Emergency Care Seminar

June 2010

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www.developinganaesthesia.org

Welcome to www.developinganaesthesia.org. This web site has been created to promote the advancement of anaesthetic practice and to empower anaesthetists in countries with limited resources. The site also hopes to foster the growth of an online community of anaesthetists throughout the world.

A web-based resource has significant advantages. The information provided can remain current and be tailored to the requirements of the community. Hard copy texts may be expensive, difficult to access and inappropriate to the delivering of anaesthesia outside of tertiary institutions. The majority of journals have similar limitations.

DevelopingAnaesthesia.org is a free, up to date resource, specifically designed to address these problems.

The authors envisage the web site will have five principle functions, though the dynamic nature of web publishing will allow the evolution of the site as directed by the anaesthesia community.

- **1. Continuing education**
  DevelopingAnaesthesia.org will provide an anaesthetic educational resource for anaesthetists. The site contains a textbook, articles, case studies and links. with time the site will contain power point and video presentations.

- **2. Anaesthetic training**
  DevelopingAnaesthesia.org will provide an anaesthetic educational resource for anaesthetic trainees. The site will contain lecture notes for physiology, pharmacology, equipment, monitoring and statistics.

- **3. Teach the teacher**
  DevelopingAnaesthesia.org will provide a resource to aid anaesthetists in educational methods.

- **4. Peer-reviewed publication**
  DevelopingAnaesthesia.org will provide a venue for peer-reviewed publication online at no cost to authors or readers. All submitted material (case studies, articles, audits etc) is welcomed and encouraged.

- **5. Discussion forums**
  DevelopingAnaesthesia.org has an open forum for discussion, exchange of ideas/experience and seeking advice. A panel of anaesthetists with experience in delivering anaesthesia and teaching in developing countries will moderate the forum but colleges in similar countries may provide the most relevant advice.

Success and the growth of www.developinganaesthesia.org will depend on feedback from the anaesthetic community it serves. Please have a look at the site and register as a user, there is no cost. Registration allows you to participate in forum discussions, submit your own articles and comments and in doing so help foster community growth.

CHEST PAIN

Chest pain is one of the commonest presenting complaints to the emergency department. It is important to exclude life-threatening causes and conditions that will require urgent treatment. The diagnosis can frequently be made on history and clinical examination, but there are some important investigations that will need to be done early if there is any doubt about the diagnosis. Mortality with acute myocardial infarction is 40% within the first 4 weeks, however, half of these deaths will occur within 2 hours of onset of symptoms, (1) thus the importance of a focused history and examination to make the diagnosis as soon as possible.

The main differential diagnoses are acute coronary syndrome (ACS) including unstable angina and myocardial infarction, musculoskeletal pain, anxiety and gastro-oesophageal pain. Other causes include pericarditis and pneumothorax, pulmonary embolus, pleurisy, pneumonia, nerve root compression and herpes zoster, aortic dissection, biliary colic and pancreatitis. Often the cause of the pain can be determined from a history of the site of the pain, its quality and duration, associated symptoms and history of risk factors for disease.

History:

The taking of the patient's history occurs concurrently with the administration of Basic Life Support (Airway – confirm its patency, Breathing – Provide oxygen by face mask & check oxygen saturation, Circulation – take the pulse, blood pressure, insert an intravenous cannula and take blood for haematology, electrolytes, urea, creatinine, cardiac enzymes and liver enzymes).

Take a focused history and perform your examination keeping the diagnosis of Acute Coronary Syndrome (ACS) in mind:
"Tell me about the pain?"
Site / Radiation
Onset & Duration of pain
Is the pain related to exertion?
Is there evidence of crescendo angina?
Are their any relieving factors, such as response to glyceryl trinitrate (GTN)
Determine the nature of the pain (colicky, burning, tightness / heaviness, related to movement / breathing).
Associated symptoms may indicate ACS. They include syncope, dyspnoea, diaphoresis, nausea and vomiting and palpitations.

Past Medical History:
The patient may be known to have existing heart disease. The risk Factors for cardiac disease should be sought. They include: advanced age, male gender, hypertension, diabetes, hypercholesterolaemia, smoking, obesity, and family history of ischaemic heart disease or vascular disease.

Medications:
The patient may already be taking nitrodilators such as GTN and isosorbide mononitrate or antiplatelet agents. Some medications may precipitate ischaemic chest pain such as cocaine.

Examination:

The cardiovascular examination begins from the moment you meet patient. It may be obvious that the patient has ischaemic chest pain. S/he may be distressed, diaphoretic, have increased respiratory effort or have an ashen or grey colour to the skin. Check for clinical signs of anaemia. Feel the pulse and assess the rate, rhythm and quality. Perform a blood pressure and count the respiratory rate.

Auscultation of the Heart:

Check the heart rate and rhythm, and listen for murmurs:

Sinus rhythm contributes up to 30% of cardiac preload. If the patient develops atrial fibrillation, there will be a significant decrease in preload. AF is found in 10% of patients aged over 80 years.

Supra-ventricular tachycardia (SVT) or other tachyarrhythmias will result in a decreased diastolic filing time (relative to systolic time). As the heart rate increases, there is less time for myocardial perfusion and ischaemia may develop or become evident in patients with coronary artery disease.

Murmurs and extra sounds:

The 3rd heart sound may be present. It may sound like a mid-diastolic, 'gallop' rhythm and it occurs due to the tautening of mitral or tricuspid papillary muscles at the end of diastolic filling. It is typically heard when there is reduced left ventricular compliance.

The 4th heart sound is a late-diastolic sound. It has a higher pitch and may be appreciated as a 'gallop' rhythm. It is always pathological and is typically due to a high-pressure atrial wave being reflected back from a poorly compliant left ventricle. It is often present in myocardial ischaemia or infarction.

A pan-systolic murmur (of mitral regurgitation) will result from a ruptured papillary muscle after myocardial infarction. It can be a cause of acute cardiac failure and death.

A loud 2nd pulmonic heart sound is suggestive of raised pulmonary pressures and occurs after a pulmonary embolus.

An infarct can lead to a pericardial rub from pericarditis secondary to necrosis of cardiac tissue under the pericardium.

There may be an ejection systolic murmur of aortic stenosis or hypertrophic cardiomyopathy. These patients may have left ventricular hypertrophy and be more susceptible to subendocardial ischaemia.

Auscultation of the chest:

Check air entry and, listen for any abnormal sounds such as expiratory wheeze, inspiratory stridor bronchial breath sounds, and crepitations (which suggest left ventricular failure).

Palpation:

Check the location of the cardiac apex beat. It may be displaced with left ventricular dilatation or left ventricular hypertrophy.

Pulses:

Check the strength, rate and nature (thready, bounding, irregularly irregular), of the peripheral pulses. There may be a disparity in the pulses in different limbs. The femoral and pedal pulses may be absent in aortic dissection or thromboembolic disease and subclavian artery disease will cause a disparity in pulses between radial pulses.

Examine the chest wall. Check for localisation or reproduction of pain if it could be traumatic or musculoskeletal in nature.

An abdominal examination is essential. Upper gastrointestinal pathology (such as peptic ulcer disease, biliary colic) often presents as chest pain.

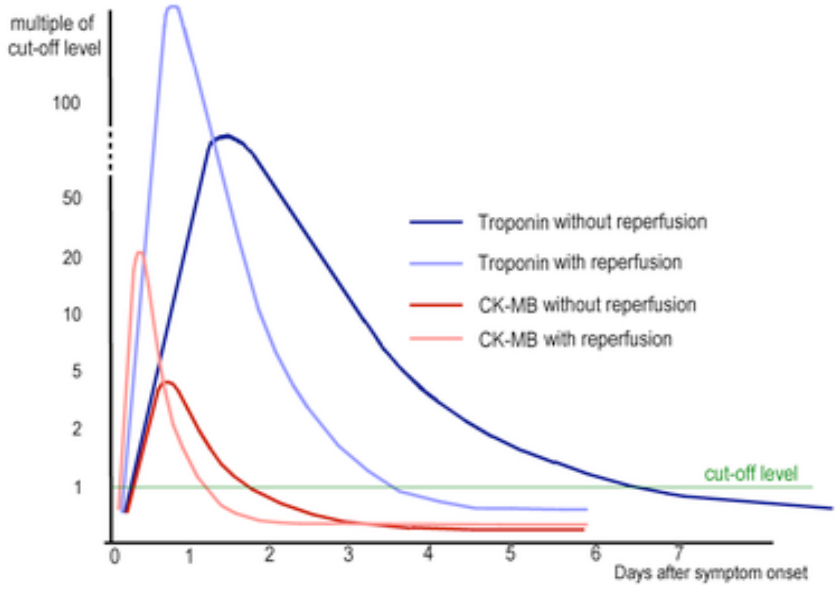
Investigations:

12 lead ECG:

The American Heart Association (AHA) recommends that an ECG be performed within 10 minutes of arrival. If it is initially normal but there is a high suspicion of acute coronary syndrome, the patient should have an ECG repeated every 10 minutes for the first 30 minutes. In a full thickness infarct there may be hyperacute T waves, ST elevation, or a new left bundle branch block may occur within hours of the infarct. In subendocardial infarction (non-Q wave infarction), there are less specific ECG changes, such as T wave inversion and ST depression. The ECG may be normal initially in 20% of patients undergoing a myocardial infarct.

Blood tests:

Blood is taken for Cardiac Enzymes as soon as intravenous access is obtained. The AHA recommends this in all patients with ACS symptoms or signs. A troponin level is preferred but creatinine kinase is appropriate if troponin cannot be measured. Blood is taken within 6 hours of the onset of symptoms, and should be repeated at 8 to 12 hours after the onset of symptoms. Troponin T and Troponin I are cardiac specific troponins. They each remain elevated after a myocardial infarction for 7-10 days (for TnI) and 10-14 days (for TnT). CK (creatinine phosphokinase) rises within 4-8 hours of an infarct and returns to normal by 48 – 72 hours. Due to the lack of specificity of CK for myocardial muscle damage, CKMB is preferred, and will peak at 20 hours after onset of pain.



Kinetics of cardiac markers in myocardial infarction with or without reperfusion treatment. (Graph made from ACC/AHA Practice Guidelines 2005 p 32)

Chest X-ray:
A chest x-ray is performed to check for other causes of chest pain and to determine if there is any pulmonary venous congestion secondary to cardiac failure.

Differential Diagnosis:

The key is to detect potentially life-threatening conditions of *Acute Coronary Syndrome, Aortic Dissection or Pulmonary embolism* early on. A useful approach is to ascertain whether pain is new, acute and ongoing (more likely to be seen with the life-threatening diagnoses), recurrent, episodic pain, or persistent pain over many days.

New acute chest pain:
Acute Coronary Syndrome:
The ECG is the best diagnostic test. If significant Q waves or new ST segment elevation is present in at least 2 leads, the probability of an acute myocardial infarct is 75%. If ST segment depression of at least 1mm, or T wave inversion is present, the probability of an acute myocardial infarct is 20%. If there are no ECG changes but there is a high level of suspicion of ACS, further observation is required with repeated ECGs and cardiac enzymes (at serial intervals of 6 or 12 hours from the initial ECG).

Aortic dissection:
Aortic dissection is suspected if there is a rapid onset of symptoms, asymmetric pulses, a history of hypertension or Marfan's syndrome. A chest x-ray that shows a dilated aortic root supports this diagnosis.

Acute Pulmonary Embolism:
Pulmonary embolism is suspected if there are associated respiratory symptoms such as dyspnoea and haemoptysis. The chest pain is pleuritic (sharp in nature, related to respiration) and is caused by the inflammatory reaction of infarcted lung against the

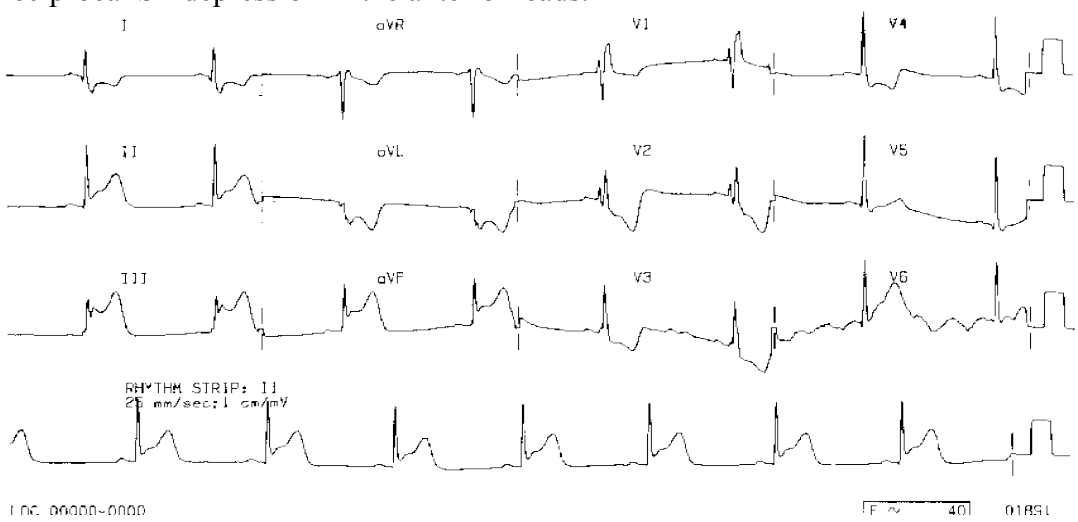
pleura. A deep venous thrombosis or risk factors for thrombosis may support the diagnosis. There may also be characteristic ECG changes of right heart strain, such as the 'S1, Q3, T3' pattern of prominent S waves in lead I, Q waves in lead III and T wave inversion in lead III, as well as a Right bundle branch block.

Other differential diagnoses to be considered:

- Cardiac – Myocarditis, Pericarditis or pericardial effusion/tamponade, Arrhythmia).
- Respiratory (Pneumonia, Pleural effusions/Malignancy, Pulmonary Embolus)
- Upper GI (Pancreatitis, Peptic Ulcer, Gastritis, Reflux, Cholelithiasis)
- Musculoskeletal

ECG example of acute inferior myocardial ischaemia:

Example of acute inferior (II, III and aVF) ST-elevation myocardial infarction with reciprocal ST depression in the anterior leads.



Immediate Management of AMI / Suspicious Chest pain:

After basic life support assessment and management has commenced, more targeted treatment of the acute coronary syndrome should be started without delay. Provide supplemental oxygen to buffer against ventilation-perfusion mismatch that may arise from left ventricular failure.

Nitrates such as glyceryl trinitrate (GTN) act as venodilators to decrease preload to the heart. The main side effect is hypotension, and thus should be used with caution in cardiac shock unless inotropic support is available. The American Heart Association recommends an infusion of GTN if more than 3 doses of GTN spray/tablets have been used. This should not preclude giving angiotensin converting enzyme inhibitors or Beta-blockers to improve mortality. To prepare a GTN infusion, staff may add 200mg of GTN to a 500ml bag of 5% dextrose (to produce an final concentration of 400 mcg/ml). Infusions typically start at 1ml/hr, increasing up to 20ml/hr.

Antiplatelet therapy is effective across the spectrum of acute coronary syndromes, and acts to prevent platelet activation and thus aggregation over the surface of the ruptured atherosclerotic plaque. This platelet plug is the chief cause of the coronary artery obstruction/occlusion that follows the disruption of the atherosclerotic, lipid filled plaque in the wall of the artery. Aspirin causes a rapid inhibition of cyclooxygenase in platelets that is irreversible, which prevents the production of thromboxane A2 by platelets. Thromboxane A2 is usually responsible for platelet aggregation and vasoconstriction. Aspirin has been shown to decrease the incidence of myocardial infarct and death in those with angina by approximately 50%. (2)

Other pharmacological agents indicated for acute coronary syndrome include morphine, which has the benefit of pain relief and lowering systemic vascular resistance (and therefore the work of the heart to eject blood which reduces myocardial oxygen demand). Heparinisation with either unfractionated heparin or low-molecular weight heparin (such as enoxaparin) should also be commenced in the acute setting.

In the first 24 hours following presentation with acute coronary syndrome, an oral Beta-blocker should be commenced, however if the patient cannot have a beta-blocker, (such as with asthmatic patients), a non-dihydropyridine Calcium Channel Antagonist should be commenced (for example ditiazem or verapamil) as long as there is no evidence of left ventricular dysfunction. (3) Similarly, an oral Angiotensin Converting Enzyme (ACE) inhibitor (or Angiotension II receptor antagonist, if ACE-inhibitor is not tolerated) should be commenced within 24 hours if pulmonary congestion or left ventricular failure is present. An adequate blood pressure must be established prior to commencing this medication.

Definitive Management plans:

Early invasive treatment options for acute coronary syndrome include thrombolysis, coronary angiography with angioplasty or percutaneous stenting of the lesion, or cardio-thoracic surgery for coronary bypass grafting. Coronary angiography and revascularisation is indicated when there is acute coronary syndrome with haemodynamic or electrical instability, or other risk factors for mortality.

If the cardiac enzymes and ECG are normal, but there is a high suspicion of acute coronary syndrome the patient should remain in hospital with cardiac monitoring and the 12 lead ECG is repeated at a specified time intervals (ideally at least 12 hours from onset of pain). If repeat ECG and cardiac enzymes are normal, the patient should have a chemical or exercise stress test performed within 72 hours (can be done as an outpatient)

Antiplatelet therapy:

Aspirin is life saving. It has been shown to improve mortality with acute coronary syndrome and decrease the risk of infarction when coronary artery disease is present. It has a good risk to benefit profile, is inexpensive, easy to administer and effective shortly after it is given. In Australia, ambulance officers routinely administer an oral dose of 300mg of aspirin to at-risk adults that have chest pain prior to reaching hospital.

Aspirin irreversibly inhibits cyclooxygenase within the platelet, to prevent the production of thromboxane-A2, a chemical responsible for vasoconstriction and increased platelet aggregation. It reduces the risk of unstable angina progressing to myocardial infarction and reduces the mortality following an acute infarction. Antiplatelet Trialist's collaboration looked at 287 studies (over 200,000 'high risk' vascular patients). This showed that antiplatelet therapy reduced the combined outcome of any serious vascular event (non-fatal stroke, myocardial infarction or vascular death) by 25%. Antiplatelet therapy reduced the incidence of a non-fatal myocardial infarction by one third and non-fatal stroke by a quarter. The dose required to achieve this effect was 150mg – 300mg of aspirin (loading), followed by 75mg -150mg daily in the long-term.

Clopidogrel is an ADP receptor antagonist, which irreversibly binds to the receptor on the platelet's surface and prevents glycoprotein IIb/IIIa receptor transforming into its active form. (4) It reduces the risk of stoke and myocardial infarction in those with vascular disease when compared with aspirin, however its full effects are not seen until 3 to 7 days after commencing treatment. When compared with aspirin, clopidogrel carries the risk of a more severe rash and greater risk of perioperative bleeding if ceased less than 10 days prior to surgery. However, clopidogrel has a lower risk of causing gastrointestinal bleeding than aspirin. A Cochrane Database Systematic Review in 2007 found Clopidogrel in combination with aspirin is associated with a reduction in the risk of cardiovascular events compared with aspirin alone in patients with acute non-ST elevated coronary syndrome. (5) The CURE trial however illustrated the significant increase in relative risk of a bleeding complication in those treated with clopidogrel. (6) Its use in acute coronary syndrome is thus limited to those who cannot tolerate aspirin, or those requiring invasive treatment such as coronary stenting.

Thrombolysis:

This treatment reduces the relative risk of in-hospital death by up to 50% if given within the first hour of the onset of symptoms. Thrombolytics act to limit the size of the infarct and left ventricular dysfunction. They reduce the incidence of septal rupture, cardiogenic shock and malignant ventricular arrhythmias. The benefits are time-critical – those that benefit the most receive thrombolysis within the first 3 hours.

These drugs are plasminogen activators – they convert the endogenous plasminogen enzyme to the fibrinolytic enzyme plasmin (fibrinolysin). They are particularly useful in acute coronary syndrome as they are more capable of dissolving newly formed clot (platelet rich and formed by weaker fibrinogen bonds) than older clots, which are more tightly bound with fibrin. (7)

Thrombolysis is not without significant risk. Intracranial haemorrhage occurs in 0.5 – 3.3% of patients undergoing thrombolytic therapy. (2)

Indications for Thrombolysis:

The AHA guidelines are only applicable to facilities with access to acute coronary angiography and stenting, and where coronary artery bypass graft surgery is available. The greatest benefit from thrombolysis is gained if it is given within 12 hrs of the onset of symptoms with:

1. ST elevation > 2mm in 2 or more CHEST leads OR
2. ST Elevation > 1mm in 2 or more limb leads OR
3. Posterior infarction (dominant R waves and ST depression in V1-3)
4. New onset Left Bundle Branch Block

Thrombolysis may be indicated 12-24 hours after the onset of pain, where there are ongoing symptoms of chest pain and/or ST elevation.

The TIMI (Thrombolysis In Myocardial Infarction) study group developed the TIMI score - a simple way of stratifying the risk of having a cardiac event in the first 14 days following non-ST elevated acute coronary syndrome or unstable angina (8). Each risk factor was equivalent to 1 point, and the cumulative score indicated those at higher risk of complications and thus an indication for more aggressive management such as thrombolysis.

The risk factors consisted of:

- Age greater than or equal to 65 years
- More than three coronary risk factors
- Prior angiographic coronary obstruction
- ST-segment deviation
- More than two angina events within 24 hours
- Use of aspirin within 7 days
- Elevated levels of cardiac biomarkers

TIMI Score:	14-day event rate
0 or 1	4.7%
2	8.3%
3	13.2%
4	19.9%
5	26.2%
6 or 7	40.9%

Contraindications to Thrombolysis:

Due to the risk of life-threatening haemorrhagic complications, patients should not receive thrombolysis if they have any of the following:

- Active gastrointestinal bleeding
- Surgery or trauma, including CPR, in previous 2 weeks
- Intracerebral or subarachnoid haemorrhage at any time in the past, or thrombotic intracerebral event in the previous 6 months.
- Pregnancy,

- Uncontrolled hypertension (Systolic > 200mmHg, diastolic > 110mmHg),
- Proliferative diabetic retinopathy
- Aortic Dissection

Other relative contraindications to thrombolysis include: menstruation, warfarin use, previous gastrointestinal bleeding, arterial puncture or central line insertion.

Thrombolytics:

Streptokinase
Mechanism of action: binds to plasminogen converting it to a plasminogen-activator complex that acts on other plasminogen molecules to generate plasmin. It is not fibrin specific, and so can produce systemic thromolysis.
Dose = 1.5 million units in 100mls 0.9% Saline intravenously over 1 hour.
Side Effects include: Nausea, vomiting, haemorrhage, stroke (1%) and allergic reactions (hypotension to anaphylaxis). Allergic reactions, including anaphylaxis, may occur because streptokinase is produced from Beta-haemolytic streptococci, and most patients will have pre-existing anti-streptococcal antibodies. Exposure to streptokinase may also stimulate antibody production. These antibodies may make repeated treatment difficult or impossible for months to years after an initial course, thus don't repeat a dose of streptokinase unless it is less than 4 days since the first administration.
T1/2 elimination = 23 minutes (stop 3 hours before surgery).
Reperfusion success rate = 50-70% (most inexpensive thrombolytic).

Alteplase (t-PA, recombinant Tissue Plasminogen Activator)
This is indicated if the patient has previously received streptokinase more than 4 days ago, or has had a previous reaction to streptokinase. It is a fibrin-specific thrombolytic drug synthesized by endothelial cells.
Dose = 100mg (1.25mg/kg infused over 3 hours).
T1/2 elimination = <5 mins (stop 1 hour before surgery)
Reperfusion success rate = 60-80%. (It is more expensive than streptokinase).

Tenecteplase:
A third generation variation of t-PA. It has improved fibrin specificity and a longer half-life, which allows for it to be given as a single bolus dose. The mortality rate is the same as with t-PA, however in patients who presented late after a myocardial infarction, there were fewer intracranial haemorrhages in those receiving tenecteplase than those treated with t-PA.

Monitoring after thrombolysis:
Thrombolysed patients should be transferred to an intensive care unit as soon as possible after initiation of treatment. Specific monitoring should include frequent measurement of blood pressure (every 15 minutes during the infusion and every 30-60 minutes after treatment), and a continuous ECG (a rhythm strip plus a 12 lead ECG 4 hours after starting the treatment). The high risk of haemorrhagic complications necessitates close observation for changes in neurological status and for bleeding, with care to avoid unnecessary trauma, invasive procedures and venous or arterial punctures.

Reocclusion post-thrombolysis occurs in approximately 20% of patients. Heparin therapy is indicated for those treated with any thrombin-specific thrombolytic agent, (such as t-PA or Tenecteplase) for the first 48 hours is recommended, along with aspirin. Activated partial thromboplastin time should be maintained at twice normal range for this time period.

Anticoagulation / Heparinisation:

All immobilised patients should receive at least prophylactic anti-thrombotic treatment with either unfractionated heparin 5,000 units subcutaneously twice daily, or clexane 40mg subcutaneously daily (or 20mg daily if there is renal impairment). Full anticoagulation may be considered on a short-term basis in selected patients, and if there has been a large ventricular wall infarction (where there is a risk of mural thrombus formation), then long-term anticoagulation with warfarin should be considered.

Early complications of Acute Myocardial Infarction / Acute Coronary Syndrome:

10% of all patients reaching hospital will die during hospitalization. Over half the patients with myocardial infarction will have at least one complication.

Arrhythmias:

These are best avoided by correcting any hypoxia, acidosis, hypokalaemia and hypomagnesemia, aiming for upper normal values for serum potassium (approximately 4.5mmol/L), and magnesium (up to 2.0 mmol/L). Atrial fibrillation is found in 22% of patients with acute coronary syndrome and if there is a rapid rate, it should be treated urgently to slow down the ventricular rate. Slow atrial fibrillation should only be treated if the blood pressure is compromised. There is no indication for prophylactic antiarrhythmics in the acute coronary syndrome. Conduction block at the AV node should be noted, as 40% of patients with 1st degree heart block will go on to develop further atrio-ventricular block. Mobitz type II block carries a higher risk of developing into complete heart block and should be paced. If trifascicular block or non-adjacent bifascicular block is present, it should also be paced.

Pulmonary Oedema / Left ventricular failure:

Pulmonary oedema can occur at any time in the course of an acute myocardial event, and close monitoring of fluid intake/ intravenous fluid therapy as well as urine output and renal function will help minimise its occurrence. Management consists of positioning of the patient upright in bed, supplemental oxygen, off-loading excess fluid with diuretics such as frusemide intravenously (40mg or twice the patient's usual oral daily dose), reducing afterload and preload with GTN as tolerated. CPAP (continuous postitive pressure ventilation with occlusive face mask) to splint alveoli open and reduce preload is indicated if the patient remains refractory to treatment. Some patients will require intubation and ventilation.

Cardiogenic Shock:

While the incidence of cardiogenic shock is around 7-8% of patients with myocardial infarction, it has a mortality rate up to 80%. Older patients with diabetes and a history of myocardial infarction in the past are particularly at risk. It may be due to reduced

ventricular compliance (diastolic failure) or a reduced stroke volume, leading to cardiac dilatation (systolic failure).

Acute mitral regurgitation resulting from ischaemia or infarction of a papillary muscle is common, presenting with abrupt severe congestive cardiac failure with a pansystolic murmur. Management includes airway management, insertion of a central venous catheter to monitor central pressures and guide fluid resuscitation, and consideration of the use of a dopamine or dobutamine infusion +/- noradrenaline if the patient has peripheral vasodilatation.

Later Complications:

Pericarditis may occur from pericardial inflammation overlying a transmural (full thickness) myocardial infarct. The signs are central chest pain relieved by sitting forwards, associated with saddle-shaped ST elevation in most leads on the 12 lead ECG.

Dressler's syndrome is an autoimmune reaction that occurs at least 1 week after the myocardial infarction. The signs include: recurrent pericarditis, pleural effusions, pericardial friction rub, fever and anaemia. Treatment consists of nonsteroidal anti-inflammatories and admission for monitoring for possible cardiac tamponade.

Cardiac tamponade will be characterised by low cardiac output, tachycardia, pulsus paradoxus, Kussmaul's sign (increase in central venous pressure with inspiration, compared to the normal decrease), raised jugular venous pressure and muffled heart sounds.

Left Ventricular aneurysm occurs in patients with large anterior infarctions. The aneurysm may be suspected if there is persistent ST elevation on ECG, left ventricular failure, angina or recurrent ventricular tachycardia. An additional concern with this complication is systemic embolisation (especially to the brain) should a mural clot dislodge from the aneurysm. If a right ventricular thrombus dislodges, then signs and symptoms of pulmonary embolus may become apparent.

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SHORTNESS OF BREATH

Shortness of breath is a symptom of most cardiac and respiratory presentations to emergency departments, including most life-threatening emergencies, however a focused history and examination can rapidly identify such time-critical conditions.

Chronic obstructive pulmonary disease (COPD) accounts for up to 1 in 8 emergency presentations in the United Kingdom. The disease is often not diagnosed, but it is estimated that approximately 9% of adults over the age of 45 meet criteria for COPD in the UK. In 1999, more than 5% of all deaths in the UK were due to COPD, however the true mortality rate is difficult to assess, as most people will die from complications of COPD rather than the disease itself. (1)

Presentations to hospitals of patients with asthma have increased significantly, particularly in the paediatric population. In the United States between 1980 and 1994, those with asthma increased by 75%, and alarmingly in children less than 5 years old this increase was more than 160%. (2)

History:

The shortness of breath in itself presents a challenge in history taking. Directed questions can help obtain the necessary information with minimal distress to the patient.

COPD and Asthma are chronic diseases with frequent exacerbations and presentations to their local emergency department, making diagnosis rapid. Key questions should address the time course of the symptom – its onset and progression – any factors that aggravate or provide relief and if there are any associated symptoms such as wheeze, chest pain, a productive cough, haemoptysis or fever.

Smoking is clearly associated with the development of COPD, and an estimation of how much the patient has smoked in his lifetime (in pack-years) is useful. The accuracy of this information may be variable. Other risk factors to be aware of include the patient's industrial exposure to aerosol pollutants, immobilisation in bed (and thus deep venous thrombosis risk), recent upper respiratory tract infection (URTI)/ lower respiratory tract infection (LRTI) and a past history of atopy (eczema, asthma) in younger patients.

A past history of COPD or asthma may distract the clinician's attention away from illnesses presenting concurrently. Don't forget to ask about recent trauma, whether new drugs have been commenced (causing allergic reactions), and take note of symptoms or a history of ischaemic heart disease and malignancy (which may present initially with shortness of breath secondary to effusions or obstruction of a bronchus).

Examination:

General appearance:

Once basic airway, breathing and circulation management has been addressed, a focused respiratory and cardiac examination should be undertaken. The patient's general appearance should be noted, taking into account overall respiratory effort -

rate, position of the patient, accessory muscle use, and the ability to talk in sentences or words or not at all. The respiratory rate is a useful marker of severity of the disease but beware of the exhausted patient who tires and develops respiratory failure. Wheeze and stridor can often be heard from the end of the bed and the timing of the stridor helps identify the site of obstruction (inspiratory if extrathoracic, and expiratory if intrathoracic). The classical 'seal-like' barking cough in young children with croup is a helpful sign that differentiates from the toxic, silent child with epiglottitis.

Adequate early exposure of the chest and abdomen in a warm environment should be done as soon as you begin your history taking. Infants should be undressed to reveal any in-drawing around the sternum, clavicles and in between the ribs. This will also reveal a distended abdomen that could be impairing movement of the diaphragm. Free movement of the diaphragm is more important for lung expansion in infants compared with adults. The ability of the baby to feed is also a useful marker of the degree of respiratory distress.

Likewise, undressing the adult will reveal previous surgical scars of cardiac or thoracic surgery, as well as obvious inequality of chest wall movement, such as with a pneumothorax or obstructed bronchus. Peripheral stigmata of disease such as finger clubbing, facial droop with Horner's disease suggesting upper lobe pathology, and kyphoscoliosis are rare but helpful indicators of likely pulmonary aetiology of the presentation.

Palpation:

Palpation of the trachea in the sternal notch for deviation and springing of the rib cage in patients with trauma will help identify a pneumothorax early. Feeling for the lateral displacement of the apex beat may reveal an enlarged heart and assessment of the height of distended jugular veins will alert the clinician to look closely for a cardiac cause of the presentation. Assessing the radial pulse gives much information within a few seconds – not only heart rate and rhythm, but also the nature and strength of the pulse – from the bounding pulse in patients with COPD and high carbon dioxide levels, to the weak thready pulse of the patient with cardiogenic shock.

Percussion and Auscultation:

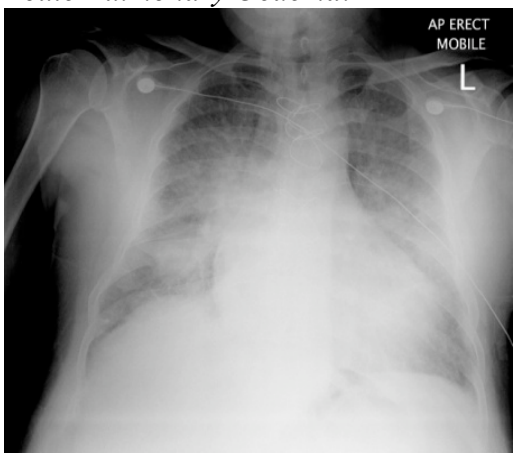
Before auscultating the lungs, take a few moments to percuss the chest, noting the hyper-resonance of a pneumothorax, and the dullness of a consolidated lobe or collapsed lung bases. Ausculation will provide much information regarding air entry, wheeze, bronchial breath sounds of a consolidated lung, inspiratory crepitations at the bases in patients with pulmonary oedema, as well as heart sounds, murmurs and rhythm.

Investigation:

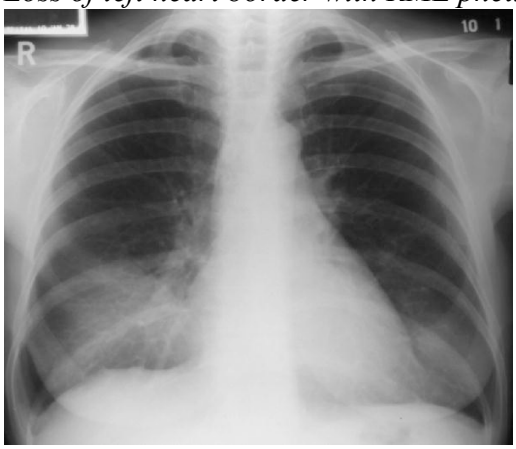
The Chest X-ray (CXR):

The CXR is important in COPD, suspected foreign body inhalation, pneumonia or pneumothorax, but of limited benefit in asthma. It is not indicated in mild to moderate asthma unless a pneumothorax is suspected.

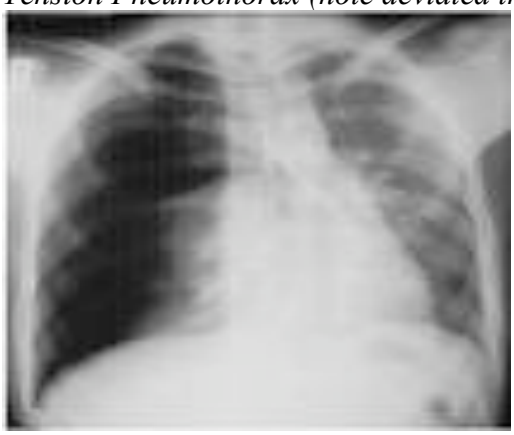
Typical CXR findings:
Acute Pulmonary Oedema:



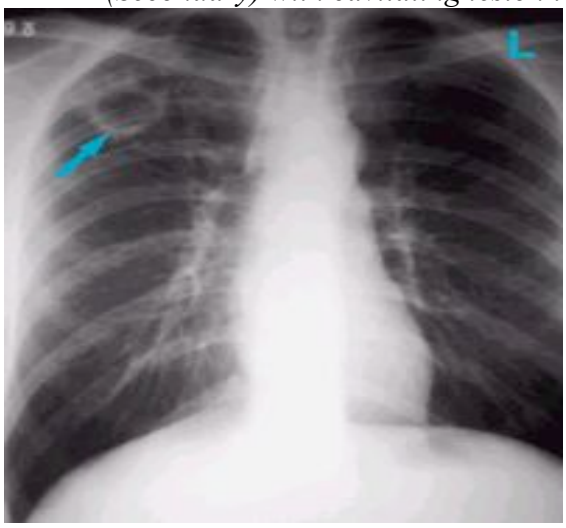
Loss of left heart border with RML pneumonia:



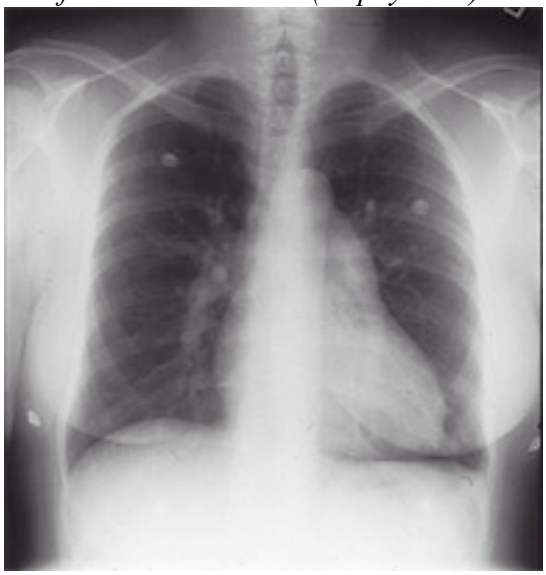
Tension Pneumothorax (note deviated trachea and absence of lung markings)



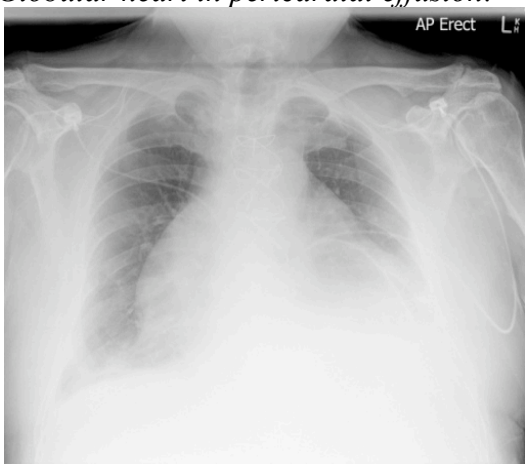
TB (Secondary) with cavitating lesion in right upper lobe:



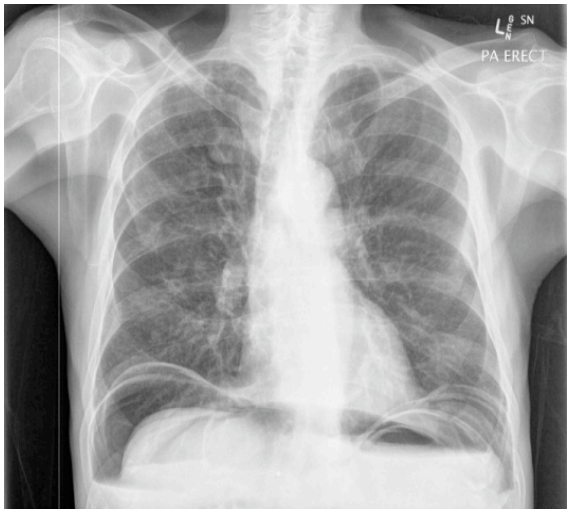
Hyperinflation with COPD (emphysema):



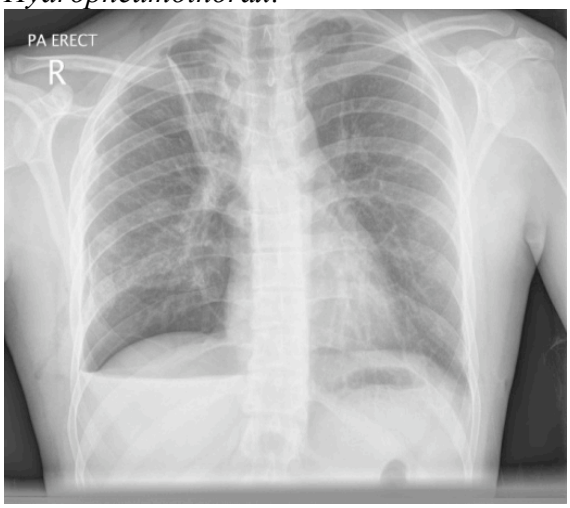
Globular heart in pericardial effusion:



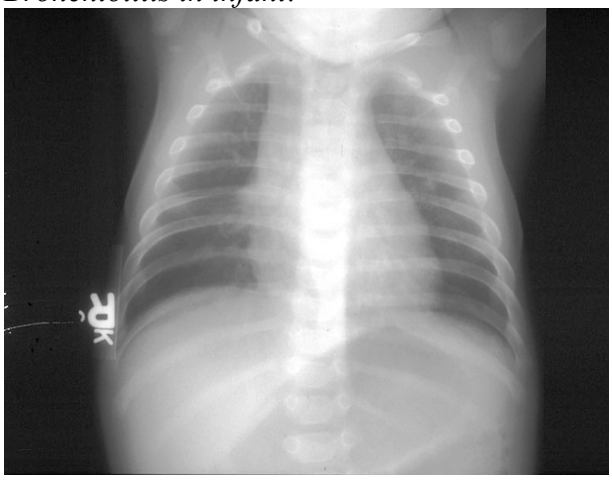
Air under diaphragm (viscous perforation):



Hydropneumothorax:



Bronchiolitis in infant:



Arterial Blood Gas (ABG):

ABG is extremely useful in assessing patients with COPD exacerbation, but of limited use in asthma, where most will be hypocapnic from the reflex hyperventilation. Arterial gases are most useful when previous blood gases are available for comparison, particularly when the patient was not acutely unwell, to establish a

baseline for their expected oxygen and carbon dioxide partial pressures. The added benefit of obtaining a blood gas is that most modern gas machines can often provide additional information such as haemoglobin, electrolytes, glucose and lactate within a minute.

Peak expiratory flows

Peak flow meters are an inexpensive and rapid assessment tool that are useful for measuring response to treatment in mild to moderate asthma, and judging the severity of the disease, when a patient's baseline measurement is available. They are of limited help in severe asthma, or with young children or patients unable to follow instructions. Peak Expiratory Flow of less than 50% of predicted indicates a severe exacerbation of asthma. The measurement of PEFR is not useful for assessment of patients with exacerbations of COPD.

Blood tests:

A full blood examination will be of use in looking for anaemia. A raised white blood cell count is elevated in an infective process. A more reliable marker of infection is a "left shift" on a blood film towards more immature leucocytes. The measurement of serum electrolytes, urea and creatinine, provide information if renal failure is suspected. This is useful in patients in with signs of fluid overload. Albumin levels provide a marker of general health and hepatic function. Cardiac enzymes should be looked for if there is a suspicion of acute coronary syndrome or pulmonary oedema, and in those with known ischaemic heart disease presenting with respiratory distress. A cardiac event may often present without chest pain, particularly in diabetic patients.

Sputum for microscopy and culturing:

It is ideal to obtain sputum specimens in those with likely infective exacerbation of COPD or pneumonia prior to starting antibiotics to assist in ensuring an appropriate antibiotic for the causative bacteria is being used, however treatment should not be delayed in order to collect an adequate specimen.

Management:

Emergency airway management and ventilator support will be addressed in other lectures.

Assist ventilation with a self-inflating bag and face mask, using jaw thrust and chin lift, as well as devices such as oropharyngeal or nasopharyngeal airways to ensure adequate chest wall excursion and oxygen saturation. Obtain assistance to set up for a rapid sequence intubation to secure the airway.

CLINICAL SCENARIOS:

Trauma

A 30-year-old male presents after being hit by a car. He has increasing shortness of breath since leaving the accident site. He is reporting pain on the left side of his chest where there was impact with the car. He has no past medical issues.

On examination his observations are:

Oxygen saturation is 92% on 15 litres of oxygen per minute, respiratory rate = 35 (per

minute), pulse = 140 (beats per minute), blood pressure = 90/60 mmHg, ECG =sinus tachycardia.

He has distended neck veins, a trachea which feels deviated to the right side, there is decreased air entry on the left side of his chest, however auscultation and palpation is difficult to assess as the patient is moaning in pain and tachypnoeic.
There is a high suspicion of let sided rib fractures and a pneumothorax.

Immediate management consists of needle decompression of a likely tension penumothorax (needle thoracocentesis), followed by insertion of a chest drain (large bore if there is the likelihood of a haemothorax). The chest tube is then attached to an underwater seal drain, whilst a large bore intravenous catheter is inserted and fluid resuscitation is commenced.

Reassess the airway, breathing, circulation whilst obtaining a chest X-ray to confirm correct placement of the chest drain, the size of the residual pneumothorax, the degree of mediastinal shift, the presence and location of the rib fractures, and other findings, which may indicate additional pathology such as a globular heart, or air under the diaphragm.

Secondary assessment and Trauma Series X-rays should then be obtained (this includes imaging of the cervical spine, lumbar spine, pelvis, and long bones suspected of fractures).

In the setting of blunt chest trauma, the clinician needs to ensure there is no coexisting cardiac injury/pericardial effusion. An ECG is the best investigation for identifying such cardiac injury in the acute setting. Raised jugular veins, hypotension and tachycardia, muffled heart sounds and widespread ST elevation will indicate cardiac tamponade, and is an emergency.

Tension pneumothorax:
The mechanism of development of a tension pneumothorax is a one way flap valve that allows air into pleural space with each inspiration, but not allowing it to escape in expiration. This causes increased intrathoracic pressure, which eventually compromises cardiac output to the point of cardiac arrest. Do NOT wait for a Chest X-ray prior to emergency decompression! If the clinical suspicion is low for a tension pneumothorax, you can obtain a chest X-ray in expiration, looking for the collapsed lung and surrounding area with a lack of lung markings.

The clinical presentation is characterised by increased respiratory effort, tachycardia, hypotension, engorged neck veins (due to mediastinal shift and compression of great veins), trachea deviated AWAY from side of pneumothorax, and reduced breath sounds on the affected side.

The treatment of a tension pneuomothorax is to take a 14G – 16G needle or intravenous cannula and insert it into the second intercostal space in the midline (just below the medial third of clavicle) on the side of the pneumothorax. The decompression provides a few more minutes for the insertion of a chest tube before catastrophic cardiovascular collapse.

Request an urgent Chest Xray at the time of needle decompression, but if there is expected to be any delay, simply insert the drain and obtain a chest X-ray after this to confirm the position and efficacy of the chest drain. An open pneumothorax should be managed by creating a one-way valve to prevent entrainment of air into the pleural cavity during expiration. An easy way of creating this is to cover the puncture site with an occlusive dressing (such as multiple layers of gauze) that is then taped to the skin on three sides. This allows air to leave during inspiration and prevents air from entering the space during expiration. This is a temporising measure until a chest drain can be inserted.

Paediatric Scenario:

A 7 year old child with recent upper respiratory tract infection, presents with worsening shortness of breath in last 2 hours.

Differential Diagnosis:

- Asthma
- Pneumonia
- Trauma/Child Abuse → fractured ribs
- Upper airway obstruction/ oedema
- Allergic reaction (Antibiotics, food allergy)

History & Examination:

Once a brief history is taken, examination of the chest should be undertaken, listening for an expiratory wheeze, and the degree of respiratory distress assessing the ability to talk, the use of accessory muscles, and intercostal and suprasternal indrawing.

The recent viral upper respiratory tract infection predisposes to hyper-responsiveness of bronchial smooth muscle, as well as increased mucosal oedema and secretions. BEWARE: A quiet chest may indicate severe obstruction to airflow or patient exhaustion. Both indicate severe asthma that is likely to require ventilatory support.

History and examination provide nearly all the information required, with peak flow meter readings requiring a compliant, non-exhausted child to be able to take maximal inspiratory effort, which is seldom achieved in acute asthma. A chest X-ray may rule out additional pathology, and show some hyperinflation, but the benefits need to be weighed against the risk of transporting an acutely unwell child to radiology. Oxygen desaturation is a late sign, and if the oxygen saturation is less than 89%, it could indicate an impending respiratory arrest.

Infant:

A 9-month-old child is brought in by his parents who report that the child feels hot, is refusing feeds, and has noisy breathing this evening.

Differential Diagnoses:

- Upper respiratory tract infection or lower respiratory tract infection

- Pneumonia
- Asthma
- Croup
- Epiglottitis
- Meningitis
- Gastroenteritis
- Foreign body aspiration (less likely at 9 months of age).

The history reveals the patient to be a term baby, who has not had vaccinations. His developmental milestones are appropriate for his age, and family members including siblings are reportedly well at home. The infant has not been taking milk or food in the last 12 hours and will not swallow paracetamol syrup. The number of wet nappies has been less in the last 24 hours, and the current nappy is dry since being changed four hours ago. There has been no diarrhoea or vomiting.

On examination, the infant appears unwell, and is drooling. There is clearly extra effort in breathing with indrawing of the chest wall in between ribs and above the sternum. The respiratory rate is 40 breaths per minute. Closer inspection reveals a soft inspiratory stridor and occasional soft, muffled cries. A temperature of 39.4 is recorded. There are no obvious wheezes or crackles on auscultation of the chest. The oxygen saturation is 96% on room air and heart rate is 150.

Acute laryngotracheobronchitis (Croup) is typically viral (mostly parainfluenza) and tends to present in children aged between 6 months and 3 years old. Typically there is a viral infection in the preceding 2-3 days, the child is not-toxic looking, has only a low grade temperature, and oxygen saturation remains normal. The barking cough and harsh inspiratory stridor develop in the middle of the night. Treatment includes management of the airway, with nebulised saline or adrenaline. A corticosteroid such as prednisolone or dexamethasone can be considered.

Epiglottitis presents typically as a septic-looking child with 2-3 days of progressive fever, throat pain and swallowing pain. The classic example is that of the quiet, drooling infant that can't swallow saliva and has a high fever. Haemophilus Influenza B vaccination being introduced in infancy has greatly decreased the incidence of epiglottitis, so now is more common in adults. The danger with epiglottitis is that a patent airway can quickly become obstructed. This is a potential problem if the child becomes distressed or is made to lie flat. Don't try to examine child. If he is stable enough and will tolerate a lateral soft tissue neck X-ray this could be obtained to demonstrate soft tissue swelling of the epiglottis.

Obtaining a skilled anaesthetist is a priority, and little should be attempted apart from providing oxygen until the anaesthetist and surgeon who is able to perform an emergency surgical airway in theatre are present. Any attempt at securing an airway should be performed in theatre so an emergency tracheostomy can be performed if endotracheal intubation is unsuccessful. Medical management with intravenous

antibiotics (such as cefotaxime) and steroids are indicated once intravenous access is established.

Adult smoker:

A 65-year-old male smoker presents with 24-hour history of chest pain and nausea, and difficulty breathing and a productive cough with brown sputum over the last 3 days.

Differential Diagnosis*:*

- COPD/ Asthma
- Pneumonia
- Acute coronary syndrome
- Pulmonary malignancy with obstruction and consolidation
- Pneumothorax
- H1N1 (Swine 'Flu)

The history is one of dull chest pain that is poorly localised but mostly on the left side of the chest. There is no radiation of the pain to the arms or throat. It is present all the time, but is worse when he is coughing or deep breathing. There has been no vomiting but he has been feeling cold and sweaty in the last 2 days.

His shortness of breath has been progressively worsening in the last 3 days, and is present at rest. He has experienced increasing wheezing and his sputum has changed colour to a yellow-brown colour (it is usually white) and has increased. He is normally short of breath walking more than 500 metres without a break. His salbutamol is providing limited relief of symptoms. His shortness of breath has never been investigated. There has been no recent weight loss or change in appetite.

He has a past medical history of hypertension, but has been non-compliant with his medication. He has also had angina in the last 5 years. He has experienced chest pain 1-2 times per week for the last year, and this has increased to daily chest pain during the past 3 days. The duration of the chest pain is typically 1-2 minutes, and always resolves with resting or if he takes a GTN tablet. He ran out of his GTN tablets last month. He has been diagnosed with asthma and takes salbutamol via a metered dose inhaler 1-2 times/day.

There is no history of recent trauma or recent travel outside his town. His wife and family members at home all in good health. There have been no periods of immobilisation recently, and no previous deep venous thromboses, pneumothorax, or surgery.

He has been unemployed for the last 2 years. He was previously employed in the markets selling fresh produce. He has had no exposure to chemicals, never worked in a mine or a factory and has a pet dog but no other animals or birds at home. He has been a smoker of 20 cigarettes a day since he was 14 years old.
His father died aged 65 from a heart attack and his mother died aged 70 in her sleep.

On examination:
Respiratory Rate = 30 breaths per minute, oxygen saturation is 92%. He is unable to finish sentences due to increased respiratory effort. His pulse is110 beats per minute and blood pressure is 140/90 mmHg. His 12 lead ECG shows a sinus tachycardia with occasional ventricular ectopics and T wave flattening in leads V5 & V6.

Auscultation of the lungs reveals decreased air entry throughout with right basal inspiratory crepitations and occasional expiratory wheeze.

A CXR shows that the right heart border is blurred and the lungs are hyperinflated, but there is an enlarged cardiac silhouette.

Arterial blood gases show: pH = 7.25, pO2 = 70 mmHg on 4 litres per minute, pCO2 = 70 mmHg, HCO3 = 29 mmol/L

Management:
Inhaled or nebulised bronchodilators such as salbutamol (5mg nebulised), can be repeated every 15 minutes. Ipratropium bromide 500mcg nebulised 6 -hourly. At least 4 litres per minute of oxygen or air is required to effectively produce aerosol.

Antibiotics are given because an acute exacerbation of COPD is nearly always due to an infective process. Antibiotics to cover atypical pneumonia with azithromycin or roxithromycin will be required.

Corticosteroid (oral prednisolone or IV hydrocortisone) will be needed in addition to the existing daily dose (if the patient is already on long term corticosteroids).

Non-invasive intermittent pressure ventilation (BiPAP) will help support ventilation and intravenous aminophylline can be considered.

Perform a CXR to rule out alternate pathology and/or coexisting pathology such as a tumour, pulmonary metastases, or pneumothorax.

Serial cardiac enzymes and repeat ECGs during the first 24 hours. A monitored (intensive care) bed is an ideal place to manage such a patient. If there is any abnormality of the serum electrolytes in association with a rise in cardiac enzymes, it needs to be corrected (particularly Ca2+, Mg2+, K+, and Na+).

Intubation and ventilation will be necessary if there is respiratory failure as evident from a rising partial pressure of carbon dioxide or if the patient is tiring.

Patient Progress:
Our patient is admitted to a monitored bed for 24 hrs.
Oxygen therapy is kept at 4 litres/minutes and salbutamol nebules are given every 2 hours with Ipratropium bromide every 6 hours. He is given intravenous hydrocortisone 50 mg every 6 hours for 24 hours. His oxygen saturations remained at 92-93% on 4 litres/minute of oxygen, and there is an improvement in his respiratory rate and shortness of breath. Arterial blood gas performed on day after admission shows: pH = 7.36, pCO2 = 50 mmHg, pO2 = 85 mmHg on oxygen. A course of

intravenous ceftriaxone and oral roxithromycin is given, followed by a two-week course of oral amoxycillin and roxithromycin.

His chest pain improved with 1 spray of GTN and paracetamol. Repeat ECGs showed improvement in T wave flattening. His tachycardia persisted and was considered to be due to nebulised salbutamol. Troponin peaked at 0.05 on day 2. He was commenced on aspirin in view of his history, and follow-up was arranged with a cardiologist for a stress test.

References:

Thorax 2004; 59(Suppl I):1-232 doi: 10.1136/thx.2004.022707

Center for Disease Control. Surveillance for Asthma - United States, 1960-1995, MMWR, 1998; 47 (SS-1).

ABDOMINAL PAIN

Abdominal pain provides a challenging diagnostic task for the clinician due to the number of organ systems potentially involved and the different ages of the patients that present with this complaint. The immediately life-threatening conditions such as a leaking abdominal aortic aneurysm can often mimic relatively benign conditions on first impression, however focusing on early identification of these time-critical illnesses within the first few minutes can be life-saving. In an adult, abdominal aortic aneurysm leak or rupture, gastrointestinal haemorrhage (from peptic ulcer or colonic tumour), ischaemic gut leading to a perforated bowel, and in women, ectopic pregnancy, are diagnoses that should be considered early. The lack of physiological reserve in both the very young and very elderly populations means that time to diagnosis is particularly important.

As with all emergency presentations, basic life support should be commenced immediately. Airway patency needs to be established and the breathing supported if required. Oxygen saturations are measured and supplemental oxygen is applied even if it appears that the oxygen saturation is normal, as a distended abdomen and opioids will invariably impair respiratory function. Intravenous access should be established with at least one large bore cannula, or two if haemorrhage is suspected. Samples of blood are obtained and sent for full blood examination, urea and electrolytes, liver function tests, amylase or lipase, cardiac enzymes if the patient elderly or at risk of ischaemic heart disease. A venous or arterial blood gas can provide a rapid estimate of the haemoglobin, pH and lactate. A beta-HCG should be taken in all women of child-bearing age. Most 'haemaccue' or finger prick haemoglobin machines are quite accurate and can provide a rapid result if it is available. The patient's pulse rate and blood pressure are measured, noting differences between limbs / absent pulses and intravenous fluids are commenced. Extra staff should be obtaining a 12 lead ECG at this point.

Once basic life support has been established, the clinician should begin history-taking whilst simultaneously making an observation of the patient's general appearance and performing a brief abdominal examination to find an obvious abdominal aortic aneurysm (AAA) or rigidity and involuntary guarding as is seen with an acute abdomen.

The patient who is shocked, with abdominal rigidity or an expansile/pulsatile mass:

If the patient is shocked, or abdominal rigidity or an expansile mass is palpated, this is an emergency that is likely to require immediate surgery. The Surgeon and operating staff should be alerted. A second intravenous large bore cannula should be inserted if not already done, and blood sent for urgent blood grouping and screening, plus 2-4 units of blood cross matched if haemorrhage is suspected. Fluid resuscitation should always begin with warm crystalloid solution, unless haemorrhagic shock is confirmed, in which case blood should be given. The use of colloids for initial fluid resuscitation in a shocked patient has not been found to have any additional benefits over crystalloid, and has the associated risk of anaphylaxis. The SAFE trial looked at outcomes at 28 days in intensive care patients who were randomised to either 4%

albumin or Normal Saline for fluid resuscitation and found no difference in outcome (1).

With the development of bedside Ultrasound machines and expertise in their use, FAST scans are becoming standard of care in most western emergency departments for trauma patients and those with suspected intra-abdominal emergencies. Often an abdominal aneurysm can be diagnosed and the amount of free fluid estimated.

Chest X-rays should be obtained to look for free gas under the diaphragm and abdominal x-rays can be considered. Abdominal x-rays are of limited assistance when the patient is supine as the image quality is poor, especially if they are taken with portable X-ray machines. Urinary catheter insertion and urine specimen collection should be performed. Discussion with the surgeon regarding naso-gastric tube insertion, and intravenous antibiotics should be had if the patient has not already proceeded to theatre. Keep in mind the pregnant woman who appears shocked – a vaginal examination with a speculum may reveal products of conception in the cervix which may be causing profound vagal stimulus and 'cervical shock'. There may be a concealed blood loss with conditions such as placental abruption.

If the patient is haemodynamically stable and does not have an acute abdomen:

There is time to take a more thorough history and physical examination. Rectal and pelvic examinations should be conducted, however this may not be always appropriate in children or those with likely perforated large bowel. Blood specimens should be taken if not already obtained, and based on a more detailed history and examination can include more specific tests such as ampylase, lipase, beta-HCG, liver function tests, blood cultures or cardiac enzymes. A specimen of urine should always be tested with a bedside 'dipstick' test, and sent to pathology if abnormal. Beta-HCG can also be performed from the urine specimen however the accuracy of this test is not the same as serological beta-HCG. If there is any doubt about pregnancy or if there is a positive urine test, a blood specimen should be sent to confirm this.

A 12-lead Electrocardiograph should be taken in adults, particularly if there is potential cardiac disease or a need for surgery. Imaging with x-rays and ultrasounds can be performed but often require the patient to be transferred to the radiology department, and so consideration must be given to the likelihood of clinical deterioration whilst the patient is being transferred or having the imaging performed away from the resources of the emergency department.

The clinician then can consider the common differential diagnoses from the above information:

Differential Diagnosis:

Vascular – Abdominal AorticAneurysm leak/rupture, Ischaemic gut:

The pain from a ruptured abdominal aortic aneurysm is an intermittent or continuous abdominal pain that will often radiate to the back or groin.The abdominal findings are an expansile mass (one that expands and contracts) rather than a pulsatile mass (such as a lymph node which transmits a pulse from an adjacent artery). The mortality from ruptured abdominal aortic aneurysm (AAA) is over 75%, (2,3) a statistic that argues strongly for early detection and prompt elective surgery. Although some AAAs are

found on careful clinical examination, the majority are impalpable, particularly in the obese patient. Because of this, imaging modalities such as ultrasound scanning are the mainstay of diagnosis. (3)

The incidence of unruptured AAA in adults aged over 50 years is approximately 3%, and these patients are usually without symptoms. Many patients will have known aneurysms of a size not necessitating surgery (usually conservatively managed if less than 6cm).

Upper and Lower Gastrointestinal System: Gastritis, Peptic ulcer disease/ perforation, Pancreatitis, Diverticulitis, Appendicitis.
Gastritis, described as a burning type epigastric pain that is often transiently relieved by eating or drinking, and aggravated by stress, alcohol or spicy foods, is a common complaint. The lifetime prevalence is approximately 10% in the USA.

Differentiating between gastritis and peptic ulcer disease is more difficult, and signs are nonspecific for both. The main concern in these patients is to exclude a cardiac cause of the pain and ulcer perforation or bleeding. Examination of faeces for occult blood should be performed; often a bedside test of stool from a rectal examination is adequate. Peptic ulcer disease, when associated with bleeding still has a mortality rate of up to 20%, hence the need for early diagnosis to prevent this complication from developing.

Acute pancreatitis can present with either gradual or sudden severe central epigastric pain radiating to the back associated with vomiting and relief on sitting forward. There are many causative factors, but a history of excess alcohol or gallstones should particularly be sought. The mortality is significant at 5-10%, and in fulminant cases, the pancreas becomes necrotic, massive fluid shifts occur, resulting in shock, renal failure, coagulopathy, hypocalcaemia and acute respiratory distress syndrome. Haemorrhage due to pancreatic enzyme erosion of a major blood vessel can result in massive intra-abdominal haemorrhage and cardiovascular collapse. A normal amylase does not exclude pancreatitis.

Diverticulosis is usually found in the sigmoid colon due to higher intraluminal pressures. It appears to be associated with a western diet, constipation and obesity. Whilst this condition is very common with advancing age (affecting over 65% of people by the age of 85 years), development of diverticulitis and complications of diverticulitis such as abscess or fistula formation, perforation or peritonitis remain much less common. The concern is that the complications are seen in the elderly who are least likely to recover from an acute abdomen. Signs and symptoms will often mimic appendicitis, but are predominantly left sided.

Hepatobiliary: Hepatitis, Cholelithiasis / Cholecystitis / Cholangitis/ Gangrenous gall bladder, Primary carcinoma or Secondary metastases
With the adoption of western diets and sedentary lifestyles, diseases endemic in the West will be appearing with greater frequency in hospital emergency departments. Diseases of the biliary tract, specifically cholelithiasis and cholecystitis (as 90% of gallstones in those on a Western diet consist principally of cholesterol), together with pancreatitis (again mostly due to impaction of gallstones or from alcohol abuse) and

peptic ulcer disease (with the use of non-steroidal anti-inflammatory drugs and alcohol abuse) may be seen more often in countries adopting Western lifestyles. Living to an older age has also seen a greater population being diagnosed with cancer ,which has the potential to spread in the circulation and seed to other organs such as the liver.

The obese middle-aged female with recurrent episodes of cramping right upper quadrant pain associated with fatty meals almost certainly has gallstones. Peptic ulcer disease has a lifetime prevalence in the USA of 12% in men and 10% in women. When associated with bleeding, mortality can be up to 20%, and this has not changed despite the introduction of Histamine 2 – receptor antagonists. (4)

Genitorurinary: Urinary Tract Infection, Renal/Ureteric colic:
Renal calculi are found in 0.2% of the population and men will be affected 4 times more frequently than women. The pain is usually described as loin pain that is severe, colicky in type and radiating to the groin (if the stone is in the ureter). Nausea and vomiting are frequent. 80% of renal calculi are visible on abdominal x-rays, and 97% will be seen on CT scans. Ultrasounds will reveal obstruction of the ureter and hydronephrosis, which may alter management.

Gynaecological: Uterine or Ovarian malignancy, fibroids, Ovarian cyst, Sexually transmitted disease / Salpingitis, Ectopic pregnancy, Miscarriage:
Any woman of reproductive age should be assessed for pregnancy with beta-HCG serology. This is useful not only in diagnosing the pregnancy (intrauterine or ectopic), and estimating the gestational age of the fetus, but also in deciding on which further imaging modalities may be used safely, in view of the high levels of radiation from CT scans and (to a lesser extent) x-rays.

The incidence of ectopic pregnancy is approximately 11 per 1,000 pregnancies in the United Kingdom. Any suspicion of an ectopic pregnancy should be managed as if there is one, and large bore intravenous access and obtaining a specimen for blood grouping and screening for antibodies should be performed without delay.

Features of infective process in sexually active females, such as high temperature and vaginal discharge, should alert the clinician towards a likely salpingitis or sexually transmitted disease, and prompt the commencement of empirical antibiotics.

Gynaecological malignancies will have a slower onset of pain that will typically be associated with other features of these malignancies such as weight loss and abnormal vaginal spotting. Similarly endometriosis will present with a history of recurrent pain associated with menses and sexual activity.

Infant: Colic, Intussuception, Urinary tract infection, Gastroenteritis, Pyloric Stenosis Paediatric: Appendicitis, Mesenteric Adenitis, Constipation, Burkitt's or other lymphoma.
Infants and older children with abdominal pain have a narrower list of potential causes, but present the challenge of taking a detailed history and ordering both appropriate and safe investigations.

Age is a big factor in making a diagnosis, and thus major diagnoses to exclude in infants are intussception (red-currant jelly-like faeces), pyloric stenosis (projectile vomiting in the first few weeks of life), constipation, gastroenteritis, urinary tract infections and vesico-ureteric reflux nephropathy. Rare lymphomas and leukaemias are often missed because the symptoms and signs are attributed to viral causes.

A history of similar pain, nausea and vomiting in siblings or other family members will often help with the diagnosis of a viral or bacterial gastroenteritis. Oral intake and urinary output should be assessed and careful fluid and electrolyte replacement provided by either the nasogastric or intravenous route. All children and infants should have a urine specimen obtained (preferably a midstream sample to avoid contamination with skin flora) and a suprapubic needle aspirate with a sterile approach may be indicated in infants.

Appendicitis incidence peaks in the second and third decades of life, and is rare at extremes of age, however if it does occur in infants it is associated with much higher rates of perforation and increased mortality. Classically the pain is initially mild and poorly localised to the periumbilical area. It then develops into more severe right lower quadrant pain as the parietal peritoneal surfaces become inflamed. Nausea and vomiting are found in up to 60% of cases.

Other differential diagnoses:

Once life-threatening diagnoses have been excluded, attention may be given to alternate reasons for the abdominal pain. These will often have an onset associated with an acute event – such as eating undercooked, reheated or poorly prepared meals, or have a long history such as with inflammatory bowel disease. Overdose of easily available medications such as paracetamol (acetaminophen) or aspirin is not uncommon, and may be potentially life threatening. Somatisation of psychiatric disorders will often present with abdominal pain, however this is a diagnosis of exclusion and will taken much time and investigation to be labelled as the cause – well outside the ability of the emergency department on first presentation.

Clinical scenario 1:

A 15 yr old female presents with 24-hour history of abdominal pain:
Differential diagnoses: Appendicitis, Constipation,Menses, Ectopic Pregnancy, Lower Lobe Pneumonia, Gastroenteritis, and Diabetic Ketoacidosis

History, examination and investigations need to be focussed on excluding life threatening diagnoses initially. In this case, ectopic pregnancy and appendicitis are the most immediately concerning diagnoses to exclude.

History:

The pain when described as being a central, nonspecific pain that changes to well localised right iliac fossa pain is very suggestive of appendicitis. Gastroenteritis can mimic the early stages of appendicitis, so it is useful to enquire about similar symptoms in family members or work colleagues. Nonspecific pain in the setting of polyuria, polydipsia and weight loss should ring alarm bells for undiagnosed diabetes or diabetic ketoacidosis particularly in this age group. A finger-prick blood glucose can assist in making this diagnosis at the bedside within seconds.

It may be difficult to take history in presence of adult relatives, or if abuse has occurred, but should be still be sought along with a history of the woman's menstrual cycle.

Unilateral pelvic pain, often cramping in nature, and unilateral adnexal tenderness occurring in early pregnancy, with or without vaginal bleeding is strongly suggestive of ectopic pregnancy. This is the leading cause of pregnancy-related death in the first trimester.

Examination:

It is essential to establish rapport with the patient. Be sensitive to exposing her and ensure adequate privacy, and provide appropriate analgesia. When palpating the abdomen, ensure warm hands or use a bed sheet to act as a thermal barrier, and commence examination at the furthest point away from painful region and work towards it.

Investigations:

Beta-HCG:

Both from urine and blood. Qualitative urine and blood tests are simple, and can detect levels as low as 10 mIU/ml. They can be performed at bedside, and are relatively sensitive and specific. A positive test should be confirmed and the level quantified with a serum beta-HCG, which can detect levels as low as 5mIU/ml. The urine specimen should also be tested with urine 'dipstick' test to look for infection and glucose.

Coagulation profile and cross-matching:

2 to 3 units of cross-matched packed red blood cells should be available if ectopic pregnancy is strongly suspected.

Blood Glucose level:

A glucometer provides accurate measurement within a few seconds. There is no need to delay this investigation until an intravenous cannula is inserted.

Full Blood examination:

Haematological disorders and malignancies can present in many ways. A raised white cell count may assist in confirming the diagnosis but can be quite nonspecific.

Electrolytes:

This test is particularly important in assessing the degree of illness in gastroenteritis, and the degree of dehydration.

Blood cultures:

Should be considered in any child who is febrile and unwell. Ideally they are taken prior to the commencement of antibiotics, with the exception of a case of suspected bacterial meningitis where no intravenous access is available. In this scenario, the clinician needs to give intramuscular penicillin immediately.

Imaging:

X-rays are helpful in looking for bowel obstruction, gross constipation, or free gas if a perforation suspected in abdominal injury. Radiation exposure to any female without checking whether she is pregnant can only be justified in trauma or life-threatening emergencies. Appendicitis may be diagnosed with ultrasound imaging or CT scanning, however this is not yet established as the gold standard test for this

diagnosis, and the radiographer may not be able to visualise appendix using ultrasonography.

Ectopic pregnancy:
Incidence is 11.1 in 1,000 pregnancies in the UK (from 2004 data). The risk factors include pelvic inflammatory disease, assisted contraception such as in-vitro-fertilisation, smoking and previous ectopic pregnancy or surgery. If it is clinically suspected, the patient should be surgically managed and ultrasound imaging should not be performed if the patient is haemodynamically unstable. In this situation resuscitation and emergency surgery is indicated. If the patient is haemodynamically stable, an ultrasound should be performed to look for an intrauterine pregnancy (although this does not always rule out a coexisting extrauterine pregnancy –this occurs in 1 in 5,000 pregnancies), free fluid in the pelvis, or an adnexal mass. If there is no evidence of any of these features and a quantitative beta-HCG is < 1,000 MIU, the patient should be observed with gynaecological review and Rhesus immunoglobulin given as required.

Clinical scenario 2

70 yr old Male presents with an acute onset of abdominal pain one hour ago.

Differential Diagnoses: ischaemic Bowel, Diverticulitis / Perforated Diverticulum / Diverticular abscess, Perforated Viscus, Ruptured or leaking AAA, Perforated Peptic Ulcer +/- active bleeding, Bowel Obstruction (due to malignancy or adhesions from previous surgery), Appendicitis (less common), Renal Colic / Urinary Tract infection / Urine retention.

In this man, one must exclude life-threatening illnesses and be hesitant to label the pathology as constipation, gastroenteritis, or urinary tract infection. Elderly patients have less cardiovascular and respiratory reserve to compensate for major illness if it is missed.

History:
The pain had an acute onset 1 hour ago, approximately an hour after finishing his dinner. He collapsed onto the ground with pain and vomited once. The pain is mostly epigastric, spreading to his central anterior chest wall, and into his left shoulder tip. There is no history of any vascular or gastrointestinal pathology.

Examination:
Abdominal examination reveals a rigid abdomen, with involuntary guarding, particularly in the upper quadrants. No palpable or expansile mass is felt, although palpation is difficult due to pain and guarding.

Mangement:
Next step….
Call the surgeon & notify theatres.
Obtain second large bore IV cannula. Blood is sent for cross matching, haematology, electrolytes, urea and creatinine, liver function tests, and coagulation studies. The blood pressure is noted to be 90/45 mmHg and the heart rate is 110 beats per minute. 1 litre of crystalloid is infused under pressure.

You are arranging for an ultrasound of the abdomen when he has a large vomit of dark brown substance resembling altered blood. The patient states that this is occurring for the second time tonight. He thinks it may be the bottle of wine he was drinking before he came into hospital. Further history reveals he drinks a bottle of vodka over 1-2 days on average plus a bottle of wine with his dinner each night. He has been troubled by lethargy, dizziness and generalised aches and pains over the last month, and has been taking ibuprofen for this intermittently.

Further investigations are arranged:

- Arterial blood gas shows a metabolic acidosis, pH = 7.23, pCO2 = 31 mmHg, HCO3 = 19, lactate elevated at 2.5, Haemoglobin is 74 g/dL,
- Erect Chest X-Ray shows gas under diaphragm.
- ECG shows a sinus tachycardia 114 beats per minute.

The most likely diagnosis is a perforated peptic ulcer aggravated by non-steroidal medication and alcohol consumption.

Management:

The airway, breathing (may be impaired by distended, painful abdomen) and circulation are assessed. Fluid resuscitation is commenced. Keep the patient fasted. Organise operating theatre and relevant anaesthetic and surgical doctors. An intravenous proton pump inhibitor such as pantoprazole can be given as an intravenous infusion. Ensure blood is sent and there is a request for at least 2 units of cross-matched blood.

References:

1. The SAFE Study Investigators. 'A Comparison of albumin and saline for fluid resuscitation in the intensive care unit'; *N Engl J Med* 2004; 350: 2247-56

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3. www.mja.com.au/public/papers/mattes/mattes.html#refbody3

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INTERPRETING ECGs

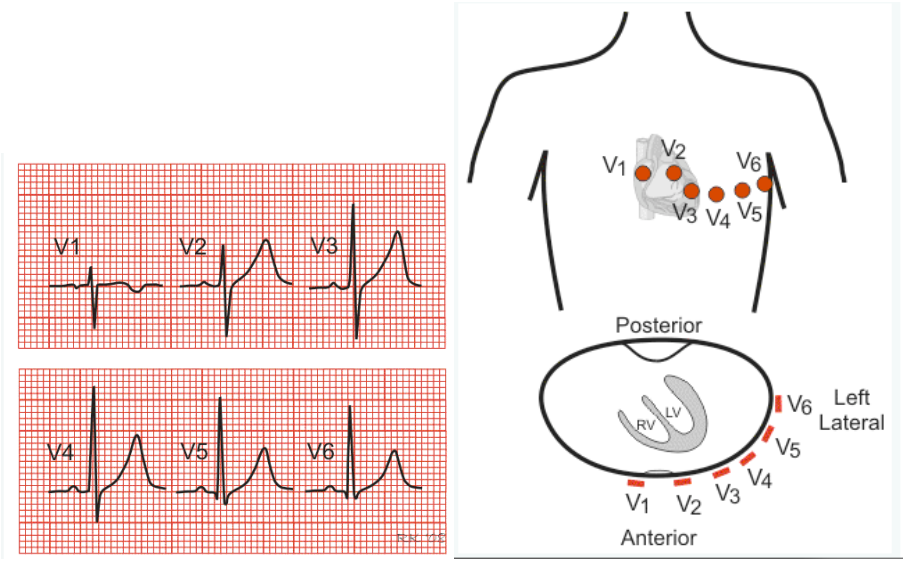
Electrical Activity of the Heart:

Cardiac pacemaker cells (typically found in the Sinoatrial SA node in the right atrium), initiate the electrical currents in the heart. The depolarisation triggered by the pacemaker cells is transmitted along conducting fibres that travel through the atria to the Atrio-Ventricular node (AV node). Here, there is a delay prior to the rapid depolarisation of specialised fibres in the ventricular septum – from the bundle of His, to the left and right bundle branches – before a slower depolarisation of the Purkinje fibres that lie in the ventricular myocardium.
Myocardial cells are polarised (carry an electric charge on their surface) due to transmembrane ion concentration differences (primarily Na+ and K+). Normal charge is negative 90mV, with the inside of the cell being negatively charged relative to the outside. When cells are stimulated to above their 'threshold' potential, they depolarise and become transiently positively charged. The process of depolarisation then spreads in one direction down the conducting pathways of the heart, followed by repolarisation, returning the electrical potential back to its resting state of –90mV.

Principles of the ECG:

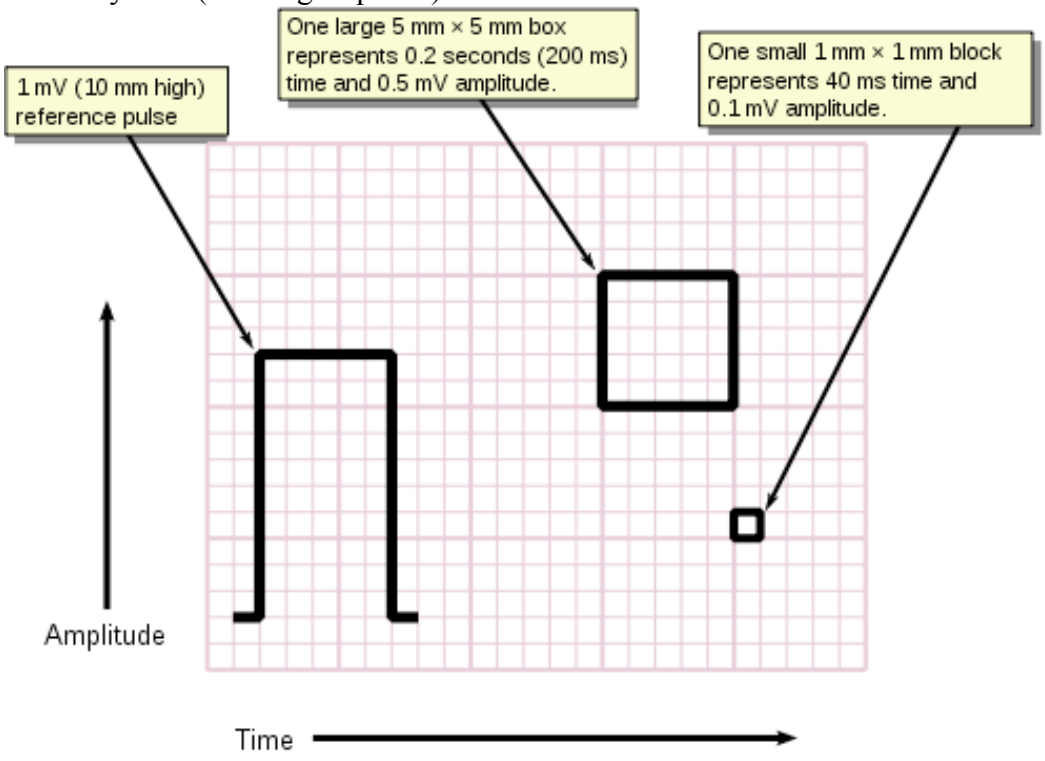
The ECG leads display the differences in electrical potential between the electrodes.

Leads:
There are six limb leads (3 bipolar leads: I, II, III and 3 unipolar leads: avR, aVL, aVF) and six chest leads (V1 to V6). Each bipolar lead measures the difference between two electrodes, whereas the unipolar leads measure the voltage at one point relative to an electrode that has zero action potential (avR looks at right arm, aVL looks at left arm, and aVF looks at left leg or foot). The letter "a" that precedes these three leads refers to the fact that these are electrically augmented by 50%. The limb leads look at the heart in a vertical plane.
The six chest leads are unipolar and positioned from V1 (fourth intercostal space adjacent to the right side of the patient's sternum) V2 same space on the left side of the sternum, V4, V5 and V6 are all in the 5th intercostal space, but in the midclavicular, anterior axillary and midaxillary lines, respectively.



These chest leads look at the heart in the horizontal plane. V1 & V2 look at the right ventricle, V3 & V4 look at the anterior wall of the left ventricle, and V5 & V6 look at the lateral wall of the left ventricle.

Calibration of the Machine is important to allow for the detection of small complexes that might indicate a pericardial effusion, and tall R waves that indicate left ventricular hypertrophy. A standard signal of 1 millivolt (mV) should move the stylus vertically 1cm (two large squares).

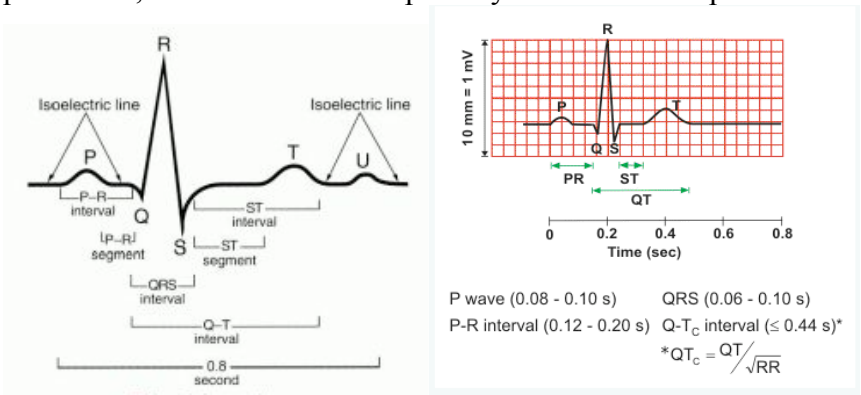


Waves & normal values:

The P wave represents atrial depolarisation. The current moves down and to the left of the patient, thus it is positive in lead II, and negative in aVR. The P wave may be bifid if there is left atrial hypertrophy when there is mitral valve pathology, or pulmonary hypertension. The normal duration of the p wave is less than 120 milliseconds.

The QRS complex represents ventricular depolarisation. The normal duration of the QRS is less than 120 milliseconds and the normal axis of the QRS complex is between -30 and + 90 degrees. If the QRS is more negative (less than -30 degrees), this reflects left axis deviation, if it is more positive (more than + 90 degrees), there is right axis deviation. The shape of the QRS differs in each lead. As depolarisation spreads towards a lead, the stylus moves upwards (positive deflection), and when it spreads away, the stylus moves downwards (negative deflection). The QRS presents the average or cumulative direction in which the wave of ventricular depolarisation is spreading. When the depolarisation is moving at right angles to the lead, the R and S waves are of equal size.

The T wave represents ventricular repolarisation. It should therefore follow the QRS in the same direction. Repolarisation is the electrically reverse process as depolarisation and it occurs in the opposite direction. The T wave may be normally inverted in young people in leads aVR and V1/V2. T wave inversion may indicate ischaemia, ventricular hypertrophy, bundle branch block or treatment with digoxin. The U wave is of unknown origin and in the normal heart it appears as a small deflection (< 1mm) following the T wave with the same polarity as the T wave. If it is of higher amplitude, it may indicate hypokalaemia. When the U wave is very prominent, is a marker of susceptibility to 'torsades de pointes'.



Intervals and segments:

The intervals represent the time taken for conduction of the electrical current through different parts of the heart.

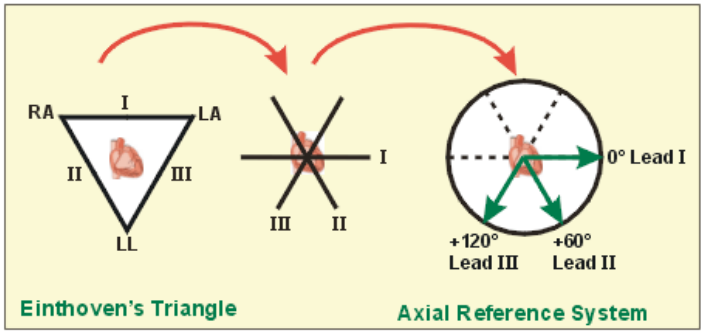
PR interval is measured from the beginning of the P wave to the beginning of the QRS complex. It represents the time taken for the spread of excitation from SA node, down through the Bundle of His to the ventricular muscle. The normal value is 0.12 – 0.2 sec. Most of this time is taken up at the AV nodal pause.

QRS duration is the time taken for the spread of excitation through the ventricular muscle. The normal duration is less than 0.12 sec.

The ST segment should be 'isoelectric', that is, the same level as the segment between the T wave and the next P wave. Elevation of the ST segment indicates infarction or, pericarditis if it is widespread. A depressed ST segment with normal upright T waves indicates ischaemia. Down-sloping ST segments may be seen in those taking digoxin.

The QT interval (corrected for rate = QTc) represents the time taken for depolarisation and repolarisation of the left and right ventricles. The normal value of the QTc is < 0.44 seconds. A prolonged QTc can be hereditary, or it can be due to drugs such as amiodarone and erythromycin. There is an increase risk of torsades de pointes (ventricular tachycardia with a twisting axis) with a prolonged QTc interval.

Bipolar Limb leads and the cardiac vector (left and right QRS axis deviation): The direction of the cardiac axis is most easily detected from the QRS complex in leads I, II & III, in which the QRS is a predominantly upward deflection when there is a normal axis. The deflection will be most positive in lead II as the wave is heading directly for this electrode. The normal cardiac axis is between negative 30 degrees and positive 90 degrees.



Right axis deviation: (QRS > positive 90 degrees) If the right ventricle becomes enlarged, the axis will shift towards the right, resulting in a more positive lead III, and a less positive (or even negative) deflection in lead I. A right axis deviation can occur after pulmonary embolism, or in a patient with pulmonary hypertension. It may be a normal finding in a tall thin person.

Left Axis deviation: (QRS < negative 30 degrees) If the left ventricle becomes hypertrophied or if there is a conduction defect, the axis may swing to the left, with lead I becoming more positive and lead III becoming predominantly negative.

Transition point: Is the point at which the QRS complex in the chest leads has equal size R and S waves. Normally this is seen in V3/ V4. If this moves to V4/V5 or V5/V6, it signifies an enlargement of the right ventricle, and a 'clockwise rotation' of the heart. This may occur with chronic lung disease.

Systematic approach to interpreting the ECG:

A systematic approach is adopted so as not to miss the diagnosis. The following is followed: Name, date, rhythm, rate, describe the P wave, PR interval, Q waves, QRS width and shape, ST position and T wave direction. Comment on the cardiac axis and the QTc interval.

Conduction defects:

1st degree heart block:
A conduction delay is present somewhere between the SA node and the AV node, causing a prolonged PR interval (> 0.2 sec). This may indicate coronary artery disease, acute rheumatic carditis, digoxin toxicity or electrolyte disturbances.

2nd degree heart block:
When there is an intermittent failure of the excitation to pass through the AV node or Bundle of His, this is called a second-degree heart block.

There are 3 types of second-degree heart block:

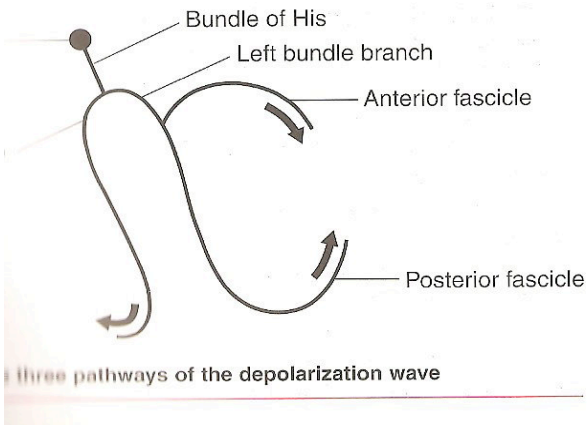
- "Mobitz type 2": Occasional failure of conduction to the ventricle.

- "Wenckebach": Progressive lengthening of the PR interval followed by a failure to conduct an atrial beat, then return of a normal PR interval, after which the cycle is repeated.
- "2:1 or 3:1" conduction: A regular pattern where only every 2nd or 3rd beat is conducted to the ventricle. For every two P waves there will be one QRS complex in 2:1 block. For every 3 P waves, there will be one QRS complex in 3:1 block.

The Wenckebach phenomenon is usually benign, however the other types of second-degree heart block may be precursors to complete heart block.

3rd degree heart block "Complete heart block":
There is complete dissociation between atrial depolarisation and ventricular depolarisation. There is no relationship between the P waves and QRS complexes. The ventricles are excited by a slow ventricular depolarising focus. The causes include acute myocardial infarction, or fibrosis of the bundle of His or of both bundle branches.

Bundle branch block:
There is a prolonged QRS duration of greater than 0.12 sec, as the myocardium supplied by the blocked bundle branch depolarises slowly and late.



Right Bundle Branch Block (RBBB):
There is an 'RSR1' or 'M' pattern in V1, and deep S waves in V6 (a 'W' pattern). RBBB may be seen in Pulmonary Embolism or Cor pulmonale. It often indicates problems with the right side of the heart, but may be seen in normal subjects. Left bundle branch block however, is always an indication of heart pathology, typically affecting the left side of the heart. In RBBB, the ventricular septum depolarises as normal from left to right, giving an R wave in V1, and a small Q wave in V6. As excitation spreads to the left ventricle there is an S wave in V1 and an R wave in V6. The prolonged time for the right ventricle to depolarise causes a second R wave (R1) in V1 and an S wave in V6, hence the 'RSR1' pattern. If the QRS is not widened in the presence of an 'RSR1' pattern, it is called a 'partial RBBB', which is non-significant.

Left Bundle branch block (LBBB):
In LBBB the septum depolarises from the right to the left, causing a small Q wave in V1 and an R wave in V6. The smaller right ventricle is depolarised before the larger

left ventricle, so there is then a small R wave in V1, and a small S wave (or notch) in V6. Finally the Left ventricle depolarises causing a deep S wave in V1 and a tall R wave in V6, giving an "M" pattern in V6, and, in a fully developed LBBB, a"W" pattern in V1. Inverted T waves in V5-6, I and aVL are associated with LBBB. In the presence of a LBBB, one CANNOT comment on the ST segment or T wave. If LBBB is present, consider aortic stenosis and ischaemic heart disease.

The Left bundle branch differs from the right bundle branch in that it divides further into the anterior and posterior fascicles. As the left ventricle is a much larger muscle compared to the right ventricle, it exerts greater influence on the cardiac axis. If there is failure to conduct down the anterior fascicle, the cardiac axis will be deviated to the left, resulting in a 'left anterior hemiblock'. If the posterior fascicle is selectively blocked, which is uncommon, there is right axis deviation. Right bundle branch block has no effect on the cardiac axis as the large bulk of the left ventricle is depolarising normally.

Bifascicular block:

This is a combination of Right bundle branch block and left bundle hemi-block (anterior fascicle block). It results in Left axis deviation and indicates significant damage to the conducting pathways.

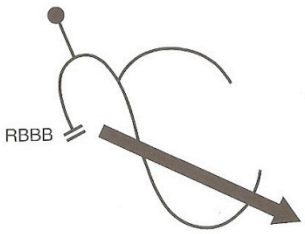


Fig. 2.21 Effect of RBBB on the cardiac axis

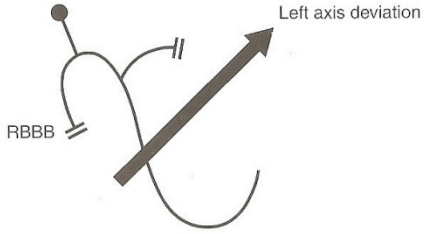


Fig. 2.22 Effect of RBBB and left anterior hemiblock on the cardiac axis

Trifascicular block:

Trifascicular block is a combination of a bifasicular block and 1st degree heart block.

Arrythmias:

Supraventricular tachycardias:

Atrial Tachycardia:

The atria depolarise at a rate of greater than 150/minute. As the AV node cannot conduct at rates greater than 200/minute, there will be an AV block if the atria depolarise faster than 200/minute. This can mimic second degree heart block.

Atrial Fibrillation:

Atrial fibrillation may occur in the normal patient and is often triggered in acute illness or sepsis. During atrial fibrillation there is an in-coordination between atrial and ventricular contraction. This impacts on stroke volume (up to 30% loss in left ventricular end diastolic volume). A loss of 30% of stroke volume may not be well tolerated in those with heart failure or stenotic heart lesions.

Atrial flutter:

The atria depolarise at a rate greater than 250/minute. There is no flat baseline between P waves, giving a 'sawtooth appearance' to the baseline of the ECG. If there is a narrow complex tachycardia with a ventricular rate of 150, be aware it may be

atrial flutter with 2:1 block, as the extra P wave may be hidden or mistaken for the T wave. Vagal manoeuvres, such as carotid sinus massage may increase the block at the AV node and slow down SA node firing, revealing the underlying rhythm as atrial flutter.

Nodal (junctional) tachycardia:
The P waves may be very close to or hidden in the QRS complex when the area around the AV node depolarises frequently. In nodal tachycardia there are narrow (normal) QRS complexes with no apparent P waves. There is normal conduction down the bundle of His.

Ventricular tachycardias:
Ventricular tachycardia (VT):
When a focus in the ventricle depolarises at a rapid rate, VT is observed. There are widened and abnormal shaped QRS complexes due to the abnormal pathway of depolarisation through the ventricular mass. No atrial activity is seen. To differentiate between VT and a supraventricular tachycardia with bundle branch block, one needs to consider the clinical presentation. VT is commonly found in the setting of acute myocardial infarction. It is also helpful to look for P waves and the relationship with the QRS complexes. The clinician should find previous ECGs and compare the shape of the QRS complexes. (They should be the same shape if LBBB was present previously). Look at the width of the QRS (if it is wider than 160 milliseconds, it is likely to be ventricular in origin), and look at previous ECGs for a change in the cardiac axis (which would suggest a ventricular origin of the tachycardia).

Ventricular fibrillation:
In ventricular fibrillation the ventricular muscle fibres contract independently from each other in a completely disorganised fashion, resulting in no QRS complexes, and thus no ability to eject blood from the ventricles.

Other ECG findings:

Long QT: (QTc >0.44 seconds):
May be congenital or acquired. It may result from medications such as amiodarone, quinidine and erythromycin, or in electrolyte disturbances such as hypocalcaemia, hypomagnesaemia or hypothermia. Prolonged QT is associated with Torsades de Pointes (polymorphic ventricular tachycardia).

Acute Cor pulmonale:
Acute cor pulmonale (typically from pulmonary embolus) is diagnosed on the ECG by the presence of a sinus tachycardia, a right shift of the QRS axis, and the classical ‘S1, Q3, T3’ pattern (prominent S waves in lead I, Q waves in lead III and T wave inversion in lead III). Acute Right Ventricular dilatation may occur with T wave inversion in V1 to V4 and poor R wave progression, which may resemble an acute anterior infarction.

Wolf Parkinson White (WPW) syndrome:
WPW is characterised by wide QRS complexes associated with a short PR interval, and a slurring of the initial part of the QRS complex (called the delta wave). These changes occur due to aberrant activation of the ventricular myocardium by a ‘bypass’

tract or ‘accessory bundle’, resulting in ‘pre-excitation’ of the ventricles. This increases the risk of re-entrant supraventricular tachy-arrhythmias. In this situation, depolarisation can spread down the Bundle of His and return back up into the atria via the accessory bundle, causing reactivation of the atria, and a sustained tachycardia.

Toxicity:

Drug toxicity from phenytoin, tricyclic antidepressants and electrolyte disturbances produce ECG changes that are typical of each condition.

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4. Pathophysiology of Heart Disease
5. Current Emergency Diagnosis and Management
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BASIC LIFE SUPPORT

Bystander resuscitation of the collapsed person is one of the most important factors in eventual long term recovery of that person.

Recently recommendations by the International Liaison Committee on Resuscitation (ILCOR) have changed slightly to facilitate:

- improved confidence of minimally trained bystanders to commence resuscitation

- the minimization of interruption to the core activity of resuscitation namely chest compression

- make resuscitation training broadly applicable and easy to learn and recall

The basic concepts in resuscitation are unchanged, and where previous protocols or training are known, and well learnt, there should be no hesitation in continuing existing practice if rescuers are unsure.

The key concepts remain

- Safety of the rescuer is paramount

- Help from others (and rescue professionals) should be called for immediately

- The victim should be moved to safety from an environment where further injury is likely

- Silent victims should be attended first

- The airway should be cleared and victim positioned to facilitate airway opening

- ***Victims that are not breathing should receive both rescue breaths and chest compressions***

- Breaths and compressions should be continued until expert assistance directs otherwise, or the victim recovers.

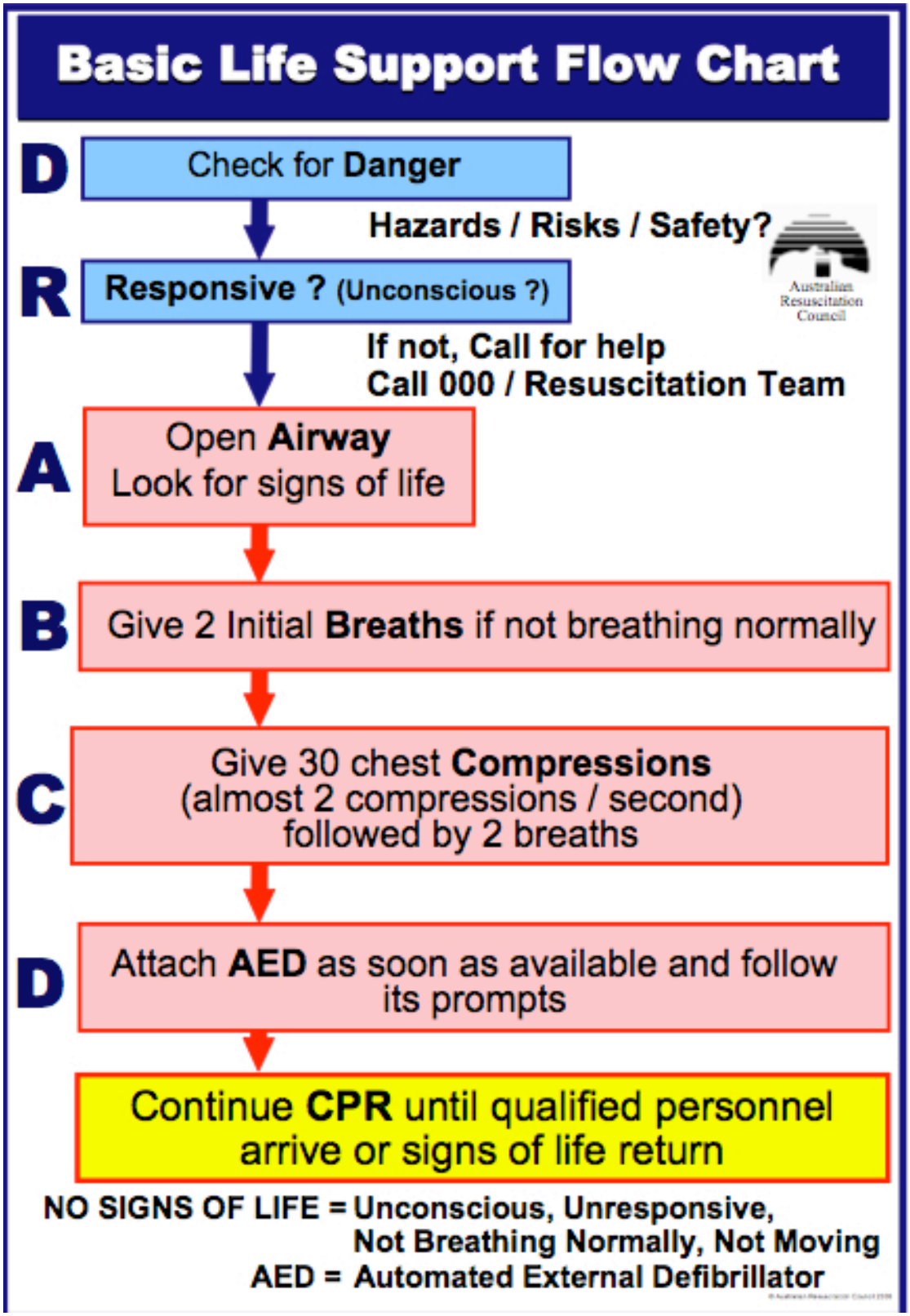
ADVANCED LIFE SUPPORT ALGORITHM

Ischaemic heart disease is a leading cause of death. It accounts for up to 40% of all deaths under the age of 75 years and sudden cardiac arrest is responsible for more than 60% of adult deaths from coronary heart disease. By 2010, cardiovascular disease (CVD) will be the leading cause of death in developing countries. CVD includes ischaemia, hypertension, heart failure and rheumatic fever.

One third of people with a myocardial infarction will die before reaching the hospital, with most of these dying within one hour of symptoms. The presenting cardiac rhythm in most of these deaths is ventricular fibrillation or ventricular tachycardia. Both of these rhythms may be treated with defibrillation. Early defibrillation is important as the chance of survival decreases by 7-10% with each minute's delay to defibrillation.

Basic Life Support:

Basic life support is the initial management of a collapsed patient. It can be administered in the community or in hospital before expert help arrives. It consists of the delivery of rescue breaths and cardiac compressions. The Australian Resuscitation Council (ARC) and other national bodies have written guidelines and have adopted a universal algorithm that is taught to health care providers including first aiders, ambulance officers, doctors, nurses, allied health providers and the general public.



CPR = Cardiopulmonary Resuscitation

The basic life support algorithm involves the recognition of a cardio-respiratory arrest, calling for help and delivery of rescue breaths and cardiac compressions before a defibrillator arrives.

The initial management of a collapsed patient involves the following steps:

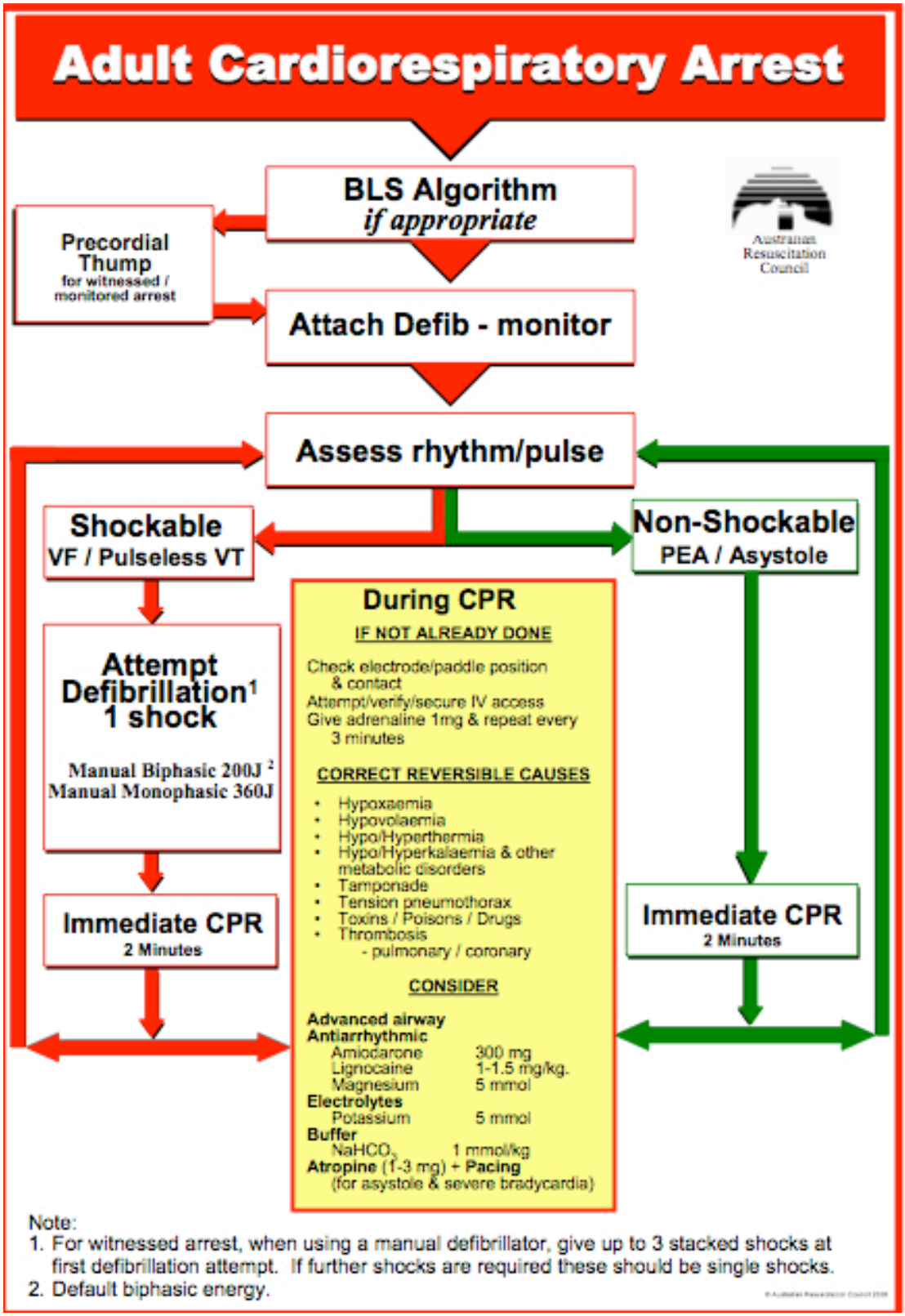
1. Ensure personal and patient safety
2. Check the patient for a response
3. Open the airway and check for breathing. A carotid pulse check is not recommended unless you are trained to do so and experienced in the assessment of sick patients
4. If you are alone, call for help
5. Give 2 rescue breaths followed by 30 chest compressions
6. As soon as the defibrillator arrives, apply the electrodes and analyse the rhythm; delivering a shock if the patient is in ventricular fibrillation (VF) or ventricular tachycardia (VT)
7. Recommence chest compressions immediately after the defibrillation attempt without pausing to check the rhythm

Cardio-pulmonary Resuscitation (CPR) needs to be commenced as soon as possible in a collapsed patient. Chest compressions and ventilation of the lungs will slow down the deterioration of the heart and lungs and for out of hospital arrest, bystander CPR can double the chance of survival. Interruptions to chest compressions must be minimized and should only occur briefly (10 seconds) during defibrillation attempts and rhythm checks. The compression to ventilation ratio is 30 compressions to 2 breaths.

Chest compressions are performed to a depth of 5 cm (one third of the antero-posterior diameter of the chest wall) over the lower half of the sternum. The rate of chest compressions is 100 beats per minute. Chest compressions can be tiring. If there are enough rescuers, change the person doing chest compression every 2 minutes.

Advanced Life Support:

The advanced life support algorithm is structured around two pathways: the shockable rhythms pathway or the non-shockable rhythms pathway. The shockable rhythms are VF and pulseless VT. The non-shockable rhythms are asystole and pulseless electrical activity (PEA). The difference in management is the need for defibrillation in patients with VT or VF. The other aspects of management are the same, including chest compressions, airway management and ventilation, venous access, administration of adrenaline and the identification of reversible factors.


Adult Cardiorespiratory Arrest
BLS Algorithm
if appropriate
Precordial Thump
for witnessed / monitored arrest
Attach Defib - monitor
Assess rhythm/pulse
Shockable
VF / Pulseless VT
Non-Shockable
PEA / Asystole
Attempt Defibrillation[1]
1 shock
Manual Biphasic 200J [2]
Manual Monophasic 360J
Immediate CPR
2 Minutes
During CPR
IF NOT ALREADY DONE
Check electrode/paddle position & contact
Attempt/verify/secure IV access
Give adrenaline 1mg & repeat every 3 minutes
CORRECT REVERSIBLE CAUSES
Hypoxaemia
Hypovolaemia
Hypo/Hyperthermia
Hypo/Hyperkalaemia & other metabolic disorders
Tamponade
Tension pneumothorax
Toxins / Poisons / Drugs
Thrombosis
- pulmonary / coronary
CONSIDER
Advanced airway
Antiarrhythmic
Amiodarone 300 mg
Lignocaine 1-1.5 mg/kg.
Magnesium 5 mmol
Electrolytes
Potassium 5 mmol
Buffer
NaHCO3 1 mmol/kg
Atropine (1-3 mg) + Pacing
(for asystole & severe bradycardia)
Immediate CPR
2 Minutes
Australian Resuscitation Council
Note:
1. For witnessed arrest, when using a manual defibrillator, give up to 3 stacked shocks at first defibrillation attempt. If further shocks are required these should be single shocks.
2. Default biphasic energy.

Shockable rhythms (VT and VF):

The sequence of events for the shockable side of the algorithm is as follows:

1. BLS (as above)
2. When VF or VT is confirmed, attempt defibrillation with one shock of 150-200 joules (biphasic defibrillator) or 360 joules (monophasic defibrillator)
3. Immediately resume CPR (30:2) without reassessing the rhythm or feeling for a pulse. Continue CPR for 2 minutes then pause briefly to check the monitor for a rhythm.
4. If VF/VT persists give a second shock
5. Resume CPR immediately and continue for 2 minutes
6. If VF/VT persists give adrenaline 1 mg intravenously followed immediately by a third shock
7. Resume CPR immediately and continue for 2 minutes. Pause briefly to check the monitor
8. If VF/VT persists, administer amiodarone 300 mg intravenously followed by a fourth shock
9. Resume CPR immediately and continue for 2 minutes. Pause briefly to check the monitor
10. If VF/VT persists, administer adrenaline 1 mg intravenously followed immediately by defibrillation for every second cycle of CPR and defibrillation (approximately every 3-5 minutes)
11. Give further shocks after each 2 minute period of CPR after confirming that VT/VF persists
12. If organised electrical activity compatible with a cardiac output is seen, check for a pulse. If there is no pulse, continue CPR and switch to the non-shockable side of the ALS algorithm. If there is asystole, continue CPR and switch to the non-shockable algorithm.

The interval between stopping chest compressions and delivering a shock must be minimised (about 10 seconds). Long interruptions to chest compression will reduce the chance of a shock restoring a spontaneous circulation.

Chest compressions are resumed immediately after a shock without checking the pulse or rhythm because even if sinus rhythm is restored, it is rare for a pulse to be palpable immediately and interruptions to chest compression will compromise myocardial perfusion.

There is no evidence for the use of any anti-arrhythmic drug routinely during human cardiac arrest to improve survival to hospital discharge, but amiodarone in shock refractory VF improves short term survival. If amiodarone is not available, lignocaine may be given (100 mg intravenously). Magnesium may be of use (5 mmol intravenous bolus) if there is a possibility of hypomagnesaemia. Often, hypokalaemia is accompanied by hypomagnesaemia.

If there is doubt about whether the rhythm is fine VF or asystole, do not attempt defibrillation. Very fine VF is unlikely to be shocked successfully into a perfusing rhythm. Continuation of good quality chest compression and ventilation may improve the amplitude and frequency of fine VF and improve the chance of successful defibrillation.

Non-shockable rhythms (PEA and asystole):

Pulseless electrical activity (PEA) is defined as organised electrical activity without any palpable pulse. There may be some mechanical contraction of the heart, but the contractions are so weak that they do not produce a palpable pulse. There may be a reversible cause and this needs to be identified and treated.

The sequence of events in a cardiac arrest where there is PEA:

1. Start CPR 30:2
2. Give adrenaline 1 mg as soon as intravenous access is secured.
3. Continue CPR 30:2 for 2 minutes.
   When the airway is secured (intubation), continue chest compressions at a rate of 100 beat per minute (with no pauses for breaths) for 2 minutes. Ventilations are delivered at a rate of 10 breaths per minute.
4. After 2 minutes, check the rhythm and for a pulse or signs of life.
5. If there are no signs of life or a pulse, continue CPR for 2 minutes.
6. Give further adrenaline 1 mg intravenously every 3 minutes (alternate 2 minute cycles).

Asystole may be caused by excessive vagal discharge. Atropine is given to a total dose of 3 mg as required to provide maximal vagal inhibition if there is asystole or slow PEA. Check the ECG carefully for the presence of p waves. Absent QRS complexes in the presence of p waves represents ventricular standstill, which is treated with cardiac pacing.

For asystole or slow (less than 60 beats per minute) PEA

1. Start CPR 30:2
2. Check the leads are attached
3. Give adrenaline 1 mg as soon as intravenous access is secured. Give atropine up to 3 mg.
4. Continue CPR 30:2 for 2 minutes
7. When the airway is secured (intubation), continue chest compressions at a rate of 100 beat per minute (with no pauses for breaths) for 2 minutes. Ventilations are delivered at a rate of 10 breaths per minute After 2 minutes check the rhythm and for a pulse (if trained to do so) or for signs of life.
8. If there are no signs of life or a pulse, continue CPR for 2 minutes.
9. Give further adrenaline 1 mg intravenously every 3 minutes (alternate 2 minute cycles).

During cardiopulmonary resuscitation:

- Emphasis is placed on good quality chest compressions
- Seek and treat reversible causes of cardiac arrest
- Obtain intravenous access
- Secure the airway
- Continue CPR during the 2 minute cycle after defibrillation, even if organised electrical activity is seen
- Continue CPR during the 2 minute cycle even if VF or VT is visible on the monitor and attempt defibrillation after the completion of the 2 minute cycle

Tracheal intubation provides a reliable airway but should only be attempted by an expert with minimal interruption to chest compressions. No intubation attempt should take longer than 30 seconds. Once the patient is intubated, continue chest compression at a rate of 100 per minute without pausing for ventilation. Continuous chest compression during ventilation produces a higher mean coronary perfusion pressure. Ventilate the lungs at a rate of 8-10 breaths per minute. Hyperventilation reduces venous return to the heart.

Intravenous access allows for the administration of drugs. Drugs administered peripherally need to be flushed with 20 ml of fluid. It may also be helpful to elevate the arm for 10-20 seconds to facilitate delivery into the central circulation.

Reversible causes of cardiac arrest:

- Hypoxia –Ventilate the lungs with 100% oxygen, ensure there is adequate chest rise and bilateral breath sounds. Check the endotracheal tube.
- Hypovolaemia –can cause PEA and is usually due to severe haemorrhage. Infuse intravenous fluids and stop the bleeding.
- Hypothermia or hyperthermia –hypothermia may occur after drowning or exposure, hyperthermia is associated with thyrotoxic storm and malignant hyperthermia is due to suxamethonium or volatile anaesthetic agents.
- Hypokalaemia or hyperkalaemia/ other electrolyte disturbances may be suspected from a history of diuretic use or renal failure. The ECG may show features of electrolyte disturbances.
- Tamponade –cardiac tamponade is difficult to diagnose but can be suspected after thoracic trauma.
- Tension pneumothorax –is diagnosed clinically by absent breath sounds and tracheal deviation. It needs to be decompressed urgently in order to release the tension and allow for a reduction in intrathoracic pressure.
- Toxins/Poisons/Drug
- Thrombosis –massive pulmonary embolus or coronary thrombosis can be treated with thrombolytic medications.

References:

1. Australian Resuscitation Council. Advanced Life Support, 5th edition, 2006.
2. Australian Resuscitation Council Website. www.resus.org.au/

TRAUMA IN THE PREGNANT PATIENT

Trauma is a common cause of non-obstetric foetal and maternal death with the risk to mother and foetus being highest in the third trimester. Effective management of the pregnant trauma patient requires knowledge of the physiological and anatomical changes of pregnancy. Adherence to the principles of "Early Management of Severe Trauma" [EMST] and the appreciation that there are two patients is fundamental.

Physiological Changes of Pregnancy

The physiological changes of particular relevance in the pregnant trauma patient include: Cardiovascular, Respiratory, Haematological, and Gastrointestinal.

Cardiovascular

Cardiac Output	↑ 30-40%	Significant blood loss may occur prior to onset of clinical markers of hypovolaemia.
Heart Rate	↑ 20-25 bpm	
Systemic Vascular Resistance	↓ 1000-1400 dyne.s.cm^{-5}	
Blood Pressure	↓ 5-15% in second Trimester. Normal at term	
Aorto-caval compression	Especially in 3rd Trimester	Left lateral Tilt

With increased intravascular volume there can be significant blood loss and uterine hypo-perfusion, without hypotension, tachycardia or other signs of shock!

Respiratory

Tidal Volume	↑ 35%	↑ Uptake of Inhalational agents
Respiratory Rate	↑ 15%	↓ O_2 reserve and more rapid desaturation
Functional Residual Capacity	↓ 25%	
O_2 Consumption	↑ 20%	
pH	7.41-7.46	Alkylosis shifts O_2 curve left increasing transfer to O_2 fetus
$PaCO_2$	27-32 mmHg	
Airway	*800% Increased risk of difficult intubation*	

The combination of: Reduced Functional residual capacity, Increased O_2 consumption and hence rapid desaturation, potentially difficult airway anatomy, increased risk of reflux and the maintenance of cervical spine precautions, ***must always be respected****. The most senior airway physician should be involved early. Airway rescue protocols and equipment need to be planned for and be immediately available.*

Haematological

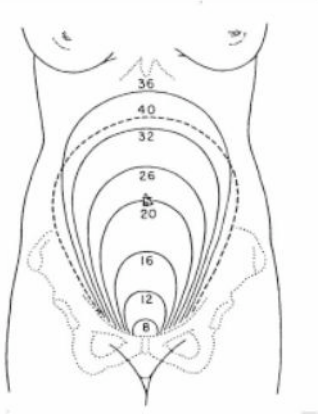
Procoagulant State: Increased coagulation Factors VII, VIII, IX, X and Fibrinogen. Five fold higher risk of pulmonary embolus and Deep Vein Thrombosis.
Physiological anaemia of pregnancy: Rise in plasma volume greater than the rise in Red cell number. Overall rise in blood volume 30-50%.

Gastro-Intestinal Tract

Increased Gastro-oesophageal reflux and reduced gastric emptying, increases the risk of aspiration.

The Expanding Uterine Target.

The expanding uterus peaks above the pelvic rim at 12-13 weeks. Up until this point, the embryo is protected by the bony pelvis, amniotic sac and soft tissues.
At 20 weeks the fundus is at the umbilicus.
The fundus reaches the Xiphisternum at 36 weeks.



Uterine blood flow is NOT subject to auto-regulation. Perfusion of the uterus, and hence foetus, is dependant upon maternal blood pressure, in particular mean arterial blood pressure.

Aortocaval compression:

From around 25 weeks the expanding uterus, under the effect of gravity in the supine position, can compress the Inferior Vena Cava causing a reduction in venous return. Most healthy women can compensate for the reduced venous return through vaso-constriction and increased heart rate. However the hypovolaemic pregnant trauma patient, when supine may become profoundly hypotensive and unstable. Lateral displacement of the uterus is imperative to facilitate venous return. Usually displacement is to the left, and achieved using a wedge [15^{o}-25^{o}], manual displacement of the uterus or tilting the entire bed/spine board. Of note up to 10% of women may have improved venous return with right lateral displacement.

Mechanism of Trauma

An assessment of the mechanism of injury in any trauma patient helps create an index of suspicion as to the nature of injuries received. Blunt force trauma can result in shearing injuries and increased risk of hepatic, splenic and retro-peritoneal injury due to increased vascularity. The most common complication of blunt trauma is placental abruption.

In motor vehicle accidents, an unrestrained pregnant woman, has a 280% increased risk of foetal death, 200% increased risk of significant haemorrhage and increased likelihood of delivery within 48 hours.

Fetal injury may be *Direct* - Organ or cord rupture, spinal injury, intra-cerebral bleed or head injury due to maternal pelvic fractures. *Indirect* mechanisms include:

Abruption, uterine rupture, feto-maternal haemorrhage and premature labour. In the second and third trimesters, the fetus has left the protective bony pelvis, and relatively minor trauma may result in significant morbidity and mortality. The highest risk is in the third trimester. The foetal head, once engaged, is at particular risk from maternal pelvic injuries and acceleration and deceleration injuries.

Determinants of Foetal Outcome

The best predictor of foetal outcome is the severity of the maternal injuries. It follows, that the best treatment for the foetus is optimising resuscitation and management of the mother.
Identified risk factors for foetal loss include:

- Significant maternal injury severity.
- Presence of severe abdominal injury.
- Presence of hemorrhagic shock: hypotension, tachycardia. The uterus lacks autoregulation and hence maternal hypotension results in hypoperfusion.
- Ejection from a vehicle
- Maternal: Hypoxia, contractions
- Abnormal foetal heart rate
- Maternal Acidosis

Trauma Management Principles: General and Obstetric Specific

The trauma management of the pregnant patient should follow the EMST/ATLS guidelines with attention to several pregnancy specific issues.

Primary Survey:
The Primary Survey mantra of **A**irway/Cervical spine immobilisation, **B**reathing, **C**irculation, **D**isability, **E**xposure [**ABCDE**] provides a robust framework for the management of trauma.

Supplemental oxygen should be applied to all trauma patients whilst the *airway* is assessed and patency secured.

Breathing assessment: Rate, depth and effort of breathing, tracheal deviation, auscultation and percussion with assessment symmetry.

Assessment of *circulation* with control of active bleeding: level of consciousness, pulse rate, rhythm and strength, skin colour, blood pressure and capillary refill time. As part of circulatory assessment and resuscitation, 15-25^{o} left lateral tilt should be performed on all pregnant women at greater than 24 weeks gestation. Spinal precautions should be adhered to wherever possible, including tilting of spine boards. Otherwise continuous manual lateral displacement of the uterus should be used.

Disability is a brief neurological examination. Either the Glasgow Coma score or AVPUP [Alert, responds to Voice, responds to Pain, Unresponsive, Pupils] scale can be used.

Exposure of the patient facilitates a thorough examination. Efforts to maintain or restore normothermia are an important part of resuscitation. Active warming devices, fluid warmers and warm blankets may all be used.

Resuscitation:

All trauma patients should receive supplemental oxygen early in their management. Given the respiratory changes of pregnancy, and the absolute foetal dependence on maternal oxygenation, all pregnant trauma patients should receive supplemental oxygen.

Fluid resuscitation with crystalloids, colloids or blood products should be aggressively pursued. Due to expansion of the maternal blood volume, (30- 50%) the clinical signs of significant hypovolaemia may appear late. **Hypovolaemia resulting in utero-placental hypoperfusion, can occur without hypotension, tachycardia or other signs of shock!**

Note: The central venous pressure shows poor correlation with left ventricular filling pressures, hence the use of a central venous catheter has a limited role in early management of trauma.

If intercostal drains need to be inserted, they should be placed 2 intercostal spaces higher than the non-pregnant patient, to allow for the upwardly displaced diaphragm and avoid intra-abdominal trauma.

Secondary Survey:

A thorough trauma *secondary survey* examines the entire body from Head to toe in a systematic way. The primary survey [ABCDE] and resuscitation should be completed, prior to commencing the secondary survey. In the pregnant patient, the secondary survey involves taking an obstetric history and examining for pregnancy specific pathology. This requires an abdominal examination to assess for uterine tenderness or contractions. Uterine contractions are common following trauma. Trauma to the uterus can cause release of products of Arachidonic Acid, stimulating uterine contractions. The fundal height should be noted and compared to expected dates. Vaginal bleeding may be from a uterine source [such as placental abruption] or localised trauma. Clear vaginal fluid may be amniotic fluid and indicate rupture of the membranes. Clear fluid should be assessed for ferning and pH. Amniotic fluid has a pH 7, whilst an acidic pH around 5 is likely to be vaginal secretions.

Summary of Trauma Management.

Primary Survey: To identify and manage immediate threats to life.
A. Airway and Cervical Spine Precautions
B. Breathing- Rate, Depth, Tracheal placement, Bilateral ventilation, Supplemental Oxygen.
C. Circulation and Control of Bleeding- Wound compression, Tilt/Uterine displacement, IV access and fluid resuscitation, Bloods [including Cross-match, Full Blood examination, electrolytes, coagulation, pregnancy, Kleihauer test
D. Disability: Brief Neurological assessment. GCS or AVPUP
E. Exposure and Temperature control
Resuscitation
Management based on Vital signs gathered during the Primary Survey: Heart rate, Respiratory Rate, Blood Pressure, Saturations
Fluid resuscitation, Intercostal Drains, ECG, Urinary Catheter, Nasogastric tube
Monitoring: O2 saturation, ECG, Blood Pressure [NIBP/IABP], Blood gas, End-Tidal CO_2
Imaging: CXR, Cervical Spine, Pelvis, Diagnostic Peritoneal Lavage/FAST
Secondary Survey
AMPLE History and Mechanism of Injury ❄
Physical Examination: Systematic review of entire body and accessible orifices.
Obstetric: *History*: Gestation, Obstetric complications, *Examination*: Fundal Height, Tenderness, contractions, Foetal heart rate assessment and lie, Vaginal fluid [blood, amniotic]
Investigations: X-ray, CT, Ultrasound, CTG
Definitive Care
Analgesia, Early involvement of Obstetric team, Possible surgery or transfer Anti-D in Rh negative mothers

❄***AMPLE***: Allergies, Medications, Past Medical History / Pregnancy, Last meal, Events leading up to injury.

Assessment of Maternal and Foetal Well-being.

If the mother has been physiologically stabilised, an assessment of foetal wellbeing can be performed as part of the secondary survey. Continuous cardiotocography [CTG] should be implemented early in patients at greater than 26 weeks. CTG monitoring should continue for at least 6 hours. Longer periods of monitoring or further investigations should be considered in the presence of: foetal compromise, contractions, vaginal bleeding, and significant uterine or maternal injury.

Pregnancy should not preclude a pregnant woman from undergoing *appropriate* trauma imaging. The risk to the foetus is highest in first trimester. Shielding can be used where possible to reduce exposure. CT scans produce significantly higher levels

of radiation than plain films. Keeping radiation exposure to less than 5 Rad, appears to minimise the risk to the foetus.

Ultra-sound FAST studies have been validated in pregnant patients, and should be used whenever the skills and technology is available. It is a sensitive diagnostic test for free peritoneal fluid in the pregnant patient. In skilled hands, ultrasound may be a useful modality for assessment of foetal wellbeing [Heart rate, Movement, Liquor] but has limited sensitivity for diagnosing intra-uterine pathology such as abruption.

Peri-mortem Caesarean Section

In non-trauma related maternal cardiac arrests, the decision to proceed with a Caesarean section is based on both maternal and foetal grounds. For the mother, evacuation of the uterus will reduce aorto-caval compression assisting in venous return, as well as improving the effectiveness of cardiac compressions. Salvage of the foetus depends on proceeding with a Caesarean 4-5 minutes after the onset of the arrest. The decision to proceed to a Caesarean must consider the viability of the foetus at that gestational age.

In trauma related cardiac arrest, the evidence is less clear, with poor maternal and foetal outcomes being reported in the situation of peri-mortem caesarean section. In the trauma situation, proceeding with a caesarean section early in the management period may interfere with maternal resuscitation. Clinical judgement remains the key in whether on not to proceed with a Peri-mortem Caesarean section.

Summary

The Primary [ABCDE] and Secondary survey approach to trauma, needs to be applied to all pregnant trauma patients. Relatively minor trauma can cause significant morbidity in pregnancy, and requires treating clinicians to maintain a high degree of clinical suspicion. A practical understanding of the physiological and anatomic changes of pregnancy is fundamental to effective treatment. The best trauma management for the foetus is prompt assessment, resuscitation and treatment of the mother by a multi-disciplinary team of physicians.

References

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4. Petrone, Asensio. Trauma in Pregnancy:assessment and treatment. *Scandinavian Journal of Surgery*. 2006;95;4-10.
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SECURING THE AIRWAY AND CERVICAL SPINE IN TRAUMA

"Airway" is always the beginning of any emergency assessment

The airway should be assessed and a patent airway established *first* in all emergency situations, before moving on to the **Breathing** or **Circulation** steps of basic or advanced life support.

Whenever there is a significant change in a patient's condition, assessment and intervention should return to the Airway-Breathing-Circulation algorithms, and again begin with assessment and management of the **Airway**.

Airway assessment may be as simple as hearing a patient talking without distress, and observing the absence of stridor or other obstruction. In this situation the patient is already *maintaining and protecting* his or her own airway and requires no airway intervention.

Cervical spine protection should be considered at the time of airway assessment

Protecting the cervical spine with a hard collar should be considered in all patients exposed to significant trauma:

- Motor vehicle accidents > 40 km per hour.
- Motor vehicle accidents involving any death.
- All motor-bike accidents.
- Falls from greater than 2m.
- Any trauma associated with a decreased level of consciousness.

Airway Intervention

Establishing and maintaining a patent and protected airway should occur before any other step in emergency management. Intervention involves:

1. Airway manoeuvres
2. Suction and debris clearance
3. Airway devices

Although endotrachial intubation offers a definitive, patent and **protected** airway, intubation should only be performed where there is appropriate equipment, trained and experienced staff and where it will not cause undue delay in oxygenation or resuscitation of the patient. No more than 60 seconds should be spent on endotracheal intubation if alternative techniques for oxygenation are available.
Alternative oxygenation techniques include: hand-mask ventilation; laryngeal mask airway; cricoid or trans-tracheal oxygenation.

Airway manoeuvres

Airway manoeuvres manipulate the upper airway to remove obstruction caused by the tongue and oro-pharynx, and so establish **airway patency**. Manoeuvres can be used in obstructed but spontaneously breathing patients, or in apnoeic patients requiring bag-mask ventilation.

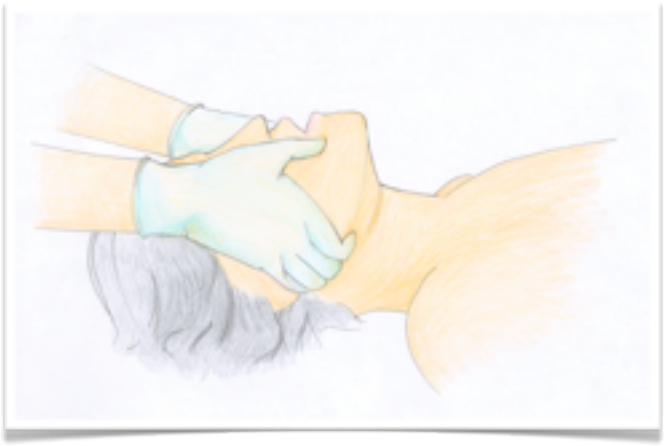
Head-tilt & chin-lift:

This manoeuvre should *only* be used if you are confident that there is no risk of injury to the cervical spine. Stand on the patient's right side, and using the left hand apply pressure to the forehead to extend the patent's neck. Using the other hand elevate the mandible, lifting the tongue from the posterior pharynx.

Do not use 'head-tilt and chin-lift' if the patient's cervical spine is unstable, as the head-tilt causes cervical spine flexion.

Jaw thrust:

This manoeuvre has the advantage of not requiring movement of the cervical spine, and should be used wherever there is risk of spinal injury or in unconscious trauma patients. Place your dominant hand at the angle of the patient's mandible (jaw) and apply upward pressure to elevate the mandible, lifting the tongue. Jaw thrust can be performed either one-sided with a single hand, or on both sides of the patient's jaw with two hands.



Suction and debris clearance

Suction of the oropharynx or the performance of a ‘finger-sweep’ with a *gloved* hand may be required in unconscious patients in order to remove vomit, gastric contents, blood, teeth or other debris from the mouth. Oral debris or foreign material will obstruct the upper airway and lead to aspiration and possible death in patients unable to protect their own airway.

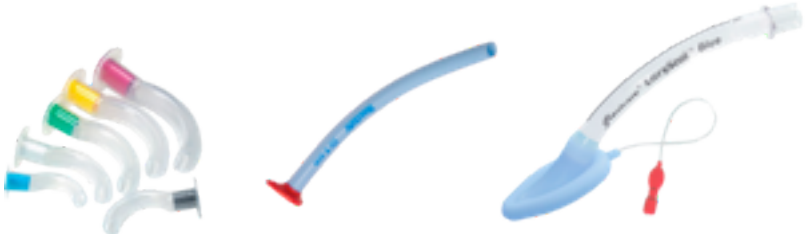
Endotracheal intubation should be considered for these patients if trained staff and appropriate equipment is available.

Airway devices

Airway devices are either **supraglottic** (placed above the ‘glottis’, or larynx) or **infraglottic** (below the larynx, such as an endotracheal tube). Supraglottic devices include the oropharyngeal Guedel’s airway, the nasopharyngeal airway, the laryngeal mask airway, and various other oral airways. While supraglottic devices *do not protect* the airway, they do assist with oxygenation of the patient by maintaining airway patency.

Oxygenation is the first priority in any airway management.
The use of supraglottic airway devices is an acceptable choice in a trauma or resuscitation scenario either to assist with oxygenation while preparing for endotracheal intubation or tracheostomy, or in situations where trained staff or equipment is not available.

Nasal airways (such as a nasopharygeal airway or nasal endotracheal intubation) should be avoided in patients with head trauma due to the risk of brain injury in the presence of base of skull fractures.



Endotracheal intubation

Endotracheal intubation secures the most definitive airway, both ensuring airway patency and protecting the airway from soiling. Nonetheless it should only be performed when it will not lead to undue delay in oxygenating or resuscitating the patient.

Intubation in unconscious non-responding patients can usually occur without either an anaesthetic agent or muscle relaxant. In conscious patients a **rapid sequence induction** should be performed using:

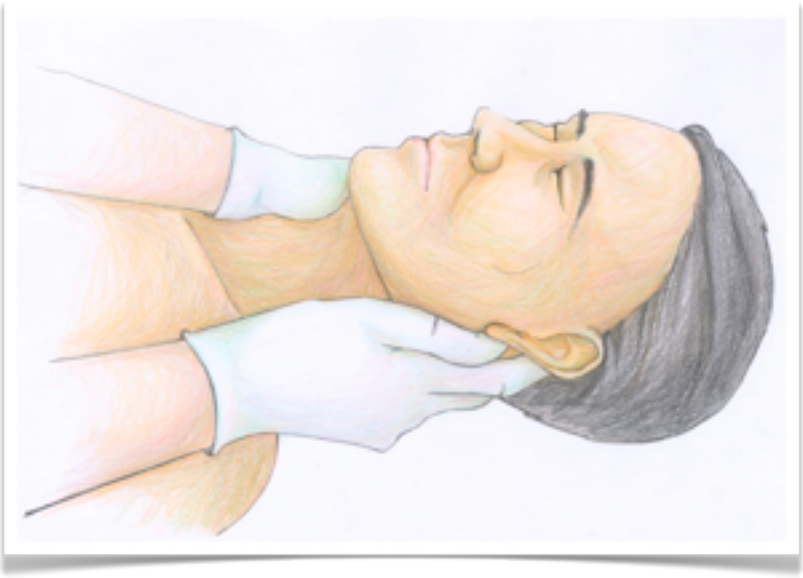
1. **Pre-oxygenation**.
2. **Thiopentone** — be careful in patients with haemodynamic compromise.
3. **Cricoid pressure** and access to oral suction.
4. Rapidly acting muscle relaxant, such as **suxamethonium**. Avoid suxamethonium if: 1. Burns older than 24 hours; 2. Neurological injury older than 24 hours; 3. Hyperkalaemia.

Manual in-line stabilisation

Special care needs to be taken when intubating patients with suspected cervical spine injuries. These patients are at risk of spinal cord trauma at the time of intubation due to neck flexion that commonly occurs with laryngoscopy.

To reduce the risk of spinal cord injury in patients with possible cervical spine trauma:

1. Carefully **remove the hard collar**.
2. Have an assistant apply "**Manual in-line stabilisation**". This requires the assistant to stand at the patient's shoulder facing the intubating doctor. The assistant grasps the head with both hands, with thumbs in front of the ears and fingers curling back around the patient's occiput. The assistant's forearms should gently rest on the patient's anterior shoulders, reducing the potential movement of the head and cervical spine in relation to the trunk.



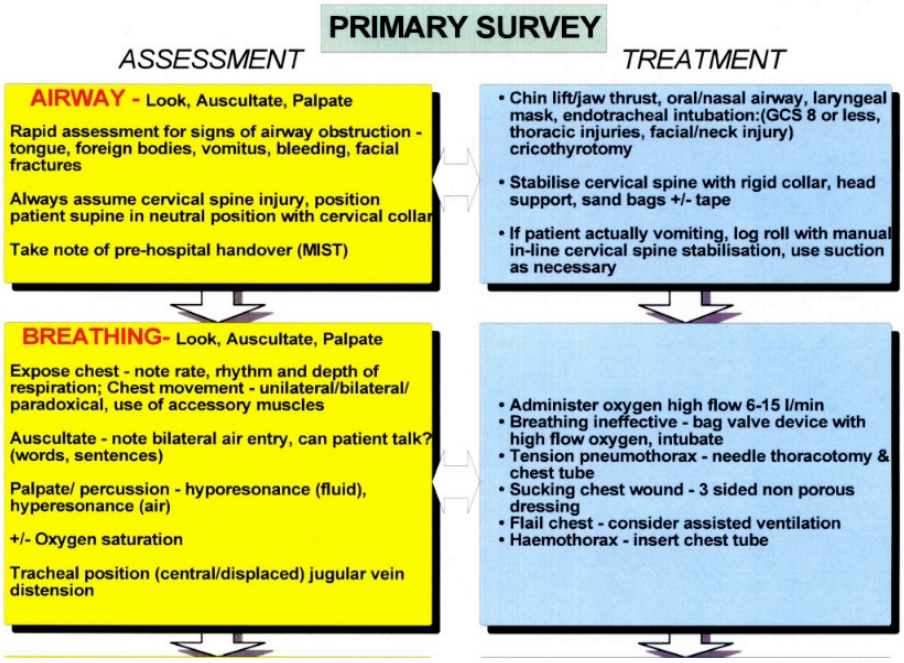
3. Perform a **rapid sequence induction**. The assistant should warn the intubating doctor if he/she feels there is any flexion of the neck at the time of intubation.

4. Manual in-line stabilisation will make intubation more difficult. If intubation cannot be achieved, manual in-line stabilisation may need to be relaxed or abandoned completely. Although manual in-line stabilisation is standard of practice in many countries, there is conflicting evidence for its benefit.

PRIMARY SURVEY IN TRAUMA: BREATHING

Breathing is the second priority in the Primary trauma survey. It is essential to assess it correctly and provide definitive support before moving on to circulation and further assessment of the patient. As with the airway, assessment and treatment occur simultaneously.

Look, Listen and Feel



Breathing assessment

Exposure: neck and chest should be adequately exposed.

Need to fully assess for the following signs:

1. Work of breathing
2. Adequacy of breathing
3. Effects of breathing

Work of Breathing:

Respiratory rate (compare with normal values):

Tachypnoea (increased rate) may be due to airway/lung disease, pain, anxiety or acidosis. Hypoventilation (decreased rate) may be due to hypoxia, brain injury or drugs. Intercostal/subcostal/sternal recession and the use of accessory muscles are markers of increasing respiratory distress and respiratory difficulties.

Stridor

Is a sign of upper airway obstruction.

Wheeze

Is a sign of lower airway obstruction.

Adequacy of Breathing:

The useful signs or clinical indicators of adequate tidal volume being moved by patient are chest movement, air entry and adequate oxygenation and a normal arterial pCO2.

Chest movement

- Expansion will reflect the tidal volume.
- Look for symmetry & variations in chest expansion, is it equal, symmetrical?
- Is the trachea centrally placed?
- Position of the apex beat
- Breath sounds, are they equal and bilateral?
- Unequal chest movement may indicate a pneumothorax
- Check the percussion note. Is it resonant equally on both sides?
- Hypo-resonance or a dull percussion note indicates fluid in the chest or consolidation and hyper-resonance or a high percussion note indicates air such as with a pneumothorax

Air entry

- Listen at the lips for adequate air movement and auscultate the chest for air entry.

Oxygenation and carbon dioxide

Oxygen saturation above 95% whilst breathing air and PaCO2 mmHg less than 40mmHg (as measured by arterial blood gasses), indicates adequate ventilation and gas exchange.

Effects of Breathing:

- Mental state

Hypoxia/hypercapnia produces agitation/drowsiness.

- Heart Rate

Hypoxia initially produces tachycardia then bradycardia (a sign of imminent death).

- Skin colour

Hypoxia produces pallor. Cyanosis is a late sign. It may be masked in the anaemic/ shocked patient.

Look for other signs of chest/airway injury such as:

- Open wound in the neck or chest
- Flail segment
- Crepitus/ surgical emphysema in the chest wall or neck indicates air outside of the lung and in the tissues. It is never normal.
- Skin bruising or abrasions to the neck or chest

Breathing management

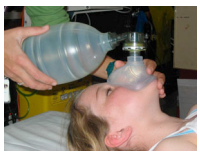
Secure the airway with cervical spine immobilisation.

If the patient's breathing is adequate, provide oxygen at 15 litres per minute via a Hudson mask. If support of the ventilation is required, use bag and mask ventilation prior to tracheal intubation.

Ensure adequate monitoring of the patient. This includes oxygen saturation, respiratory rate, pulse, ECG, and blood pressure monitoring. Continually reassess and monitor trends.

Bag and Mask Ventilation

If the airway is stable with or without an oropharngeal airway, and the patient requires breathing support, bag and mask ventilation, with a self-inflating bag, can be used. Attach oxygen to the apparatus from a supply of oxygen and set the flow to 15 litres/min and provide insufflations or positive pressure ventilation sufficient to inflate the chest. Auscultate the chest and ensure equal bilateral air entry and chest movement on insufflations. Allow 2- 3 times longer than the duration of inspiration for passive expiration. Reassess ventilation continuously whilst using bag and mask ventilation. If there is any further inadequacy, consider tracheal intubation.



Bag & mask ventilation

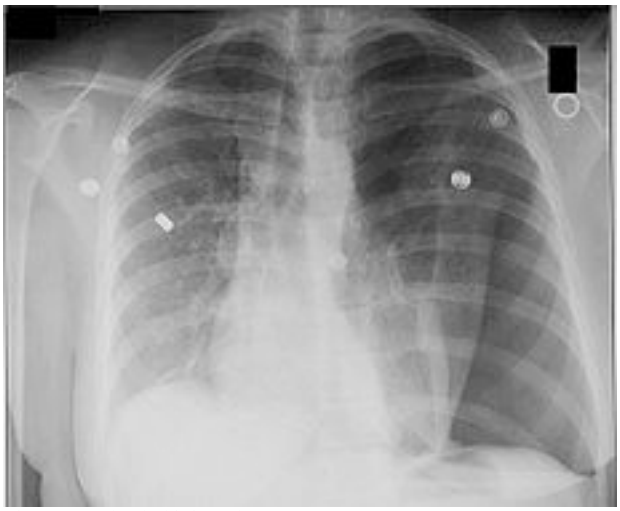
Choose a suitable size mask and suitable size bag. There are 3 sizes of bag available: 250 ml, 500 ml and 1500 ml.

Indications for Intubation:

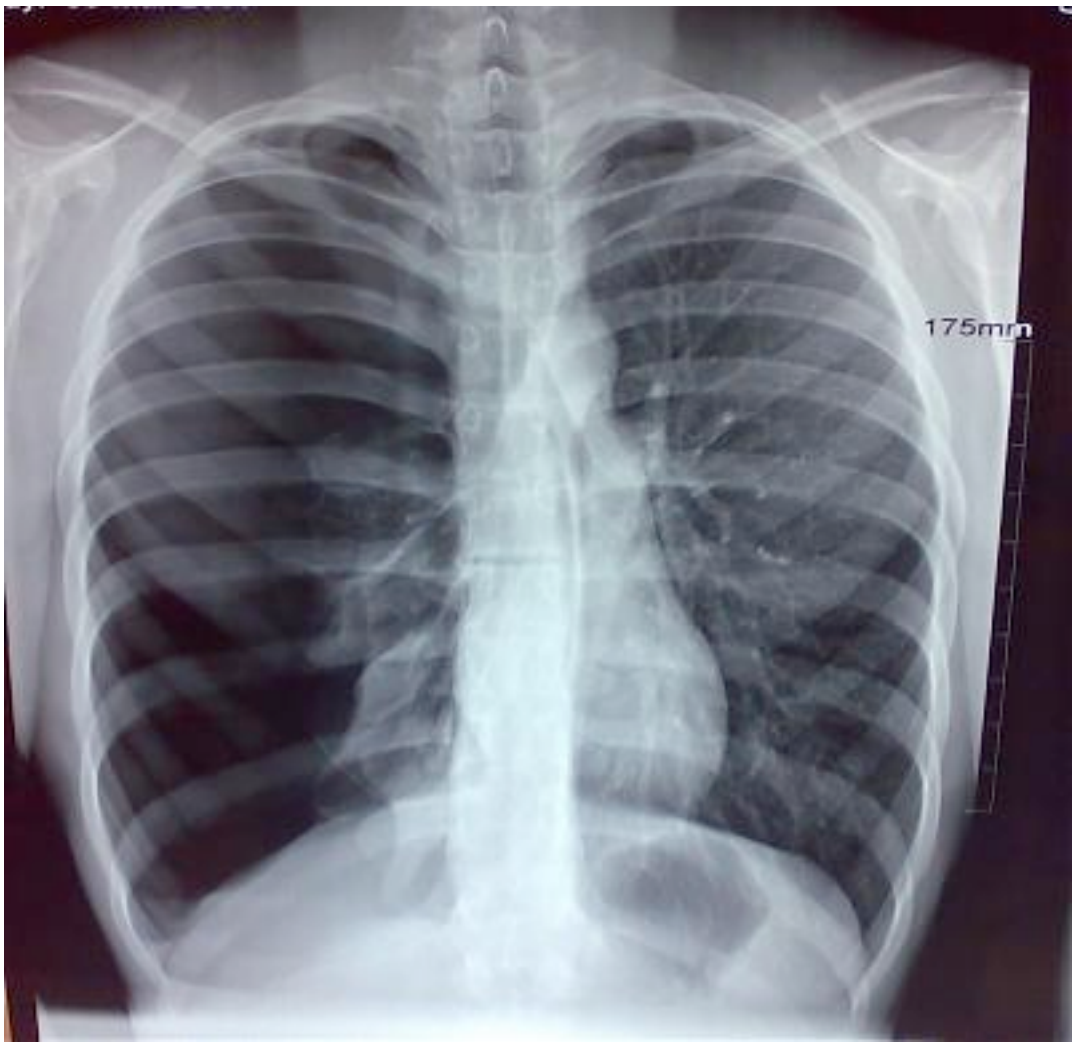
- Airway obstruction that persists despite simple opening maneuvers and placement of an oropharyngeal (Guedel) airway or for definitive airway stabilisation;
- Adequate ventilation is not possible via bag and mask ventilation;
- The exists a need for definitive airway protection (from blood, vomitus, foreign bodies);
- The patient is unresponsive to pain with a Glasgow Coma Score of less than 8;
- There is evidence of significant head injury (such as abnormal posture);
- There will be a need for prolonged ventilation;
- Respiratory tract burn injury.

During the survey of breathing in a trauma patient, there is a need to identify and initiate treatment for tension pneumothorax, open pneumothorax, massive haemothorax and a flail chest.

Tension pneumothorax:



Chest X-ray of Left-sided Tension



A tension pneumothorax is the progressive build-up of air within the pleural space, which allows air to escape into the pleural space but not to return. This creates a “one-way value” air leak. Progressive build-up of pressure in the pleural space pushes the mediastinum to the opposite hemithorax, and obstructs venous return to the heart. This leads to circulatory instability and may result in traumatic cardiac arrest. Positive pressure ventilation may exacerbate this 'one-way-valve' effect.

Diagnosis

The classic signs of a tension pneumothorax are deviation of the trachea away from the side with the tension, a hyper-expanded chest, with an increased percussion note that moves little with respiration and a raised jugular venous pressure. The central venous pressure is usually raised, but will be normal or low in hypovolaemic states.

Trachea	→
Expansion	↓
Percussion Note	
Breath sounds	↓
Neck veins	↑

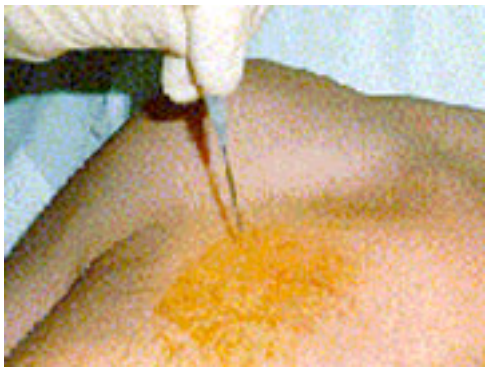
These classical signs are not always present. More commonly, the patient is tachycardic and tachypnoeic, and may be hypoxic. This can be followed by circulatory collapse and a pulseless electrical activity (PEA) cardiopulmonary arrest.

In patients receiving positive pressure ventilation, a tension pneuomothorax may develop insidiously. Positive pressure ventilation can worsen the “one-way” valve effect. An unexplained tachycardia, hypotension and rise in airway pressure are strongly suggestive of a developing tension pneumothorax in a ventilated patient.

Management

The classical management of a tension pneumothorax is emergency chest decompression with needle thoracostomy. A 14-16G intravenous cannula is inserted into the second rib space in the mid-clavicular line. The needle is advanced until air can be aspirated into a syringe connected to the needle. The needle is withdrawn and the cannula is left open to air.

An immediate rush of air out of the chest indicates the presence of a tension pneumothorax. The maneuver essentially converts a tension pneumothorax into a simple pneumothorax. This will then require further management with the insertion of a chest drain tube.



Needle thoracostomy is probably not a benign an intervention. There is a possibility of lung laceration with the needle and damage to other structures. In addition, needle thoracostomies are also prone to blockage, kinking, dislodgement and falling out. Be aware that a tension pneuomothorax may re-accumulate undetected if the needle shifts.

Chest tube placement is the definitive treatment of traumatic pneumothorax. The controlled placement of a chest tube is preferable to blind needle thoracostomy provided the patient's respiratory and haemodynamic status will tolerate the extra minutes it takes to insert the chest tube.

Massive Haemothorax:

The classic signs of a haemothorax are decreased chest expansion, dullness to percussion and reduced breath sounds in the affected hemithorax. There is no mediastinal or tracheal deviation unless there is a massive haemothorax.

Management:

Chest tube placement is the first step in the management of traumatic haemothorax. The majority of haemothoraces have already stopped bleeding and simple drainage is all that is required. All chest tubes placed for trauma should be of sufficient calibre to drain haemothoraces without clotting. Therefore, the smallest acceptable size of a chest drain for an adult patient is 32 F. 36F tubes are the preferred size if available.

Fluid resuscitation will be required if there is ongoing bleeding into the chest cavity to replace the losses.

The complications of chest drain insertion for a haemothorax include a retained haemothorax and infection with resultant empyema.

Flail Chest:

A flail chest, by definition, involves 3 or more consecutive rib fractures in 2 or more places, which produces a free-floating, unstable segment of chest wall. Separation of the bony ribs from their cartilaginous attachments, termed costo-chondral separation, can also cause flail chest.

Diagnosis:

Patients report pain at the fracture sites, pain upon inspiration, and dyspnoea. Physical examination reveals paradoxical motion of the flail segment with respiration. The chest wall moves inward with inspiration and outward with expiration. Tenderness at the fracture sites is present and there may be shortness of breath and a high respiratory rate and tachycardia. If a flail segment is present, there is a high incidence of associated thoracic injuries such as pulmonary contusions and closed head injury.

Management:

The management of chest wall injury is directed towards protecting the underlying lung and allowing adequate oxygenation, ventilation and pulmonary toilet. This strategy is aimed at preventing the development of pneumonia, which is the most common complication of chest wall injury. All patients should initially be placed on 100% oxygen via a non-rebreathing facemask. Further respiratory support with intubation and assisted ventilation is required for major chest wall injury.

Analgesia is the mainstay of therapy for rib fractures. There are several options including local anaesthesia (which provides the best analgesia), opioids and non-steroidal anti-inflammatory medications. The options for local anaesthetic block include intercostal nerve block, paraverterbral block and epidural block. Opioids are useful but should be used with caution because they will cause respiratory depression, especially in the elderly.

Intubation and ventilation are required if the patient is unable to maintain adequate spontaneous ventilation.

Open pneumothorax

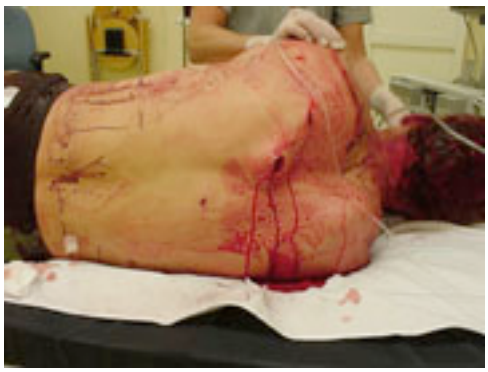
An open pneumothorax occurs when there is a pneumothorax associated with a chest wall defect.

The negative intra-thoracic pressure generated during inspiration results in air entering into the chest cavity through the hole in the chest wall rather than the trachea. This is because the chest wall defect is much shorter than the trachea, and hence provides less resistance to flow. Once the size of the hole is more than 0.75 times the diameter of the trachea, air preferentially enters through it into the thoracic cavity.

This results in inadequate oxygenation and ventilation, and a progressive build-up of air in the pleural space. The pneumothorax may create a tension (be under pressure) if a flap has been created in the chest wall that allows air in, but not out again.

Diagnosis:

A wound in the chest wall is identified that appears to be 'sucking air' into the chest and may be visibly bubbling. This is diagnostic. The patient’s breathing is rapid, shallow and laboured. There is reduced expansion of the hemithorax, accompanied by reduced breath sounds and an increased percussion note.



Management:

100% oxygen should be delivered via a facemask and intubation is performed when oxygenation or ventilation is inadequate. Intubation should not delay placement of a chest tube and closure of the wound.

Definitive management of the open chest wound involves the placement of an occlusive dressing and insertion of an intercostal chest tube.

Rarely, if a chest drain is not available and the patient is a long way from a definitive care facility, a bandage may be applied over the wound and taped on 3 sides. This, in theory, acts as a flap-valve to allow air to escape from the pneumothorax during expiration, but not to enter during inspiration. This dressing may be difficult to apply to a large wound and its effect is very variable. A chest drain should be placed and the wound closed as soon as possible.

References:

1. ATLS – Students Course Manual 7th edition
2. EMAC – Emergency Management of Anaesthetic Crisis Course notes
3. Trauma.org

HEAD INJURY

Traumatic brain injury is a leading cause of death from trauma. Patients with severe traumatic brain injury have a high mortality rate of 30-50% (7). Early death at the scene can result from the injury itself, apnoea and loss of airway patency. After resuscitation, death can result from cerebral oedema and loss of cerebral blood flow. (1) Many survivors will have persistent severe neurological disability.

Road safety and public health measures are important in the primary prevention of traumatic brain injury. These consist of the use of restraints in cars (seatbelts), legislation to prevent speeding and alcohol intoxication in drivers. Helmets are useful to prevent the occurrence of head injury and have become mandatory in many countries for motorcycle and bicycle riders.

Once the brain injury has occurred, treatment is supportive to prevent a secondary brain injury from hypoxia, hypoperfusion and cerebral oedema. Hypoxia and hypotension will increase the mortality after a head injury.

There are two potentially life-threatening conditions that need to be recognised and treated early. They are acute extradural haematoma and acute subdural haematoma.

A patient with an acute extradural haematoma typically has an initial loss of consciousness followed by a lucid interval (when they regain consciousness) and then rapid deterioration. The haematoma occurs secondary to bleeding from the middle meningeal artery. A rapid rise in intracranial pressure occurs and the patient usually develops hemiparesis on the side opposite the injury and a fixed pupil on the same side as the injury.

An acute subdural occurs due to tearing of a bridging vein between the cortex and dura mater. Blood accumulates in the subdural space and there is usually severe contusion of the underlying brain.

The management of acute extradural and acute subdural haematomata consists of urgent surgical decompression. This is typically done via a burr hole. This may need to be done in remote centres if transport to a larger neurosurgical centre will be delayed. A patient with acute intracranial hypertension may display signs of “coning”, which is characterised by downward displacement of the brain and the herniation of the uncus of the temporal lobe under the tentorium cerebelli with compression of the third cranial nerve and brainstem. (4) The clinical signs of severely elevated intracranial pressure are bradycardia, hyertension and decreased respiratory rate.

Assessing the severity of head injury

During the primary survey, a rapid assessment of conscious state is performed based on whether the patient is **A**lert; responding to **V**erbal stimuli, responding to **P**ainful stimuli or **U**nresponsive (AVPU). The pupillary response is checked.

The Glasgow coma score (GCS) is a more detailed neurologic evaluation and is used to classify the severity of the head injury. It was first published in 1973. It is usually performed during the secondary survey of the trauma patient.

	Best Response	**Score**
Eye opening	Spontaneous	4
	To voice	3
	To pain	2
	None	1
Best verbal response	Orientated	5
	Confused	4
	Inappropriate words	3
	Incomprehensible	2
	None	1
Best motor response	Obeying commands	6
	Localizing pain	5
	Withdrawal to pain	4
	Abnormal flexion	3
	Extension	2
	None	1

Reference: (3)

The GCS is used as a tool to identify how serious the head injury is. In conjunction with the papillary response and localizing signs, it can be used to identify those patients who need computerized tomography of the head and urgent transfer to a facility where neurosurgery can be performed to decompress a mass lesion (usually a haematoma).

GCS	**Loss of consciousness**	**Severity of injury**	**Action**
15	No	Minimal	Suitable for discharge in the care of a relative
14-15	Yes	Mild	CT brain if available, otherwise observe for at least 4 hours
9-13		Moderate	CT brain immediately and contact neurosurgeon with results
<9		Severe	Intubate and transport to a neurosurgical facility immediately

Reference: (1)

The other use for a GCS is to track any changes in the patient's condition. If there is a fall in the score of more than 2 points, this indicates a significant deterioration in the patient's condition.

The secondary survey should also include an inspection of the face and scalp checking for lacerations, cerebrospinal fluid (CSF) leakage from the nose or ears (occurs with a base of skull fracture) feeling for depressed skull fractures, and checking for underlying debris or brain tissue under lacerations. Bruising of the eyelids or over the mastoid process may indicate a base of skull fracture.

Alteration of the conscious state is the hallmark of brain injury and one should never assume that alcohol or drugs is the cause of drowsiness in a confused patient after a head injury. Sedation is avoided unless intubation is required because it interferes with the conscious state and will increase hypercarbia due to respiratory depression.

Physiology

The intracranial space is thought of as a non-compliant "box". This "box" contains the brain, which takes up 88% of the intracranial volume, blood in the vessels (2-3%) and cerebrospinal fluid (CSF) (9%). Most of the blood is in veins, but 15% is in the arteries and another 15% in the sinus system. (2)

The normal intracranial pressure (ICP) is less than 15 mmHg. Elevations of the ICP can result from changes in the volume of the brain (oedema, tumour), blood (vaso and veno-dilatation secondary to metabolic activity, bleeding) and the CSF (hydrocephalus secondary to obstruction to CSF flow, or reduced absorption of CSF). When gradual changes in the volume of one component occur, there is some degree of compensation by reduction in volume of the other contents of the skull to limit the rise in intracranial pressure.

The CSF volume is easiest to alter. An increase in intracranial pressure will cause the displacement of CSF into the spinal subarachnoid space through the foramen magnum and the arachnoid villi will absorb more CSF until the pressure reaches 30 mmHg.

Cerebral blood volume can be reduced by compression of the venous system, but further rises in ICP will cause the compression of capillaries that will lead to cerebral ischaemia and brain oedema.

The cerebral vasculature will respond to changes in pCO2. Hypocapnia will cause vasoconstriction, and hypercapnia will cause cerebral vasodilatation.

Because brain cellular function relies on adequate oxygenation and delivery of nutrients, cerebral perfusion is very important. The mean arterial pressure (MAP) and the intracranial pressure (ICP) determine the cerebral perfusion pressure (CPP). In order to maintain cerebral perfusion after injury, raised intracerebral pressure is reduced to less than 20 mmHg with the aim of maintaining a cerebral perfusion pressure of 60-70 mmHg. (2) Therefore, to maintain adequate brain function, it is

important to avoid a drop in mean arterial pressure (MAP) and to prevent hypoxaemia.

$$CPP = MAP - ICP$$

To correctly measure intracranial pressure, a dedicated ICP monitor is used. This is inserted by the neurosurgeons. There are several types of monitor. A ventricular catheter is ideal as it is accurate and allows for the therapeutic drainage of CSF in case the ICP rises.

Management considerations for traumatic brain injury

The initial primary injury to the brain includes contusion and axonal injury at the time of the injury. This is aggravated by secondary injury to the neurons. Subdural and extradural haematomata will cause compression of the brain and may accumulate after the primary injury. Secondary injury occurs due to ischaemia of the brain. This is chiefly due to hypoxia, hypotension and raised intracranial pressure.

Ischaemia occurs when there is an imbalance of cerebral blood flow and metabolism, disruption of cerebral autoregulation, loss of cerebral vascular reactivity to carbon dioxide and vasogenic fluid accumulation that leads to brain swelling. (6)

Pre hospital management of severe traumatic brain injury

KEY POINTS:
(adapted from Hammel CL and Henning JD ref. 7)

- Management of severe traumatic brain injury includes the rapid transfer to secondary care whilst preventing secondary brain injury.
- Airway compromise and inadequate ventilation are common and need to be addressed immediately.
- Pre hospital endotracheal intubation may be required. This needs to be done with due consideration of the fact that intracranial pressure can rise markedly with laryngoscopy and that hyperventilation may be detrimental. (8)
- Hypotension is an independent risk factor for mortality. It is reasonable to assume that it is due to hypovolaemia in the trauma patient and therefore it is treated with small boluses of isotonic crystalloid fluids.
- Patients are best managed in a centre where they can receive definitive neurosurgical treatment within 4 hours of injury.
- There is NO ROLE for the routine use of corticosteroids in patients with head injury.

Measures to reduce secondary brain injury (adapted from ref 1)

Providing adequate oxygenation and airway protection is important. Airway compromise after severe traumatic brain injury leads to cerebral hypoxia and increases mortality. Basic airway adjuncts and high flow oxygen are administered initially. Laryngeal masks can be useful where access to the patient is restricted and endotracheal intubation is difficult or not possible. (7) Early intubation is inevitably required for severe injury (GCS <9), but evidence for its benefit in the prehospital

environment is not strong. This is probably because of the fact that laryngoscopy without the use of anaesthetic agents causes high intracranial pressure and that hyperventilation is frequent during transport.

Hyperventilation and positive pressure ventilation will reduce cardiac output due to a reduction in venous return. This is especially true when there is hypovolaemia due to blood loss. Increased intrathoracic pressure with hyperventilation may also lead to a rise in intracranial pressure by increasing the jugular venous pressure, which is transmitted through the venous system into the skull. (8)

Pre-hospital rapid sequence induction is beneficial if done by an appropriately trained physician. (7) The indications for pre-hospital rapid sequence intubation are:

- Airway problems that cannot be managed with simple manoeuvres.
- Respiratory insufficiency (oxygen saturations <92%) despite high flow oxygen (15 l/min) or impending respiratory failure due to other injuries.
- GCS less than 9 or a rapidly falling GCS.
- Patients at risk of respiratory deterioration during transport (for example, those with facial burns)
- Patients needing sedation for transfer to hospital because they present a danger to themselves or for complete analgesia. (7)

Providing adequate cerebral perfusion involves the maintenance of an adequate blood pressure and avoiding severe cerebral vasoconstriction.

- Maintenance of blood pressure: (1)
    - An adequate mean arterial blood pressure is required to maintain cerebral perfusion. It needs to be supported by fluids and vasopressors, particularly if sedative agents are in use, as they tend to lower blood pressure. Arterial hypertension is undesirable as it will cause worsening of any cerebral oedema and aggravate raised intracranial pressure.
    - The ideal resuscitation fluid in not known. Glucose containing solutions are detrimental, as they will lead to worsening of cellular oedema and hyperglycaemia. The use of small boluses of isotonic crystalloids to correct hypotension seems reasonable.

Lowering the intracranial pressure after head injury is just as important. Monitoring of intracranial pressure is indicated in patients with severe head injury (GCS of 3-8). Treatment to lower ICP should be implemented when the ICP reaches an upper threshold of 20-25 mmHg. (6) The following measures can be used to lower ICP.

- Elevation of the patient's head:
    - This allows for drainage of CSF from the head into the spinal subarachnoid space and for adequate drainage of venous blood from the head.
- Sedation and analgesia:
    - Control of pain and agitation reduces catecholamine release and cerebral metabolism. Muscle relaxation is only required for intubation, during transport to hospital or for specific procedures. It is not recommended that they be used for prolonged periods in head injured patients because of the risk of permanent polyneuropathy.

    - Propofol is suitable for sedation of patients with elevated intracranial pressure. It has a rapid onset and short recovery time, even after prolonged infusion. This is useful because it allows for neurologic assessment when the infusion is ceased. It is also useful because it reduces cerebral metabolic demand and helps control intracranial hypertension. It may also have neuroprotective and anticonvulsant properties. (5) The main disadvantages of propofol are hypotension and the rare instance of propofol infusion syndrome after prolonged infusion. The propofol infusion syndrome causes lactic acidosis, high serum lipid levels, ECG abnormalities, cardiovascular collapse, rhabdomyolysis, hyperkalaemia, arrhythmias and renal failure. (5)
    - Ketamine increases cerebral blood flow without any effects on the cerebral metabolic rate in healthy volunteers. This has the potential to increase ICP. However, studies in ventilated patients show that ICP is not changed by ketamine. It is useful to control pain and will help to maintain blood pressure. The use of ketamine can be justified in head injured patients as long as the ventilation is controlled. (2)
- Normothermia:
    - Therapeutic hypothermia is thought to reduce the cerebral metabolic rate and therefore protect the brain by decreasing intracranial blood flow and lowering the intracranial pressure. Hypothermia also decreases free radical production, reduces brain oedema, lower intracellular calcium concentration, suppresses nitric oxide production, increases gamma-amino butyric acid release and blunts the release of glutamate. It has been proposed as a treatment for severe traumatic brain injury, but the most recent trials have NOT shown a benefit of lowering the patient's temperature and currently, there is not enough evidence to support the routine use of hypothermia for traumatic brain injury. (6) Hyperthermia is detrimental but evidence for the use of antipyretics for a fever is not strong. The current recommendation is to keep the patient's temperature normal after traumatic brain injury and not to actively re-warm a patient with severe traumatic brain injury who is already cold on presentation to hospital. Passive re-warming of a cold patient is probably safer. Hypothermia is a key factor in the development of traumatic coagulopathy in the patients with multiple injuries. (7)
- Normocapnia:
    - Both high and low arterial carbon dioxide levels are detrimental in the setting of traumatic brain injury. Hypocapnia ($pCO_2 < 30$ mmHg) and hypercapnia ($pCO_2 > 45$ mmHg) have been associated with increased mortality and less favourable outcomes. (8)
    - Hyperventilation leads to cerebral vasoconstriction and ischaemia and should be avoided in patients with head injury. Hypercapnia on the other hand will lead to cerebral vasodilatation, increased cerebral blood flow and a rise in intracranial pressure. (7)
    - Hyperventilation reduces intracranial pressure and is useful in the short term to treat rapid elevations in ICP and prevent herniation. It is not recommended that hypocapnia be induced routinely as it will reduce cerebral blood flow and cause ischaemia.

    - In the multiple trauma patients with chest as well as head injury who need ventilation, it is safe to use modest degrees of positive end expiratory pressure (PEEP) to improve oxygenation. In these situations, it is wise to keep PEEP at levels below 10-15 cmH2O due to the risk of increasing intracranial pressure. (2)
- Osmotic diuresis:
    - Intermittent boluses of mannitol or hypertonic saline can reduce ICP but may cause fluid overload. The dose of mannitol is 0.25 to 0.5 g/kg but an initial dose of 1.4 g/kg has been reported to improve the 6 - month clinical outcomes after head injury. (2) Mannitol acutely expands the plasma volume and reduces haematocrit and blood viscosity. This will improve cerebral blood flow and cerebral oxygen delivery. The osmotic effect removes water from the brain. The onset of the osmotic effect of mannitol is 15-20 minutes and the duration of the effect is 1-6 hours. It is recommended that it be administered as boluses rather than via an infusion due to the risk of rebound raised intracranial pressure. Mannitol is excreted in the urine and there is a risk of acute tubular necrosis if the serum osmolarity rises above 320 mOsmol/l. Frusemide can be administered as a supplemental treatment to reduce brain water even further. (2)
- Drainage of cerebrospinal fluid:
    - Drainage of CSF is achieved with the use of an intraventricular catheter. This catheter is also capable of serving as an ICP monitor. It is placed by the neurosurgeon and drainage of CSF is done in a sterile system to avoid meningitis.
- Induced coma:
    - A deep level of anaesthesia can be induced with barbiturates or propofol to reduce the cerebral metabolic rate. Unfortunately, this may induce hypotension. The patient may require vasopressors to support the blood pressure and there is a small risk of fatal rhabdomyolysis associated with the prolonged use of propofol infusion in head injured patients.
- Decompressive craniotomy:
    - In selected patients, the removal of part of the skull allows for the brain to swell without raising ICP. This therapy is not well established yet.

Monitoring of patients with head injury in the pre-hospital environment

- Patients with severe traumatic brain injury need to be monitored for hypoxaemia (arterial desaturation <90%) and hypotension (systolic blood pressure <90 mmHg).
- Continuous pulse oximetry is ideal
- Blood pressure should be measured with the most accurate method available.
- End-tidal carbon dioxide monitoring is recommended for the transfer of ventilated patients (7)

Spinal cord injury

Head injury is a strong risk factor for the presence of a cervical spine injury. It is important to assume that spinal injury is present until it can be excluded with X-Rays

or CT imaging. In the field, a hard collar is placed to keep the spine aligned and avoid movement, which may lead to a spinal cord injury. Because hard collars can impede venous return from the head, it is a priority to exclude a cervical spine injury as soon as practical so that the collar may be removed. The alternative is to immobilize the head with tape and sandbags in the sedated and intubated patients. (7)

Hospital management of the head injured patient

It is recommended that the patient with a GCS less than 9 be transported to a trauma centre with the capacity to perform an immediate CT scan, provide neurosurgical care, have the ability to provide intensive care support and monitoring of intracranial pressure. (9) The receiving hospital needs receive information about the patient arriving with severe head injury so that the CT scanner can be made ready and a neurosurgeon can be contacted.

Intubation and controlled ventilation should be instituted either in the field or within one hour of arrival to hospital for patients with a GCS of less than 9. In-line cervical spine stabilization needs to be maintained during intubation attempts. Ventilation should be guided by end-tidal carbon dioxide monitoring to achieve normocapnia.

During the initial treatment of brain injury, it is important to avoid hypotension. The majority of patients will require a systolic blood pressure close to 120 mmHg and certainly over 90 mmHg. Normal saline is the fluid of choice in the emergency room unless there has been significant blood loss, when blood products should be used. If the patient shows signs of a high ICP, mannitol or hypertonic saline can be used.

Monitoring should be frequent with an assessment of the GCS (or similar) evidence of lateralizing signs (such as unilateral weakness) and pupil size. Changes need to be communicated urgently to the neurosurgeon. ICP monitoring is ideal, but may not always be available, as it requires neurosurgical expertise to place. It is recommended that the intracranial pressure be kept below 20 mmHg. The ideal ICP is not known, but at a pressure less than 7 mmHg, brain herniation is rare, whereas an ICP over 10 mmHg is sometimes associated with cerebral swelling and herniation. (9) The clinical signs of brain herniation are dilated, asymmetrical or unreactive pupils, extensor motor response in the limbs and/or a decrease in the GCS score of more than 2 points when the previous GCS was less than 9.

Convulsions need to be treated urgently, as they will increase intracranial pressure and increase the metabolic demand of the brain.

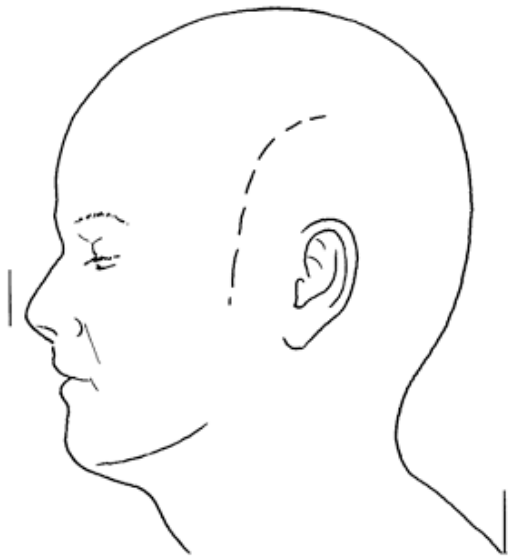
In patients with severe brain injury, it is recommended to monitor intracranial pressure.

Extradural haematomas over 30 ml should be evacuated immediately. Acute subdural haematomas should go directly to surgery if the clot is thicker than 10 mm and/or there is midline shift of over 5 mm on the CT scan or if there are clinical signs such as a fall in GCS of more than 2 points, asymmetric or fixed dilated pupils and/or an intracranial pressure of over 20 mmHg. Intracerebral lesions over 50 ml in size should be evacuated and smaller lesions may need an operation if there is deterioration in the patient's condition. (9)

Anaesthesia for a brain-injured patient may be required for surgery on the brain or to correct other injuries. Nitrous oxide can stimulate cerebral metabolism and is best avoided. Total intravenous anaesthesia is recommended. Thiopentone or propofol are suitable agents. Opioids should be use cautiously in the spontaneously breathing patients as they will cause hypoventilation. They are very useful to blunt the neuroendocrine response to intubation, but they can lower the blood pressure significantly. If they are used, it is important to treat hypotension. Muscle relaxants are recommended for tracheal intubation and can be useful for transport to avoid coughing (which raises the ICP). Ketamine can cause cerebral vasodilatation and had previously been avoided for neurosurgery, however, it is probably safe to use in the absence of an alternative agent if the patient's ventilation is controlled to achieve normocapnia.

Appendix:
Performing a burr hole (10)

Shave and prepare the skull over the temporal region between the ear and orbit on the side of the injury. Infiltrate the scalp with local anaesthetic and make a 3 cm incision through skin and temporal fascia. Separate the temporalis muscle and incise the cranial periosteum. Use retractors or cautery to control bleeding. Make the burr hole 2cm above and behind the orbital process of the frontal bone. Make a hole through the inner table of bone with a drill cutter using very little pressure. Using cautery or a ligature, control bleeding from the anterior branch of the middle meningeal artery. Control venous bleeding with a sponge. Wash out the haematoma with a syringe. Close the scalp in two layers.



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SPINAL CORD INJURY

In the absence of a depressed level of consciousness, paraplegia or quadriplegia indicates spinal cord injury.

The likelihood of a vertebral injury in unconscious trauma victims is 10% and 2% of trauma victims with altered conscious state have a spinal cord (neurologic injury). (1)

Neurological Assessment

The elements of the neurologic examination are frequently completed during the regional portions of the secondary survey; however, include a formal assessment of the spine to complete the neurologic assessment. (2)

Log roll the patient with inline stabilization of the head and neck.
Inspect the entire spine from the cranium to the sacrum for bony abnormalities, deformities, and tenderness. A detailed survey of the back to identify penetrating injuries, bruising, or other injuries is performed. Back injuries are frequently missed. (2)

The patient with a head injury should be considered to have a cervical spine injury until proven otherwise. There is a significant incidence of spinal cord injury that develops and/or progresses after a patient has presented to hospital after spinal trauma. This may be due to the fact that the injury was not recognized initially, the secondary effects of the spinal cord injury (oedema and ischaemia of the spinal cord), aggravation of the initial injury by hypoxaemia or hypotension or inadequate vertebral immobilization. Care needs to be taken during procedures and patient transfers to avoid conversion of patient with bony spinal injury with no neurologic injury into a patient with a spinal cord injury with significant paraplegia or quadriplegia.

The level of the spinal cord injury is related to the level of the spinal column injury. Most injuries (>70% in adults and >60% in children) (3) occur at the level of the 4^{th}, 5^{th} and 6^{th} cervical vertebrae, where the spine is most mobile. Approximately 8-10% of patients with a vertebral fracture have a secondary fracture at another level. Many patients with spinal injury have associated injuries such as head, thoracic and abdominal injuries. Pain from other injuries in a multiply injured patient may divert attention from the spine and cause the patient and those treating him to miss a spinal injury. (1)

Assessment of the level of injury needs to be performed. The following is a guide to determining the level of the spinal injury: (from PTC)
Motor Response:

- Diaphragm C3, C4, C5
- Shrug shoulders C4
- Elbow flexion C5
- Wrist extension C6
- Elbow extension C7
- Wrist flexion C7
- Finger flexion C8
- Finger abduction T1

- Chest Expansion T1-T12
- Hip Flexion L2
- Knee Extension L3-4
- Ankle dorsiflexion L4
- Ankle plantarflexion S1-S2

Sensory Response:

- Anterior thigh L2
- Anterior knee L3
- Anterolateral ankle L4
- Dorsum of great toe and 2nd toe L5
- Lateral aspect of foot S1
- Posterior calf S2
- Peri-anal sensation S2-5

Loss of autonomic function causes hypotension, bradycardia and loss of temperature control. There may be urinary retention and delayed gastric emptying. Urinary catheterization and a nasogastric tube are indicated. Care must be taken when a gastric tube is placed if there is the possibility of a base of skull fracture as the nasogastric tube can enter the cranial vault.

A thoracic spinal injury may cause diaphragmatic breathing, which manifests as paradoxical abdominal and chest wall movement. The patient may develop respiratory failure and require ventilation.

Spinal reflexes may be flaccid or absent.

The unconscious victim is more challenging to examine but there are some signs that indicate a spinal cord injury. They include:

- Paradoxical breathing or chest wall movement
- Priapism (persistent male erection)
- Preserved facial grimace in the absence of response to painful stimulus in the limbs
- Lower limb flaccidity in the presence of normal upper limb tone
- Upper limb movement without lower limb movement
- Persistent bradycardia and hypotension despite volume challenge
- Flaccid rectal sphincter (1)

Radiologic evaluation

When can we be confident that the cervical spine is safe?
X Rays are indicated if any of the following are present:

1. Neck pain
2. Loss of consciousness at the scene of the injury
3. A neurologic abnormality or symptoms, including a head injury.
4. Intoxication
5. Severe pain in another site that distracts the patient from his neck. For example severe pain in a broken leg. (4)

If there is an urgent need to exclude cervical spine injury, perform a portable lateral

cervical spine (C- spine) film during the resuscitation phase. An adequate lateral C-spine x-ray (visualizing from the skull base to T1) helps identify most C-spine fractures and subluxations. Ultimately, a full C-spine series (Antero-Posterior, lateral, and odontoid views) must be performed to exclude injury, and many clinicians will request a CT if any doubt exists. (2)

Formal x-rays include a lateral film (NO FLEXION OR EXTENSION VIEWS), an Antero-posterior view and an open mouth or odontoid view if possible. CT scanning is required if there is still a strong suspicion of spinal injury. (4)

CT scanning is replacing plain radiographs in many patients being evaluated for spine trauma. Current scanners offer the capability to reconstruct spine images at the same time that scans are obtained of the chest, abdomen, and pelvis. Many clinicians will scan the cervical spine in patients with other indications for scans of the head or the torso.

Obtain plain x-ray films of the spine in patients with high-energy blunt trauma or those with suspected cervical spine injury. In some patients, x-rays of the spine may need to be deferred in order to complete resuscitation or live-saving surgery. In these patients, it is wise to protect the spine with immobilization until formal x-rays are available.

Management

If the possibility of a spinal cord injury exists, full spinal precautions are required. The administration of intravenous corticosteroids within 8 hours of injury can be considered but remains a controversial treatment.

As with head trauma, oxygenation and adequate perfusion are important to avoid extension of the neurologic injury.

Spinal cord oedema and haemorrhage tend to resolve within 10-14 days after the primary injury and there may be some improvement of spinal cord function that may result in local segmental recovery. (1)

Immobilization techniques include a cervical collar; sandbags and tape, inline cervical immobilization, a spine board or Jordan frame and log roll for moving the patient.
Full clinical assessment should follow initial stabilization. Including palpation of the entire spine, the bladder (catheterization may be required) and signs of spinal shock (hypotension, bradycardia). Beware of overloading patient with fluid.

If inspiratory efforts are weak or when a high cervical cord lesion is suspected, perform an endotracheal intubation with inline stabilization.

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BURNS MANAGEMENT

The incidence of burns in Australia is similar to that of other western countries, and is approximately 1% per head of population per year. In the adult population, burns due to explosions or flames are most common, followed by scalds, and then contact burns. In children, scalds are the most common burn injury. Approximately 10% of burns will require hospitalisation, and 10% of these will be classed as severe burns.

First Aid in Burns

The first step is to stop the burning process. This process depends on the type of burn. Always consider your own safety, and avoid harm that would impede further assistance to the victim.

If on **FIRE**:

Stop-Drop-Roll

Clothes should be extinguished by smothering that is, with a heavy woollen blanket.

Do not beat at the flames as this will cause 'fanning' and the flames will continue.

Remember that flames travel upwards.

If **ELECTRICAL**:

Turn off any current.

If **CHEMICAL**:

Remove the burning agent and irrigate with water.

Once the burning process has been halted, remove clothing, jewellery and other items from the burn area that may continue the injury process. Any part of the clothing not stuck to the burn site can retain heat and increase the severity of the injury. Clothing can retain chemical contaminants and continue burning. In children, always remove nappies or plastic pants, as hot liquid can accumulate and remain there. Remove all jewellery and watches, as they may retain heat and act as a tourniquet.

Burns should be cooled as rapidly as possible, to limit the burn injury and to provide some pain relief. Use running cold tap water for 20 minutes. This is useful for up to 3 hours after the burn. Beware of causing hypothermia, especially in children. Do not use ice, as there is significant risk of both hypothermia and cold injury. If a large proportion of the body is burnt, immerse the patient in a tepid bath. Do not leave the patient unattended, and do not leave the patient in the bath for more than a few minutes.

Once the burning process has stopped and the burn has been cooled, the area should be dressed to minimise contamination of the wound, and reduce the impact of fluid and heat loss. Where possible use a clean dressing or plastic cling film (such as that used to cover food stuffs) applied directly to the burn wound. Clean sheets or other lint-free material can also be used to cover burns. Cover the non-burnt areas with blankets and keep the patient warm. Children can become hypothermic quickly, and many patients will develop a degree of shock requiring care.

Specific burns types require specific first aid management.

Electrical Burns

Ensure that any current has been disconnected, and that it is safe to approach the patient.
Remove the patient from danger.
Monitor the patient's Airway, Breathing and Circulation (ABC).
Electrical injury, especially high voltage, is likely to cause serious internal injuries that may not be immediately visible:
Look for both an ENTRY and EXIT point.

- Large amounts of tissue, substructures and organs beneath the skin can be injured.
- Myocardial damage may result in arrhythmias, both immediate & late, and require ECG monitoring for 24 hours after injury

Chemical Burns

Chemical burns are very unpredictable and can be very deep. Tissue destruction will continue as long as the chemical agent remains in contact with the skin or membrane.
Tissue damage from chemical burns depends on:

- The strength or concentration of the agent;
- The site of contact (eye, skin, mucous membrane);
- Whether swallowed or inhaled;
- Amount of the agent the patient came into contact with;
- The duration of exposure;
- How the chemical works.

Some of the common chemical agents that result in burns:
<u>Alkalis</u>

- Lime, sodium hypochlorite
- May be present in household cleaning agents (disinfectants, bleaches), cement

<u>Acids</u>

- Hydrofluouric acid, sulphuric acid (car battery, toilet bowl cleaners) and hydrochloric acid

<u>Phosphorus</u>

- Found in fireworks, fertilisers and military supplies

If the burns are external, wash the area with cold water for at least 10 - 30 minutes. This will dilute the effects of the chemical on the skin. If the eye is affected, flush the eye continuously until medical review.

If taken internally, burns will be apparent on the lips and inside the mouth. Determine the type of chemical ingested, and refer to specific instructions about management. These instructions may appear on the container or packaging. Closely monitor airway status, and consider intubating the patient early before airway oedema and trauma makes intubation difficult.

Depth of Burn

Epidermal e.g. sunburn
The skin appears red and painful, and there will be no blisters. Healing occurs in less than 1 week, and will not result in scarring.

Superficial Dermal
The skin will appear red and painful, and blisters will form. Capillary refill remains present. Healing is slower, occurring over a few weeks, and may result in some scarring.

Deep Dermal
The skin will be dark red and not painful. Blisters may be seen. There will be no capillary refill. There will be minimal or no healing, and scarring will occur.

Full Thickness
A white, waxy, charred appearance to the skin, with no sensation. There will be no blisters, no capillary refill, and no healing.

Electrical Burns
There will often be an entry and exit point, with an underlying path of tissue destruction caused by the electrical current. High resistance tissues, such as bone, produce excessive heat, which will burn deep muscle and nerves adjacent to bone. Muscle burns require more fluid resuscitation because of the risk of myoglobinuria and renal failure, and are also more likely to require a fasciotomy.

Anatomy of a Burn

The classic description of the burn wound and surrounding tissues is a system of several circumferential zones radiating from primarily burned tissues:

Zone of coagulation
A nonviable area of tissue at the epicenter of the burn.

Zone of ischaemia or stasis
Surrounding tissues (both deep and peripheral) to the coagulated areas, which are not devitalized initially but, due to microvascular insult, can progress irreversibly to necrosis over several days if not resuscitated properly.

Zone of hyperaemia
Peripheral tissues that undergo vasodilatory changes due to neighbouring inflammatory mediator release but are not injured thermally and remain viable.

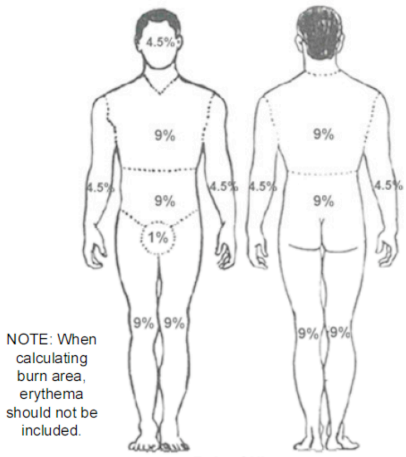
Assessment of Burns

A burn is a dynamic injury that changes with time and management. Good first aid and appropriate early fluid resuscitation may limit the size of the burn and improve outcome. Appropriate resuscitation and management requires assessment of the burn,

which requires measurement of the size and the depth of the burn. As with all trauma, appreciate the presence of other injuries that may affect patient management.

The Rule of Nines
The percent of a burned adult body can be calculated as:

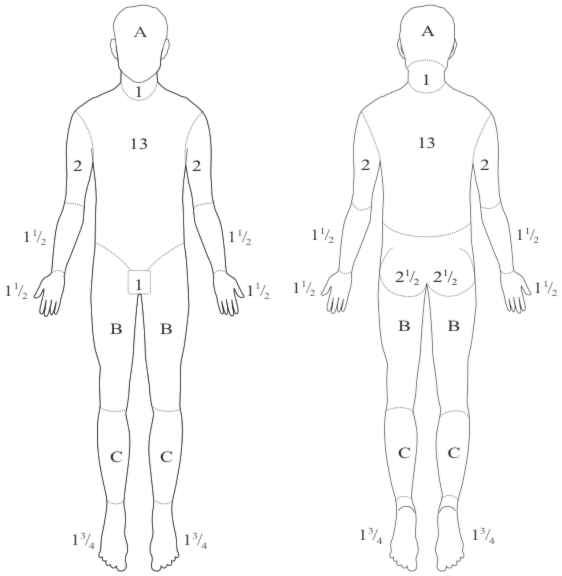
Head (face and scalp)	9%
Chest (front)	9%
Abdomen (front)	9%
Upper/mid/low back and buttocks	18%
Each arm	9%
Each palm	1%
Groin	1%
Each leg	18%



Rule of Nines
Adult Burn Injuries *

Note that the palm of the hand is approximately 1% of the Total Body Surface Area (TBSA), and provides a ready guide for estimating the size of smaller burns.

Lund & Browder Chart
The 'Rule of 9s' is inaccurate for children because of the varying rates of growth in the head, thigh and lower leg of different age groups. The Lund & Browder chart allows for these differences, to provide more accurate assessment of the burn size.



Region	Partial thickness (%) [NB1]	Full thickness (%)
head		
neck		
anterior trunk		
posterior trunk		
right arm		
left arm		
buttocks		
genitalia		
right leg		
left leg		
Total burn		
NB1: Do not include erythema		

Area	Age 0	1	5	10	15	Adult
A = half of head	9½	8½	6½	5½	4½	3½
B = half of one thigh	2¾	3¼	4	4½	4½	4¾
C = half of one lower leg	2½	2½	2¾	3	3¼	3½

The accurate assessment of the burn injury enables appropriate management of the burn to optimise outcome.

Management of a Burn Injury

The Australia & New Zealand Burns Association criteria (2004) for admission to a specialist hospital burns unit for management are:

- Burns greater than 10% of Total Body Surface Area (TBSA)
- Burns of special areas - face, hands, feet, genitalia, perineum, and major joints
- Full-thickness burns greater than 5% of TBSA
- Electrical and chemical burns
- Burns with an associated inhalation injury
- Circumferential burns of the limbs or chest
- Burns in the very young or very old
- Burns in people with pre-existing medical disorders that could complicate management, prolong recovery, or increase mortality
- Burns with associated trauma

Accurate assessment of the burn patient using the Lund & Browder chart will determine if the patient requires transfer to hospital for further management. As with all trauma patients, examine for the presence of other injuries.

Early management during transfer of the burn patient will improve outcome. Keep the patient warm, particularly if the patient is small or paediatric, and be aware that the patient may already by hypothermic from first aid management. Observe vital signs and manage Airway, Breathing and Circulation as appropriate. The establishment of intravenous access is imperative, as it allows the commencement of fluid resuscitation (which may reduce the size of burn), and the administration of appropriate analgesia. Intravenous fluids are mandatory for burns greater than 10% TBSA. Patients with facial or inhalational burns are best managed sitting upright if possible, with supplemental oxygen. Consider the tetanus status of the patient, and administer tetanus toxoid where appropriate.

Fluid Resuscitation
Fluid resuscitation is life saving and can limit the size of the developing injury. Burns deaths were commonly due to hypovolaemia and renal failure, but have decreased in incidence with appropriate fluid management. The most commonly used guide to fluid resuscitation is the Parkland Formula (Baxter, 1971). This regimen is suitable for burns larger that 20% BSA (Body Surface Area) in adults, and 10% BSA in children, and uses Hartmann's Solution or Lactated Ringer's Solution. Be aware that it is not for superficial burns

Parkland Formula
Total fluid requirement in 24 hours
= 4 ml x (total burn surface area (%)) x (body weight (kg))
50% of the fluid is given in first 8 hours
50% of the fluid is given in next 16 hours

Children also receive maintenance fluid:
4 ml/kg/hr for first 10 kg of body weight plus
2 ml/kg/hr for next 10 kg of body weight plus
1 ml/kg/hr for every next kg of body weight

Resuscitation is only a guide, and requires monitoring and titration. A urine output 0.5-1.0 mls/kg/hr in adults, or 1.0-1.5 ml/kg/hour in children, indicates adequate fluid administration. Accurate measurement is achieved with an indwelling urinary catheter, which is mandatory if the burn is greater than 15-20% BSA. More aggressive fluid administration may be indicated in electrical burns (where the small % BSA burn does not reflect the deep tissue damage), inhalational injury, or where there is delayed resuscitation. In these instances, Baxter also gave plasma 24-36 hours after the burn injury, at the rate of 0.4 ml/kg/% BSA.

Excessive Fluid Resuscitation
The importance of monitoring and titration of fluid resuscitation cannot be overstated. While the administration of fluid parallels the survival after a major burn, it is also possible to fluid overload the patient. The problems with excessive fluid include:

- Abdominal compartment syndrome, resulting in ischaemia to abdominal and retroperitoneal organs
- Limb compartment syndrome
- Orbital compartment syndrome
- Impaired wound healing and graft take

Acute Burns and the Airway

Where burns involve any part of the airway, from lips to lower respiratory tract, management is critical and interventions must be performed early. Singed hairs around the mouth or nose, or carbonaceous sputum may indicate the presence of an airway burn. If the burn has occurred within a closed space, such as a small room, higher temperatures are reached and an inhalational injury is more likely. Direct visualisation of the airway is useful in the primary care setting, looking for the evidence of burns to tissues in the pharynx and upper respiratory tract. Evaluation of blood gases, chest x-ray and pulse oximetry will provide an insight to the degree of lower respiratory tract involvement. Any airway burn should be intubated early, prior to the onset of oedema that may make intubation difficult or impossible. Facial burns will make mask oxygenation difficult, and subsequent oedema may make ventilation difficult.

Also consider the need for intubation in those with large burns. A large volume of fluid resuscitation may contribute to generalised oedema, and may affect the airway. If there is a need to transfer the patient, be aware of the time required to complete the transfer. It may be safer to intubate the patient early. Suxamethonium is safe to use in the acute setting, and usual endotracheal tube sizings (not larger) are recommended.

Burn Surgery

Burn surgery occurs in two phases:
Acute Surgery

- Escharotomy
- Excision of dead tissue and surface coverage with grafting

Later Surgery

- Cosmetic or functional for scars and contractures

Burn Escharotomy
Burnt dermis, such as deep dermal and full thickness burns, forms a non-elastic eschar. The eschar will inhibit underlying tissue expansion, causing different problems in different anatomical areas. The most severe eschars occur with circumferential burns, where underlying tissue oedema will cause an increase in tissue pressure beneath the non-elastic burn. In limbs, this can result in circulatory loss and ischaemia distal to the burn. Circumferential abdominal burns may result in an abdominal compartment syndrome, with ischaemia of peritoneal and retroperitoneal structures (gut, kidneys). A circumferential burn of the chest may produce an eschar that impedes ventilation, and can result in respiratory failure.

Treatment of an eschar requires surgical incision down to viable tissue, allowing the underlying tissues to "spring back", relieving the tissue pressure. The incision needs to be extended into non-burnt tissue proximally and distally. Eschars are insensate, but the surrounding viable tissues retain sensation. Escaharotomy can be done with local anaesthetic infiltration to the proximal and distal tissue incisions, with or without sedation. The procedure is best done in the operating theatre with diathermy to minimise blood loss. At an appropriate time, the eschar will need excision and grafting, as it is non-viable non-healing tissue.

Debridement of burns and skin coverage
Burn debridement removes contamination and non-healing tissues from a burn, leaving a raw surface that requires skin coverage, to prevent infection and fluid losses. Skin from unburnt areas of the patient is utilised. The most common method is split-skin grafting, where fine slices of skin are shaved from the patient. These slices contain dermal material for cell growth in the transplant, but leave some dermal material in the donor site that will regenerate. Making small incisions in the slice, which is called meshing, stretches the donor skin. The meshed skin is placed over the debrided burn, which must provide an adequate blood supply for the donor skin to grow. It is not suitable for areas of bone or cartilage, due to the poor vascularity of these tissues. These areas may require more extensive surgery, such as a muscle flap. Use of inotropes perioperatively may result in poor tissue perfusion and poor graft take. Beware of large volume fluid resuscitation and the development of oedema, which may collect beneath the graft and prevent vascularisation of the graft.

Burns Anaesthesia

Airway
A difficult airway can occur at all stages of burn management. Facial burns may make mask anaesthesia or preoxygenation painful and difficult. Airway oedema, due to airway burns or fluid resuscitation may make intubation difficult, and extubation may need to be delayed. Later scar contractures can make for a difficult airway and intubation. Release of contractures around the head, neck and face, under local anaesthetic is sometimes required prior to induction of anaesthesia, to improve airway conditions.

Intravenous Access
The nature and extent of a burn may make intravenous (IV) access and monitoring difficult. Adequate IV access will be dictated by the size of burn and amount of fluid

resuscitation required, as well as the anticipated surgical blood loss. The use of sheath introducers or rapid infusion catheters will facilitate rapid blood replacement. Femoral vein catheters or surgical cut-downs may be required.

All fluids should be given via fluid warmers. Rapid infusion devices that also provide warming are ideal for these patients.

Use of Muscle Relaxants
Suxamethonium can be safely used for intubating a burns patient within the first few days of injury. It is not safe to use after this time. Following a severe burn, there is an increase in the expression of extrajunctional acetylcholine receptors. The receptor numbers are markedly increased by 7-10 days after a burn. Life-threatening hyperkalaemia and cardiac arrest can occur when a depolarizing muscle relaxant, such as suxamethonium, is used. Most cardiac arrests are documented from 10 days after a burn. The safe time to use suxamethonium again is unclear, but it is suggested that waiting until the last graft has healed may be appropriate.

The increase in extrajunctional acetylcholine receptors is thought to be the cause of resistance to non-depolarizing muscle relaxants. Burns patients may require larger or more frequent dosing. The use of a neuromuscular monitor is recommended to optimise relaxation and recovery when muscle relaxants are to be used.

Blood Loss
Most burns surgery produces massive rapid blood loss. This occurs during debridement of burnt areas and from donor sites. Blood loss in excess of 2500 ml within 45 - 60 minutes is not uncommon. The amount of loss is difficult to predict, and is very often difficult to estimate during surgery. Expect a loss of one unit of packed Red Blood Cells (RBC) for every 3% BSA burnt. Full thickness burns need complete excision to viable underlying tissues, and will bleed more. Older burns (> 3-4 days) will have more revascularization, and result in more blood loss. Head, neck and trunk surgery will often result in a greater blood loss. Use of tourniquets on limbs will reduce blood loss.

Standard massive transfusion principles apply. Along with an estimation of red blood cells (RBCs) required, use more Fresh Frozen Plasma (FFP), cryoprecipitate and platelets. Use less crystalloid to avoid excessive oedema. Always maintain a good relationship with the blood bank – they will help where possible!

Temperature Control
It is difficult to prevent hypothermia during surgery. There is limited opportunity to cover the patient, there may be insufficient area available to use forced warm air blankets, and large fluid volumes are often transfused quickly. The result may be difficulty in waking and extubating the patient, and the development of a coagulopathy. Hypothermia may be the limiting factor to terminate surgery.

Measures taken to avoid hypothermia include maintaining a hot (> 28^{o} C) and humid theatre to conserve body heat, and the use of IV fluid warmers and warmed fluids where possible. Quick surgery is good surgery, with haemostasis a priority. Ensure the patient is covered as much as possible, and that the patient is warmed prior to theatre.

Analgesia
Most burns will cause initial acute pain, with severe burns also causing recurrent post-operative pain from debridement, grafting and dressing. Particularly painful sites include donor sites and the hands. Opiate requirements are large, and development of opioid tolerance often occurs.

Multi-modal analgesia is used when possible. The use of paracetamol is appropriate for almost all patients. Consider the use of a COX-2 selective Non-Steroidal Anti-Inflammatory Drug (NSAID), such as Parecoxib (there is less platelet inhibition than a non-selective NSAID). The use of Tramadol, Ketamine and Clonidine may be appropriate both intra-operatively and post-operatively.

Local anaesthetic blocks are especially useful. Easy blocks that cover large surfaces include wrist blocks, lateral cutaneous nerve of thigh block and fascia illiaca or femoral nerve block. Local infiltration of other donor sites is often easy. The use of long-acting local anaesthetic agents can provide excellent analgesia for some hours post-operatively.

Analgesia for burns dressings
Burns dressings are painful, unpleasant, and often dreaded by the patient. The use of Patient Controlled Analgesia (PCA), using midazolam and ketamine, has improved this situation. Midazolam 0.5 mg and Ketamine 10 mg are provided together as a bolus with a 3 minute lock-out period. Supplemental analgesia can be provided with inhaled Entonox (N_2O & O_2), and the use of oxycodone as a premed.

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ENVENOMATION

Australia is ill-fated in having many species of venomous creatures, both on land and in the sea, including: 38 terrestrial snakes and 23 sea snakes, 22 spiders, 4 ants, the honey bee, 3 wasps, 2 beetles, 6 scorpions, 2 caterpillars, centipedes, millipedes, mosquitoes, sandflies, and other insects as well as the platypus and the echidna.

In the coastal waters there are 2 blue-ringed octopuses, 7 jellyfish, cone shells, 23 fish species, 11 rays, starfish, corals, anemones, urchins, stinging sponges, marine worms, leeches, frogs and toads.

Terrestrial snakes

Australia is home to the ten most lethal snakes in the world.

The major Australian snakes are the brown snakes, the tiger snake group, mulga/black snakes, taipans and death adders. Snake envenoming is uncommon but potentially life-threatening. Estimates suggest that there are between 500 and 3000 snakebites annually. In about 200 to 500 cases antivenom is required. Envenomation is characterised by systemic effects including coagulopathy, neurotoxicity, myotoxicity and renal impairment. Between one and four deaths occur each year with most resulting from brown-snake bites.

Pressure immobilisation bandaging is safe and appears to be effective first aid if applied correctly soon after the bite.

Snake venom detection kits are available to help identify the causative snake. Antivenoms are available for the five major groups of snakes and are the mainstay of therapy in patients with systemic envenoming. Anti-venom should be administered by slow intravenous infusion in a critical care area. Serious adverse reactions to anti-venoms are uncommon.

Clinical effects:

Many types of venom have multiple components or toxins that lead to a complicated clinical picture.

Often the snakebite site is painless. It may have classical paired fang marks, but this is not the most common picture. Usually there are just a few lacerations or scratches, and sometimes these may be painless or go unnoticed. Bruising, bleeding, and local swelling may be present, but significant local tissue destruction is uncommon in Australia.

With many snakebites the only effects observed are local effects because insufficient venom is injected or the snake is non-venomous. Regional lymphadenopathy may be marked, even with non-venomous snakebites. With more significant envenomation there may be local or systemic effects. These range from non-specific effects (nausea, vomiting, headache, abdominal pain, diarrhoea, dizziness and collapse) to major organ effects (coagulopathy, neurotoxicity, rhabdomyolysis or renal damage).

Coagulopathy

The majority of dangerous Australian snakes cause a procoagulant coagulopathy. The venom contains a prothrombin activator that leads to consumption of major coagulation factors including fibrinogen, resulting in a coagulopathy characterised by low fibrin levels. This is referred to as a venom-induced consumptive coagulopathy. This is characterised by very high d-dimers, undetectable fibrinogen, and unrecordable prothrombin time and activated partial thromboplastin time. Recovering from this takes many hours after venom neutralisation has been achieved with antivenom.

Some black snakes cause an anticoagulant coagulopathy, probably due to an inhibitor, that is rapidly reversed with antivenom. It is not associated with consumption of clotting factors, so fibrinogen and d-dimer levels are normal.

Neurotoxicity

Paralysis is a classic effect of snakebite and is due to presynaptic or postsynaptic neurotoxins in the venom. Neurotoxic envenoming causes a progressive descending flaccid paralysis. Ptosis is usually the first sign, then facial and bulbar involvement progressing to paralysis of the respiratory muscles and peripheral weakness in severe cases.

Post-synaptic: Australian snakes have at least one low molecular weight postsynaptic neurotoxin. These snake toxins block post-synaptic acetylcholine receptors causing paralysis. They are rapid in onset and their action often causes the predominant clinical picture. Fortunately, anti-venoms usually rapidly reverse these effects.

Presynaptic neurotoxins disrupt neurotransmitter release from the terminal axon. Presynaptic toxins are generally modified phospholipase A2 toxins, which specifically target the terminal axon of the neuromuscular junction, causing first release of neurotransmitter, then extensive damage to the axonal structure, completely disrupting transmitter synaptic vesicle production. Paralysis, when it occurs, usually commences with cranial nerves, then skeletal muscle, and finally, the muscles of respiration. Clinically this causes a progressive flaccid paralysis with the onset of the first signs usually within one-hour post bite, and full respiratory paralysis taking 3 - 24 hours. Once paralysed, the recovery rate is determined by axonal repair and is not influenced by antivenom therapy. Complete recovery may take days or weeks.

Myotoxicity

Some Australian snakes, such as the mulga snakes and tiger snakes, have venom containing myotoxins that cause rhabdomyolysis with muscle pain, tenderness and weakness, a rapidly rising creatine kinase and myoglobinuria.

Renal damage

Acute renal failure can occur secondary to severe rhabdomyolysis, in association with microangiopathic haemolytic anaemia (reported with brown snakes) or can occur rarely in isolation.

Major types of Australian snakes: clinical syndromes

This sequence of events is highly variable although the usual sequence of systemic symptom development is:

(<1hr) Headache, irritability, photophobia, nausea, vomiting, diarrhoea, confusion; coagulation abnormalities; occasionally sudden hypotension with loss of consciousness.

(1-3 hrs) Cranial nerve paralysis (ptosis, diplopia, dysphagia etc), abdominal pain, haemoglobinuria, hypertension, tachycardia, haemmorrhage.

(>3hrs) Limb and respiratory muscle paralysis leading to respiratory failure, peripheral circulatory failure with pallor and cyanosis, myoglobinuria, and eventually, death.

Each Australian snake causes a characteristic clinical syndrome, which can be used with information about the geographical distribution of snakes to determine which snake is involved when a patient is envenomed.

There are five major groups of medically important Australian snakes, which cause characteristic clinical syndromes. Identifying the snake is important for diagnosis and determining the appropriate antivenom to be administered, but is not always possible. Venom detection kits are available to assist with identification.

Brown snakes

Brown snakes occur widely throughout mainland Australia. They are fast moving and easily alarmed snakes that strike readily. However, they have a high rate of dry bites with envenoming occurring in less than half of bites. Bites cause minimal local effects and non-specific systemic effects are uncommon. Severe envenoming is characterised by an early collapse, within an hour of the bite, usually with spontaneous recovery within 5 to 10 minutes. Collapse appears to occur at the time of onset of the coagulopathy, but the mechanism is unclear. The major clinical feature is a venom-induced consumption coagulopathy. Renal damage and microangiopathic haemolytic anaemia have been reported and neurotoxicity is rare.

Tiger snake groups

Tiger snakes occur in southern and eastern Australia. The major clinical effects are a venom-induced consumption coagulopathy, presynaptic neurotoxicity and rhabdomyolysis. An early collapse can occur and initially the only detectable effect may be a coagulopathy.

Rough-scaled snakes are closely related to tiger snakes and cause similar effects. Copperheads are less well characterised, but appear to cause neurotoxicity and coagulopathy. The Hoplocephalus genus cause coagulopathy and are clinically similar to brown snakes except that tiger snake antivenom is used for treatment.

Mulga and black snake groups

Mulga snakes occur across Australia except the south and east. They cause severe rhabdomyolysis and anticoagulant coagulopathy associated with non-specific symptoms.

The red-bellied black snake is common in southern and eastern Australia, but only causes non-specific systemic effects, mild rhabdomyolysis and local effects, which are usually managed without antivenom.

Taipans

Taipans occur in northern Australia and are very dangerous with a high envenoming rate. The mortality rate is high in untreated cases. Clinical effects include a venom-induced consumption coagulopathy, presynaptic neurotoxicity and mild rhabdomyolysis. Thrombotic microangiopathy, haemolytic anaemia and renal failure have been rarely reported.

Death adders

Death adders are widespread but secretive ambush predators that have a characteristic 'viper-like' appearance. The major clinical effect is a postsynaptic neurotoxicity associated with non-specific systemic features.

Diagnosis

In the majority of cases there is a history of a snakebite or suspected snakebite. History, examination and investigations focus on whether the patient is envenomed or not and by which snake so that the correct antivenom can be given. Occasionally the diagnosis is not obvious if a snake is not seen or the patient presents with coagulopathy or neurotoxicity and no history of a bite.

A careful history is required to determine the circumstances of the bite and what first aid has been applied. Early symptoms suggest severe envenoming. The examination should include the bite site, and palpation of the lymph nodes draining the site for tenderness. In addition to standard observations, the examination includes looking for signs of paralysis (ptosis, bulbar palsy, respiratory effort and peripheral weakness), any evidence of coagulopathy or evidence of rhabdomyolysis (muscle tenderness and weakness).

Investigations should include a full blood count, coagulation studies including d-dimer, and biochemical tests including creatine kinase. A urine analysis is helpful for detecting blood or myoglobin.

A whole blood clotting time may be useful if coagulation studies are not available. Blood is collected in a clean glass tube and the time to clot is measured. The normal clotting time is less than 10 minutes. If the clotting time is greater than 20 minutes, this is highly suggestive of a procoagulant coagulopathy. If the clotting time is between 10 and 20 minutes, the result is indeterminate, but may be consistent with an anticoagulant coagulopathy. This test may be useful in remote situations to determine if a patient has significant coagulopathy.

Management.

<u>Do not wash the area of the bite</u>.

It is extremely important to retain traces of venom for use with venom identification kits. Venom identification kits can often accurately identify the type of snake in 30 minutes, and thus reliably and safely reduce the need for administration of polyvalent antivenom. Snake-specific antivenoms are less hazardous to the patient than polyvalent antivenoms.

Stop lymphatic spread. Bandage firmly, splint and immobilise.

The lymphatic system is responsible for systemic spread of most venom. This can be reduced by the application of a firm bandage (as firm as you would put on a sprained ankle) over a folded pad placed over the bitten area. While firm, it should not be so tight that it stops blood flow to the limb. Start bandaging directly over the bitten area, ensuing that the pressure over the bite is firm and even. If you have enough bandages you can extend towards more central parts of the body, to delay spread of any venom that has already started to move centrally.

Antivenom

Antivenom should be administered to all patients who exhibit signs of systemic spread.

Antivenom is created by injecting a small amount of the targeted venom into an animal such as a horse, sheep, goat or rabbit. The subject animal will undergo an immune response to the venom, producing antibodies against the venom's active molecule, which can then be harvested from the animal's blood and used to treat envenomation.

Antivenoms bind to and neutralize the venom, halting further damage, but do not reverse the damage already done.

Antivenoms are purified by several processes but will still contain other serum proteins that can act as antigens. Some individuals may react to the antivenom with an immediate hypersensitivity reaction (<1%) or a delayed hypersensitivity reaction (characterized by fever, rash, generalized lymphadenopathy, aching joints and renal impairment up to 14 days later). Antivenom should therefore be used with caution.

Premedication with subcutaneous adrenaline is currently recommended prior to the intravenous administration of Australian snake antivenoms. Adults should receive 0.25-0.3mg of adrenaline by the subcutaneous route. Although traditional, the role of antihistamines in premedication is unclear.

Most antivenoms are given by the intravenous route, although redback spider antivenom and box jellyfish antivenom are more often used intramuscularly.

Antivenoms that are given intravenously should be diluted in at least 100ml of normal saline, 5% dextrose or Hartmann’s solution immediately prior to administration. It should initially be administered slowly while the patient is observed for signs of allergic reaction. If no reaction is observed, then the rate of infusion may be increased.

Critically ill patients:

Maintain immobilisation, splint and bandage until the situation is under control
Support the airway, breathing and circulation. Intubate and ventilate with 100% oxygen.

Give antivenom immediately.

Volume expansion may be necessary.

Severe coagulation disturbances, electrolyte abnormalities, and muscle damage leading to acute renal failure are likely. Take blood for group and X-match, coagulation screen (including fibrinogen levels, and tests for DIC), full blood count, electrolytes and calcium, creatinine kinase and arterial blood gases. Perform ECG. Repeat at appropriate intervals.

Collect urine for microscopy to detect haematuria and for free protein, haemoglobin and myoglobin measurement. Record urine output. Freeze the first sample for venom detection.

Repeat antivenom as clinically indicated.

Less seriously ill patients - no signs of systemic spread

Admit to ICU for non-invasive monitoring, strict bed rest and full head injury (neurologic) observations.

Leave bandages in place until antivenoms and a venom detection kit is available. Obtain intravenous access.

When ready, cut a hole over the wound site, inspect and take swabs for use with the venom detection kit. Once the results of the venom detection kit are known, slowly and progressively remove the bandages. If systemic symptoms occur, reapply bandages and give antivenom.

Usually, if there are no signs of envenomation four hours after removal of the bandages, and if repeat blood tests taken at that time are normal, then it is probable that significant envenomation has not occurred. If laboratory tests are not available, 12 to 24 hours is a reasonable period of observation.

OBSTETRIC EMERGENCIES

Obstetric emergencies can be related to the pregnancy or be incidental to the pregnancy. In obstetrics, the challenge that presents itself is the fact that there are two patients to consider and many emergencies may be primarily maternal or foetal. Naturally, maternal compromise leads to foetal compromise. The treating team needs to be aware that there are significant changes in physiology that may impact on the management of the mother.

Pulmonary veno-thromboembolism, haemorrhage and hypertensive disorders are the major causes of maternal mortality. Failed intubation and oxygenation are an important cause of death related to anaesthesia.

Emergencies during delivery include severe foetal distress, foetal malpresentation and obstructed labour (shoulder dystocia). The anaesthetist will be asked to provide anaesthesia and uterine relaxation to help manage these conditions. In the case of foetal distress, there are some "intra-uterine foetal resuscitation" manoeuvres that can help to improve the foetal condition before delivery. The management of cardiac arrest is similar to adult cardiac arrest management with some important differences due to the physiologic and anatomic changes of pregnancy.

Physiologic changes of pregnancy

There are several important physiological changes that occur with pregnancy. Most changes begin in early pregnancy and are complete by the 12^{th} week. These changes help prepare the mother for delivery and help her adapt to pregnancy, but will reduce her physiologic reserve. The causes of theses changes are hormonal, mechanical and related to the presence of the placenta (a low resistance high flow vascular bed) and increased metabolic demand.

The cardiac output increases by 40-50% by 32 weeks. This results from an increase in heart rate of 15% and a 35% increase in stroke volume. Systemic vascular resistance decreases by 35%, which results in a fall in blood pressure (diastolic BP drops more than the systolic BP).

The gravid uterus causes aortic artery and inferior veno-caval compression. This reduces venous return to the heart and can reduce uterine perfusion. Compression is worst in the supine position. The normal patient will compensate with a tachycardia and vasoconstriction of the vessels in the upper half of the body. Blood will be diverted to the right atrium via the epidural and azygos system. Some women will experience bradycardia, nausea, sweating, pallor and fainting in the supine position as a result of veno-caval compression.

The respiratory changes of pregnancy include elevation of the diaphragm by 4 cm and an increase in the anterior-posterior diameter of the chest wall. The angle of the ribs decreases so that they become more horizontal. Chest wall compliance is reduced by 30%, so it is harder to ventilate the pregnant woman. The functional residual capacity (FRC) of the woman will be reduced and in some women, the closing capacity is greater than the FRC resulting in closure of the small airways, particularly in the supine position. Because the metabolic rate and oxygen consumption are increased in

pregnancy, this reduction in FRC will lead to hypoxia during periods of apnoea. Capillary and soft tissue engorgement in the upper airways leads to nasal obstruction, and oedema of the upper airway. Nasal bleeding is more common and difficult intubation is more likely in the pregnant patient.

Pregnancy leads to an increase in total blood volume. The relative increase of the red blood cell volume is lower than the increase in the plasma volume and this leads to what is known as the physiologic anaemia of pregnancy. Although the platelet count drops by up to 20%, there is an increase in clotting factors and decrease in plasminogen and anti-thrombin III activity, leading to a hypercoagulable state and an increased risk of thrombosis.

After delivery, there is a loss of the placental shunt and an "auto-transfusion" will occur with uterine contraction. Most of the physiologic changes of pregnancy will reverse over the first five days post partum, but there is still a high risk of thromboembolism in the immediate post partum period.

The physiologic changes that occur during pregnancy render the patient vulnerable during periods of physiologic stress. There is a higher risk of hypoxia, failed intubation, acid aspiration, pulmonary oedema, and thromboembolism. The patient with pre-existing cardiac disease will be at risk of cardiac failure during pregnancy, particularly during the second trimester, when the cardiac changes are greatest.

Haemorrhage

Death due to maternal haemorrhage is an ongoing problem around the world. In developed countries the death rate from haemorrhage has fallen since the 1960s, but has remained steady since the early 1970s. The rate per million maternities in 2000-2002 in the United Kingdom was 8.5, with placental abruption, placenta praevia and post-partum haemorrhage being identified as the causes of life-threatening haemorrhage.

All the deaths from haemorrhage in the UK Confidential Enquiry 2000-2002 associated with placenta praevia were in women who had a previous caesarean section.

The UK Enquiry identified catastrophic haemorrhage as a persisting problem. Of note, was the fact that high-risk patients were still being delivered in isolated obstetric units with no access to specialists or blood products and that anaesthetic and obstetric care was sub-standard in many of the women that died.

This highlights the need for identification of women at risk and for improvement in management.

The predictors of post partum haemorrhage include, previous post partum haemorrhage, multiple gestation, macrosomia, polyhydramnios, and prolonged use of oxytocin and coagulation disorders. Women who are identified as high risk of haemorrhage need to be managed in a specialist centre. Outpatient consultation with the anaesthetic team should occur at about 30 weeks. Intravenous access, full blood count, coagulation studies and cross matching should be performed on arrival to the

delivery ward. Good communication between caregivers is vital to ensure rapid and successful management.

The anaesthetist's role involves resuscitation, intravenous access, the administration of blood and blood products, invasive monitoring, provision of oxygenation and providing anaesthesia for delivery (if necessary) or evacuation of a retained placenta. Intensive care management following maternal haemorrhage is likely to be for the ongoing correction of clotting abnormalities, warming the patient and ventilatory support.

Sepsis

Puerperal sepsis (infection during or shortly after childbirth, miscarriage or abortion) is a significant cause of maternal mortality worldwide. The commonest sites of sepsis are the urogenital tract, wounds, breast infection (mastitis) peritonitis and respiratory tract. Puerperal fever is now rare due to improved hand hygiene during delivery and antibiotic therapy but infection occurs in 1-8% of all deliveries in the USA and 3 women will die for every 100,000 deliveries. The most important risk factor is caesarean section. Current antibiotic guidelines recommend the use of a first generation cephalosporin such as cephalothin 2G intravenously or cephazolin 1G intravenously after clamping of the umbilical cord for both emergency and elective caesarean sections.

Sepsis and septic shock are a response to infection. Sepsis is the leading cause of maternal mortality in intensive care. Most deaths are due to multiple organ dysfunction.

The obstetric patient is vulnerable to pyelonephritis, chorioamnionitis, endometritis, wound infection and necrotizing fasciitis and cholecystitis. Gram-negative aerobic bacilli were once the predominant organisms associated with maternal sepsis, but gram-positive infections now make up half of the infections.

Group A Streptococci (most commonly Streptococcus pyogenes) is responsible for severe haemolytic streptococcal illness. Group B streptococcus GBS (Streptococcus agalacticae) can cause less severe maternal illness but a mother carrying it in her genital tract can pass it on to the foetus during birth and it can cause pneumonia or meningitis in the neonate. Women who are GBS positive on vaginal swabs (routinely taken between 35-37 weeks of pregnancy) are treated with penicillin in labour after rupture of their membranes. The other organisms that cause maternal infection are staphylococci, coliform bacteria, anaerobic bacteria, Chlamydia, myoplasma and rarely, Clostridium welchii.

Septic shock is sepsis associated with a systemic inflammatory response that includes hypotension in spite of volume replacement and perfusion abnormalities that lead to low urine output, lactic acidosis and altered mental state. There is a reduction in vascular tone and an increased cardiac output occurs after the volume state is restored to normal with fluid administration. Myocardial dysfunction also occurs.

The systemic inflammatory response syndrome includes two of: a temperature above 38 degrees Celcius or below 36 degrees, a respiratory rate over 20 breaths per minute

or arterial carbon dioxide below 32 mmHg, pulse over 90 beats per minute and white count over 12,000/cc or less than 4000/cc or bands > 10%.

Management of sepsis

Initial assessment includes assessment of the likely source of the infection. Tests include chest x-ray, abdomino-pelvic CT scanning looking for abscesses, uterine necrosis and pyometrium, wound swabs or fluid cultures and blood cultures.

General treatment of sepsis includes:

1. The administration of broad-spectrum antibiotics,
2. Aggressive fluid replacement guided by central venous pressure monitoring,
3. Blood products to treat anaemia or coagulation disorders,
4. Vasopressors and ionotropes,
5. Removal of the infection source,
6. Ventilatory support
7. Supportive care of the intensive care patient (Deep Venous Thrombosis prophylaxis, nutrition, stress ulcer prophylaxis, haemofiltration)
8. Delivery of the baby (necessary if there is chorioamnionitis).

Early administration of antibiotics reduces mortality and morbidity. For pregnancy related infection, a combination of penicillin, aminoglycoside and either clindamycin or metronidazole for anaerobes is appropriate. Alternatively a carbapenum or 3rd or 4th generation cephalosporin may be used. Vancomycin is used for suspected methicillin-resistant Staphylococcus Aureus infection.

Fluids are administered to restore blood volume and are administered according to blood pressure, aiming for a systolic of at least 90 mmHg, and urine output of over 0.5 mg per kilogram per hour. Boluses of crystalloids of 250 to 1000 millilitres over 5-15 minutes are recommended. The pregnant state and sepsis both decrease intravascular oncotic pressure. Fluid overload will result in pulmonary oedema. It is recommended that central venous pressure (CVP) monitoring be used as a guide to fluid replacement aiming for a CVP of 5-8 mmHg. Red blood cell replacement is recommended to maintain the haemoglobin between 9-10 mg/dL in the septic patient.

Vasopressors are required when volume replacement does not restore the blood pressure. Recent data suggest that noradenaline is the best choice to increase blood pressure and cardiac index in sepsis, with less tachycardia. It is more potent than dopamine and is capable of increasing cardiac output, renal blood flow and urine output. Most patients have an increased cardiac output in sepsis, but if cardiac output is low, dobutamine may be considered (2.5 mcg/kg/min, increasing every 30 minutes until the cardiac index is over 3). If organ perfusion remains low, vasopression in modest doses is added to prevent splanchnic and coronary ischemia.

Early endotracheal intubation and mechanical ventilation are recommended in severe sepsis. The indications for mechanical ventilation are severe tachypnoea (respiratory rate over 40), use of accessory muscles of breathing, altered mental status and severe hypoxaemia in spite of oxygen therapy. The tidal volumes are limited to 6 ml per kg and the ventilator pressures are limited to less than 30 cm of water and positive end expiratory pressure (PEEP) is used to improve oxygenation.

In all cases of septic shock, early management is important to prevent multiple organ dysfunction. If there are fluid collections, or abscesses and devitalized tissue, this needs to be drained and removed. Myometrial necrosis will require a hysterectomy. If chorioamnionitis is likely or confirmed on amniocentesis, delivery of the baby will be required.

Pregnant septic patients are at risk for utero-placental insufficiency and pre-term labour. The decision to deliver the baby should be based on gestational age and the patient's condition. Long periods of maternal hypoxemia and acidosis may result in permanent foetal damage or progression into active labour. Indications for delivery are chorioamnionitis, a non-reassuring foetal heart rate pattern or labour.

Cardiac arrest

Although rare, cardiac arrest in the pregnant patient needs to be managed carefully, in order to improve the chances of survival for both mother and foetus.
The causes of cardiac arrest include, pre-eclampsia, eclampsia, pulmonary thromboembolism, haemorrhage, amniotic fluid embolism, peripartum cardiomyopathy or pre-existing cardiac disease, and sepsis and anaesthetic complications. In women with primary heart disease, a cardiac arrest in pregnancy is associated with a high mortality rate.

The incidence of arrest is 1 in 30,000 pregnancies. The fundamentals of management are the same as for a non-pregnant patient. Once recognized, a call for help is made, the airway is cleared and two rescue breaths are given before cardiac compression of the lower half of the chest are commenced at a rate of 100 per minute. The ratio of cardiac compression to ventilation is 30 compressions to two breaths regardless of the number of rescuers involved.

Cardiac compressions generate 30% of cardiac output in the non-pregnant patient. In the supine position of a woman in her third trimester of pregnancy, compression of the inferior vena cava and aorta occurs. As a result, there is a fall in the venous return to the heart and cardiopulmonary resuscitation (CPR) is less effective and chest compressions generate only 10% of cardiac output. Left lateral tilt of 27 degrees allows for a compression force of 80% during external cardiac massage whilst still relieving aorto-caval compression.
The airway is at risk due to aspiration. The mother has higher oxygen requirements than a non-pregnant patient and intubation can be difficult due to weight gain, breast tissue, a change in chest wall shape and tissue oedema. High flow oxygen should be used in the resuscitation of a pregnant patient. Intubation with a 0.5-1 mm smaller than usual cuffed tube should occur early in the resuscitation. A difficult intubation is predicted. After intubation mild hyperventilation to a maternal paCO2 of 30-32 mmHg is performed.

If direct cardioversion is required for ventricular fibrillation or unstable ventricular tachycardia, the usual protocols are used as for a non-pregnant patient. Foetal heart rate monitoring is discontinued during defibrillation.

There are case reports of successful maternal resuscitation after delivery of the foetus. If a woman suffers a cardiac arrest in the third trimester of pregnancy and CPR has not been successful within 4 minutes, the current recommendation is to deliver the foetus to increase the chances of maternal and neonatal survival. It is not practical to transfer the patient to the operating room during a cardiac arrest. If there is a need to do a peri-mortem caesarean delivery it is recommended that it be performed at the scene of the arrest by the most senior obstetric person available. Minimal equipment is required. A betadine antiseptc solution, a disposable pre-loaded scalpel and packs for the uterus and abdomen are all that is required. A plan is made to transfer the patient to theatre should she be successfully resuscitated. (13)

If the gestational age is less than 24 weeks, it is unlikely that the foetus will survive if delivered; so advanced cardiac life support of the mother is continued without delivery. After 24 weeks, surgical delivery can be considered. This cut off date is arbitrary and will depend on the level of neonatal care available.

In such a situation, CPR is continued if maternal survival it thought to be likely. Time is not wasted preparing a surgical field, but delivery of the foetus is expedited to improve the chances of intact (that is with no neurologic injury) survival for the infant.

Perimortem Caesarean Section and Infant survival (Clark et al, Critical Care Obstetrics, 1997)

Time from arrest to delivery	Neurologically intact infant survival
< 5 minutes	98%
6-15 minutes	83%
16-25 minutes	33%
26-35 minutes	25%

In the situation where resuscitation of the mother has been successful before caesarean section, it is better to wait before delivering the foetus, as successful in-utero resuscitation is more likely to produce a neurologically intact infant.

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HAEMATEMESIS AND VARICEAL BLEEDING

Causes of Upper Gastrointestinal Tract (GIT) bleeding vary between countries, although the two most common causes are **peptic ulcer disease** and **oesophageal varices.**

Causes of Upper GIT bleeding, USA (1996)

Peptic ulcer disease	**55%**
Oesophagogastric varices	**14%**
Arteriovenous malformations	6%
Mallory-Weiss tears	5%
Tumors and erosions	4%
Dieulafoy's lesion	1%
Other	11%

Varices and Variceal bleeding

Variceal bleeding occurs most commonly where there is a high incidence of chronic liver disease due to **hepatitis** and **alcohol-induced cirrhosis.** Liver cirrhosis causes increased portal venous pressures, distending superficial oesophogeal veins as portal blood returns to the systemic circulation through collateral circulation. The distended oesophageal veins, or 'varices', are fragile, thin-walled and bleed easily. One third of all patients with oesophageal varices will experience variceal bleeding.

Variceal bleeding stops spontaneously in more than 50% of patients, but there is a mortality of 70-80% in those with continued bleeding. Each episode of variceal hemorrhage has a 30% risk of mortality. The risk of rebleeding is high (60-70 %) until gastroesophageal varices are obliterated, usually with endoscopic banding or diathermy.

Over 50% of early rebleeding occurs within the first 10 days, with the first 48-72 hours being the time of greatest risk. Risk factors for rebleeding include:

1. Age over 60 years
2. Renal failure
3. Large varices
4. Severe initial bleeding, with haemoglobin < 8 g/dL at admission.

Long-term survival depends upon the severity of liver disease and *may not* improve even after variceal obliteration. There is only a 30% 2-year survival after the first episode of variceal bleeding regardless of treatment.

The administration of a nonselective beta blocker such as propranolol can decrease the risk of rebleeding by reducing portal hypertension.

The onset of massive upper gastrointestinal bleeding from gastroesophageal varices usually signifies advanced liver disease (Child class B or C). Liver transplantation is the only treatment that significantly improves the long-term prognosis in these patients.

Approach to the emergency patient with haematemesis

Emergency assessment and resuscitation of the patient presenting with haematemesis follows similar principles as other emergency assessments. The patient's **Airway**, **Breathing** & **Circulation** should first be reviewed with particular attention paid to the vital signs (heart rate, blood pressure, pulse oximetry). Any significant compromise in these areas should be corrected before progressing to other management.

A focused history should be collected, with special attention given to past liver disease, alcohol consumption, hepatitis, and any history of gastroeosophogeal reflux or ulcers.

Special attention should be given to high-risk patients. Consider ICU admission for these patients:

Systolic BP < 100 mmHg	Over 60 years old
Postural BP > 20 mmHg decrease	Renal failure
Heart rate > 100 bpm	Hemoglobin < 8 g/dL
Blood transfusion > 2 units	

Resuscitation follows principles similar to managing any patient with haemodynamic compromise. Place two large-bore intravenous cannulae. Begin with up to 2 litres of crystalloid or colloid fluid intravenously, and if there is ongoing hypotension then transfuse blood.

For young and fit patients aim for a haematocrit > 20%. For older or infirm patients aim for haematocrit > 30%. Ongoing bleeding with an INR >1.5 requires fresh frozen plasma, and if the platelet count is < 50,000/μL, the patient requires platelets. Be careful not to over-resuscitate as increased venous pressures may worsen bleeding. A systolic blood pressure of 90 mmHg or Mean Arterial Pressure (MAP) of 60 mmHg is acceptable.

Ongoing management

Ongoing management aims to either decrease portal hypertension (beta-blockers, surgical portal decompression, or transjugular intrahepatic shunts) or to directly treat the varices themselves (variceal ligation).

Propanolol, a non-selective beta-blocker, reduces the risk of variceal re-bleeding. Start with propanolol 40 mg/bd, and aim for a resting heart rate of 55-60 beats per minute. Alternatively use nadolol 40 mg/d (or begin with 20 mg/day if the patient is hypotensive). Do not administer a beta-blocker to the haemodynamically unstable or under-resuscitated patient.

Somatostatin or octreotide can be used to reduce portal vessel pressures and thus bleeding from varices, and also reduce gastric acid secretion.

Intubation of patients

Intubation should be considered for patients with respiratory depression or depressed consciousness, as they are at greater risk of aspiration and for patients with active bleeding requiring endoscopy. Intubation should always be performed as a **rapid sequence induction** in these situations. Special care should always be taken when inducing haemodynamically unstable patients — reduce the dose of induction agent appropriately.

Ideally **two airway suction devices** should be available to the anaesthetist at the time of intubation. Blockage of a sucker with blood clot is a common problem and can have critical consequences.

Managing Endoscopy

Endoscopy is the mainstay of managing gastrointestinal bleeding, allowing both the diagnosis of varices and ulcers, as well as treatment. Variceal banding or diathermy can control variceal bleeding, and injecting ulcers with adrenaline (epinephrine) or clipping vessels can control ulcer bleeding.

A single dose of erythromycin intravenously of 3 mg/kg over 30 min or metoclopramide 20 mg intravenously 20-120 minutes before endoscopy will improve visibility for the endoscopist by increasing gastric emptying.

Omeprazole 40 mg intravenously or another proton pump inhibitor will reduce the risk rebleeding if it is due to a gastric ulcer.

Antibiotics should be considered in all cases of variceal bleeding as bacterial infections are present in 20% of those with cirrhosis, and up to 50% will develop infection during admission. Antibiotics may reduce mortality in these patients by up to 25%. Broad-spectrum antibiotics should be chosen: quinolones: 400 mg of ofloxacin, 400 mg twice daily of norfloxacin, or 400 to 1000 mg of ciprofloxacin; or cephalosporins (ceftriaxone 1g twice daily), for 7 days.

In the presence of hepatic encephalopathy give consideration to:

- Lactulose 30 mL orally twice daily to reduce intestinal bacterial production of ammonia.
- Aggresssively correcting hypokalaemia, which can promote the development of hepatic encephalopathy via increased renal ammonia production.
- Be alert for renal failure.
- Give thiamine to alcoholics.

Patients with significant risk factors (more than 2 units blood transfused; age > 60 years; renal failure; Hb < 8 g/dL) should be monitored in an intensive care or high dependency environment for the first 24-48 hours after endoscopy, if facilities are available.

PAEDIATRIC EMERGENCY CARE: A CASE STUDY

A 17-month-old male infant weighing 13.5 kg presents to the Accident and Emergency department with shortness of breath.

He had been eating three hours previously and his mother gave a history of a sudden bout of coughing. On arrival in the emergency department, he was distressed, crying and coughing intermittently. He had no stridor, however his respiratory rate was 40 breaths per minute with some intercostal and sternal recession. Auscultation revealed a mild wheeze that was worse on the right side of the chest. His oxygen saturation was 94% on room air.

Differential Diagnosis:

The most likely diagnosis is an inhaled foreign body, but although less likely, an infection or trauma are possible.

Foreign body (FB) aspiration is one of the leading causes of death young children; for example, FB aspiration has been responsible for more than 300 deaths per year in the USA (Black RE *et al.* 1994). It most commonly occurs in the 1-3 year-old age group, tending to occur in boys more than girls.

Most deaths occur at the time of aspiration due to complete upper airway obstruction. Of those children who reach hospital, the mortality is low. Despite this, significant peri-operative morbidity and mortality can still occur and can potentially be reduced, prevented or anticipated with appropriate management and precautions.

The diagnosis of FB aspiration can be difficult; frequently the inhalation event is not witnessed, and the history may be less clear than expected. The presentation is variable, and dependent on:

- Early versus late presentation
- Size and shape of the object inhaled
- Site of object within the airway (trachea/main bronchus/distal airways)
- Time of presentation since event

The early presentation can therefore range from severe acute upper airway obstruction through to a well, pink child often with a cough. The children presenting late often present with signs of secondary lower respiratory tract infection. More than 90% of foreign bodies lodge in a main bronchus – occurring only slightly more commonly on the right side in children.

The diagnosis is therefore based on the degree or ‘index of suspicion’. In turn, this is based on a careful history, examination and special investigations.

History:

- A classical history may comprise a sudden onset of choking, followed by coughing and wheezing.
- A persistent cough

Examination:

Signs such as cyanosis, stridor, severe intercostal/sternal recession and evidence of reduced conscious level are consistent with upper airway obstruction requiring emergency management

A history of voice change or barking cough may indicate laryngeal oedema or upper airway foreign body obstruction. There may be a persistent cough.

A late presentation may allow for the production of signs of pneumonia, such as fever, cough, tachycardia, tachypnoea and focal chest signs.

It must be remembered that none of the features above are 100% specific, and the differential diagnoses must be considered such as asthma, pneumothorax, croup or other infective respiratory tract infections.

You decide to do a chest Xray:

A plain Anterior-Posterior chest X-ray was clear with no evidence of pneumothorax, collapse, consolidation or hyperinflated areas. Does this exclude an inhaled foreign body?

Special investigations:

Apart from oxygen saturations, most cases will have an antero-posterior chest x-ray and lateral chest x-ray. The films should extend to the entire neck (Rovin JD & Rogers BM 2000). It must be remembered that the majority of inhaled material is organic in origin, and therefore a plain radiograph may fail to demonstrate an abnormality, especially in the first 24 hours. The absence of radiographic abnormalities does not exclude the diagnosis of an inhaled FB.

The sensitivity of the plain radiograph as a diagnostic tool in these patients has been cited at only 67 -82%, with a specificity of 44-74% (Farrell PT 2004). Of note, another study found the rate of **normal** chest X-ray findings in children with known FB to be 56% if tested in the first 24 hours after inhalation, but only 33% were normal if more than 24 hours had passed (Mu L *et al.* 1993).

The most frequent radiological findings seen in FB aspiration are:

- Normal – no abnormality
- Gas Trapping (due to ball-valve effect of foreign body with respiration)
- Mediastinal shift
- Atelectasis
- Lobar collapse/consolidation

Management:

How do you manage this child with a suspect inhaled foreign body?

Most common areas of confusion:

1) Should anaesthesia be delayed until the child is adequately fasted?

2) A sedating premedication such as oral midazolam must be prescribed to reduce anxiety.
3) How to manage this patient in the emergency department and in the theatre.
4) If the child develops complete airway obstruction in the emergency department, the child should be immediately anaesthetised for bronchoscopic foreign body removal.

The initial management strategy is **immediate Basic Life Support.**

If the child is acutely unwell, they should initially be assessed and managed as per the 'choking child' Basic Life Support Algorithm.

This involves **2 questions** in the assessment process:

1) Does the child have an effective cough?

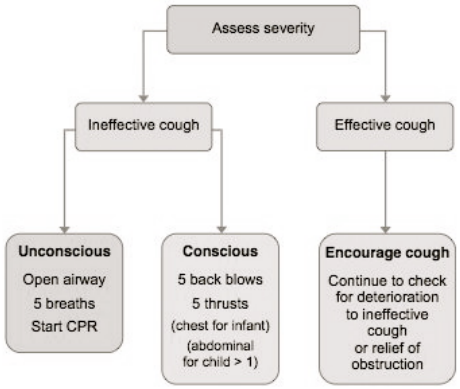
That is, is he or she crying, talking, and can he or she take a breath between coughs? Is the conscious state normal and is there any cyanosis present? If there is an adequate cough, the child should be closely observed and encouraged to cough by themselves.

If the cough is not effective, the next question is:

2) Is the child conscious?

If the child is conscious, the rescuer may intervene with 5 back blows followed by 5 chest thrusts

If the child is not conscious, the child should have airway, breathing and circulation assessed and managed as per the basic life support guidelines, with assisted ventilation and chest compressions.



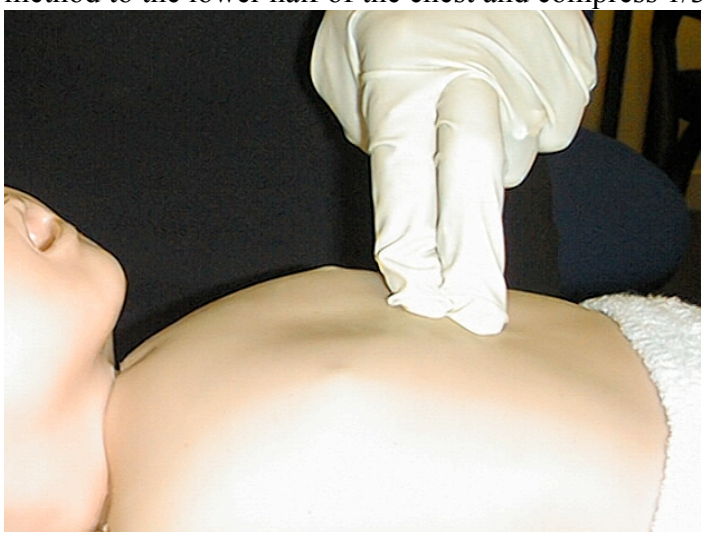
If the child is conscious deliver 5 back blows between the shoulder blades with the child over your arm or leaning forward or 5 chest thrusts to the lower half of the chest. The Heimlich manoeuvre and abdominal thrusts are no longer recommended as there is a risk of damaging internal organs.

If the child is unconscious, start basic life support. Check for a response, open the airway and check for breathing. If the child is unresponsive and is not breathing, give 5 rescue breaths then check for a pulse or signs of a circulation. If there is no circulation, commence chest compressions to the lower half of the chest at a ratio of

30 compressions to 2 breaths (for a single rescuer) or 15 compressions to 2 breaths for two rescuers.

Chest compressions are started if there is no pulse, a slow pulse (less than 60 beats per minute with poor perfusion) or no signs of a circulation (such as the absence of movement, coughing and normal breathing).

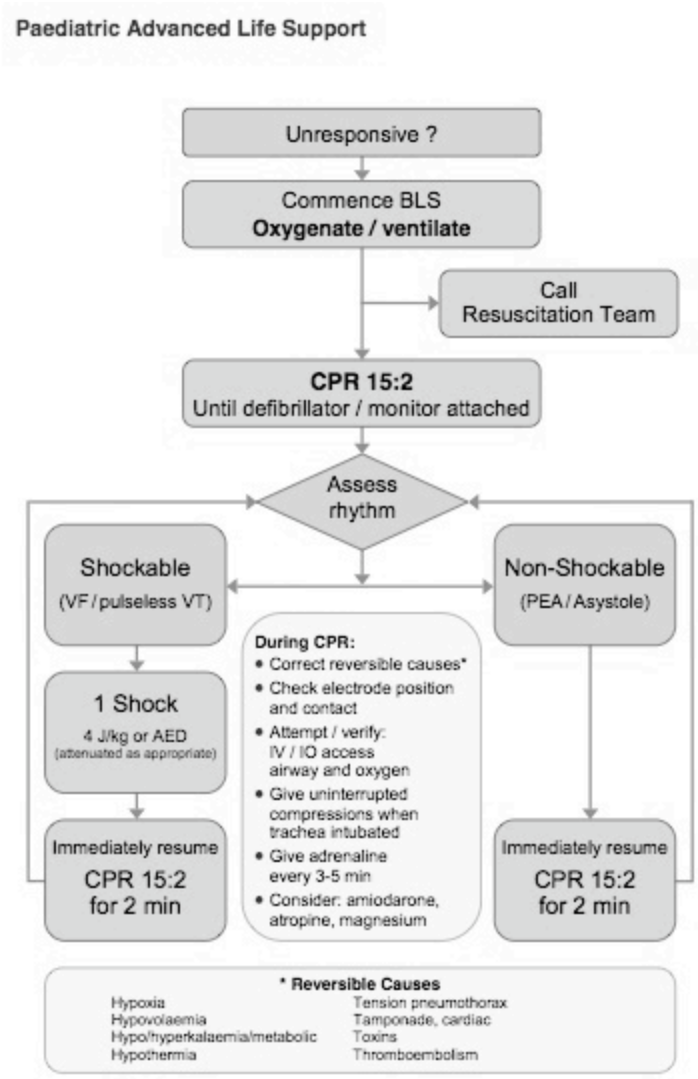
The method of chest compression for an infant is to use 2 fingers or an encircling method to the lower half of the chest and compress 1/3 – ½ of the depth of the chest.



DO NOT INTERRUPT CHEST COMPRESSIONS FOR MORE THAN 10 SECONDS EXCEPT TO PERFORM DEFIBRILLATION.

The compression rate at all ages is 100 per minute.

- For single rescuers the ratio of compressions to ventilations is 30:2.
- For two rescuers the ratio of compressions to ventilations is 15:2.
- If no help has arrived the emergency services must be contacted after 1 minute of cardiopulmonary resuscitation with no reassessment of ABC.
- With pauses for ventilation there will be less than 100 compressions per minute although the *rate* is 100 per minute.
- The rate of 100 chest compressions and 20 ventilations per minute should be the goal.



- Important aspects of Paediatric advanced life support:
  - Defibrillation
    - Initial 2 J/kg, then 4 J/kg
    - Same joules in both monophasic and biphasic defibrillation
  - Dose of resusciation drugs:
    - Adrenaline 10mcg/kg
    - Amiodarone 5mg/kg for refractory VF
  - CPR
    - 15:2 in hospital setting

Subsequent management of the child with an inhaled foreign body:
Most children who reach hospital with an inhaled FB will have an effective cough. Once the child has been assessed, arrangements need to be made for transfer to theatre with appropriately skilled senior personnel for a bronchoscopy under general anaesthesia.

The key to success is communication between all medical staff with regard to planning and equipment. The anaesthetist should be familiar with all initial and contingency plans, and should also have checked the bronchoscope and that it

connects to the breathing system. It is also suggested that two anaesthetists should be present – one of whom should be a paediatric anaesthetist (Farrell PT 2004).

Technique of Anaesthesia

Preparation:

Once the assessment has been made and the personnel in place, a number of considerations need to be made before theatre.

Fasting

Ideally, children should be fasted for at least 6 hours for solids and 2 hours for clear fluids to reduce the probability and severity of aspiration under anaesthesia. As in the scenario above, a compromise sometimes has to be made, in view of the urgency of the case. Depending on the clinical state of the child, children are often transferred to theatre without full 'ideal' fasting.

Premedication

Careful clinical judgement needs to be exercised with regard to sedative premedication. If possible, the child should be kept calm up to the moment of induction, to avoid dislodgement and worsening of the obstruction. However, many clinicians often avoid sedative premedication to improve perioperative respiratory drive.

Medical therapy

Antibiotics may be given to those presenting late with clinical features of pneumonia. Steroids are also often employed to treat and prevent laryngeal oedema, although there is little documented evidence for its efficacy (Weir PM 2004).

Intravenous access

Despite the potential full stomach, it may be reasonable to insert the cannula in the distressed child with difficult veins once the child is induced with anaesthetic. Sensible clinical judgement needs to be employed.

Induction of Anaesthesia

Most anaesthetists will use an inhalational induction with either sevoflurane or halothane in order to maintain spontaneous ventilation.

Although Halothane is cheap, smooth at induction and easy to maintain depth with spontaneous ventilation, its lack of familiarity amongst many anaesthetists and propensity for cardiac dysrhythmias renders it less popular than in previous years. Many now favour sevoflurane, with the advantage of relative stability of the cardiovascular system.

Nitrous oxide is usually avoided for induction and maintenance, particularly if there is radiological evidence of gas trapping. It is also advisable to be able to preoxygenate with anaesthetic agent/O_2 mix before instrumentation by the proceduralist.

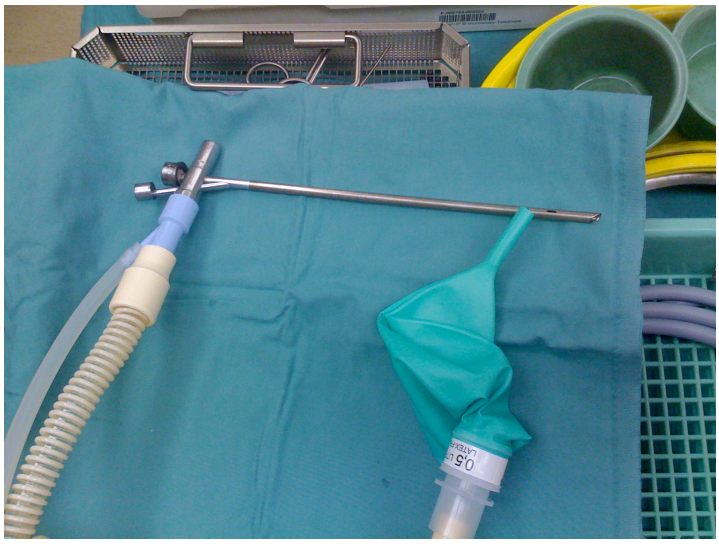
Once at adequate depth (using eye signs, respiration pattern and abdominal wall tone), the cords and upper trachea are sprayed under direct laryngoscopy with 4mg/kg lignocaine (lidocaine). This reduces the cardiavascular and tussive response to bronchoscopy.

Maintenance of Anaesthesia

This is challenging for the anaesthetist – especially in the presence of reduced gas exchange in many of these children. Considerable controversy exists as to how to conduct ventilation and oxygenation during rigid bronchoscopy in children. Traditionally, it is felt that a spontaneously breathing technique is superior, as it reduces the chance of distal movement or dislodgement of the foreign body, and may be less likely to worsen distal air trapping.

Connection of circuit to bronchoscope can be achieved via a port on the rigid bronchoscope. It is then possible to use a combination of inhaled and intravenous agents to maintain anaesthesia.

If a technique of neuromuscular paralysis and intermittent positive pressure ventilation is used, there is a risk of dislodgement of the FB and complete airway obstruction.



References:

1. Dr Adam Skinner (Paediatric Anaesthetist Royal Children's Hospital) Tutorial of the Week – Inhaled Foreign Body
2. Advanced Paediatric Life Support course notes
3. http://www.resus.org.uk

CARDIAC COMPRESSIONS

Greater emphasis is now placed on compressions over ventilation in improving outcome after cardiac arrest. Since 2005, the AHA (American Heart Association) has given greater weight to cardiac compressions over ventilation, with the introduction of compression to ventilation ratio from 15:2 to 30:2. No studies have compared outcomes of compression alone versus combined compression-ventilation resuscitation, however it is hoped that the removal of the mouth-to-mouth component for non-trained bystanders in the community will increase the likelihood of Cardio-Pulmonary Resuscitation (CPR) being commenced. This comes in the wake of community concern about the transmission of communicable disease such as H1N1 influenza.

The European Resuscitation Council also changed their guidelines to the 30:2 ratio but felt less strongly about promoting compression-only CPR. Their guidelines comment on the importance of combined compression-ventilation in particular circumstances such as in paediatric arrest (where hypoxia is the commonest cause), where there is a non-cardiac cause for arrest such as drowning/asphyxia, for most in-hospital arrests and where CPR is prolonged > 4 minutes. The focus is thus on improving the quality of the compressions with minimal interruption for ventilation/intubation or defibrillation, and the maintenance of a rate of 100 compressions per minute, at a 30:2 ratio.

To perform effective external cardiac compression the rescuer needs to ensure the patient has a firm surface behind them to ensure effective compressions (such as on the floor).

Hand Position:

The heel of the hand is used to direct pressure downwards. The fingers are raised off the chest so there is no compression of the ribs. There is no evidence that use of one or two hands is more efficacious. The rescuers elbows should be locked in full extension, vertically over the sternum and the weight of the resuscitator's body directly over the patient should be used to achieve the compressions. In infants it is best to use both thumbs to compress the lower half of the sternum with the hands encircling the chest. Alternately in infants, the end of two fingers directly compressing the lower half of the sternum is acceptable.

Site:

The lower half of sternum is the site for chest compression in all patients regardless of age. Compressions are ineffective if they are performed too high on the sternum, and there is a risk of regurgitation or trauma to abdominal contents if compressions are performed below the sternum.

Rate:

Equal time should be allowed for the compression and the recoil of the chest wall. A steady rate should be achieved with equal duration of each compression, at 100 compressions per minute. This should equate to approximately 2 compressions per

second. Due to interruptions for two breaths, there may not be 100 compressions every minute, however it is important that the rate be maintained.

Depth:

One third of chest wall depth should be compressed. This is at least 4-5cm in adults.

References:

1. Australian Resuscitation Council Guidelines (2006).
2. Advisory Statement of the European Resuscitation Council of Basic Life Support. 31st March 2009 (updated November 2009).

VASCULAR ACCESS

Intravenous access:

Access to the circulation is required for; drug administration, fluid administration, blood sampling transvenous pacing.

During cardio-pulmonary resuscitation, the circulation time from the central veins is 30 seconds compared with up to 5 minutes after peripheral administration. It is best to achieve more central access where possible and to flush any drugs given with 20 ml of fluid and raise the limb.

Sizing of cannulae:

There are two common ways of sizing cannulae, the standard wire gauge and French gauge. Using standard wire gauge or gauge (G), the diameter of the cannula increases with a decrease in number. Using French gauge (F), the diameter of the cannula increases with an increase in number. A larger bore (diameter) and short cannula allows for rapid infusion rates.

Peripheral cannulation:

Peripheral cannulae are usually inserted with the introducer-in-needle technique but may be inserted using a Seldinger technique (where a small cannula is inserted first and then a wire is used followed by a dilator over the wire and followed by the larger bore cannula). The common sites for peripheral cannulation during circulatory collapse are: the external jugular vein, the femoral vein and the long saphenous vein.

Central cannulation:

Central venous cannulae are usually inserted using a Seldinger technique. The sites for central venous access are the internal jugular vein and the subclavian vein. Possible complications of central venous cannulation are: arterial puncture, haematoma, haemothorax, pneumothorax, venous air embolism, cardiac arrhythmias, damage to the brachial plexus and sepsis.

Intraosseous access:

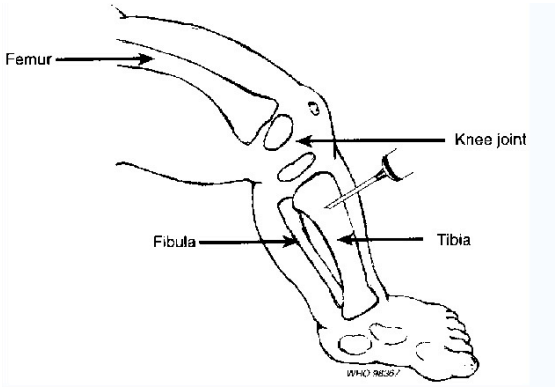
If intravenous access is not achieved within two or three attempts in an emergency situation, intraosseous access is preferred. It is an acceptable method of gaining access to the circulation in all age groups. A rigid cannula is inserted into the intramedullary cavity of a long bone. There are dedicated intraosseous access devices, but a short 16-18 G spinal needle with a stylet is an alternative. The tibia is the preferred site but a fractured limb should be avoided.

In the case of cardiac arrest, access to the circulation is difficult as the peripheral vessels constrict. The intraosseous space is not collapsible and is therefore more readily accessible. Any medication or fluid that can be delivered via a central venous route can be delivered via the intraosseous route.

The intraosseous site for access to the circulation is used as a temporary measure until suitable intravenous access is achieved. The rate of complications from intraosseous access is low (<1%) and is associated with prolonged duration of access. It is not

recommended that it be used for over 24 hours. Possible complications include, fracture, ostoemyelitis, epiphyseal plate injury and abscess formation. Extravasation of fluid and subperiosteal infusion are possible if the cannula is not in the medulla. A compartment syndrome or skin necrosis may result if the cannula is not correctly positioned.

The technique for intraosseous access is:
Find the insertion point on the surface of the tibia 2-3 cm below and medial to the tibial tuberosity.
Clean and prepare the skin, use a drape and sterile gloves.
Immobilise the limb with your non-dominant hand.
Puncture the tibia at a 90-degree angle to the skin and advance the needle and its stylet in a screwing motion into the medullary cavity. A rocking motion is avoided during insertion to avoid fracture. The entrance into the medulla is felt as a loss of resistance and the needle is then stable in this position.
Remove the stylet and inject 10 ml of Normal Saline. If there is no resistance to injection or swelling at the site, it is then confirmed to be in the medullary cavity and is ready to use.



The technique for removal of the intraosseous cannula is:
Use an aseptic technique to minimize the risk of infection.
Loosen and remove any devices attached to the cannula.
Use gentle rotation to remove the cannula smoothly.
Apply a sterile gauze pad and firm pressure for several minutes to minimize the risk of haematoma formation.
Remove the pad and apply a sterile dressing.

References:

1. Grevstad U, Gregersen P, Rasmussen LS. Intravenous access in the emergency patient. *Current Anaesthesia and Critical Care*, 2009; 20: 120-127
2. Australian Resuscitation Council. Advanced Life Support, 5th edition, 2006.
3. Clinical guideline for intraosseous access. Great Ormond Street Hospital for Children.

CERVICAL SPINE INJURY: IMMOBILISATION

The aim of immobilization is to maintain neutral alignment for the entire body. This is achieved via an appropriate sized and fitted hard-collar or manual inline immobilisation when the collar is temporarily removed and by using a log-roll manoeuvre for any movement of the head or torso. Cervical Spine immobilisation should occur during the assessment and the process of securing the airway.

Who to immobilise:

Cervical spine injury is associated with 5% of all blunt forces to the head. There is a proportional increase in incidence of injury with the increase in the size of the force. Any unconscious patient with a history of possible trauma must be immobilised, and any conscious patient with one or more of the following mechanisms of injury that may indicate risk of spinal injury:

Pedestrian / cyclist hit > 30km/hr.
Passenger - collision > 60km/hr.
Fall - more than 3 meters.
Kicked / fall from a horse.
Backed over by a car.
Thrown from vehicle.
Thrown over handlebars of bike.
Severe electric shock.
Multiple trauma
Significant injury above clavicles
Trauma & Unexplained hypotension
History of neck trauma
Neck tenderness
Limitations of neck movement due to pain.
Neurological deficit
Other major injuries (e.g. fractured limbs, abdominal injury)

How to immobilise the cervical spine:

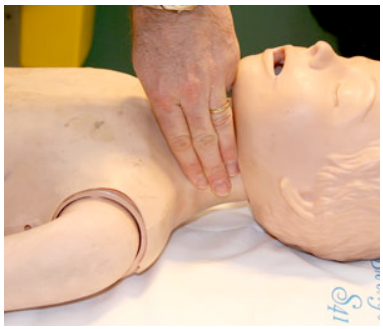
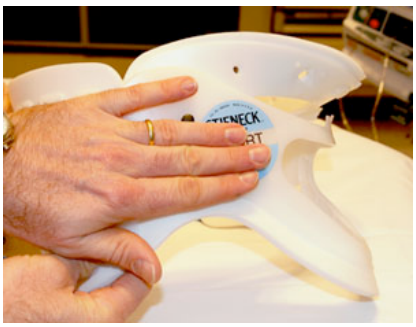
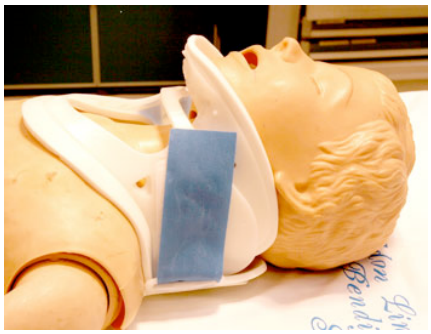
Fit a once-piece hard collar (see sizing below)
Children <3yo are especially difficult. Rigid cervical collars do not usually fit children <6 years of age. They should be immobilised with parents or staff holding the head and body, or sandbags or towels in situ and, if cooperative, the head taped to the board.

If uncooperative, avoid rigidly fixing the head to trolley or spinal board unless the body is also strapped to the board, as a patient who is thrashing their body around while their head is strapped to the board can do more damage.

If the patient's head is attached to the bed, be particularly aware of vomiting and the risk of aspiration - someone must be with the patient at all times.

In the acute phase there is no place for sedation without intubation to aid cervical spine immobilisation. However analgesia is an important consideration in trauma patients.

Sizing a one piece hard collar ("Stiffneck" collar):
A one piece hard collar is used in the initial stage. Measure the distance from the top of the patient's shoulder to the angle of the jaw with your hand. On the "Stiffneck" collar, measure from the bottom of the rigid plastic to the "measuring post". This should correspond to the above measurement .

		
Measuring collar	Measuring neck	Appropriately fitting collar

Management of the patient with a possible spinal injury:

Spinal boards are to be left in place for as minimal time as possible. Beyond 2 hours, pressure sores of the occiput, scapulae, sacrum and heels may develop. An ideal time to remove the board and logroll patient back onto a firm, well-padded trolley is when the patient is about to have their back examined and a rectal examination performed.

Pressure area care:
The greatest area for a pressure ulcer in children < 3 yrs old is the occiput and for those > 3 years old is the sacrum and heels.
Hard cervical collars must be removed and pressure areas from bony prominences assessed every 2 hours.

Positioning:
The ideal position of the patient is flat on their back. If the head needs to be elevated in the case of suspected raised intracranial pressure, the whole bed should be tilted (reverse trendelenberg). If the entire spine apart from the cervical spine has been cleared, the bed may be tilted from the hips.

The log-roll is the standard manoeuvre to allow examination of the back and transfer. The number and degree of rolls should be kept to a minimum. Rigid transfer slides using a slide board are useful for transferring the patient from one surface to another (such as to and from the CT scanner or operating table).

Patients who are agitated or restless due to shock, hypoxia, head injury or intoxication may be impossible to immobilise adequately. Forced restraints or manual fixation of the head may risk further injury to the spine. It may be necessary to remove immobilisation devices and allow the patient to move unhindered. No traction should be applied to the cervical spine.

Anaesthesia may be necessary to manage the patient. Intubation of the trauma victim is best achieved via rapid sequence induction. The collar should be removed and manual, in-line immobilisation re-instituted for the intubation. The collar is replaced once the endotracheal tube has been secured. The lack of neck extension will make laryngoscopy more difficult, thus a gum elastic bougie and other difficult airway devices should be at hand.

The spinal log roll:

The objective of the log roll procedure is to maintain correct anatomical alignment in order to prevent the possibility of further neurologic injury when patient transfer, examination or nursing care is required. The log rolling procedure is implemented at various stages of the trauma patient's management including: during the primary and secondary survey to examine the patient's back, during a bed to bed transfer (such as from an ambulance trolley to the emergency trolley) and to facilitate pressure area care, change bedding and chest physiotherapy.

At least four staff members will be required to assist in the log roll procedure as outlined below:

1. One to maintain manual inline mobilisation of head and neck (with Cervical collar insitu)
   2. One to move the torso (hands across posterior shoulders and hips)
   3. One to move pelvis (hands across abdomen and thighs – overlapping with the arm of the person responsible for the torso).
   4. One to move the spine board and examine the back, plus an additional person if there is an obese patient or one with limb injuries.

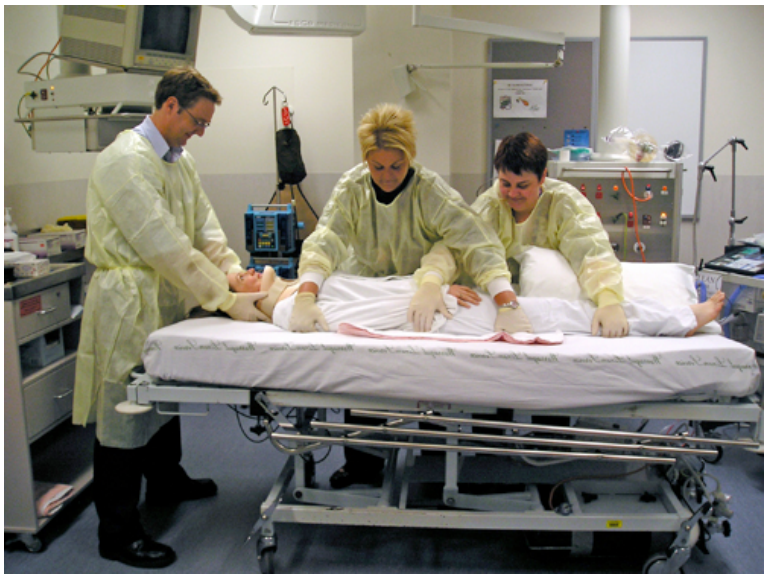
The steps in the spinal log roll procedure are as follows:
Explain the procedure to the patient regardless of conscious state and ask the patient to lie still and to refrain from assisting. Ensure that the collar is well fitting prior to commencement.

Ensure that devices such as indwelling catheters, intercostal catheters or ventilator tubing are repositioned to prevent overextension and possible dislodgement during repositioning.

The bed must be positioned at a suitable height for the head holder and assistants and the patient must be anatomically aligned prior to the procedure.monitoring devices such as arterial lines or peripheral intravenous lines. The patient’s distal arm should be extended in alignment with the thorax and abdomen or bent over the patient’s chest if appropriate and the arm is uninjured. A pillow should be placed between the patient’s legs.

The assistant supporting the patient’s upper body places one hand over the patient’s shoulder to support the posterior chest area, and the other hand around the patient’s hips.

The assistant supporting the patient's abdomen and lower limbs, overlaps with assistant 1 to place one hand under the patient's back, and the other hand over the patient's thighs.



On direction from the person managing cervical immobilisation, the patient is turned in anatomical alignment in one smooth action.

On completion of the planned activity, the head holder will direct the assistants to either return the patient to the supine position or to support the patient in a lateral position with wedge pillows. The patient must be left in correct anatomical alignment at all times.

DEFIBRILLATION

Cardiac defibrillation is indicated for the treatment of ventricular fibrillation or pulseless ventricular tachycardia. Early successful defibrillation is one of the few interventions that have been shown to improve outcome from VF/VT cardiac arrest.

For every minute that passes between collapse and attempted defibrillation, mortality increases 7-10% in the absence of cardiopulmonary resuscitation (CPR). If there is any delay in obtaining a defibrillator, start chest compressions and ventilation immediately. If CPR is provided, the decrease in survival form collapse to shock is more gradual (3-4% per minute).

Mechanism of defibrillation:

During defibrillation, a current is passed through the myocardium to depolarise a critical mass of the cardiac muscle simultaneously to allow the natural pacemaker tissue to resume control.

A defibrillator has 3 main features. A power source capable of providing a direct current, a capacitor that can be charged to a pre-determined energy level and two electrodes to place on the patient's chest through which the capacitor discharges.

Successful defibrillation requires enough current to be delivered to the myocardium, but the actual delivered current is difficult to determine because it is influenced by thoracic impedance and the position of the electrodes. As little as 4% of the current actually reaches the heart.

Safe defibrillation:

Defibrillation can present a risk of electric shock to the rescuers, burns, sparking and fire. The hazard is increased in the presence of water, metal fixtures, flammable substances and oxygen. **All bystanders and rescuers need to be clear of the patient during defibrillation.**

Sparks from poorly applied defibrillator pads can cause a fire. The use of self-adhesive pads may minimise the risk of sparks. An open oxygen source should be removed to at least one metre from the patient. If a circuit is attached to an endotracheal tube, even at high flow, the danger is eliminated if it is not disconnected from the patient.

Foil-backed nitrate patches need to be removed from the patient's chest to avoid arcing during defibrillation. Similarly, avoid placing the defibrillator pads over ECG electrodes and an implanted device. Permanent pacemakers and implanted defibrillators may have their circuits damaged if defibrillator electrodes are placed over them.

It is recommended that the defibrillator be charged only with the paddles on the chest and not whilst they are held in the air. If it is decided that a shock is no longer required after charging, it can be discharged safely by altering the energy setting.

Sequence of events for the use of manual defibrillator:

1. Confirm cardiac arrest, checking breathing (and pulse simultaneously if you are trained to do so.)
2. Commence rescue breathing and chest compressions until a defibrillator is available
3. Attach defibrillator pads or monitor. Do not interrupt chest compressions.
4. Confirm ventricular fibrillation (VF) or pulseless ventricular tachycardia (VT) from the ECG monitor or from adhesive pads or defibrillator paddles.
5. Place pads on the patient's chest with one below the clavicle to the right of the sternum and one at the 5th intercostal space in the mid-axillary line. The pads need to be clear of any breast tissue, and they are best placed longitudinally. It is possible to place the pads on the lateral chest walls (one left and one right) or one over the left precordium and one on the back behind the heart if access to the chest is restricted. Self-adhesive pads are preferable to standard defibrillation paddles. The pads allow the operator to defibrillate the patient from a safe distance rather than leaning over the patient. When gel pads are used with paddles, the electrolyte gel becomes polarised and can be a poor conductor after defibrillation. They may need to be replaced.
6. Place the paddles over the gel pads firmly.
7. Select the correct energy level. Use 360 Joules (with a monophasic defibrillator) or 150-200 Joules (with a biphasic defibrillator) for the first shock. Use 360 Joules (monophasic) or 150-360 Joules (biphasic) for subsequent shocks.
8. Ensure that no high flow oxygen is passing over the patient's chest or within one metre of the zone of defibrillation.
9. Warn everyone to stand clear and charge the defibrillator.
10. Check there is no one in danger by performing a quick visual check to ensure everyone is clear.
11. Deliver the shock.
12. Recommence chest compressions and ventilation with minimal delay. The interruption to chest compressions should not exceed 10 seconds.

Synchronised cardioversion:

Cardioversion can be used to convert atrial or ventricular tachycardia to sinus rhythm. To perform electrical cardioversion, it is important to synchronise the shock first so as to avoid shocking during the T wave of the ECG. If a shock is delivered during the refractory period, there is a risk of inducing VF. Conscious patients must be sedated or anaesthetised before performing cardioversion.

References:

3. Australian Resuscitation Council. Advanced Life Support, 5th edition, 2006.
4. Australian Resuscitation Council Website. www.resus.org.au/

INTERCOSTAL CHEST TUBE INSERTION

Drainage of the pleural space by means of a chest tube is the commonest intervention in thoracic trauma, and provides definitive treatment in the majority of cases. Whilst it is a relatively simple procedure, it carries a significant complication rate, (reported as being between 2% and 10%).

Indications

A chest tube is indicated to drain the contents of the pleural space. Usually this will be air or blood, but may include other fluids such as chyle or gastric/oesophageal contents.

- Pneumothorax (open, simple, tension)
- Haemothorax
- Significant chest injuries in patients who is undergoing a general anaesthesia or being transferred by air.
- Traumatic arrest (bilateral)
- Empyema and parapneumonic pleural effusion
- Malignant pleural effusion
- Post operative for thoracotomy and oesophagectomy

Equipment needed:

- Sterile drapes, gown, gloves,
- Sterile skin wash – chlorohexidine or betadine (iodine)
- Mask and protective eye wear
- Local anaesthetic 10-20mls, with 23 gauge needle and 10ml syringe
- Large bore chest tube 32 or 36 Fr and remove the stylet (a smaller tube will suffice for a pneumothorax)
- Curved clamp
- Underwater seal device or closed drainage system and sterile water, connecting tubing
- Suture material (1” silk) and Dressings

Technique:

The procedure is explained to the patient (or relative) and consent is obtained.

Pre-medication is considered. Intercostal catheter insertion is a very painful technique, and an analgesic is usually required. Prophylactic antibiotics are recommended for insertion of an ICC in a trauma patient. This is usually a cephalosporin. There is a real risk of empyema after ICC insertion in a trauma patient (2.4%)

The patient is positioned for the procedure. Preferably the patient is sitting up slightly, with monitoring including ECG, pulse oximetry and oxygen by a facemask. A vasovagal reaction may occur due to intense vagal stimulation, so caution must be exercised in the sitting position. The patient’s hand is placed behind the head to expose the axilla.

The site of insertion is usually the 4th or 5th intercostal space (approximately at the nipple line), just anterior to the mid-axillary line on the affected side. On expiration, the diaphragm rises to the 5th rib at the level of the nipple, and thus chest drains should be placed above this level. Rib spaces are counted down from the 2nd rib at the sterno-manubrial joint.

There is a safe triangle for insertion of the ICC. Its boundaries are the anterior border of latissimus dorsi and the lateral border of the pectoralis major muscle, an anterior line superior to the horizontal level of the nipple and the apex of the triangle is below the axilla.

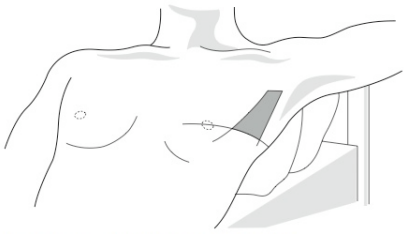
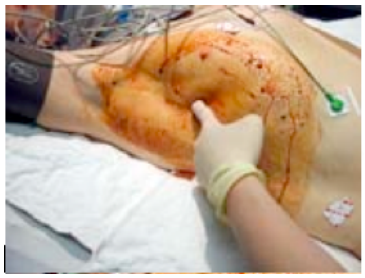


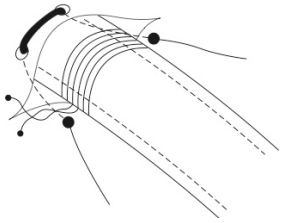
Figure 3 Diagram to illustrate the "safe triangle".



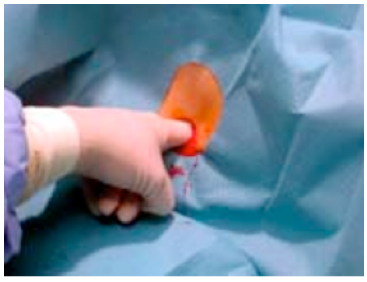
Prepare the equipment – clamp the proximal end of the chest tube and remove the stylet. Prepare and drape the skin.

Infiltrate the skin and deeper tissues including the pleura with local anaesthetic. The needle is then directed perpendicular to the skin and local anaesthetic infiltrated through the layers of the chest wall down onto the rib below the actual intercostal space. Here, local anaesthetic is injected around the periosteum of the rib. The needle is then angled above the rib and advanced slowly until air is aspirated.

Make a 2-3cm incision transversely through the skin and subcutaneous tissues (in the line of the intercostal space along the upper border of the rib). A wound closure suture is inserted before blunt dissection is performed.

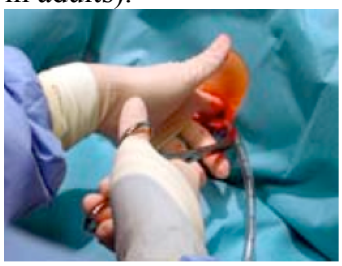


Blunt dissection is performed using a curved clamp along the track. The clamp is inserted into muscle tissue and is spread to split the fibres.

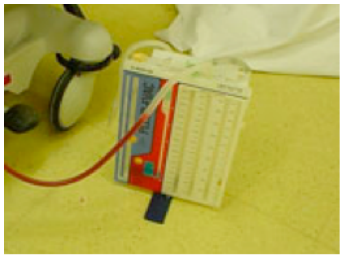


Insert a gloved finger to confirm the track, and that you are in the chest cavity and other organs are not present.

A large-bore (28-30 F) chest tube is mounted on the clamp and passed along the track into the pleural cavity. It is directed posteriorly and basally to a desired length (15cm in adults).



The tube is connected to an underwater seal.



Confirm correct placement by observing bubbling in the drain bottle, as well as swinging of the fluid in the tubing with inspiration.

Suture the tube in place and apply a sterile dressing.

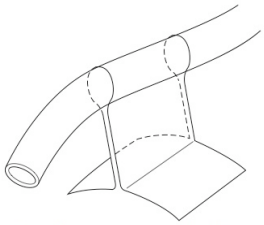


Figure 5 Omental tag to support the tube while allowing it to lie a little away from the chest wall.

A chest X-ray is taken to confirm placement & position.

Complications

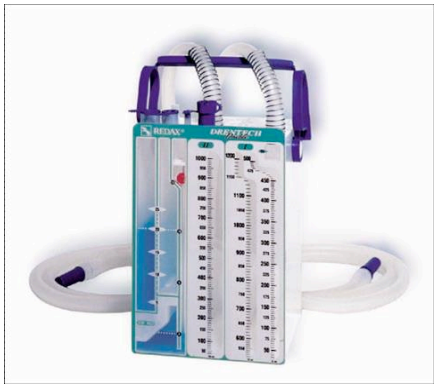
"There is no organ in the thoracic or abdominal cavity that has not been pierced by a chest drain."

- Laceration of the lung or other intrathoracic or abdominal organs
- Introduction of pleural infections
- Damage to intercostals nerves, arteries or veins
- Incorrect position
- Chest tube kinking, clogging or disconnection
- Persistent pneumothorax
- Subcutaneous emphysema

Chest drain (underwater seal)

An underwater seal is used to allow air to escape through the drain but not to re-enter the thoracic cavity.

Generally the device has 3 main chambers, however newer devices have up to 5 chambers.



The basic requirements are a suitable chest drain with minimal resistance, an underwater seal and a collection chamber. It should also contain a release vent, in order to prevent the chamber becoming pressurized.

The drainage tube is submerged to a depth of 1-2 cm in a collection chamber of approximately 20 cm diameter. This ensures minimum resistance to drainage of air and maintains the underwater seal even in the face of a large inspiratory effort. The chamber should be 100 cm below the chest because sub-atmospheric pressures up to -80 cmH_2O may be produced during obstructed inspiration. Drainage can occur under gravity, or LOW PRESSURE suction may be applied.

Key points regarding chest drains:

- The underwater seal acts as a one-way valve through which air is expelled from the pleural space and prevented from re-entering during the next inspiration
- Retrograde flow of fluid may occur if the collection chamber is raised above the level of the patient
- Absence of oscillations may indicate obstruction of the drainage system by clots or kinks, loss of sub-atmospheric pressure or complete re-expansion of the lung
- Persistent bubbling indicates a continuing broncho-pleural air leak
- The collection chamber should be kept below the level of the patient at all times to prevent fluid being siphoned into the pleural space. Clamping a pleural drain in the presence of a continuing air leak may result in a tension pneumothorax

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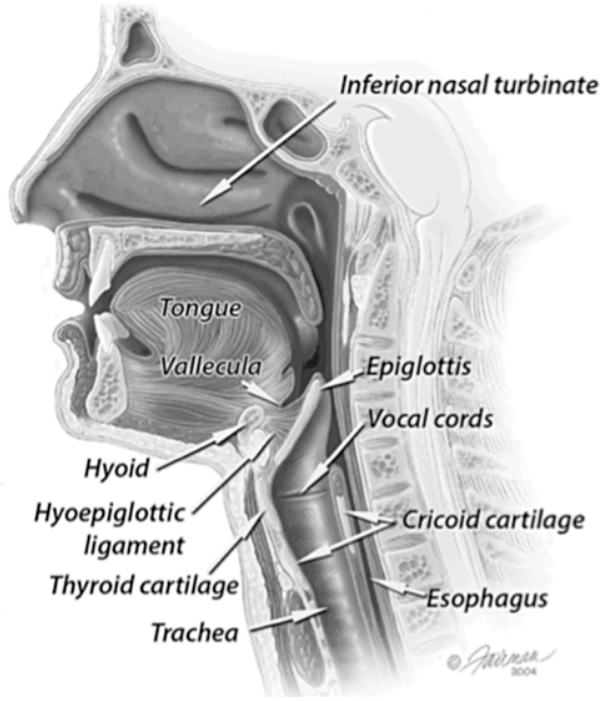
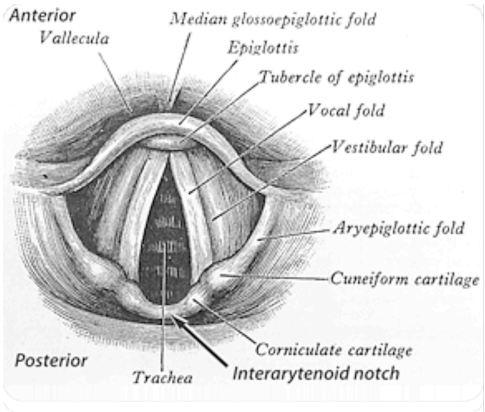
LARYNGOSCOPY AND INTUBATION

Laryngoscopy is a predictable sequence of progressively visualised structures. The anatomy will be viewed in the order of:

(1) tongue and uvula
(2) epiglottis
(3) posterior cartilages and interarytenoid notch
(4) glottic opening
(5) vocal cords

Anatomy



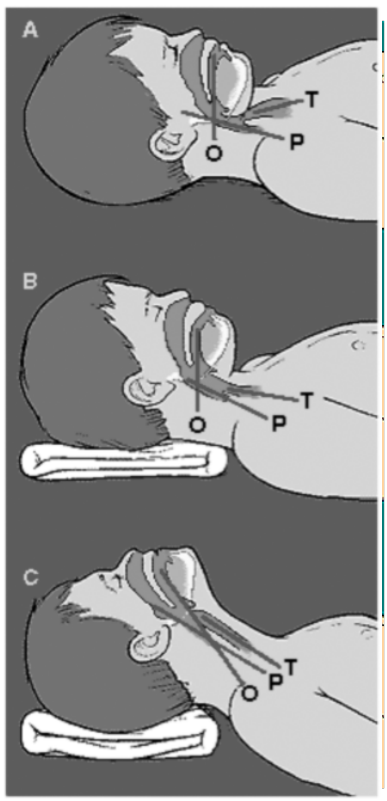


Patient Positioning

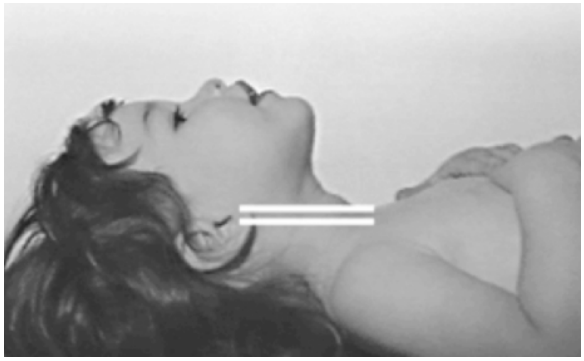
Traditional teaching has suggested that the airway superior to the larynx can be thought of in three planes:

T - Trachea
P - Pharynx
O - Oropharynx

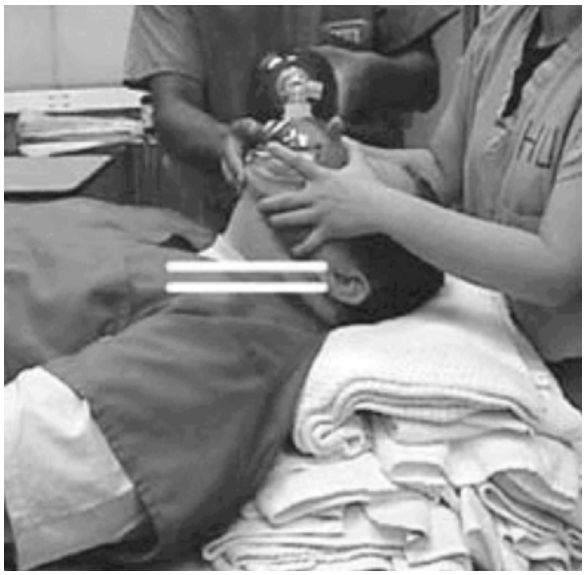
Traditional positioning suggests bringing all three planes into relative alignment. From the neutral position (A), note that flexion of the neck (achieved by head support) improves the alignment of the Tracheal and Pharyngeal axes (B). Extending the head (the atlanto-occipital joint) aims to bring the Oropharyngeal axis into line (C). This final position, often called the "sniffing" position, is more correctly known as the Magill Intubating Position.

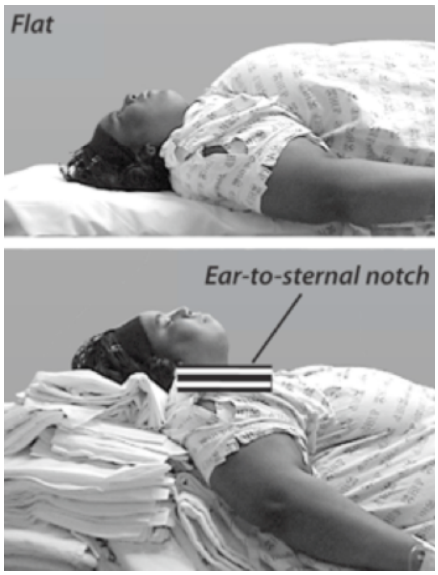


With correct positioning, the auditory meatus of the ear will horizontally align with the sternal notch, when viewed from the side of the patient. Some head and neck elevation may be required to achieve this. Although referred to as “Ear-to-Sternal Notch Position”, it reflects an assessment of the patient in the Magill position.



Head and neck elevation may be required to achieve good positioning.





This position is called the “Ramped Position”, or the “Head Elevated Laryngoscopy Position” (HELP). The advantages of the elevated or back up position are greatest in the obese, and include:

- improving upper airway patency
- better mechanics of ventilation, both with spontaneous breathing, and with mask ventilation
- improved laryngoscopy

A 25^{o} back-up or elevation of the patient will alter the position of the upper airway structures and improve view at laryngoscopy. A supine-horizontal patient normally requiring a 45 ^{o} direction of lift with a laryngoscope will find this reduced to only 20 ^{o} in the elevated position. Thus, a vertical force is changed to a more hoizontal force, which is much easier for the anaesthetist.

Note that all these positions maintain the basic elements of the “sniffing” or Magill Intubating Position.

Equipment

The layryngoscope is the most commonly used instrument that allows the anaesthetist to visualize the vocal cords and achieve intubation. The laryngoscope consists of a blade and a handle. There are a variety of blades available, the most common being the Macintosh curved blade and the Miller straight blade.

The Macintosh is a curved blade that allows the tip to be inserted into the vallecula (the space between the base of the tongue and the pharyngeal surface of the epiglottis). Pressure on the underlying hypoepiglottic ligament causes the epiglottis to lift upward (indirect epiglottic elevation), improving the view of the glottic opening. Most adults require a Macintosh number 3 or 4 blade.

The Miller is a straight blade that is passed into the oropharynx so that the tip of the blade lies beneath the laryngeal surface of the epiglottis. The epiglottis is then lifted to expose the vocal cords. Most adults require a Miller number 3 blade. The Miller blade is considered a better blade for visualisation of the larynx, but the Macintosh blade is better for intubation.

The laryngoscope handle allows force to be applied to the blade. The direction of this force should always be along the handle. Never use the laryngoscope as a lever, as this will result in damage to teeth and/or gums, and will only push the larynx further anterior and out of sight.

Laryngoscopy Technique

Hold laryngoscope in left hand.

Open the patient's mouth with a right-handed scissor technique.

With the laryngoscope handle pointed toward the patient’s feet, insert the laryngoscope blade on the right side of the mouth and use it to sweep the tongue toward the midline and further to the left

The tip of the blade gets around the base of tongue, permitting a change in the angle of lifting and a better mechanical advantage.

Lift (not lever) the laryngoscope in the direction of the handle to lift the tongue and floor-of-mouth structures off the posterior pharyngeal wall.

Advance the blade in the midline until the edge of the epiglottis is recognised.

Laryngoscopy Technique – Macintosh

1. Position the tip of the Mac blade in the vallecula (between the epiglottis and base of tongue) and lift upwards and away from yourself.
2. Pressure on the underlying hyoepiglottic ligament causes the epiglottis to lift upward (indirect epiglottic elevation).
3. Positioning of a Mac blade tip in the vallecula is a more important determination of glottic opening than force applied along the laryngoscope.

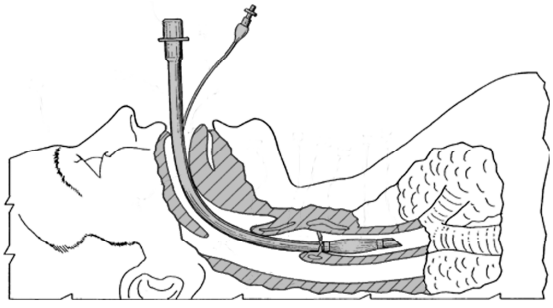
Laryngoscopy Technique – Miller

1. Position the tip of the Miller blade posterior to the epiglottis,
2. The epiglottis is lifted directly up to expose the glottic opening

Intubation

The epiglottis is the bridge from the tongue to the glottic opening. Elevation of the epiglottis will reveal the posterior cartilages and the inter-arytenoid notch. The glottic opening and vocal cords are more anterior inside the larynx.

When the vocal cords or the arytenoid cartilages are clearly seen, advance the tube down the right side of the mouth, keeping the vocal cords in view until the last possible moment. Advance the tube through the vocal cords and position the cuff below the vocal cords. Inflate the cuff of the endotracheal tube while ventilating the patient, to ensure that an adequate seal is achieved with minimal cuff pressure. Secure the endotracheal tube and support the ventilating circuit, to prevent accidental dislodgment.



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