- Paralysis of the muscle group may occur

Diagnosis

- Is made by measurement of intracompartmental pressure measurements. Pressures in excess of 20mmHg are abnormal and >40mmHg is diagnostic.

Treatment

- This is essentially prompt and extensive fasciotomies
- In the lower limb the deep muscles may be inadequately decompressed by the inexperienced operator when smaller incisions are performed
- Myoglobinuria may occur following fasciotomy and result in renal failure and for this reason these patients require aggressive IV fluids
- Where muscle groups are frankly necrotic at fasciotomy they should be debrided and amputation may have to be considered
- Death of muscle groups may occur within 4-6 hours

Fluid Resuscitation Burns

Indication: >15% total body area burns in adults (>10% children)

- The main aim of resuscitation is to prevent the burn deepening
- Most fluid is lost 24 hours after injury
- First 8-12 hours, fluid shifts are from intravascular to interstitial fluid compartments
- Therefore, circulatory volume can be compromised. However fluid resuscitation causes more fluid into the interstitial compartment especially colloid (therefore avoided in first 8-24 hours)
- Protein loss occurs

Fluid resuscitation formula

Parkland formula
(Crystalloid only e.g. Hartman's solution / Ringers' lactate)

Total fluid requirement in 24 hours =
4ml x total burn surface area (%) x body weight (kg)

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

Resuscitation endpoint: Urine output of 0.5-1.0 ml/kg/hour in adults (increase rate of fluid to achieve this)

Points to note:

- Starting point of resuscitation is time of injury
- Deduct fluids already given

After 24 hours

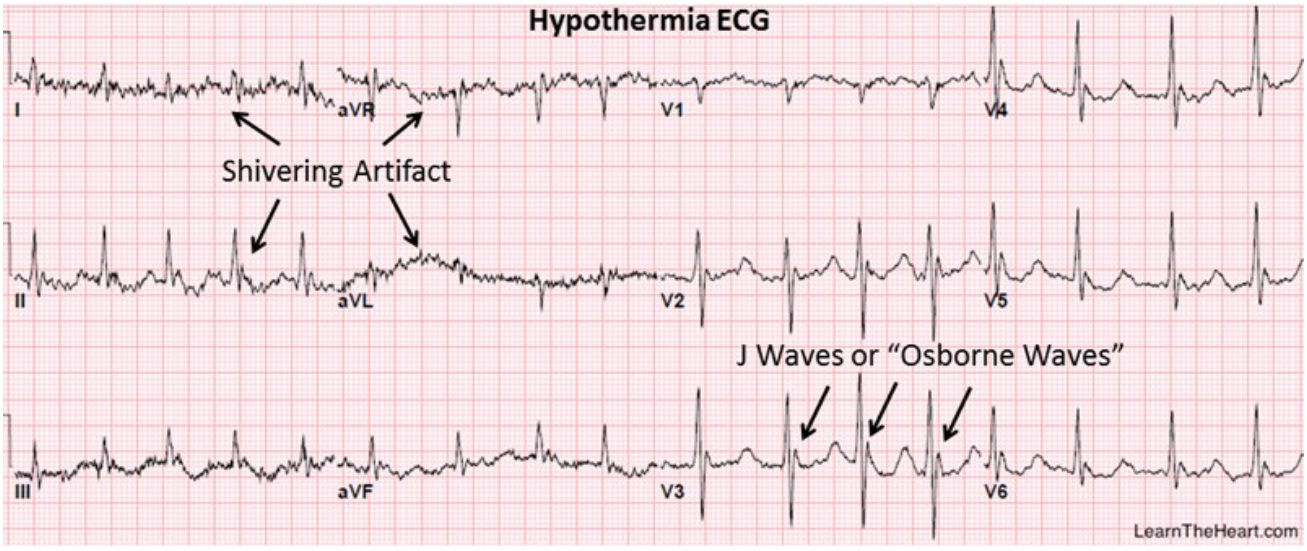
- Maintenance crystalloid (usually dextrose-saline) is continued at a rate of *1.5 ml x (burn area) x (body weight)*
- Colloids are rarely used (e.g. albumin)
- Antioxidants, such as vitamin C, can be used to minimize oxidant-mediated contributions to the inflammatory cascade in burns
- High tension electrical injuries and inhalation injuries require more fluid
- Monitor: packed cell volume, plasma sodium, base excess, and lactate

Hypothermia

Core body temperature below 35°C. Severe hypothermia is present when the core temperature is below 30°C. Hypothermia is associated with a reduction in both respiratory and cardiac activity.

Management

An organised cardiac rhythm may be converted to fibrillation if CPR is attempted inappropriately so ECG should be analysed with care. The rewarming technique used depends upon the degree of hypothermia and the physiological state of the patient. ***Mild hypothermia*** may respond to external rewarming devices. ***Severe hypothermia*** may require active core rewarming techniques such as peritoneal lavage, haemodialysis or cardiac bypass. Patients who develop cardiac arrhythmias who are severely hypothermic may respond to bretylium tosylate (sadly no longer available in most centres), but do not generally respond to standard therapies or DC cardioversion.



Local Anaesthetic Toxicity

Toxicity results from either accidental intravascular injection (rapid onset of symptoms-usually correct dose), or from excessive dosage (slower onset). Local anaesthetic agents not only exert a membrane stabilising effect on peripheral nerves but will also act on excitable membranes within the CNS and Heart. The sensory neurones in the CNS are suppressed before the motor ones. As a result, the early symptoms will typically be those of circumoral paraesthesia and tinnitus, followed by falling GCS and eventually coma.

Management of toxicity

- Stop injecting the anaesthetic agent
- High flow 100% oxygen via face mask
- Cardiovascular monitoring
- Administer lipid emulsion (**Intralipid** 20%) at 1.5ml/Kg over 1 minute as a bolus
- Consider lipid emulsion infusion, at 0.25ml/ Kg/ minute
- If toxicity due to **prilocaine** then administer **methylene blue**

Safe doses

10ml of lignocaine 1% contains 100mg of drug, this would constitute 70% of the maximum safe dose in a 50 kg patient. Up to 7mg / kg can be administered if adrenaline is added to the solution.

Doses of local anaesthetics

Agent	Dose plain	Dose with adrenaline
Lignocaine	3mg/Kg	7mg/Kg
Bupivicane	2mg/Kg	2mg/Kg
Prilocaine	6mg/Kg	9mg/Kg

These are a guide only as actual doses depend on site of administration, tissue vascularity and co-morbidities.

Chest Pain in Pregnancy

Aortic dissection

- Predisposing factors in pregnancy are hypertension, congenital heart disease and Marfan's syndrome
- Mainly *Stanford type A* dissection
- Sudden tearing chest pain, transient syncope
- Patient may be cold and clammy, hypertensive and have an aortic regurgitation murmur
- Involvement of the right coronary artery may cause inferior myocardial infarction

Surgical management

Gestational timeframe	Management
< 28/40	Aortic repair with the fetus kept in utero
28-32/40	Dependent on fetal condition
> 32/40	Primary Cesarean section followed by aortic repair at the same operation

Mitral stenosis

- Most cases associated with rheumatic heart disease
- Becoming less common in British women; suspect in Immigrant women
- Commonest cardiac condition in pregnancy
- Commonly associated with mortality
- Valve surgery; balloon valvuloplasty preferable

Pulmonary embolism

- Leading cause of mortality in pregnancy
- Half dose scintigraphy; CT chest if underlying lung disease, should aid diagnosis
- Treatment with low molecular weight heparin throughout pregnancy and 4-6 weeks after childbirth
- Warfarin is contra indicated in pregnancy (though may be continued in women with mechanical heart valves due to the significant risk of thromboembolism)

Imaging in the Pregnant Trauma Patient

Sonography and FAST scanning are established in pregnancy and have the advantage of avoiding ionising radiation. However, the sensitivity of the FAST scan is reduced in pregnancy especially with advanced gestational age. Sensitivity of FAST scanning is 60-80% across all trimesters and 90% in the first. CT scanning remains the first line investigation in major trauma where significant visceral injury is suspected. The maximum permitted safe dose of radiation in pregnancy is 5mSv. A pelvic CT scan would fall below this level. That said, early exposure to radiation will increase the risk of developmental anomalies and foetal loss. Late exposure increases the risk of childhood cancer twofold. CT scanning remains the most sensitive test for identifying complications such as placental abruption in this group.

Management of Acute Coronary Syndrome

NICE produced guidelines in 2010 on the management of unstable angina and non-ST elevation myocardial infarction (NSTEMI). They advocate managing patients based on the early risk assessment using a recognised scoring system such as GRACE (Global Registry of Acute Cardiac Events) to calculate a predicted 6 month mortality.

All patients should receive

- Aspirin 300mg
- Nitrates or morphine to relieve chest pain if required

Whilst it is common that non-hypoxic patients receive oxygen therapy there is little evidence to support this approach. The 2008 British Thoracic Society oxygen therapy guidelines advise not giving oxygen unless the patient is hypoxic.

Antithrombin treatment. Low molecular weight heparin should be offered to patients who are not at a high risk of bleeding and who are not having angiography within the next 24 hours. If angiography is likely within 24 hours or a patients creatinine is > 265 umol/l unfractionated heparin should be given.

Clopidogrel 300mg should be given to patients with a predicted 6 month mortality of more than 1.5% or patients who may undergo percutaneous coronary intervention within 24 hours of admission to hospital. Clopidogrel should be continued for 12 months.

Intravenous **glycoprotein IIb/IIIa receptor antagonists**(eptifibatide or tirofiban) should be given to patients who have an intermediate or higher risk of adverse cardiovascular events (predicted 6-month mortality above 3.0%), and who are scheduled to undergo angiography within 96 hours of hospital admission.

Coronary angiography should be considered within 96 hours of first admission
to hospital to patients who have a predicted 6-month mortality above 3.0%. It should also be performed as soon as possible in patients who are clinically unstable.

Thrombolysis or Percutaneous Intervention in Myocardial Infarction

Thrombolytic drugs activate plasminogen to form plasmin. This in turn degrades fibrin and help breaks up thrombi. They in primarily used in patients who present with a ST elevation myocardial infarction. Other indications include acute ischaemic stroke and pulmonary embolism, although strict inclusion criteria apply.

Examples

- Alteplase
- Tenecteplase
- Streptokinase

Indications for thrombolysis or PCI: (Any of the following ECG changes):

- ST elevation of > 2mm (2 small squares) in 2 or more consecutive anterior leads *(V1-V6)*
- ST elevation of > 1mm (1 small square) in greater than 2 consecutive inferior leads *(II, III, avF, avL)*
- New Left Bundle Branch Block

Contraindications to thrombolysis

- Active internal bleeding
- Recent haemorrhage, trauma or surgery (including dental extraction)
- Coagulation and bleeding disorders
- Intracranial neoplasm
- Stroke < 3 months
- Aortic dissection
- Recent head injury
- Pregnancy
- Severe hypertension

Side-effects

- Haemorrhage
- Hypotension - more common with streptokinase
- Allergic reactions may occur with streptokinase

Ventricular Tachycardia

Ventricular tachycardia (VT)is broad-complex tachycardia originating from a ventricular ectopic focus. It has the potential to precipitate ventricular fibrillation and hence requires urgent treatment.

There are two main types of VT:

- monomorphic VT: most commonly caused by myocardial infarction
- polymorphic VT: A subtype of polymorphic VT is torsades de pointes which is precipitated by prolongation of the QT interval. The causes of a long QT interval are listed below

Causes of a prolonged QT interval

Congenital

- Jervell-Lange-Nielsen syndrome (includes deafness and is due to an abnormal potassium channel)
- Romano-Ward syndrome (no deafness)

Drugs

- Amiodarone, sotalol, class 1a antiarrhythmic drugs
- Tricyclic antidepressants, fluoxetine
- Chloroquine
- Terfenadine
- Erythromycin

Other

- Electrolyte: hypocalcaemia, hypokalaemia, hypomagnesaemia
- Acute myocardial infarction
- Myocarditis
- Hypothermia
- Subarachnoid haemorrhage

Ventricular Tachycardia: Management

Whilst a broad complex tachycardia may result from a supraventricular rhythm with aberrant conduction, the European Resuscitation Council advise that in a peri-arrest situation it is assumed to be ventricular in origin

If the patient has adverse signs (systolic BP < 90 mmHg, chest pain, heart failure or rate > 150 beats/min) then immediate cardioversion is indicated. In the absence of such signs antiarrhythmics may be used. If these fail, then electrical cardioversion may be needed with synchronised DC shocks

Drug therapy

- Amiodarone: ideally administered through a central line
- Lidocaine: use with caution in severe left ventricular impairment
- Procainamide

Verapamil should NOT be used in VT

If drug therapy fails

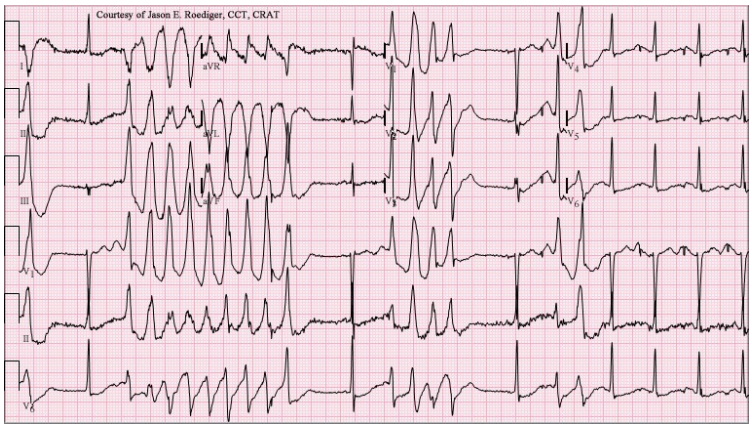
- Electrophysiological study (EPS)
- Implant able cardioverter-defibrillator (ICD) - this is particularly indicated in patients with significantly impaired LV function

Torsades De Pointes

Torsades de pointes ('twisting of the points') is a rare arrhythmia associated with a long QT interval. It may deteriorate into ventricular fibrillation and hence lead to sudden death

Causes of long QT interval

- Congenital: Jervell-Lange-Nielsen syndrome, Romano-Ward syndrome
- Antiarrhythmics: amiodarone, sotalol, class 1a antiarrhythmic drugs
- Tricyclic antidepressants
- Antipsychotics
- Chloroquine
- Terfenadine
- Erythromycin
- Electrolyte: hypocalcaemia, hypokalaemia, hypomagnesaemia
- Myocarditis
- Hypothermia
- Subarachnoid haemorrhage



Management

- IV magnesium sulphate

Management of Hyperkalaemia

Untreated hyperkalaemia may cause life-threatening arrhythmias. Precipitating factors should be addressed (e.g. acute renal failure) and aggravating drugs stopped (e.g. ACE inhibitors). Management may be categorised by the aims of treatment

Stabilisation of the cardiac membrane

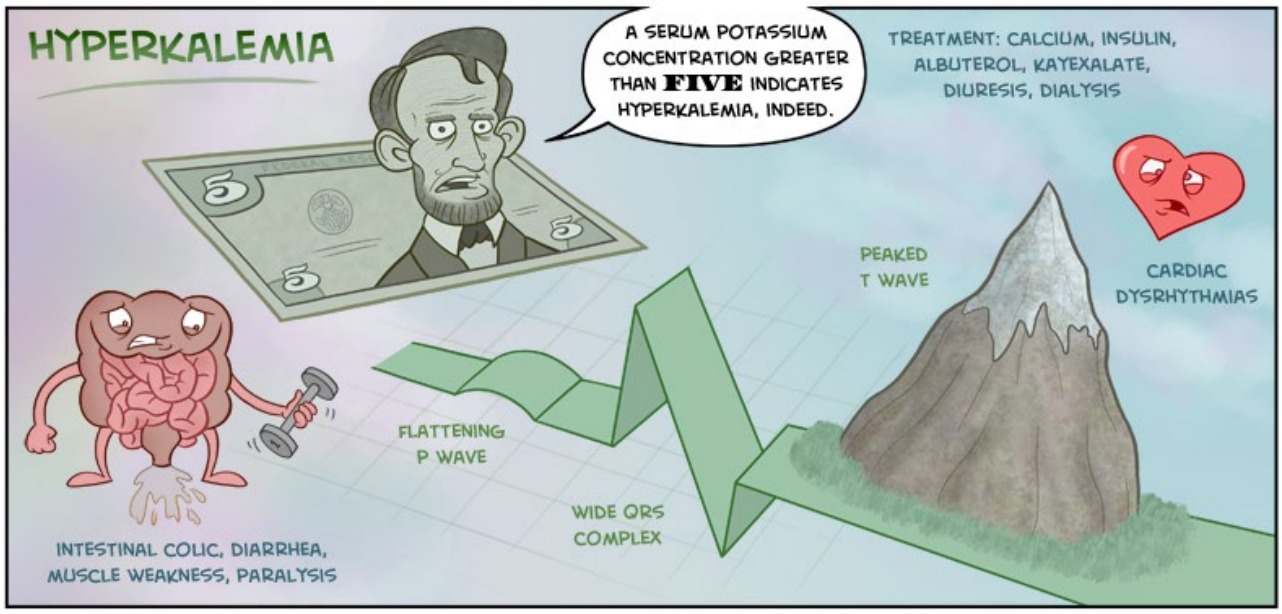
- Intravenous calcium gluconate

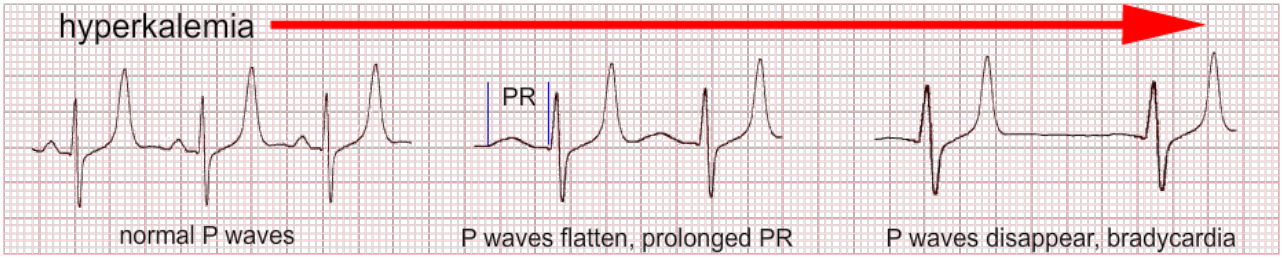
Short-term shift in potassium from extracellular to intracellular fluid compartment

- Combined insulin/dextrose infusion
- Nebulised salbutamol

Removal of potassium from the body

- Calcium resonium (orally or enema)
- Loop diuretics
- Dialysis





Pulmonary Embolism (PE)

Investigations

The British Thoracic Society (BTS) published guidelines in 2003 on the management of patients with suspected PE

Key points from the guidelines include:

- Computed tomographic pulmonary angiography (**CTPA**) is now the recommended initial lung-imaging modality for non-massive PE. Advantages compared to V/Q scans include speed, easier to perform out-of-hours, a reduced need for further imaging and the possibility of providing an alternative diagnosis if PE is excluded
- If the CTPA is negative, then patients do not need further investigations or treatment for PE
- Ventilation-perfusion scanning may be used initially if appropriate facilities exist, the chest x-ray is normal, and there is no significant symptomatic concurrent cardiopulmonary disease

Some other points

Clinical probability scores based on risk factors and history and now widely used to help decide on Ix & Mx

Modified Wells Criteria for predicting PE

A score of <4 means PE is unlikely (12.4%), >4 is suggestive of PE (37.1%).

Variable	Score
Clinical signs and symptoms of deep vein thrombosis (DVT) *(minimum of leg swelling and pain on palpation of deep veins)*	3
Alternative diagnosis less likely than PE	3
Heart rate >100 bpm	1.5
Immobilisation > 3 days or surgery within past 4 weeks	1.5
Previous DVT or PE	1.5
Haemoptysis	1
Malignancy (treatment or palliation within past 6 months)	1

D-dimers

- Sensitivity = 95-98%, but poor specificity

V/Q scan

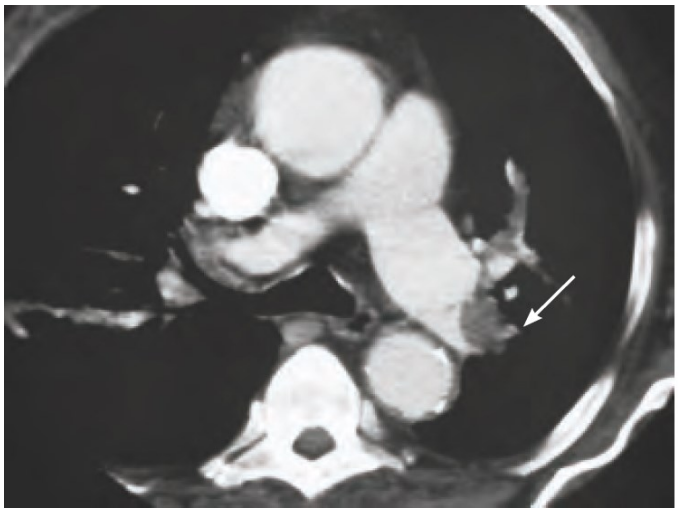
- Sensitivity = 98%; specificity = 40% - high negative predictive value, i.e. if normal virtually excludes PE
- Other causes of mismatch in V/Q include old pulmonary embolisms, AV malformations, vasculitis, previous radiotherapy
- COPD gives matched defects

Pulmonary angiography

- Significant complication rate compared to other investigations

CTPA

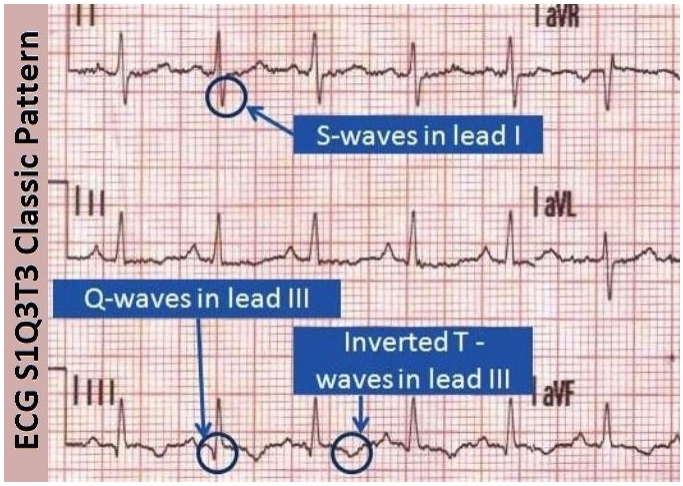
- **Current gold standard**
- Peripheral emboli affecting subsegmental arteries may be missed



A computed tomography pulmonary angiogram showing pulmonary emboli as filling defects (arrow) in the pulmonary artery.

ECG Changes

- No changes
- **S1, Q3, T3**
- Tall R waves: V1
- **P** pulmonale (peaked P waves): inferior leads
- **R**ight axis deviation, Right bundle branch block
- **A**trial arrhythmias
- **T** wave inversion: V1, V2, V3
- Right ventricular strain: if identified is associated with adverse short-term outcome and adds prognostic value to echocardiographic evidence of right ventricular dysfunction in patients with acute pulmonary embolism and normal BP



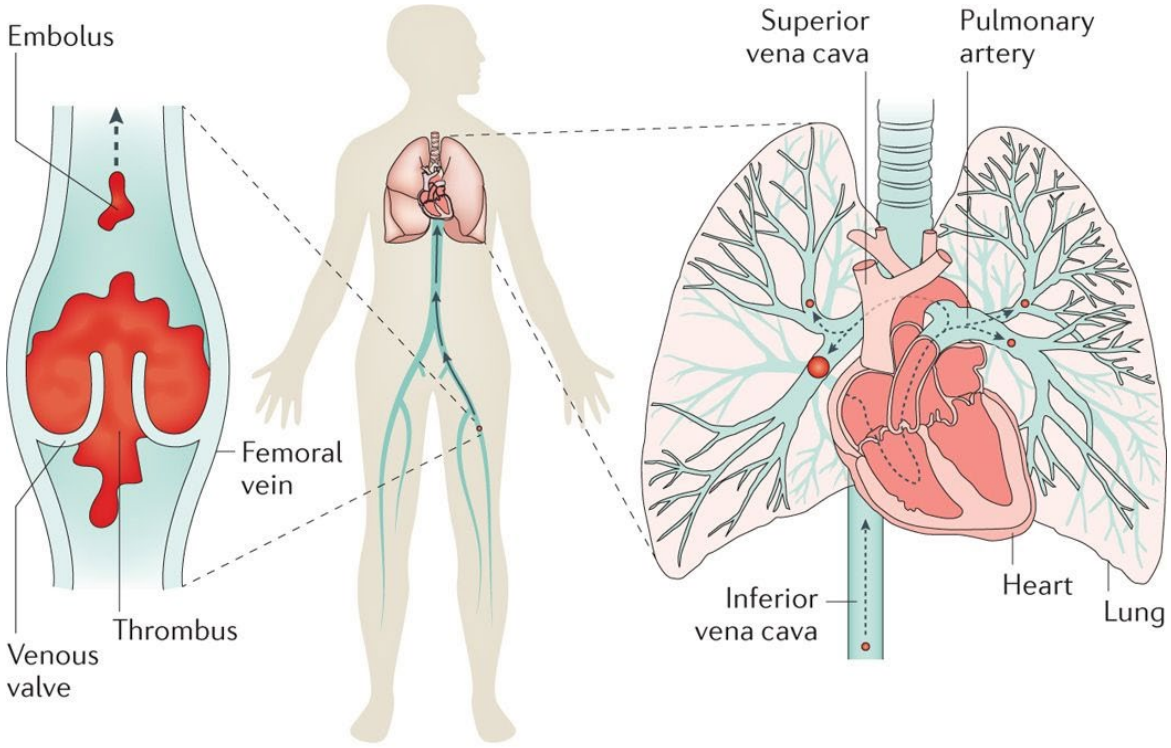


Diagram of femoral vein thrombus causing PE

Management of pulmonary embolism

A summary of the British Thoracic Society guidelines

- Heparin should be given if intermediate or high clinical probability **before** imaging.
- Unfractionated heparin (UFH) should be considered ***(a)*** as a first dose bolus, ***(b)*** in massive PE, or ***(c)*** where rapid reversal of effect may be needed.
- Otherwise, low molecular weight heparin (LMWH) should be considered as preferable to UFH, having equal efficacy and safety and being easier to use.
- Oral anticoagulation should only be commenced once VTE has been reliably confirmed.
- The target INR should be 2.0-3.0; when this is achieved, heparin can be discontinued.
- The standard duration of oral anticoagulation is: 4 to 6 weeks for temporary risk factors, 3 months for first idiopathic, and at least 6 months for other; the risk of bleeding should be balanced with that of further VTE.

Massive PE

- **CTPA** or echocardiography will reliably diagnose clinically massive PE.
- **Thrombolysis is 1st line for massive PE** (i.e. circulatory failure) and may be instituted on clinical grounds alone if cardiac arrest is imminent; a 50 mg bolus of alteplase is recommended.
- Invasive approaches (thrombus fragmentation and IVC filter insertion) should be considered where facilities and expertise are readily available.

Indications of IVC filter

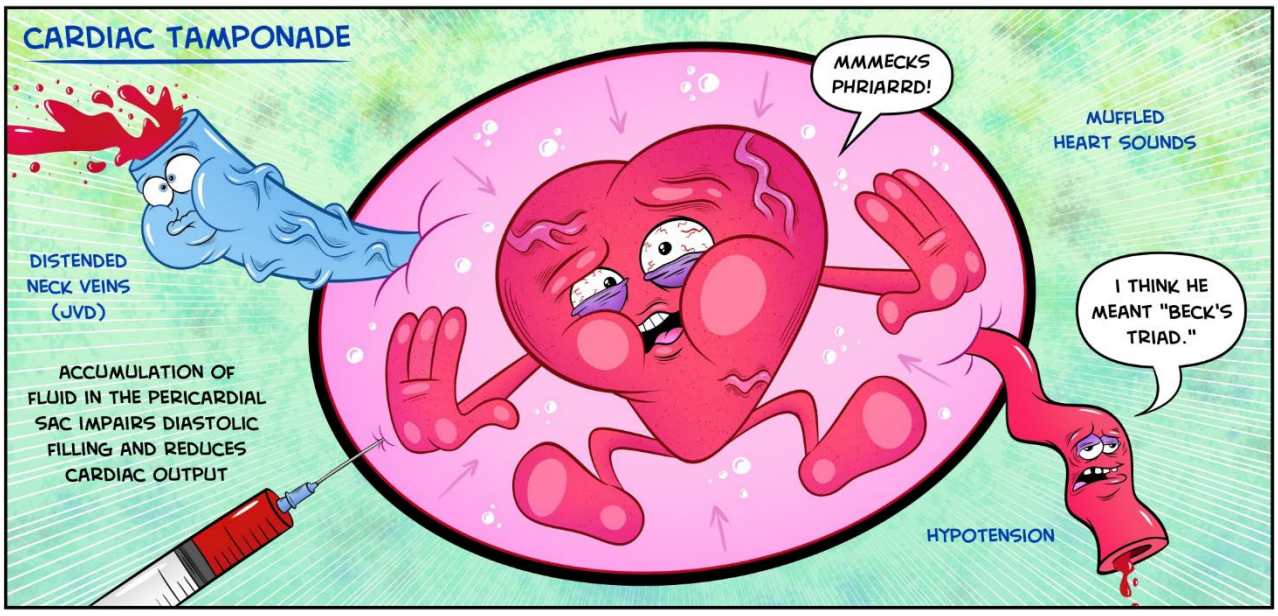
- Contraindications to anticoagulation
- Failure of anticoagulation
- Massive PE with residual DVT in a patient at risk for further PE
- Recurrent PE
- Trauma patient with high risk of VTE

Also see Thromboprophylaxis in Peri-operative Care

Thoracic Trauma

Types of thoracic trauma

Tension pneumothorax	• Often laceration to lung parenchyma with flap • Pressure develops in thorax • Most common cause is mechanical ventilation in patient with pleural injury • Symptoms overlap with cardiac tamponade, hyper-resonant percussion note is more likely in tension pnemothorax
Flail chest	• Chest wall disconnects from thoracic cage • Multiple rib fractures (at least two fractures per rib in at least two ribs) • Associated with pulmonary contusion • Abnormal chest motion • Avoid over hydration and fluid overload
Pneumothorax	• Most common cause is lung laceration with air leakage • Most traumatic pneumothoraces should have a chest drain • Patients with traumatic pneumothorax should never be mechanically ventilated until a chest drain is inserted
Haemothorax	• Most commonly due to laceration of lung, intercostal vessel or internal mammary artery • Haemothoraces large enough to appear on CXR are treated with large bore chest drain • Surgical exploration is warranted if >1500ml blood drained immediately
Cardiac tamponade	• Beck's triad: elevated venous pressure, reduced arterial pressure, reduced heart sounds • Pulsus paradoxus • May occur with as little as 100ml blood
Pulmonary contusion	• Most common potentially lethal chest injury • Arterial blood gases and pulse oximetry important • Early intubation within an hour if significant hypoxia
Blunt cardiac injury	• Usually occurs secondary to chest wall injury • ECG may show features of myocardial infarction • Sequelae: hypotension, arrhythmias, cardiac wall motion abnormalities
Aorta disruption	• Deceleration injuries • Contained haematoma • Widened mediastinum
Diaphragm disruption	• Most due to motor vehicle accidents and blunt trauma causing large radial tears (laceration injuries result in small tears) • More common on left side • Insert gastric tube, may pass into intrathoracic stomach
Mediastinal traversing wounds	• Entrance wound in one hemithorax and exit wound/foreign body in opposite hemithorax • Mediastinal haematoma or pleural cap suggests great vessel injury • Mortality is 20%



Tension Pneumothorax

Tension pneumothorax is a state of positive pressure within a pneumothorax throughout the respiratory cycle. A breach in the pleura allows air into the intra pleural space via a one way valve. The initial pressure pneumothorax expands until positive pressure is present throughout the respiratory cycle. The risk is greatest in the ventilated trauma patient as positive pressure is used. Undiagnosed tension pneumothorax accounts for 3.8% of trauma deaths.

Clinically, the classic features include chest pain, dyspnoea, hypoxia, hypotension, tracheal deviation, ipsilateral hyperpercussion note, decreased air entry. In ventilated patients, cardiovascular disturbance and sub cutaneous emphysema are relatively common and more so than in a case where the patient is breathing spontaneously.

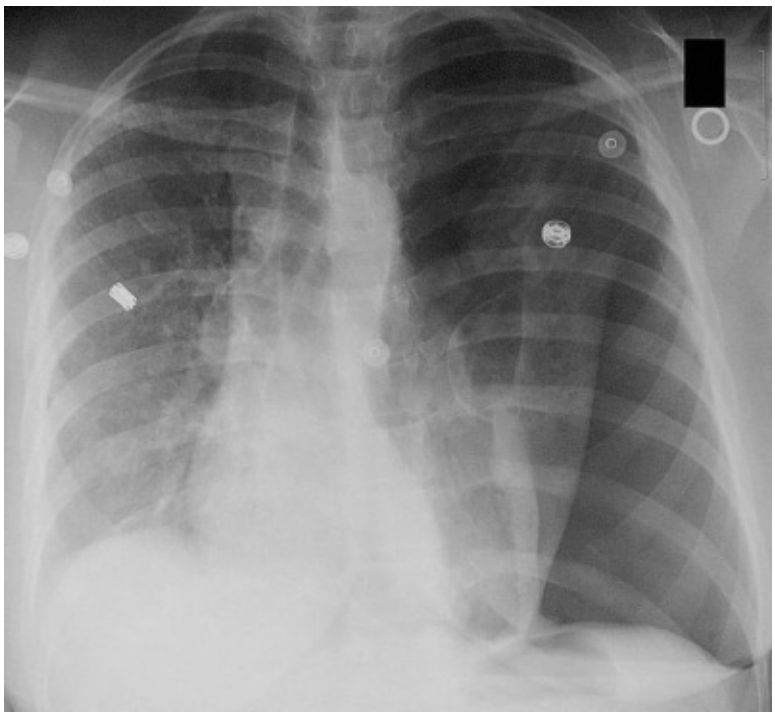


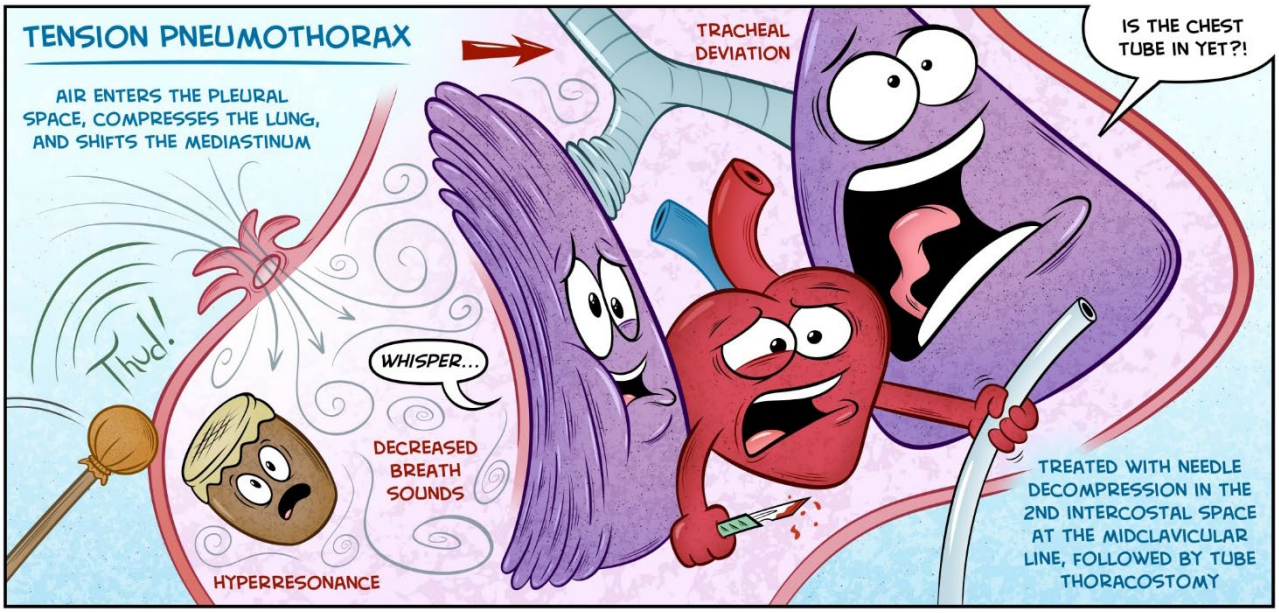
Image showing mediastinal shift with a tension pneumothorax

Chest x-ray features

- Lung collapse towards the hilum
- Diaphragmatic depression Increased rib separation
- Increased thoracic volume
- Ipsilateral flattening of the heart border
- Contra lateral mediastinal deviation

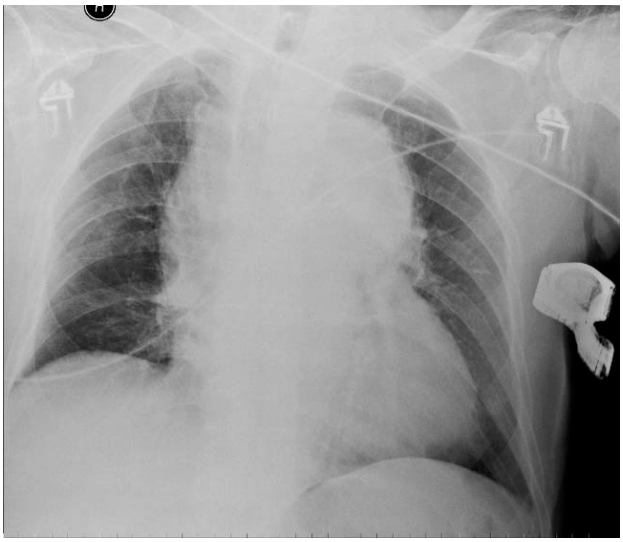
Management

Immediate needle decompression followed by definitive wide bore chest drain insertion



Thoracic Aorta Rupture

- Mechanism of injury: Decelerating force i.e. RTA, fall from a great height
- Most people die at scene
- Survivors may have an incomplete laceration at the ligamentum arteriosum of the aorta.



Clinical features

- Contained haematoma: persistent hypotension
- Detected mainly by history, CXR changes

CXR changes

- Widened mediastinum
- Trachea/Oesophagus to right
- Depression of left main stem bronchus
- Widened paratracheal stripe/paraspinal interfaces
- Space between aorta and pulmonary artery obliterated
- Rib fracture/left haemothorax

Diagnosis

Angiography, usually CT aortogram.

Treatment

Repair or replacement. Ideally they should undergo endovascular repair.

Vascular Trauma

Assessment

- Check for signs of distal perfusion
- Doppler signal distally (monophasic/ biphasic or triphasic)
- Anatomical location (which vessel is likely to be involved)
- Duplex scanning and angiography are "gold standard" tests but may not be immediately available in the trauma setting

Management

- Almost always operative.
- Obtaining proximal and distal control of affected vessels is crucial.
- Simple lacerations of arteries may be directly closed, or a vein patch applied if there is a risk of subsequent stenosis.
- Transection of the vessel should be treated by either end to end anastomosis (often not possible) or an interposition vein graft.
- Use of PTFE in traumatic open injuries will invariably result in infection.

Stroke: Types

Primary intracerebral haemorrhage (PICH, c. 10%)	• Presents with headache, vomiting, loss of consciousness
Total anterior circulation infarcts (TACI, c. 15%)	• Involves middle and anterior cerebral arteries • Hemiparesis/hemisensory loss • Homonymous hemianopia • Higher cognitive dysfunction e.g. Dysphasia
Partial anterior circulation infarcts (PACI, c. 25%)	• Involves smaller arteries of anterior circulation e.g. upper or lower division of middle cerebral artery • Higher cognitive dysfunction or two of the three TACI features
Lacunar infarcts (LACI, c. 25%)	• Involves perforating arteries around the internal capsule, thalamus and basal ganglia • Present with either isolated hemiparesis, hemisensory loss or hemiparesis with limb ataxia
Posterior circulation infarcts (POCI, c. 25%)	• Vertebrobasilar arteries • Presents with features of brainstem damage • Ataxia, disorders of gaze and vision, cranial nerve lesions
Lateral medullary syndrome (posterior inferior cerebellar artery)	• Wallenberg's syndrome • Ipsilateral: ataxia, nystagmus, dysphagia, facial numbness, cranial nerve palsy • Contralateral: limb sensory loss
Weber's syndrome	• Ipsilateral III palsy • Contralateral weakness

Anterior cerebral artery *(branch of ICA)*
- Contralateral hemiparesis and sensory loss, lower extremity > upper
- Disconnection syndrome

Middle cerebral artery *(branch of ICA)*
- Contralateral hemiparesis and sensory loss, upper extremity > lower
- Contralateral hemianopia
- Aphasia (Wernicke's)
- Gaze abnormalities

Posterior cerebral artery *(terminal branch of Basilar artery)*
- Contralateral hemianopia with macular sparing
- Disconnection syndrome

Indications for hemicranieotomy include:
- Age under 60 years
- Clinical deficit in middle cerebral artery territory
- Decreased consciousness
- > 50% territory infarct

Lacunar
- Present with either isolated hemiparesis, hemisensory loss or hemiparesis with limb ataxia

Lateral medulla (posterior inferior cerebellar artery)
- Ipsilateral: ataxia, nystagmus, dysphagia, facial numbness, cranial nerve palsy e.g.

Horner's
- Contralateral: limb sensory loss

Pontine
- VI nerve: horizontal gaze palsy
- VII nerve
- Contralateral hemiparesis

If patient is within 3h of symptom onset of a stroke. Therefore, he should be **urgently referred to the medical team for thrombolysis, BEFORE Aspirin is given**. There are concerns that high dose aspirin would increase the risk of intracerebral haemorrhage if thrombolysis is undertaken.

Head Injury Management - NICE Guidelines

Summary of guidelines

- All patients should be assessed within 15 minutes on arrival to A&E
- Document all 3 components of the GCS
- If GCS <8 or = to 8, consider stabilising the airway
- Treat pain with low dose IV opiates (if safe)
- Full spine immobilisation until assessment if:
    - GCS < 15
    - neck pain/tenderness
    - paraesthesia extremities
    - focal neurological deficit
    - suspected c-spine injury

If a c-spine injury is suspected a 3 view c-spine x-ray is indicated. CT c-spine is preferred if:

- Intubated
- GCS <13
- Normal x-ray but continued concerns regarding c-spine injury
- Any focal neurology
- A CT head scan is being performed
- Initial plain films are abnormal

Immediate CT head (within 1 hour) if:

- GCS < 13 on admission
- GCS < 15 2 hours after admission
- Suspected open or depressed skull fracture
- Suspected skull base fracture (panda eyes, Battle's sign, CSF from nose/ear, bleeding ear)
- Focal neurology
- Vomiting > 1 episode
- Post traumatic seizure
- Coagulopathy

Contact neurosurgeon if:

- Persistent GCS < 8 or = 8
- Unexplained confusion > 4h
- Reduced GCS after admission
- Progressive neurological signs
- Incomplete recovery post seizure
- Penetrating injury
- Cerebrospinal fluid leak

Observations

- 1/2 hourly GCS until 15

Head Injury - Paediatrics

Criteria for immediate request for CT scan of the head (children)

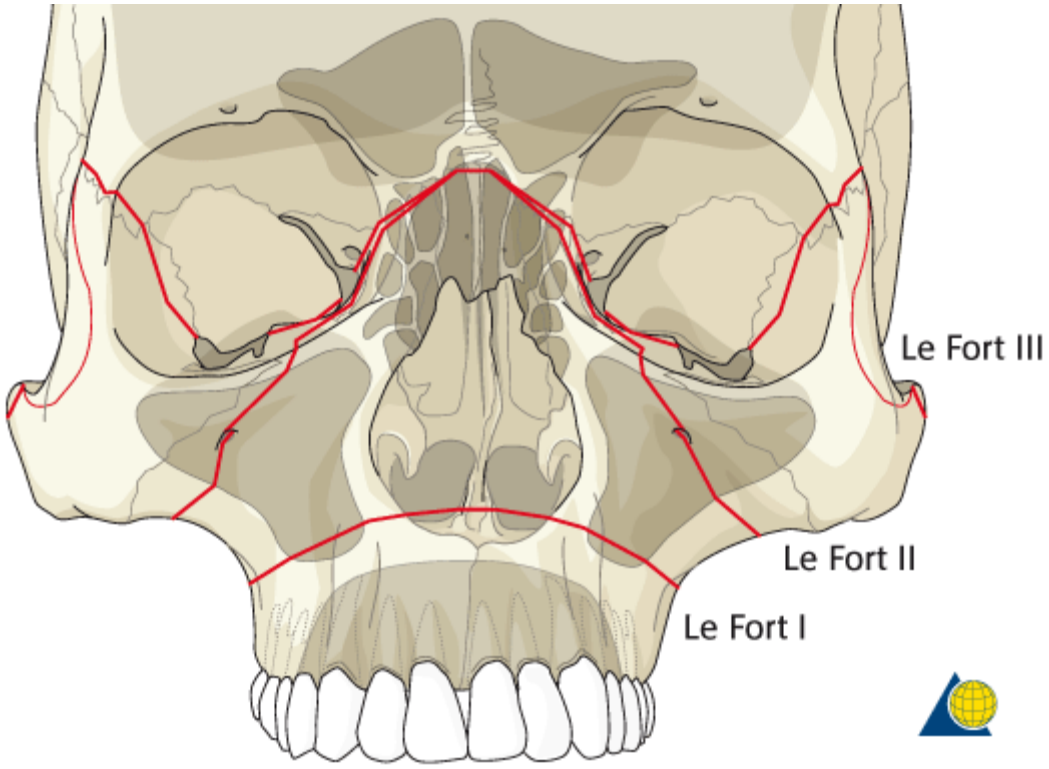
- Loss of consciousness lasting more than 5 minutes (witnessed)
- Amnesia (antegrade or retrograde) lasting more than 5 minutes
- Abnormal drowsiness
- Three or more discrete episodes of vomiting
- Clinical suspicion of non-accidental injury
- Post-traumatic seizure but no history of epilepsy
- GCS less than 14, or for a baby under 1 year GCS (paediatric) less than 15, on assessment in the emergency department
- Suspicion of open or depressed skull injury or tense fontanelle
- Any sign of basal skull fracture (haemotympanum, panda' eyes, cerebrospinal fluid leakage from the ear or nose, Battle's sign)
- Focal neurological deficit
- If under 1 year, presence of bruise, swelling or laceration of more than 5 cm on the head
- Dangerous mechanism of injury (high-speed road traffic accident either as pedestrian, cyclist or vehicle occupant, fall from a height of greater than 3 m, high-speed injury from a projectile or an object)

Craniomaxillofacial Injuries

In the UK are due to: Interpersonal violence (52%), Motor vehicle accidents (16%), Sporting injuries (19%), Falls (11%)

Le Fort Fractures

Grade	Feature
Le Fort 1	The fracture extends from the nasal septum to the lateral pyriform rims, travels horizontally above the teeth apices, crosses below the zygomaticomaxillary junction, and traverses the pterygomaxillary junction to interrupt the pterygoid plates.
Le Fort 2	These fractures have a pyramidal shape and extend from the nasal bridge at or below the nasofrontal suture through the frontal process of the maxilla, inferolaterally through the lacrimal bones and inferior orbital floor and rim through or near the inferior orbital foramen, and inferiorly through the anterior wall of the maxillary sinus; it then travels under the zygoma, across the pterygomaxillary fissure, and through the pterygoid plates.
Le Fort 3	These fractures start at the nasofrontal and frontomaxillary sutures and extend posteriorly along the medial wall of the orbit through the nasolacrimal groove and ethmoid bones. The thicker sphenoid bone posteriorly usually prevents continuation of the fracture into the optic canal. Instead, the fracture continues along the floor of the orbit along the inferior orbital fissure and continues superolaterally through the lateral orbital wall, through the zygomaticofrontal junction and the zygomatic arch. Intranasally, a branch of the fracture extends through the base of the perpendicular plate of the ethmoid, through the vomer, and through the interface of the pterygoid plates to the base of the sphenoid. This type of fracture predisposes the patient to CSF rhinorrhea more commonly than the other types.



Ocular injuries

Superior orbital fissure syndrome

Severe force to the lateral wall of the orbit resulting in compression of neurovascular structures. Results in:

- Complete opthalmoplegia and ptosis (Cranial nerves 3, 4, 6 and nerve to levator palpebrae superioris)
- Relative afferent pupillary defect
- Dilatation of the pupil and loss of accommodation and corneal reflexes
- Altered sensation from forehead to vertex (frontal branch of trigeminal nerve)

> Orbital apex syndrome
> This is an extension of superior orbital fissure syndrome and includes compression of the optic nerve passing through the optic foramen. It is indicated by features of superior orbital fissure syndrome and ipsilateral afferent pupillary defect.

Orbital blow out fracture

Typically occurs when an object of slightly larger diameter than the orbital rim strikes the incompressible eyeball. The bone fragment is displaced downwards into the antral cavity, remaining attached to the orbital periosteum. Periorbital fat may be herniated through the defect, interfering with the inferior rectus and inferior oblique muscles which are contained within the same fascial sheath. This prevents upward movement and outward rotation of the eye and the patient experiences diplopia on upward gaze. The initial bruising and swelling may make assessment difficult and patients should usually be reviewed 5 days later. Residual defects may require orbital floor reconstruction.

Nasal Fractures

- Common injury
- Ensure new and not old deformity
- Control epistaxis
- CSF rhinorrhoea implies that the cribriform plate has been breached and antibiotics will be required.
- Usually best to allow bruising and swelling to settle and then review patient clinically. Major persistent deformity requires fracture manipulation, best performed within 10 days of injury.

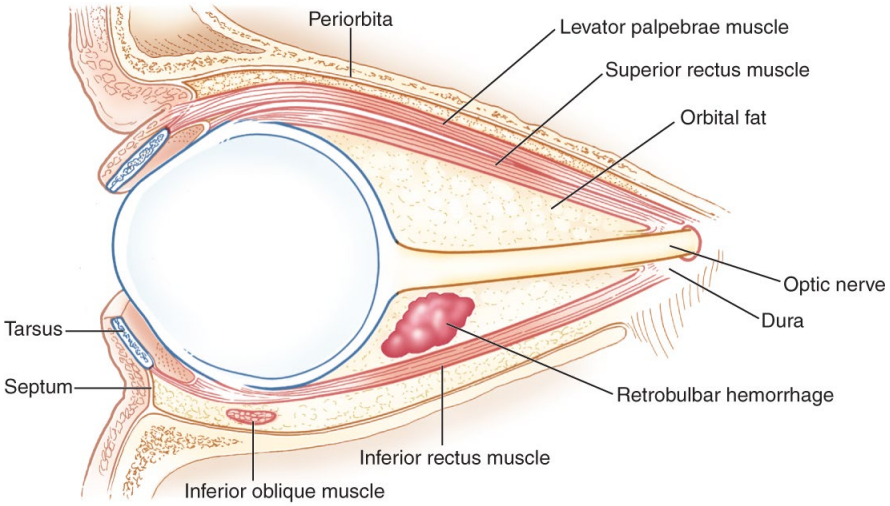
Retrobulbar haemorrhage

Rare but important ocular emergency.

Presents with:

- Pain (usually sharp and within the globe)
- Proptosis
- Pupil reactions are lost
- Paralysis (eye movements lost)
- Visual acuity is lost (colour vision is lost first)

May be the result of Le Fort type facial fractures.



Management:

- Mannitol 1g/Kg as 20% infusion, Osmotic diuretic, Contra-indicated in congestive heart failure and pulmonary oedema
- Acetazolamide 500mg IV, (Monitor FBC/U+E) Reduces aqueous pressure by inhibition of carbonic anhydrase (used in glaucoma)
- Dexamethasone 8mg orally or intravenously
- In a traumatic setting an urgent cantholysis may be needed prior to definitive surgery.

Oculogyric Crisis

An oculogyric crisis is a dystonic reaction to certain drugs or medical conditions

Features

- Restlessness, agitation
- Involuntary upward deviation of the eyes

Causes

- Phenothiazines
- Haloperidol
- Metoclopramide
- Postencephalitic Parkinson's disease

Management

- Procyclidine

Opioid Misuse

Opioids are substances which bind to opioid receptors. This includes both naturally occurring opiates such as morphine and synthetic opioids such as buprenorphine and methadone.

Features of opioid misuse

- Rhinorrhoea
- Needle track marks
- Pinpoint pupils
- Drowsiness

Complications of intravenous opioid misuse

- Viral infection secondary to sharing needles: HIV, hepatitis B & C
- Bacterial infection secondary to injection: infective endocarditis, septic arthritis, septicaemia, necrotising fasciitis, groin abscess
- Pseudoaneurysm
- Venous thromboembolism
- Osteomyelitis
- Overdose may lead to respiratory depression and death

Emergency management of opioid overdose

- IV or IM naloxone: has a rapid onset and relatively short duration of action

Sickle Cell Anaemia

- Autosomal recessive
- Single base mutation
- Deoxygenated cells become sickle in shape
- Causes: short red cell survival, obstruction of microvessels and infarction
- Sickling is precipitated by: dehydration, infection, hypoxia
- Manifest at 6 months of age
- Africans, Middle East, Indian
- Diagnosis: Hb electrophoresis

A combination of a high reticulocyte count and severe anaemia indicates sickle cell anaemia, however another differential can be of a transient aplastic crisis due to parvovirus. This is less likely as this causes a reticulocytopenia rather than a reticulocytosis.

Sickle crises

- Bone pain
- Pleuritic chest pain: acute sickle chest syndrome commonest cause of death
- CVA, seizures
- Papillary necrosis
- Splenic infarcts
- Priapism
- Hepatic pain

Hb does not fall during a crisis, unless there is

- Aplasia: *Parvovirus*
- Acute sequestration
- Haemolysis

Parvovirus B19 infects erythroid progenitor cells in the bone marrow and causes temporary cessation of red blood cell production, patients who have underlying hematologic abnormalities are at risk of cessation of red blood cell production if they become infected. This can result in a transient aplastic crisis. Thus, patients with sickle cell anaemia are at risk. Typically, these patients have a viral prodrome followed by anaemia, often with haemoglobin concentrations falling below 5.0 g/dL and reticulocytosis.

Long-term complications

- Infections: *Streptococcus* pnemoniae
- Chronic leg ulcers
- Gallstones: haemolysis
- Aseptic necrosis of bone
- Chronic renal disease
- Retinal detachment, proliferative retinopathy

Surgical complications

- Bowel ischaemia
- Cholecystitis
- Avascular necrosis

Management

- Supportive
- Hydroxyurea
- Repeated transfusions pre operatively
- Exchange transfusion in emergencies

Sickle cell trait

- Heterozygous state
- Asymptomatic
- Symptoms associated with extreme situations ie anaesthesia complications
- Protective against *Plasmodium falciparum*